THE AMERICAN JOURNAL OF GASTROENTEROLOGY
Vol. 94, No. 11, 1999

ISSN 0002-9270/99/$20.00
Published by Elsevier Science Inc.
PII S0002-9270(99)00560-2

PRACTICE GUIDELINES

Diagnosis and Management of Diverticular Disease of the Colon in Adults

Neil H. Stollman, M.D., F.A.C.P., and Jeffrey B. Raskin, M.D., F.A.C.P, F.A.C.G., for and on behalf of the *Ad Hoc* Practice Parameters Committee of the American College of Gastroenterology*
Division of Gastroenterology, University of Miami School of Medicine, Miami, Florida

PREAMBLE

Guidelines for clinical practice are intended to suggest preferable approaches to particular medical problems as established by interpretation and collation of scientifically valid research, derived from extensive review of published literature. When data are not available, which will withstand objective scrutiny, a recommendation may be made based on a consensus of experts. Guidelines are intended to apply to the clinical situation for all physicians without regard to specialty. Guidelines are intended to be flexible, not necessarily indicating the only acceptable approach, and should be distinguished from standards of care that are inflexible and rarely violated. Given the wide range of choices in any health care problem, the physician should select the course best suited to the individual patient and the clinical situation presented. These guidelines are developed under the auspices of the American College of Gastroenterology and its Practice Parameters Committee. These guidelines are also approved by the governing boards of the American Gastroenterological Association and the American Society for Gastrointestinal Endoscopy. Expert opinion is solicited from the outset for the document. Guidelines are reviewed in depth by the committee, with participation from experienced clinicians and others in related fields. The final recommendations are based on the data available at the time of the production of the document and may be updated with pertinent scientific developments at a later time. The following guidelines are intended for adults and not for pediatric patients.

*Members of the Practice Parameters Committee of the American College of Gastroenterology: Nimish Vakil, M.D., F.A.C.G (Chairman), Milwaukee, WI; W. Scott Brooks, Jr., M.D., F.A.C.G., Atlanta, GA; William D. Carey, M.D., F.A.C.G., Cleveland, OH; Kenneth R. DeVault, M.D., F.A.C.G., Jacksonville, FL; Simon K. Lo, M.D., F.A.C.G., Torrence, CA; Douglas M. Simon, M.D., F.A.C.G., New Rochelle, NY; J. Patrick Waring, M.D., F.A.C.G., Atlanta, GA; Freda L. Arlow, M.D., F.A.C.G., Bloomfield Hills, MI; William J. Caddick, M.D., Gastonia, NC; Christopher P. Cheney, M.D., F.A.C.G., Washington, DC; Francis A. Farraye, M.D., F.A.C.G., West Roxbury, MA; Daniel S. Pratt, M.D., Boston, MA; Amy M. Tsuchida, M.D., F.A.C.G., Tacoma, WA; John M. Wo, M.D., Louisville, KY; Alan N. Barkun, M.D., Montreal, Canada; Stephen H. Caldwell, M.D., Charlottesville, VA; Sita S. Chokhavatia, M.D., Paramus, NJ; Kris Kowdley, M.D., Seattle, WA; Dawn Provenzale, M.D., F.A.C.G., Durham, NC; Thomas R. Viggiano, M.D., F.A.C.G., Rochester, MN; and Marc J. Zuckerman, M.D., F.A.C.G., El Paso, TX.

INTRODUCTION

Diverticular disease of the colon is very common in developed countries and its prevalence increases with age. To refer to an acquired deformity present in perhaps two-thirds of the elderly as a 'disease' may be inaccurate, particularly as the large majority of those affected will remain entirely asymptomatic. Nonetheless, an estimated 20% of patients may manifest clinical illness (1). The purpose of this guideline is to briefly review the epidemiology, etiopathogenesis, and clinical presentations of diverticular disease, and then propose recommendations regarding appropriate diagnostic and therapeutic strategies supported by the best available current evidence.

EPIDEMIOLOGY

The true incidence of colonic diverticulosis is difficult to measure, mainly because most patients are asymptomatic. The incidence clearly increases with age, varying from less than 10% in those under 40 yr, to an estimated 50–66% of patients over age 80 (2–4). There is no apparent sex predilection. Diverticulosis has been termed a "disease of Western Civilization" because of its striking geographic variability. The disorder is extraordinarily rare in rural Africa and Asia; conversely, the highest incidence rates are seen in the United States, Europe, and Australia (2).

PATHOLOGICAL ANATOMY

Diverticula have been observed to occur typically in two or four parallel rows. This pattern is likely related to the penetration of the small arteries supplying the colonic mucosa (the vasa recta), with relative weakness of the muscular wall at these sites allowing for herniation of the mucosa and submucosa. In Western societies, diverticula occur mainly in the distal colon, with up to 90% of patients having involvement of the sigmoid colon, and only 15% having right-sided involvement (3, 5, 6). This is in contrast to Asian countries, in which right-sided involvement is more prominent (7, 8). Diverticula can vary in number from solitary findings to literally hundreds. They are typically 5–10 mm in diameter, but can exceed 2 cm in size.

ETIOLOGY/PATHOGENESIS

Gross examinations of diverticular colons report thickening of the muscle wall and shortening of the taenia, with a resulting accordion-like bunching of the folds. Routine histology does not, however, reveal muscle hypertrophy. More recently, electron microscopic studies have confirmed that the colonic wall in diverticulosis has structurally normal muscle cells, but contains a >2-fold increase in elastin deposition between the muscle cells in the taenia (9). The elastin is laid down in a contracted form, presumably causing shortening of the taenia and the resulting bunching of the circular muscle.

The possibility of altered colonic motility playing an etiological role in diverticular disease has been explored for many years, with the repeated demonstration of higher resting, postprandial, and neostigmine-stimulated pressures in diverticular patients compared to controls (10–13). Painter postulated a theory of 'segmentation' in which contraction of the colon at haustral folds causes the colon to act as a series of discrete 'little bladders', rather than as a continuous single-chambered lumen (11, 14). He proposed that this segmentation might play a physiological role in delaying transport and augmenting water reabsorption, but could also generate excessively high pressures within each segment or 'bladder', forcing the mucosa to herniate.

The wide geographic variability of diverticular disease and its correlation with a Westernized diet has long suggested a dietary factor, most likely fiber, in its pathogenesis. Burkitt and Painter in fact labeled diverticulosis a 'deficiency disease', which, like scurvy, should be avoidable with dietary changes (2). They rigorously demonstrated that individuals in the UK, eating a refined Western diet low in fiber, had colonic transit times of ~80 h, and mean stool weights of ~110 gm/day. This was contrasted with rural Ugandans, eating very high fiber diets, who had significantly shorter transit times (~34 h), and greater stool weights (>450 gm/day) (15). The longer intestinal transit times and smaller volume stools in the UK patients were felt to increase intraluminal pressures and predispose to diverticular herniation. As reasonable as this postulate seems, studies in Western populations comparing patients with and without diverticular disease have generally failed to show significant differences. Nonetheless, corroborative animal data does exist. Most notable is a study of rats fed diets of varying fiber content throughout their natural lifespan; 45% of the rats on the lowest fiber diet developed diverticula, compared with only 9% of those fed the highest fiber diet (16).

PREVENTION OF DIVERTICULOSIS

Recommendation: *Diets high in fruit and vegetable fiber may decrease the development of colonic diverticulosis.*

The observations above, that low fiber diets are associated with colonic diverticulosis, has led to the postulate that active therapy with higher fiber diets might prevent diverticular disease. This theory of diverticular 'prophylaxis' is supported by results from the Health Professionals Follow-up Study, which prospectively followed 51,529 US male health professionals (17). Over a 6-yr period, 385 (0.75%) new cases of symptomatic diverticular disease were identified. A significant inverse association was found between insoluble dietary fiber intake (especially fruit and vegetable, *e.g.*, cellulose fiber) and the risk of subsequently developing symptomatic diverticular disease (Relative Risk = 0.63, 95% CI 0.44–0.91). Although the results were fairly linear, suggesting increasing benefit with increasing fiber intake, the greatest benefit was seen in those individuals consuming an average of 32 g/day of total fiber. Further analysis of this large epidemiological study also described a similar protective effect of physical activity on the development of symptomatic diverticulosis (18), and no effect seen with alcohol, smoking, or caffeine consumption (19). Although detection and recall biases must be considered in interpreting these results, they provide support for a more general recommendation that patients may benefit from increasing their fruit and vegetable fiber intake. Patients should be cautioned to gradually increase their fiber intake and maintain adequate hydration, to avoid transiently worsening their symptoms.

UNCOMPLICATED DIVERTICULOSIS

Recommendation: *Incidentally identified diverticulosis in an asymptomatic patient does not require further diagnostic evaluation. It is reasonable to recommend a diet high in fruit and vegetable fiber to patients with uncomplicated diverticulosis. There is little data supporting a role for antispasmodic agents in these patients, and no role for antibiotics in the absence of signs or symptoms suggesting diverticulitis.*

The large majority of patients with diverticulosis will remain entirely asymptomatic. There is no data to support any therapeutic recommendations or routine follow-up in this large population. An unquantified subset of the remainder will have bothersome symptoms attributed to their diverticular disease, so-called symptomatic uncomplicated diverticulosis. Patients with nonspecific symptoms, such as pain, bloating, and/or changed bowel habits, will be discussed in this section. Patients with well-defined complications of their disease, such as diverticulitis, stricture, obstruction, or bleeding, will be discussed in the following section on complicated diverticulosis.

Patients may come to clinical attention because of nonspecific abdominal complaints, and are found to have diverticulosis coli; a causative relationship is often difficult to establish. Most will present with pain, typically in the lower abdomen, and more frequently, but not invariably, left-sided. By definition, such patients do not manifest signs of inflammation, such as pyrexia or neutrophilia, which might indicate diverticulitis. The pain is often exacerbated by

eating and diminished with defecation or the passage of flatus. It has been suggested, although this is purely conjecture, that this pain reflects colonic wall tension because of increased intraluminal pressure. Patients may also report other symptoms of colonic dysfunction, including bloating, constipation, diarrhea, or the passage of mucus, although the relationship of these symptoms to their diverticula is speculative. Physical examination may reveal fullness or mild tenderness in the left lower quadrant, but frank rebound or guarding should be absent. A stool positive for fecal occult blood in this setting should never be attributed to diverticulosis without a further complete colonic evaluation. Laboratory studies should be normal.

In a patient with nonspecific symptoms of colonic dysfunction, the demonstration of diverticula radiographically or colonoscopically adds little to the diagnostic probabilities or management because of the high prevalence of this finding in the general population. One must be careful to consider alternative diagnoses before attributing the symptoms solely to diverticulosis. These nonspecific symptoms obviously overlap considerably with those of the Irritable Bowel Syndrome (IBS). It has been postulated that diverticula may in fact be a late consequence of the IBS. Otte *et al.* reviewed 69 patients with IBS, 24 of whom had diverticula (20). Over a 7-yr follow-up period, symptoms or prognosis were no different between those with or without diverticula. Ritchie has reported on the similarity of pain sensation from rectal balloon distention in patients with IBS and diverticulosis (21). Whether or not these two disorders are in fact distinct entities is unknown and probably not clinically important, as both are treated in a similar fashion with equally good prognoses.

DIAGNOSTIC MODALITIES

For many years, the barium enema (BE) examination had been the standard investigation in patients with symptoms suggesting colonic disease. Barium studies can provide information on the number and location of colonic diverticula, but obviously cannot discern their clinical significance. More recently, caution has been expressed about a significant diagnostic error rate for BE examinations in patients with sigmoid diverticulosis. Boulos *et al.* described 65 patients with bowel symptoms who had double-contrast barium enemas revealing sigmoid diverticulosis (22). All underwent subsequent colonoscopy. Nineteen neoplastic lesions (17 polyps, two carcinomas) were reported on BE; colonoscopy revealed no polyps in nine of 17 (53%), and confirmed only one of the two carcinoma readings. In the 46 barium examinations showing only diverticulosis and no neoplasia, colonoscopy revealed polyps in eight and carcinomas in three, an error rate of 24%. Overall, the BE interpretation was inaccurate in 32%. The authors recommend routine colonoscopy in all patients with symptomatic diverticular disease, particularly to exclude neoplasia. Until further prospective data are available, however, no firm recommendation can be made regarding the relative utilities of these two diagnostic modalities, and the choice of testing should depend on the clinical scenario and the local expertise available.

Previously, the presence of diverticulosis was felt by some to be a relative contraindication to colonoscopy, for fear of an increased likelihood of perforation. Numerous series and many years of clinical experience, however, have demonstrated the relative safety of colonoscopy in patients with uncomplicated diverticular disease. One must remain cautious, however, about the possibility of undiagnosed or subclinical diverticulitis with undetected microperforation, and be judicious with air insufflation. This is particularly true if the tip of the colonoscope might be in the neck of a diverticulum, as very high 'local' pressures can develop within the diverticulum itself. The diverticular colon may also be difficult to examine colonoscopically because of spasm, luminal narrowing from prominent enlarged folds, fixation from prior inflammation and pericolic fibrosis, as well as confusion between luminal and diverticular openings. The use of a smaller diameter colonoscope may be helpful in this situation (23, 24).

The incidental identification of diverticulosis coli by barium examination, colonoscopy, or computerized tomography (CT) scanning does not require any further diagnostic evaluation.

TREATMENT

Dietary Fiber

The possible 'protective' effect of fiber in asymptomatic patients has already been mentioned (17). Although there have been multiple uncontrolled studies demonstrating the salutary effect of fiber supplements in patients with intestinal symptoms and diverticulosis, lack of a placebo group makes such data suspect. Brodribb published the first randomized, double-blind, placebo-controlled trial of a high fiber diet in patients with symptomatic diverticular disease, but reported results for only 18 patients (25). Although a significant placebo effect was observed at 1 month, by 3 months there was a statistically significant decrease in bowel symptoms in treated patients. A larger study of fiber supplementation in symptomatic patients with diverticulosis has been reported by Ornstein *et al.*, in which patients took bran or placebo in a double-blind, cross-over study for 4 months (26). Unlike Brodribb's data, no symptomatic improvement was demonstrated. Despite the conflicting data and the near certainty that diverticula do not *regress* with increased fiber intake, some amelioration of symptoms in patients with uncomplicated disease can be expected with a high fiber diet, which is reasonable to prescribe for its other potential health benefits as well. Historically, whole pieces of fiber (such as nuts, corn, seeds) have been excluded from such diets for fear they might become entrapped in diverticula. Controlled studies that support this belief are lacking.

Further, there is no data to support a role for any specific 'fad' or 'elimination' diet in this disorder.

Medications

The documented hypermotility of the colon in this disorder suggests that anticholinergic and antispasmodic agents may improve symptoms by diminishing muscular contraction. Nonetheless, there are no adequately controlled therapeutic trials documenting such a benefit. Intravenous glucagon has been reported in one study to offer short-term relief of pain, presumably as a result of smooth muscle relaxation (27).

COMPLICATED DIVERTICULOSIS

Diverticulitis

Diverticulitis, defined as inflammation and/or infection associated with diverticula, is the most common clinical complication of this disorder, affecting an estimated 10–25% of patients with colonic diverticula (3). The process through which a diverticulum becomes inflamed has been likened to that causing appendicitis, in which the diverticulum becomes obstructed by inspissated stool in its neck, ultimately leading to perforation of a single diverticulum (28, 29). The extent and localization of this perforation then determines its clinical behavior. Microperforations may remain very well localized, contained by the pericolic fat and mesentery, leading to small pericolic abscesses. Larger macroperforations can result in more extensive abscess formation, which may track longitudinally around the bowel wall forming a large inflammatory mass, extend to other organs, and/or cause fistulous disease. In its later stages, fibrosis and strictures may result. Free perforations into the peritoneum causing frank bacterial peritonitis can be life threatening, but are fortunately very uncommon. Hinchey *et al.* have described a grading system reflecting the degree of perforation (30):

Stage I: Confined pericolic abscess.
Stage II: Distant abscess (retroperitoneal or pelvic).
Stage III: Generalized peritonitis caused by rupture of a pericolic or pelvic abscess, "noncommunicating" with bowel lumen because of obliteration of diverticular neck by inflammation.
Stage IV: Fecal peritonitis caused by free perforation of a diverticulum ("communicating").

Clinical Features

Patients with acute diverticulitis classically present with left lower quadrant pain, reflecting the marked propensity for this disorder to occur in the sigmoid colon in Western countries. Patients with redundant sigmoids may well manifest suprapubic or even right-sided pain. Asian patients, as previously mentioned, have predominantly right-sided diverticula, and may also manifest right-sided pain. The pain may be intermittent or constant, and is frequently associated with a change in bowel habits, either diarrhea or constipation (31). Hematochezia is rare. Anorexia, nausea, and vomiting may occur. Dysuria and urinary frequency may be reported by patients, reflecting a 'sympathetic cystitis' induced by bladder irritation from the nearby inflamed sigmoid colon.

Table 1. Differential Diagnosis of Acute Diverticulitis

Differential Diagnosis	Clinical Scenarios and Diagnostic Considerations
Acute appendicitis	Suspect if RLQ symptoms or nonresolution with medical therapy
Crohn's disease	Suspect if apthous ulcers, perianal involvement, or chronic diarrhea
Colonic carcinoma	Suspect if weight loss, bleeding. Diagnose with colonic evaluation **after** acute inflammation resolved
Ischemic colitis	Suspect if high-risk patient, bloody diarrhea, or thumbprinting. Diagnose with limited flexible sigmoidoscopy
Pseudomembranous colitis	Suspect with antibiotic use or diarrhea. Diagnose with stool toxin or limited flexible sigmoidoscopoy
Complicated ulcer disease	Suspect if pneumoperitoneum or peritonitis, or with clinical history, NSAID use, or dyspepsia
Ovarian cyst, abscess, torsion	Suspect in female patient with unilateral pain. Diagnose with pelvic or transvaginal ultrasound
Ectopic pregnancy	Suspect in female patient of childbearing age. Diagnose with pregnancy test and ultrasound

RLQ = right lower quadrant; NSAID = nonsteroidal anti-inflammatory drug.

Physical exam usually discloses localized tenderness, generally in the left lower quadrant, although as noted above, right-sided signs do not preclude diverticulitis. Guarding and rebound tenderness may be present, as may a tender, cylindrical palpable mass. Bowel sounds are typically depressed, but may be normal in mild cases, or increased in the presence of obstruction. Rectal exam may disclose tenderness or a mass, particularly with a low-lying pelvic abscess. Fever is present in the majority of patients, although hypotension and shock are unusual. The white blood cell (WBC) count is frequently elevated, although not invariably so, with one study reporting a normal WBC count in 45% of patients with acute diverticulitis (31). No other laboratory abnormalities are routinely helpful.

Differential Diagnosis

The differential diagnosis of acute diverticulitis is extensive, with acute appendicitis the most common misdiagnosis. Other considerations include Crohn's Disease, colonic neoplasms, ischemic or pseudomembranous colitis, complicated peptic ulcer disease, and gynecologic pathology (Table 1).

Diagnostic Modalities

Recommendation: *The diagnosis of diverticulitis may be made on clinical grounds. Plain radiographs should be performed in all patients with significant abdominal pain and suspected diverticulitis. Select patients with severe ill-*

ness, atypical presentations, clinical deterioration, or suspected complications should be evaluated further, with CT scanning the best initial diagnostic modality. Contrast enema examinations, ultrasonography, and limited sigmoidoscopy are appropriate in some patients.

PLAIN RADIOGRAPHY. An erect chest radiograph, together with erect and supine abdominal radiographs should generally be performed on most patients with clinically significant abdominal pain. The erect chest film has the dual purpose of detecting a pneumoperitoneum, which has been reported to be present in up to 12% of patients with acute diverticulitis (32), and to assess cardiopulmonary status in a generally elderly population with frequent comorbid illness. Abdominal X-rays have been reported to be abnormal in 30–50% of patients with acute diverticulitis (32). Findings include small or large bowel dilation or ileus, bowel obstruction, or soft tissue densities suggesting abscesses.

COMPUTERIZED TOMOGRAPHY. As diverticulitis is mainly an extraluminal disease, *e.g.*, peridiverticulitis, luminal contrast studies, which for many years were the diagnostic standard, may be inaccurate. In recent years, CT scanning has been assuming an increasing role. Most now consider it the procedure of choice, both for its ability to image transmural/extraluminal disease and adjacent structures, as well as its therapeutic potential in the drainage of abscesses.

Abdominal and pelvic scanning is generally performed with water-soluble contrast, both orally to opacify the small bowel, and rectally to better evaluate the rectosigmoid. If not contraindicated, intravenous contrast is generally used as well. CT criteria suggestive of diverticulitis include the presence of diverticula with pericolic infiltration of fatty tissue, thickening of the colonic wall, and abscess formation. A number of prospective investigations have reported a sensitivity of 69–95% and a specificity of 75–100% for CT scanning, generally superior to contrast examinations (33–36). Most recently, a very large prospective study in Switzerland evaluated 423 patients over a 10-yr period (37). The sensitivity of CT scanning was excellent (97%). Further, the presence of severe disease, defined by abscess and/or extraluminal air or contrast, was prognostically very useful, accurately predicting failure of medical treatment and risk of secondary complications. Overall, and respecting issues of cost, availability, and local expertise, the data suggest that in cases where the patient is seriously ill, the diagnosis is in doubt, or clinical deterioration occurs, CT has become the most suitable primary investigation today. Nonetheless, a negative CT scan does not completely exclude this diagnosis.

CONTRAST ENEMA EXAMINATIONS. Contrast enemas still remain a useful complementary study to CT scanning, yielding additional and clinically useful information in some cases. The choice of contrast material to be used remains somewhat controversial. Although barium is less expensive than water soluble contrast media and provides better mucosal detail, the possibility of a perforation is a relatively strong contraindication to its use, for fear of fecal/barium peritonitis. In these situations, water-soluble contrast media should be used, a gentle single-contrast study should be performed and terminated once significant findings have been discovered. An attempt to visualize the entire colon should be deferred to a later date when the acute attack has resolved. Air (double) contrast studies are not indicated when acute diverticulitis is suspected, for fear that insufflation may dislodge a fecalith and result in perforation.

Findings considered *diagnostic* for diverticulitis include demonstration of extravasated contrast material outlining an abscess cavity, an intramural sinus tract or fistula (5, 33). Extensive diverticulosis, spasm, mucosal thickening or 'spiking', or deformed sacs are suggestive but nonspecific signs. An extraluminal mass compressing or displacing the bowel is said to be the most common finding in severe diverticulitis (32), although this finding is clearly not specific for this diagnosis. Obviously, the *absence* of any diverticula or the above findings should provoke a reconsideration of the diagnosis.

ULTRASONOGRAPHY. Based on its relatively low cost, convenience, and noninvasive nature, ultrasonography (US) has been advocated by some as a useful modality in diverticulitis. Characteristic findings have included hypoechoic bowel wall thickening, presence of diverticula or abscesses and hyperechogenicity surrounding the bowel wall, implying active inflammation. Three prospective studies have reported a sensitivity of 84–98% and a specificity of 80–98% (38–40). Two recent small studies have compared CT and US directly for acute colonic diverticulitis; one reported comparable accuracy (41), whereas the other found CT to be superior (42). Despite the above reports, the examination remains very operator dependent and in the absence of larger, well-designed prospective comparative studies, it remains a second line diagnostic tool to be used in selected clinical circumstances or research endeavors. Sonography may be most useful in female patients to exclude pelvic/gynecologic pathology.

ENDOSCOPY. Because of the risk of perforation, either from the instrument itself or insufflation of air, endoscopy is generally avoided in the initial evaluation of the patient with acute diverticulitis. Its use should be limited to situations in which the diagnosis of diverticulitis is unclear, in which case a limited rigid or flexible sigmoidoscopy with minimal air insufflation may be helpful to exclude other diagnoses, such as inflammatory bowel disease, carcinoma, or ischemic colitis.

Treatment

Recommendation: *Selected patients with mild diverticulitis (with good oral intake and a supportive social structure) can be treated as outpatients with broad-spectrum oral antibiotics. Patients with more severe illness or comorbid*

disease should be hospitalized and treated with bowel rest and intravenous antibiotics.

One of the initial decisions in uncomplicated diverticulitis involves a determination of the need for hospitalization. This ultimately depends on an individual patient's initial clinical presentation and the impression of the physician. No hard and fast generalizations are appropriate. Factors to be considered include the patient's ability to tolerate oral intake, severity of illness, comorbid disease and available outpatient support systems (*e.g.*, a reliable family). An appropriate candidate for outpatient management would be one with mild symptoms, no peritoneal signs, the ability to take oral fluids, and a supportive home network. In general, the very elderly or immunosuppressed patients, those with severe comorbid disease and those with high fevers or significant leukocytosis should be hospitalized. Appropriate patients for ambulatory care should be treated with a clear liquid diet and a broad-spectrum oral antibiotic with activity against anaerobes and Gram-negative rods (particularly *E. coli* and *B. fragilis*). Amoxicillin plus clavulanic acid, sulfamethoxazole-trimethoprim with metronidazole, or a quinolone with metronidazole have been recommended as reasonable choices (43). Patients so treated should obviously have close follow up. They should be instructed to call the physician for increasing pain, fever, or inability to tolerate oral fluids, which may necessitate hospitalization. In general, symptomatic improvement should be evident within 2–3 days, at which time the diet may be slowly advanced. Antibiotic treatment should be continued for 7–10 days.

Patients requiring hospitalization with acute diverticulitis should, in general, have their bowels 'rested' with clear liquids or nothing by mouth. Intravenous fluid therapy to maintain or restore intravascular volume, balance electrolytes, and ensure adequate urinary output should be initiated. Intravenous antibiotics should be started, aimed principally at the colonic anaerobic and Gram-negative flora, especially *E. coli* and Bacteroides species. Recommended combination regimens, based more on clinical consensus than randomized trials, include antianaerobic coverage with metronidazole or clindamycin and Gram-negative coverage with an aminoglycoside (*e.g.*, gentamicin, tobramycin), monobactam (*e.g.*, aztreonam), or third-generation cephalosporine (*e.g.*, ceftazidime, cefotaxime, ceftriaxone) (43). Single agent coverage with intravenous second-generation cephalosporins, such as cefoxiten (44) or cefotetan, or B-lactamase inhibitor combinations such as ampicillin-sulbactam or ticarcillin-clavulanate are reasonable alternatives. Similarly to outpatients, one should expect improvement with decreasing fever and leukocytosis within 2–4 days, at which point the diet may be advanced. If improvement continues, patients may be discharged but should complete a 7- to 10-day oral antibiotic course. Failure to improve with conservative medical therapy warrants a diligent search for complications, consideration of alternative diagnoses and surgical consultation.

Treatment Outcome

Recommendation: *A colonic evaluation is indicated after resolution of a clinically diagnosed case of presumptive diverticulitis to exclude other diagnostic considerations such as colonic neoplasia. Elective (prophylactic) surgery may be reasonable in patients with recurrent attacks of diverticulitis, patients with complicated disease (e.g., fistulas, abscess), or in young or immunocompromised patients. As diverticulitis may recur in as few as one of four patients, surgery is not generally indicated after a single uncomplicated episode.*

Most patients hospitalized with acute diverticulitis will respond to conservative medical therapy, but it has been estimated that 15–30% will require surgery during that admission (1, 5, 45, 46). Not surprisingly, such patients have a higher mortality rate, up to 18% in one series (47). Free perforation with generalized peritonitis, although uncommon, carries a high mortality rate (up to 35%) and requires urgent surgical intervention (5, 46). For the majority who do respond well to conservative therapy, a complete colonic evaluation is indicated after resolution of a clinically diagnosed case of presumptive diverticulitis, to exclude other diagnostic considerations such as colonic neoplasia. Once this has been performed, an important clinical question subsequently revolves around the likelihood of recurrence and the role of elective 'prophylactic' surgical resection to prevent further attacks. The risk of recurrent symptoms after an attack of acute diverticulitis has been variously reported to range from as low as 7% to as high as 62%, with most authors accepting an approximation of one-third to one-quarter as a reasonable estimate of recurrence risk (1, 5, 45, 46, 48–50). Recurrent attacks are less likely to respond to medical therapy and have a higher mortality rate (45, 46); therefore, most authorities agree that elective resection is indicated after two attacks of uncomplicated diverticulitis, although this recommendation has been questioned (51). The risk/benefit analysis of such an approach must be individualized for a specific patient, and consideration must be given to the severity and responsiveness of the attack, general health of the patient and the risk to the patient of a subsequent attack compared to that of surgery itself. The surgical morbidity of such 'elective' resections for recurrent disease may be changing as well, as laparoscopic techniques become more widely applied in this setting. Although a detailed review of such data (*i.e.*, laparoscopic resections for diverticular disease) is beyond our scope here, a number of fairly large series of such patients has been published, with generally favorable results (52–55).

Diverticulitis is relatively uncommon in patients <40 yr old, representing 2–5% of all diverticulitis patients (5, 45); however, there seems to be a significant male predominance in these patients (5, 56, 57). Further, the disease is felt to be more virulent in younger patients, with 25–80% of patients reportedly requiring urgent surgery during their initial attack (5, 56, 58–60). Additionally, it has been reported that among young patients with diverticulitis (<50 yr old), those

who initially responded to conservative medical measures had a significantly higher risk of recurrences or complications than older patients (31, 61, 62). The accuracy of this observation has been questioned, however. Nonetheless, based both on the low operative risk of an elective procedure in an otherwise healthy young person and the many years of risk for recurrence awaiting such patients, surgical resection may be reasonably considered after one well-documented episode of uncomplicated diverticulitis in the younger patient.

Physicians are seeing an increasing number of immunosuppressed patients, who may present with much more subtle signs and symptoms than immunocompetent patients and represent a more difficult diagnostic challenge. Perkins *et al.* (63), in comparing the clinical course of acute diverticulitis in 10 immunosuppressed patients with 76 nonimmunosuppressed ones, found that 24% of the immunocompetent patients failed medical therapy, whereas all of the immunosuppressed patients required surgery. A more recent series compared 40 immunocompromised patients with 169 immunocompetent patients with acute diverticulitis (64). The immunocompromised patients had a higher rate of free perforation (43% *vs* 14%), need for surgery (58% *vs* 33%), and postoperative mortality (39% *vs* 2%) than the noncompromised patients. Because of this high risk of complicated disease, some have advocated elective resection after one attack in an immunosuppressed patient (65).

Up to 10% of patients will have symptomatic recurrent diverticulitis after surgical resection. Re-operation may be required in up to 3% of patients (46, 66, 67), which is often technically more difficult because of inflammation and adhesions. In a series of 501 patients from the Mayo Clinic who had resection and reanastomosis for diverticular disease, a higher recurrence rate was found when the sigmoid colon was used for the distal resection margin as compared to the rectum (66). These authors and others (67) have advocated that the entire distal colon be routinely removed during resections for diverticular disease with a rectal (rather than sigmoid) anastomosis.

COMPLICATIONS OF DIVERTICULITIS

Abscess

***Recommendation**: An abscess should be suspected when clinical improvement is slow to occur, and it is managed based on its size and complexity. Small pericolic abscesses can frequently be managed conservatively, whereas larger abscesses require drainage. CT-guided percutaneous drainage may allow for rapid stabilization and a later, elective, single-stage surgical resection. Multiloculated, inaccessible or poorly responsive abscesses may require initial surgical drainage.*

When perforation of a colonic diverticulum occurs, the ability of the pericolic tissues to control the spread of the inflammatory process will determine subsequent clinical behavior and treatment. When very limited spread occurs, a localized phlegmon develops. Further spread can lead to the formation of larger local or distant abscesses. Generalized peritonitis uncommonly occurs, but requires urgent surgical intervention.

Clinical signs suggesting abscess formation include persistent fever and/or leukocytosis despite an adequate trial of appropriate intravenous antibiotics, or a tender mass on physical examination. Once suspected, radiographic evaluation with CT scanning is the best modality to make the definite diagnosis of an abscess and follow its course over time; CT scanning is also valuable as a guide for percutaneous drainage.

The management of diverticular abscesses should be individualized to their size and complexity. Small pericolic abscesses (Stage I) can often be treated conservatively with antibiotics and bowel rest (68). Their favorable prognosis may be attributed to a persistent fistula between the abscess and the colon, permitting spontaneous internal drainage. In cases where surgery is required, a single-stage en bloc resection and primary re-anastomosis can generally be performed.

For patients with distant abscesses (Stage II) or unresolving pericolic abscesses, drainage is indicated. In prior years, surgery was the sole option, and is still widely used; readers are directed to recently published guidelines from the American Society of Colon and Rectal Surgeons (46). In cases where an adequate bowel prep is possible and there is no significant peritoneal contamination, a single-stage resection and reanastomosis can be performed. When this is not possible, a Hartmann resection is indicated. In general, the single-stage procedure is associated with lower morbidity and mortality than a two-stage procedure.

First introduced in the 1980s (69), CT-scan guided percutaneous drainage of abdominal abscesses has assumed a prominent complementary role with surgery. The immediate advantage of percutaneous catheter drainage is rapid control of sepsis and patient stabilization, without the need for and risk of general anesthesia. More generally, it will often eliminate the need for a two-stage procedure with colostomy, instead allowing for temporary palliative drainage and subsequent single-stage resection in 3–4 wk. Two retrospective series have reported success rates of 74% and 80% in stabilizing patients and safely allowing for subsequent single-stage procedures (70, 71). An initial surgical procedure is required in the 20–25% of patients in whom the abscess is multiloculated, anatomically inaccessible for drainage, or not responding to drainage. A single-stage procedure is preferable, although not always possible (72). Successful laparoscopic resections have also been described for treatment of abscesses (52), although this technique is not yet widely applied.

Pyogenic liver abscesses may also occur as a complication of colonic diverticulitis (73–75). Antibiotics, percutaneous drainage, and surgery each have a role in their management.

Fistulas

Recommendation: *Diverticular fistulas are generally managed surgically.*

When a diverticular phlegmon or abscess extends or ruptures into an adjacent organ, fistulas may occur. In a review of 84 patients with internal fistulas caused by diverticular disease (76), 65% were colovesicular. There was a 2:1 male predominance, attributed to the protection given the bladder by the uterus, and supported by the observation that 50% of women with such fistulas have had a previous hysterectomy. Pneumaturia and fecaluria are common symptoms (77). Cystoscopy, cystography, and BE are useful diagnostically. Single-stage operative resection with fistula closure and primary anastomosis can be performed in ~75% of patients (76, 77).

Colovaginal fistulas are the next most common internal fistula, representing approximately 25% of all cases (76). The passage of stool or flatus per vagina is pathognemonic. Treatment is surgical resection with fistula closure. Coloenteric, colouterine, and coloreteral fistulas occur, but much less commonly. Spontaneous colocutaneous fistulas are very rare and more frequently follow prior surgical repair.

Obstruction

Recommendation: *Acute obstruction during an episode of acute diverticulitis is usually self-limited and responds well to conservative therapy. Chronic strictures usually require colonoscopy to exclude neoplasia, and if symptomatic, may be managed endoscopically or surgically.*

Obstruction may accompany diverticular disease either acutely or chronically. During an attack of acute diverticulitis, partial colonic obstruction can occur because of relative lumenal narrowing from the pericolic inflammation and/or compression from abscess formation. Complete obstruction is unusual. Colonic ileus or pseudo-obstruction can also occur. These conditions usually improve with effective medical therapy. Should obstruction fail to resolve, prompt surgical consultation is indicated. Acute diverticulitis can cause small bowel obstruction as well, either mechanically if a loop of small intestine becomes incorporated into the inflammatory mass, or by localized irritation and the development of an ileus. The obstruction should improve as the inflammation subsides. Surgical intervention may be required if persistent.

Recurrent attacks of diverticulitis, which may be subclinical, can initiate progressive fibrosis and stricturing of the colonic wall in the absence of ongoing inflammation. In such cases, high grade or complete obstruction can occur requiring surgical therapy. A more insidious presentation with nonspecific symptoms is not uncommon. Frequently, a stricture is demonstrated on BE, with uncertain etiology. The critical issue is to distinguish between a diverticular stricture and a stenosing neoplasm, as an accurate diagnosis will guide the correct surgical procedure if needed. Colonoscopy has been reported to be diagnostic in up to two of three of such patients (78). Strictures in which malignancy cannot be excluded despite colonoscopic and radiographic examinations should undergo surgical resection. In those in which neoplasm is felt to be sufficiently excluded, a trial of endoscopic therapy can be attempted. A number of reports have described the relative safety and efficacy of such therapy for strictures of various etiologies, utilizing varied techniques such as bouginage, balloon, laser, electrocautery, and a blunt dilating endoscope (79–82).

HEMORRHAGE

Diverticula and vascular ectasias are responsible for the majority of episodes of lower GI bleeding (83–86), although a precise determination of each of these lesions' respective contributions is difficult. Nonetheless, three recent large series of patients with lower gastrointestinal (GI) bleeding have recently reported their results, comprising over 500 patients in total (87–89). In each study, diverticular bleeding was the most common etiology identified, comprising 24–42% of episodes.

Severe hemorrhage has been reported to occur in 3–5% of all patients with diverticulosis (83, 90). Despite the fact that most diverticula are located in the left colon in Western individuals, a number of series suggest that bleeding diverticula may occur more often in the proximal colon (83, 90–94). Together, these reports argue strongly against empiric left-hemicolectomy or segmental sigmoid-colectomy in patients with severe rectal bleeding in the absence of definitive localization of the bleeding.

The association of nonsteroidal anti-inflammatory drug (NSAID) use with peptic ulcer disease and bleeding is well documented. Recent data have also implicated these agents in diverticular bleeding (95, 96). Whether patients with diverticulosis should be counseled to avoid NSAIDs, as is done for ulcer patients, is still conjecture, as is the potential role of selective COX-2 antagonist NSAIDs.

Pathophysiology

Diverticular bleeding is arterial, and thought to be caused by medial thinning of the vasa recta as it courses over the dome of a diverticulum (91). The factor(s) that initiate this isolated, asymmetric arterial change or the precipitating events leading to its rupture are unknown. Inflammation does not seem to be a contributing factor as it is absent histologically in resected bleeding diverticula. This is in concordance with the general clinical impression that bleeding rarely, if ever, complicates diverticulitis.

Clinical Features

The clinical presentation of patients with diverticular hemorrhage is usually one of an abrupt, painless onset. The patient may have mild lower abdominal cramps and the urge to defecate, followed by the passage of voluminous red or maroon blood or clots. Melena is uncommon. The presence of colonic diverticula should not be considered an adequate

explanation for a positive fecal occult blood test or iron deficiency anemia.

The natural history of diverticular hemorrhage has been well described. Bleeding will cease spontaneously in 70–80% of patients. Rebleeding rates range from 22 to 38% (90, 93). The chance of a third bleed after a second episode may be as high as 50% (90), leading some to recommend surgical resection after a second bleeding episode.

Diagnosis and Management

Recommendation: *The diagnosis and treatment of lower GI bleeding from diverticula requires a coordinated approach by gastroenterologists, radiologists, and surgeons. After resuscitation, diagnostic options include nuclear bleeding scans, angiography, and colonoscopy; the choice of a particular modality will depend on the clinical scenario and local expertise. Angiography and colonoscopy may be therapeutically useful in patients with ongoing bleeding, and surgery may be required in those in whom these are unsuccessful.*

The comprehensive management of patients with lower GI bleeding is beyond the scope of these guidelines; the reader is referred to the recently published American College of Gastroenterology Practice Guidelines on lower GI bleeding (97). Fluid and blood product resuscitation requires immediate attention. Excluding an upper GI source by nasogastric lavage or upper endoscopy is warranted, as 10–15% of patients with hematochezia will have an upper tract etiology. Urgent flexible sigmoidoscopy is an appropriate initial approach. If no obvious etiology is found, then further evaluation with noninvasive (nuclear scintigraphy) or invasive techniques (angiography, colonoscopy) can be undertaken in an attempt to localize and/or treat the bleeding source.

The role of endoscopic therapy in acute diverticular bleeding is being refined. A case report in 1985 first described cessation of hemorrhage from an actively bleeding diverticulum by local irrigation with 1:1000 epinephrine (98). Later reports have demonstrated the hemostatic abilities of the heater probe (99), bicap probe (100), injection therapies (101, 102), and fibrin sealant (103) in patients with bleeding diverticula. Foutch recently reported on 13 patients with acute lower GI bleeding in whom a specific diverticulum was 'unequivocally' identified as the cause of bleeding (104, 105), and described endoscopic 'stigmata' thought to have prognostic values, similar to those associated with peptic ulcers. Cumulative results from 22 patients in nine studies of endoscopically treated diverticular bleeding reveal a 95% hemostasis rate with no morbidity (106). Although this intervention is promising, more controlled data will be required before endoscopic therapy becomes a standard intervention in this setting.

Surgery in acute lower GI bleeding is usually reserved until medical, endoscopic, or angiographic therapies fail. Segmental resection is most commonly performed if the bleeding site is definitively known from a therapeutically unsuccessful angiographic or endoscopic procedure. The rebleeding rate compiled from seven series was 6% in 167 patients who underwent segmental resections for angiographically documented bleeding sites (107). In patients with persistent bleeding, and no angiographic or endoscopic identification of a definite bleeding site, a subtotal colectomy may be required.

Treatment Outcome and Follow-up

Recommendation*: Colonoscopy should generally be performed in patients after an episode of lower GI bleeding to elucidate the bleeding source and exclude neoplasia.*

For the majority of patients, diverticular bleeding is self-limited. Subsequent colonoscopy should generally be performed to potentially elucidate the bleeding source, but more importantly to exclude neoplasia. In a review of over 2000 colonoscopies for overt or occult rectal bleeding, neoplastic polyps were found in 32% and carcinoma in 19% (108). Another retrospective study looked at the yield of colonoscopy in 258 patients with rectal bleeding who had had negative proctosigmoidoscopies, and single-contrast BEs that were normal or showed only diverticulosis (109). The overall incidence of significant findings was 41%, including carcinoma in 29 patients (11%) and telangiectasias in 17 (7%). A more recent prospective study from Ontario found colonoscopy more sensitive than the combination of sigmoidoscopy and barium examination for the diagnosis of adenoma, carcinoma, and angiodysplasia (110). The increased ability to diagnose vascular ectasias and diverticula, which cause the majority of bleeding, as well as the therapeutic potential of colonoscopy all support its utility as a primary investigative modality in patients with lower GI bleeding.

Reprint requests and correspondence: Neil H. Stollman, M.D., F.A.C.P., 1201 N.W. 16th Street, Gastroenterology (D-1007), Miami, FL 33125.

Received July 13, 1999; accepted July 21, 1999.

REFERENCES

1. Almy TP, Howell DA. Diverticular disease of the colon. N Engl J Med 1980;302:324–31.
2. Painter NS, Burkitt DP. Diverticular disease of the colon: A deficiency disease of western civilization. Br Med J 1971;2:450–4.
3. Parks TG. Natural history of diverticular disease of the colon. Clin Gastroenterol 1975;4:53–69.
4. Painter NS, Burkitt DP. Diverticular disease of the colon, a 20th century problem. Clin Gastroenterol 1975;4:3–21.
5. Roberts PL, Veidemheimer MC. Current management of diverticulitis. Adv Surg 1994;27:189–208.
6. Slack WW. The anatomy, pathology and some clinical features of diverticulitis of the colon. Br J Surg 1962;50:185–90.
7. Lee Y-S. Diverticular disease of the large bowel in Singapore: An autopsy study. Dis Colon Rectum 1986;29:330–5.
8. Chia JG, Wilde CC, Ngoi SS, et al. Trends of diverticular

disease of the large bowel in a newly developed country. Dis Colon Rectum 1991;34:498–501.
9. Whiteway J, Morson BC. Elastosis in diverticular disease of the sigmoid colon. Gut 1985;26:258–66.
10. Arfwidsson S, Kock NG, Lehmann L, et al. Pathogenesis of multiple diverticula of the sigmoid colon in diverticular diseases. Acta Chir Scand 1964;342(suppl):1–68.
11. Painter NS. The aetiology of diverticulosis of the colon with special reference to the action of certain drugs on the behavior of the colon. Ann R Coll Surg Engl 1964;34:98–119.
12. Trotman IF, Misiewicz JJ. Sigmoid motility in diverticular disease and the irritable bowel syndrome. Gut 1988;29:218–22.
13. Sugihara K, Muto T, Morioka Y. Motility study in right sided diverticular disease of the colon. Gut 1983;24:1130–4.
14. Painter NS, Truelove SC, Ardran GM, et al. Segmentation and localization of intraluminal pressures in the human colon, with special reference to the pathogenesis of colonic diverticula. Gastroenterology 1965;49:169–77.
15. Burkitt DP, Walker ARP, Painter NS. Effect of dietary fibre on stools and transit times, and its role in the causation of disease. Lancet 1972;2:1408–11.
16. Fisher N, Berry CS, Fearn T, et al. Cereal dietary fiber consumption and diverticular disease: A lifespan study in rats. Am J Clin Nutr 1985;43:788–804.
17. Aldoori WH, Giovannucci EL, Rockett HRH, et al. A prospective study of dietary fiber types and symptomatic diverticular disease in men. J Nutr 1998;128:714–9.
18. Aldoori WH, Giovannucci EL, Rimm EB, et al. Prospective study of physical activity and the risk of symptomatic diverticular disease in men. Gut 1995;36:276–82.
19. Aldoori WH, Giovannucci EL, Rimm EB, et al. A prospective study of alcohol, smoking, caffeine, and the risk of symptomatic diverticular disease in men. Ann Epidemiol 1995;5:221–8.
20. Otte JJ, Larsen L, Anderson JR. Irritable bowel syndrome and symptomatic diverticular disease—different diseases? Am J Gastroenterol 1986;81:529–31.
21. Ritchie J. Similarity of bowel distension characteristics in the irritable colon syndrome and diverticulosis. Gut 1977;18: A990 (abstract).
22. Boulos PB, Karamanolis DG, Salmon PR, et al. Is colonoscopy necessary in diverticular disease? Lancet 1984;1:95–6.
23. Williams CB, Saunders BP. Technique of colonoscopy. In: Raskin JB, Nora HJ, eds. Colonoscopy: Principles and techniques. New York: Igaku-Shoin Medical Publishers, Inc., 1995:121–42.
24. Kozarek RA, Botoman VA, Patterson DJ. Prospective evaluation of a small caliber upper endoscope for colonoscopy after unsuccessful standard examination. Gastrointest Endosc 1989;35:333–5.
25. Brodribb AJM. Treatment of symptomatic diverticular disease with a high fibre diet. Lancet 1977;1:664–5.
26. Ornstein MH, Littlewood ER, Baird IM, et al. Are fiber supplements really necessary in diverticular disease of the colon? A controlled clinical trial. Br Med J 1981;282: 1353–6.
27. Daniel O, Basup K, Al-Samarrae HM. Use of glucagon in the treatment of acute diverticulitis. Br Med J 1974;3:720–2.
28. Berman LG, Burdick D, Heitzman ER, et al. A critical reappraisal of sigmoid peridiverticulitis. Surg Gyn Obstet 1968;127:481–91.
29. Williams RA, Davis IP. Diverticular disease of the colon. In: Haubrich WS, Schaffner F, eds. Bockus gastroenterology, 5th ed. Philadelphia: WB Saunders, 1995:1637–56.
30. Hinchey EJ, Schaal PH, Richards MB. Treatment of perforated diverticular disease of the colon. Adv Surg 1978;12: 85–109.
31. Ambrosetti P, Robert JH, Witzig J, et al. Acute left colonic diverticulitis: A prospective analysis of 226 consecutive cases. Surgery 1994;115:546–50
32. McKee RF, Deignan RW, Krukowski ZH. Radiological investigation in acute diverticulitis. Br J Surg 1993;80:560–5.
33. Doringer E. Computerized tomography of colonic diverticulitis. Crit Rev Diag Imag 1992;33:421–35.
34. Hulnick DH, Megibow AJ, Balthazar EJ, et al. Computed tomography in the evaluation of diverticulitis. Radiology 1984;152:491–5.
35. Cho KC, Morehouse HT, Alterman DD, et al. Sigmoid diverticulitis: Diagnostic role of CT-comparison with barium enema studies. Radiology 1990;176:111–5.
36. Stefansson T, Nyman R, Nilsson S, et al. Diverticulitis of the sigmoid colon: A comparison of CT, colonic enema, and laparoscopy. Acta Radiologica 1997;38:313–9.
37. Ambrosetti P, Grossholz M, Becker C, et al. Computed tomography in acute left colonic diverticulitis. Br J Surg 1997;84:532–4.
38. Verbanck J, Lambrecht S, Rutgeerts L, et al. Can sonography diagnose acute colonic diverticulitis in patients with acute intestinal inflammation? A prospective study. J Clin Ultrasound 1989;17:661–6.
39. Schwerk WB, Schwarz S, Rothmund M. Sonography in acute colonic diverticulitis: A prospective study. Dis Colon Rectum 1992;35:1077–84.
40. Zielke A, Hasse C, Nies C, et al. Prospective evaluation of ultrasonography in acute colonic diverticulitis. Br J Surg 1997;84:385–8.
41. Pradel JA, Adell J-F, Taourel P, et al. Acute colonic diverticulitis: Prospective comparative evaluation with US and CT. Radiology 1997;205:503–12.
42. Eggesbo HB, Jacobsen T, Kolmannskog F, et al. Diagnosis of acute left-sided colonic diverticulitis by three radiological modalities. Acta Radiol 1998;39:315–21.
43. Chow AW. Appendicitis and diverticulitis. In: Hoeprich PD, Jordan MC, Ronald AR, eds. Infectious diseases: A treatise of infectious processes. Philadelphia: JB Lippincott, 1994: 878–81.
44. Kellum JM, Sugerman HJ, Coppa GF, et al. Randomized, prospective comparison of cefoxitin, and gentamicin-clindamycin in the treatment of acute colonic diverticulitis. Clin Ther 1992;14:376–84.
45. Parks TG. Natural history of diverticular disease of the colon: A review of 521 cases. Br Med J 1969;4:639–45.
46. The Standards Task Force of the American Society of Colon and Rectal Surgeons. Practice parameters for sigmoid diverticulitis-supporting documentation. Dis Colon Rectum 1995; 38:126–32.
47. Elliot TB, Yego S, Irvin TT. Five-year audit of the acute complications of diverticular disease. Br J Surg 1997;84: 535–9.
48. Munson KD, Hensien MA, Jacob LN, et al. Diverticulitis. A comprehensive follow-up. Dis Colon Rectum 1996;39:318–22.
49. Larson DM, Masters SS, Spiro H. Medical and surgical therapy in diverticular disease: A comparative study. Gastroenterology 1976;71:734–7.
50. Parks TG, Connell AM. The outcome in 455 patients admitted for treatment of diverticular disease of the colon. Br J Surg 1970;57:775–8.
51. Lorimer JW. Is prophylactic resection valid as an indication for elective surgery in diverticular disease? Can J Surg 1997; 40:445–8.
52. Franklin ME, Dorman JP, Jacobs M, et al. Is laparoscopic

surgery applicable to complicated colonic diverticular disease? Surg Endosc 1997;11:1021–5.
53. Stevenson ARL, Stitz RW, Lumley JW, et al. Laparoscopically assisted anterior resection for diverticular disease: Follow-up of 100 consecutive patients. Ann Surg 1998;227:335–42.
54. Eijsbouts QAJ, Cuesta MA, de Brauw LM, et al. Elective laparoscopic-assisted sigmoid resection for diverticular disease. Surg Endosc 1997;11:750–3.
55. Sher ME, Agachan F, Bortul M, et al. Laparoscopic surgery for diverticulitis. Surg Endosc 1997;11:264–7.
56. Konvolinka CW. Acute diverticulitis under age forty. Am J Surg 1994;167:562–5.
57. Acosta JA, Grebenc ML, Doberneck RC, et al. Colonic diverticular disease in patients 40 years old or younger. Am Surg 1992;58:605–7.
58. Freischlag J, Bennion RS, Thompson JE. Complications of diverticular disease of the colon in young people. Dis Colon Rectum 1986;29:639–43.
59. Vignati PV, Welch JP, Cohen JL. Long-term management of diverticulitis in young patients. Dis Colon Rectum 1995;38:627–9.
60. Spivak H, Weinrauch S, Harvey JC, et al. Acute colonic diverticulitis in the young. Dis Colon Rectum 1997;40:570–4.
61. Ambrosetti P, Robert J, Witzig JA, et al. Prognostic factors from computed tomography in acute left colonic diverticulitis. Br J Surg 1992;79:117–9.
62. Cunningham MA, Davis JW, Kaups KL. Medical versus surgical management of diverticulitis in patients under age 40. Am J Surg 1997;174:733–6.
63. Perkins JD, Shield CF, Chang FC, et al. Acute diverticulitis: Comparison of treatment in immunocompromised and nonimmunocompromised patients. Am J Surg 1984;148:745–8.
64. Tyau ES, Prystowsky JB, Joehl RJ, et al. Acute diverticulitis: A complicated problem in the immunocompromised patient. Arch Surg 1991;126:855–9.
65. Schoetz DJ. Uncomplicated diverticulitis: Indications for surgery and surgical management. Surg Clin N Am 1993;73:965–74.
66. Benn PL, Wolff, BG, Ilstrup DM. Level of anastomosis and recurrent colonic diverticulitis. Am J Surg 1986;151:269–71.
67. Frizelle FA, Dominguez JM, Santoro GA. Management of post-operative recurrent diverticulitis: A review of the literature. J R Coll Surg Edinb 1997;42:186–8.
68. Ambrosetti P, Robert J, Witzig JA, et al. Incidence, outcome, and proposed management of isolated abscesses complicating acute left-sided colonic diverticulitis: A prospective study of 140 patients. Dis Colon Rectum 1992;35:1072–6.
69. Gerzof SG, Robbins AH, Johnson WC, et al. Percutaneous catheter drainage of abdominal abscesses. N Engl J Med 1981;305:653–7.
70. Schecter S, Eisenstat TE, Oliver GC, et al. Computed tomographic scan-guided drainage of intra-abdominal abscesses. Dis Colon Rectum 1994;37:984–8.
71. Stabile BE, Puccio E, Van Sonnenberg E, et al. Preoperative percutaneous drainage of diverticular abscesses. Am J Surg 1990;159:99–105.
72. Wedell J, Banzhaf G, Chaoui R, et al. Surgical management of complicated colonic diverticulitis. Br J Surg 1997;84:380–3.
73. Yoshida M, Mitsuo M, Kutsumi H, et al. A successfully treated case of multiple liver abscesses accompanied by portal venous gas. Am J Gastroenterol 1996;91:2423–5.
74. Nosher JL, Guidici M, Needell GS, et al. Elective one-stage abdominal operations after percutaneous catheter drainage of pyogenic liver abscess. Am Surg 1993;59:658–63.
75. Read DR, Hambrick E. Hepatic abscesses in diverticulitis. South Med J 1980;73:881–3.
76. Woods RJ, Lavery IC, Fazio VW, et al. Internal fistulas in diverticular disease. Dis Colon Rectum 1988;31:591–6.
77. McBeath RB, Schiff M, Allen V, et al. A 12 year experience with enterovesical fistulas. Urology 1994;44:661–5.
78. Forde KA, Treat MR. Colonoscopy in the evaluation of strictures. Dis Colon Rectum 1985;28:699–701.
79. Kozarek RA. Hydrostatic balloon dilation of gastrointestinal stenoses: A national survey. Gastrointest Endosc 1986;32:15–9.
80. Manten HD, Zara J, Raskin JB, et al. Balloon dilation of recto-sigmoid strictures: Transendoscopic approach. Gastrointest Endosc 1986;32:A164 (abstract).
81. Oz MC, Forde KA. Endoscopic alternatives in the management of colonic strictures. Surgery 1990;108:513–9.
82. Blomberg B, Rolny P, Jarnerot G. Endoscopic treatment of anastomotic strictures in Crohn's disease. Endoscopy 1991;23:195–8.
83. Reinus JF, Brandt LJ. Vascular ectasias and diverticulosis: Common causes of lower intestinal bleeding. Gastroenterol Clin N Am 1994;23:1–20.
84. Potter GD, Sellin JH. Lower gastrointestinal bleeding. Gastroenterol Clin N Am 1988;17:341–55.
85. Boley SJ, DiBiase A, Brandt LJ, et al. Lower intestinal bleeding in the elderly. Am J Surg 1979;137:57–64.
86. Gostout CJ, Wang KK, Ahluist DA, et al. Acute gastrointestinal bleeding: Experience of a specialized management team. J Clin Gastroenterol 1992;14:260–7.
87. Peura DA, Lanza FL, Gostout CJ, et al. The American College of Gastroenterology Bleeding Registry: Preliminary findings. Am J Gastroenterol 1997;92:924–8.
88. Bramley PN, Masson JW, McKnight G, et al. The role of an open-access bleeding unit in the management of colonic haemorrhage. A 2-year prospective study. Scand J Gastroenterol 1996;31:764–9.
89. Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: A population-based study. Am J Gastroenterol 1997; 92:419–24.
90. McGuire HH, Haynes BW. Massive hemorrhage from diverticulosis of the colon: Guidelines for therapy based on bleeding patterns observed in 50 cases. Ann Surg 1972;175:847–55.
91. Meyers MA, Alonso DR, Gray GF, et al. Pathogenesis of bleeding colonic diverticulosis. Gastroenterology 1976;71:577–83.
92. Casarella WJ, Kanter IE, Seaman WB. Right-sided colonic diverticula as a cause of acute rectal hemorrhage. N Engl J Med 1972;286:450–3.
93. McGuire HH. Bleeding colonic diverticula: A reappraisal of natural history and management. Ann Surg 1994;220:653–6.
94. Wong S-K, Ho Y-H, Leong APK, et al. Clinical behavior of complicated right-sided and left-sided diverticulosis. Dis Colon Rectum 1997;40:344–8.
95. Wilcox CM, Alexander LN, Cotsonis GA, et al. Nonsteroidal antiinflammatory drugs are associated with both upper and lower gastrointestinal bleeding. Dig Dis Sci 1997;42:990–7.
96. Aldoori WH, Giovannucci EL, Rimm EB, et al. Use of acetaminophin and nonsteroidal anti-inflammatory drugs. A prospective study and the risk of symptomatic diverticular disease in men. Arch Fam Med 1998;7:255–60.
97. Zuccaro G. Practice guidelines. Management of the adult patient with acute lower gastrointestinal bleeding. Am J Gastroenterol 1998;93:1202–8.
98. Mauldin JL. Therapeutic use of colonoscopy in active diverticular bleeding. Gastrointest Endosc 1985;31:290–1.

99. Johnston J, Sones J. Endoscopic heater probe coagulation of the bleeding colonic diverticulum. Gastrointest Endosc 1986; 32:A160 (abstract).
100. Savides TJ, Jensen DM. Colonoscopic hemostasis for recurrent diverticular hemorrhage associated with a visible vessel: A report of 3 cases. Gastrointest Endosc 1994;40:70–3.
101. Bertoni G, Conigliaro R, Ricci E, et al. Endoscopic injection hemostasis of colonic diverticular bleeding: A case report. Endoscopy 1990;22:154–5.
102. Kim YI, Marcon NE. Injection therapy for colonic diverticular bleeding: A case study. J Clin Gastroenterol 1993;17: 46–8.
103. Andress HJ, Mewes A, Lange V. Endoscopic hemostasis of a bleeding diverticulum of the sigma (sic) with fibrin sealant. Endoscopy 1993;25:193.
104. Foutch PG. Diverticular bleeding: Are nonsteroidal anti-inflammatory drugs risk factors for hemorrhage and can colonoscopy predict outcome for patients. Am J Gastroenterol 1995;90:1779–84.
105. Foutch PG. Diverticular bleeding and the pigmented protuberance (sentinel clot): Clinical implications, histopathological correlation, and results of endoscopic intervention. Am J Gastroenterol 1996;91:2589–93.
106. Foutch PG. Utility of endoscopic hemoclipping for colonic diverticular bleeding (Response to Dr. Hokama). Am J Gastroenterol 1997;92:543–5 (letter).
107. Browder W, Cerise EJ, Litwin MS. Impact of emergency angiography in massive lower gastrointestinal bleeding. Ann Surg 1986;204:530–6.
108. Shinya H, Cwern M, Wolf G. Colonoscopic diagnosis and management of rectal bleeding. Surg Clin N Am 1982;62: 897–903.
109. Tedesco FJ, Waye JD, Raskin JB, et al. Colonoscopic evaluation of rectal bleeding: A study of 304 patients. Ann Intern Med 1978;89:907–9.
110. Irvine EJ, O'Connor J, Frost RA. Prospective comparison of double contrast barium enema plus flexible sigmoidoscopy versus colonoscopy in rectal bleeding. Gut 1988;29:1188–93.