GI

MEDICAL GRAND ROUNDS

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# DIVERTICULAR DISEASE OF THE COLON

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## I. Introduction and definition

Traditionally, diverticulosis of the colon was considered to be an anatomical abnormality which was associated with no symptoms unless bleeding or inflammation supervened. Diverticulitis was thought to be inflammation within a diverticulum, and medical treatment of "resolved" or chronic diverticulitis consisted of the institution of low residue diets. However, as a result of a number of pathological, radiological, physiological, and epidemiological studies, these traditional concepts of the pathogenesis, manifestations, and treatment have undergone revision (1-11).

The phrase "diverticular disease of the colon" has been used to encompass and emphasize three apparently overlapping stages in the formation and complications of diverticulosis (12-15).

I. Prediverticular disease has many of the pathological, physiological, and clinical features of diverticulosis but without obvious diverticula.
II. Diverticulosis is the presence of uninflamed diverticula of the colon.
III. Diverticulitis is the association of diverticula with inflammation which probably occurs only after a diverticulum is perforated.

## II. Incidence and epidemiology

Most studies indicate that in Western Europe and the United States the incidence of diverticular disease increases with advancing age (16-20). A radiological survey of healthy subjects demonstrates colonic diverticula in about 8 percent of the adult population and in about one-third of individuals over age 60. The overall incidence of diverticulosis in necropsy series in patients over 60 years of age ranges from 20 to 50 percent (17,19).

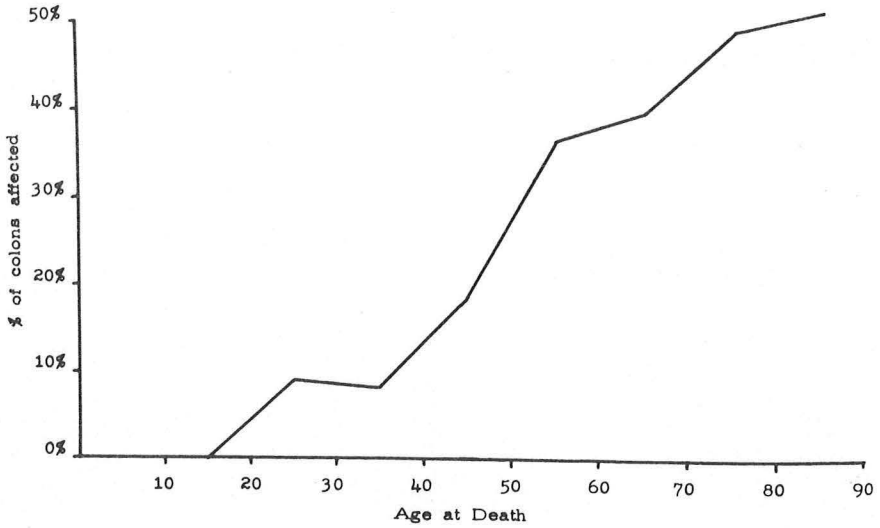



Figure 1. Increasing incidence of diverticular disease found at autopsy. From Reference 20.

Diverticula are rarely found in individuals less than 30 years of age, but recent reports indicate that complicated diverticular disease occurs in young individuals (21,22).

The world-wide distribution of diverticular disease has been a focus of considerable attention. Many anecdotal reports and several systematic studies indicate that diverticular disease is rare in underdeveloped countries, especially in Africa and Asia (4,11,23-25). Diverticular disease is reported to be increasing in incidence in the Western world. (Figure 2)

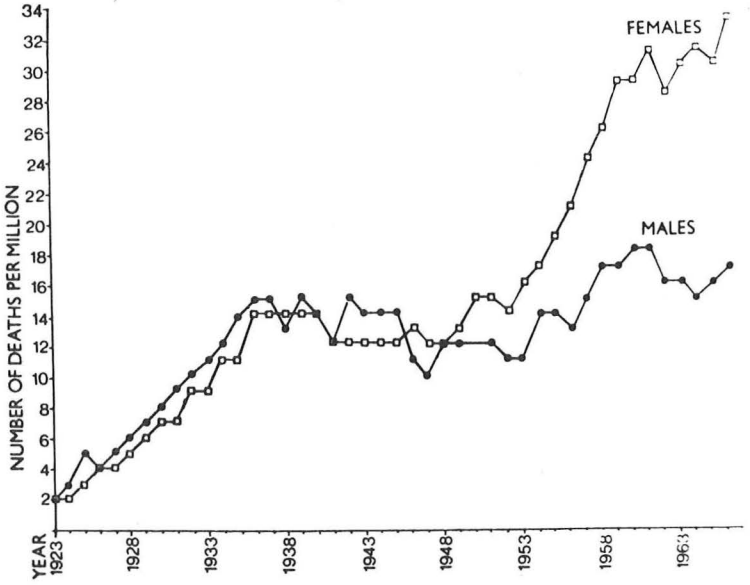



Figure 2. Crude death rate from diverticular disease. From Lancet, Sept. 13, 1969.

There does not appear to be a racial predilection because the incidence of diverticular disease in native Africans or Japanese who settle in Western countries increases with time (24-28). Furthermore, in the industrialized areas in Africa, diverticular disease is now being recognized with increasing frequency.

## III. Diverticulosis

A. Pathology. Diverticula are herniations of the mucosa and submucosa between the muscle fibers of the inner circular muscle of the colon and are pulsion or false diverticula (Figure 3). The out-pouchings are usually found between the mesenteric (taenia mesocolica) and lateral taeniae (taenia omentalis and taenia libera).

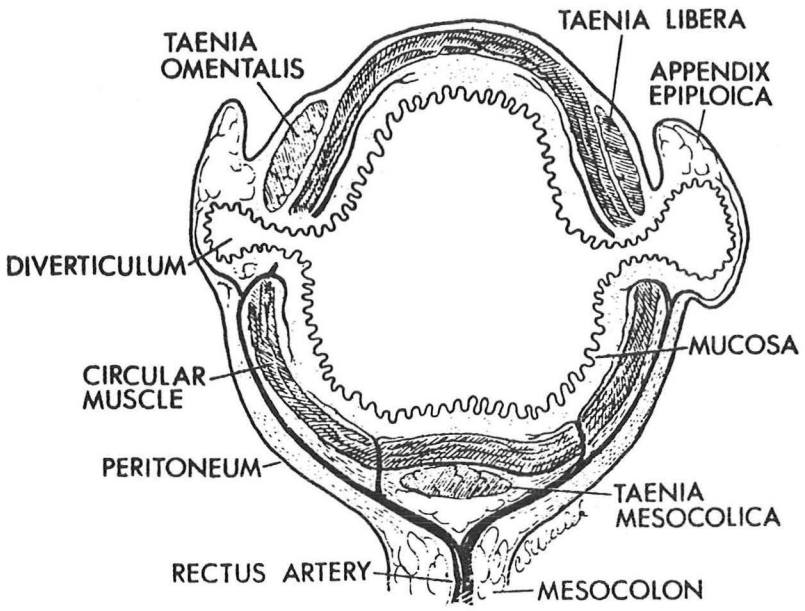



Figure 3. From Berman and Kirsner (2).

95%. in Sigmoid

The sigmoid colon is the site of diverticula in about 95 percent of patients (19,29). Involvement of the proximal colon with diverticula occurs less frequently, appears to increase in incidence with the duration of the disease, and is almost always associated with contiguous involvement distally.

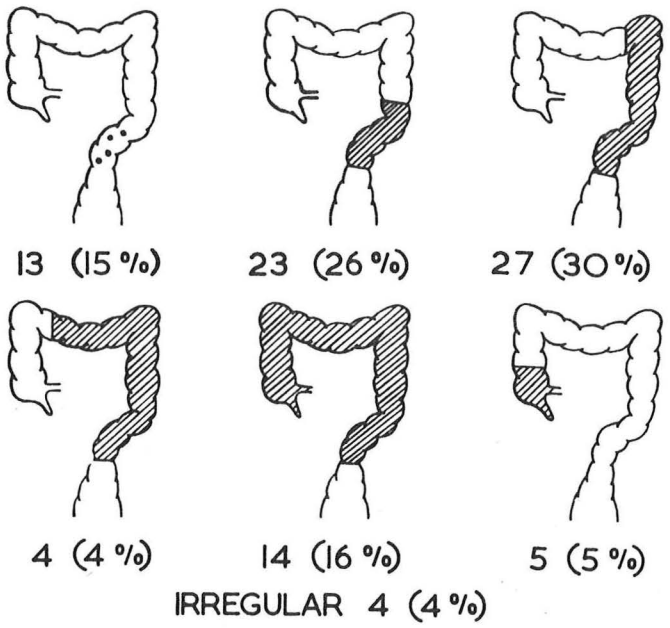



Figure 4. Site of diverticula at necropsy. In 200 cases diverticula were present in 90. From Parks (20).

The diverticula vary in size from 1 mm to 7 cm, and they may vary in number from a few to hundreds. Occasionally a solitary diverticulum may be found, particularly in the cecum or sigmoid colon. Rarely, a giant diverticulum is present, usually in the sigmoid colon. Because the longitudinal muscle completely encircles the rectum, diverticula in this area are rare (30).

Careful dissections in many resected and necropsy specimens of colon containing diverticula demonstrate that there is high incidence (50 to 70 percent) of thickening or enlargement of both the circular and longitudinal muscles (13,19,31,32). The results of studies performed to determine if this muscular enlargement is due to hyperplasia, hypertrophy, or muscular shortening are conflicting but the best studies suggest that hypertrophy is not present (13,32-34). It is certain, however, that colonic muscular enlargement can be present without associated pathological evidence or a clinical history of diverticulitis.

Many patients who have had radiological demonstration of diverticula are subsequently found to have no diverticula at post-mortem examination (3). Careful histological studies in the colons of these patients sometimes demonstrate wide interfascicular connective tissue gaps or clefts in the circular muscle through which mucosal herniations may have previously passed. Thus, diverticula of the colon may sometimes be reducible. A prediverticula state has been identified in a number of patients. Radiographic and pathological studies in these patients revealed thickening or spasm of colonic musculature but no diverticula (12,13,35,36).

Despite the widespread emphasis on the muscle abnormality of diverticular disease, some patients have diverticula in great numbers without muscle thickening (19,33). Fleischner coined the term "spastic colon diverticulosis" or myochosis coli for the condition in which diverticula are associated with muscular enlargement (3,33). He distinguishes a second type of diverticulosis, "simple massed diverticulosis" in which shortening and narrowing of the colon is produced by multiple herniations of the mucosa and muscular thickening is absent. This is a poorly defined entity, and it has not been confirmed if muscular abnormalities are absent as suggested by Fleischner.

Vascular relation. Intra-arterial barium-gelatin injections with high resolution radiography, microdissection, and histologic sections have provided detailed information concerning the angioarchitecture of colonic diverticula (37). The vasa recta reach the bowel wall at the mesenteric taenia and divide into branches which course subserosally (Figures 3 & 5). These vessels penetrate the circular muscle obliquely from the mesenteric side of bowel between the mesenteric and lateral taeniae and provide a rich submucosal plexus around the circumference of the colon. The largest vasa recta

penetrate the colon in four loci; on either side of the mesenteric taenia and adjacent to the mesenteric side of the two lateral taeniae.

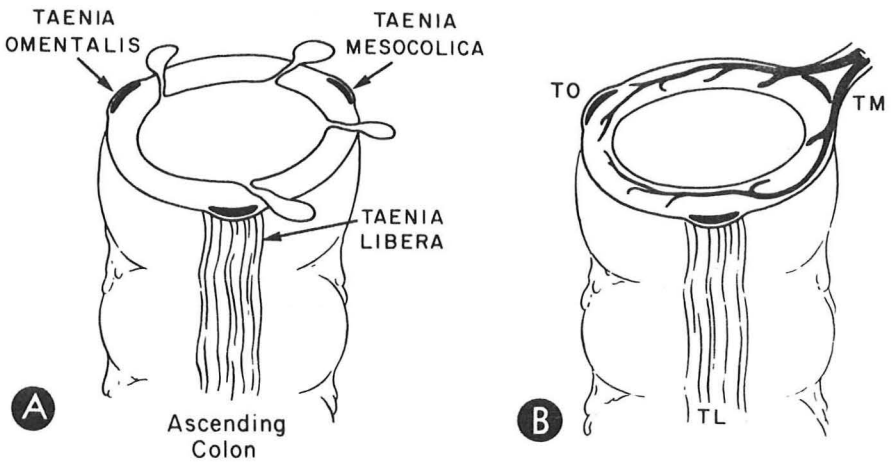



Figure 5. Site of diverticula formation.
From Meyers et al (37).

The vasa recta penetrate the circular muscle (cm) through connective tissue clefts which are obliquely oriented and separate the muscle fascicles. The mucosa herniations occur adjacent to these vessels through the clefts. This suggests that the inter-fascicular connective tissue clefts are the path of least resistance for herniation (3,37-39). As the hernia sac protrudes through these clefts it carries the obliquely penetrating vasa recta over its dome. (Figure 6)

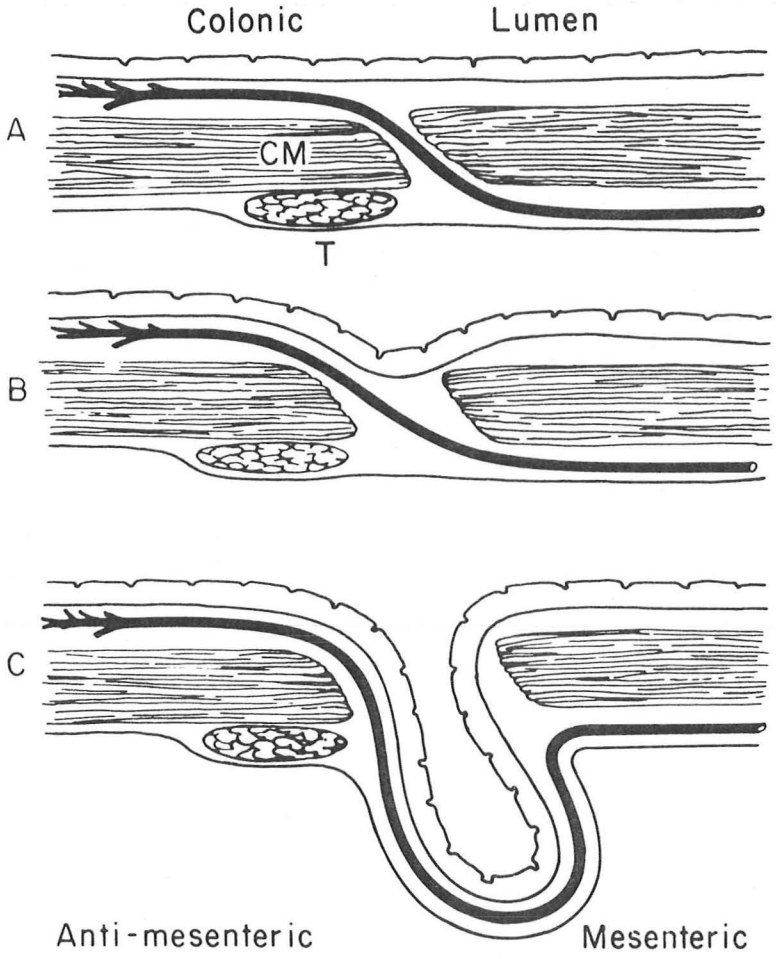



Figure 6. Structural dynamics of diverticula formation and vascular relationships. From Meyers et al (37).

Once herniation occurs, there is prominent vasculature associated with a diverticulum over its dome, and at the antimesenteric border of its orifice. The submucosal vascular plexus also crowds around the neck of the diverticulum. In some cases, four rows of obliquely oriented diverticula can be demonstrated at the sites of penetration of the largest vessels. (Figure 5)

Right sided diverticula. One or more diverticula are found in the cecum or right colon in one to ten percent of patients (40-42). Most are associated with left-sided diverticulosis and/or muscular enlargement of the sigmoid colon. These are usually false diverticula since they do not contain the circular muscle layer in their wall. However, other types of cecal or right-sided diverticulosis have been identified. Solitary cecal diverticula can be true diverticula containing all layers of the bowel wall and may be congenital. Traction diverticula also occur, usually in the ascending colon related to

calcified tuberculous mesentery lymph nodes (43). In Oriental races, diverticulosis confined to the cecum and ascending colon has been reported. A peculiar thickening of the orifices of diverticula due to mucosa lymphoid hyperplasia is described in a few of the cases (44).

B. Pathogenesis. The pathogenesis of diverticular disease of the colon has not been completely elucidated. However, the results of epidemiological, pathological, radiological, and manometric studies have provided a framework upon which several interesting theories have been constructed. In order for a mucosal herniation to occur, colonic wall stretch must exceed the focus of least resistance in the colonic wall. Thus, herniations could result from a primary degeneration in the connective tissue clefts between the muscle bundles. Alternatively, excessive muscle contractions could separate and weaken the connective tissue clefts. Finally, increased intraluminal pressure could result in increased stretch or tension on the colonic wall.

1. Degen of clefts
2. ↑ muscle contr
3. ↑ pressure

1. Colonic wall abnormalities. As stated previously, diverticula occur at specific locations in the colonic wall where the larger vessels penetrate through interfascicular connective tissue in the circular muscle layer of the colon. Separation or degeneration of this connective tissue could allow herniation to occur. No vascular abnormalities have been found (19), but the abnormally thickened or spastic muscles might distort or stretch these potentially weaker connective tissue fibers between the muscle bundles and allow mucosal herniation. The reported association of diverticula with Marfan's syndrome is likely related to connective tissue abnormalities in the colon wall (45).

2. Motor abnormalities. Measurements of motor activity in the sigmoid colon have been most successfully performed with open-ended catheters filled with air or fluid (46).

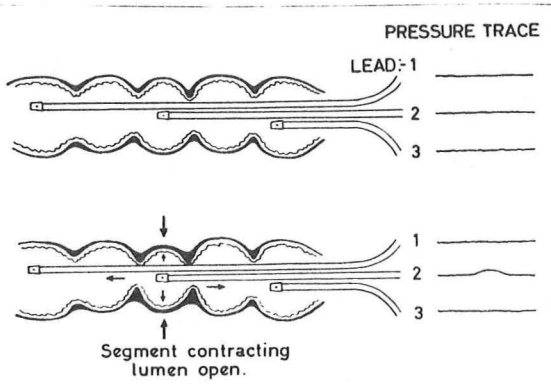



Figure 7. Two opened-end recording tubes in the sigmoid colon with corresponding pressure tracings on the right. The lower panel illustrates that partial segmentation around the second catheter causes a slight rise in pressure. From Painter (66).

Activity is generally expressed as a multiple of the amplitude, duration, and number of pressure waves recorded over a given period of time (colonic motility index) (47,48) or by quantitating the area under the pressure curves (13). Most of the published studies reveal that the sigmoid colon in selected patients with diverticulosis generate higher, more prolonged, and more frequent segmental contractions than normals in response to neostigmine or to morphine (13,47,48). Likewise, after food ingestion, the segmental pressure response is greater in selected patients with diverticulosis than in normal individuals.

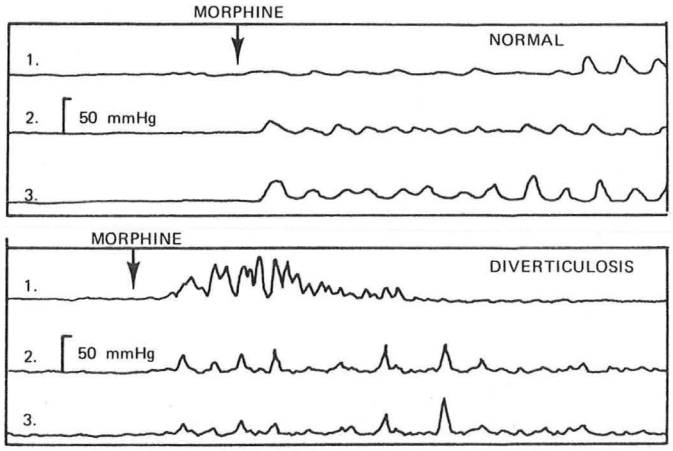



Figure 8. Effect of intravenous morphine on segmental pressure in the sigmoid colon in normals (upper panel) and patients with diverticulosis (lower panel). From Painter and Truelove (49).

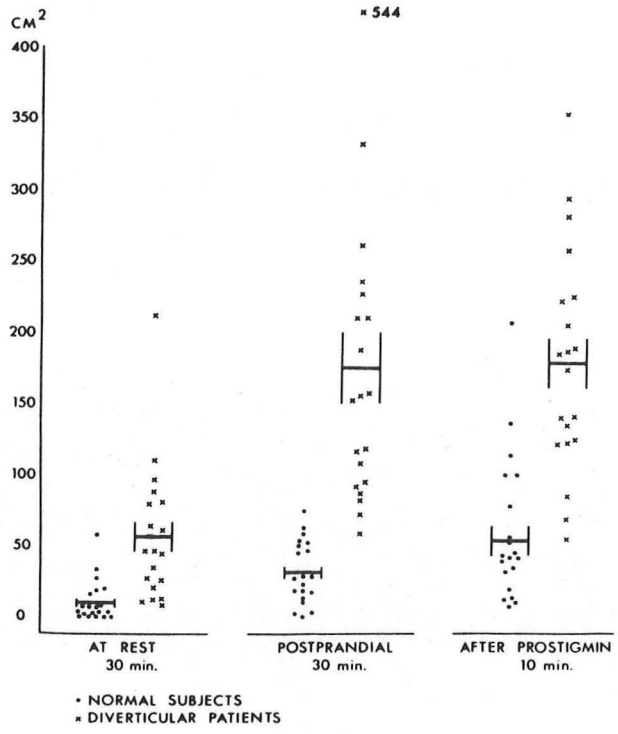



Figure 9. Motility index ($CM^2$) at rest, postprandial, and after prostigmin in normals (•) and diverticular patients (x). From Arfwidsson (13).

excessive segmental contractions

Studies reported by Painter and Truelove combined manometric measurements with simultaneous cineradiography in selective patients and found that excessive segmental contractions occurred primarily when a catheter opening was localized to a segment of colon containing diverticula (49). After administration of morphine, the diverticula in these segments became markedly distended with barium.

Most manometric studies have been performed in selected patients with colonic symptoms, previous diverticulitis, or in patients in whom symptoms are not well described (13,48-50). More recently, Weinreich and Anderson successfully studied the colonic motility index in response to neostigmine in 200 of 221 consecutive patients who had barium enema examinations (51). One hundred thirty-nine of these patients were found to have diverticulosis of which one-third had minimal or no colonic symptoms. In the 200 patients an elevated colonic motility index correlated with a history of abdominal colic rather than the presence of diverticula. Unfortunately, the motility tracings were not interpreted in a blinded manner.

MOTILITY INDEX IN 200 CONSECUTIVE PATIENTS

| | No. of Patients | Average Motility Index |
|---|---|---|
| Normal Subjects | 12 | 597 |
| Diverticulosis--Asymptomatic | 51 | 641 |
| Symptomatic | 88 | 1841 |
| Vague Dyspepsia or Diarrhea | 18 | 381 |
| Colicky Abdominal Pain | 31 | 1195 |

Adapted from Reference 51.

This study and others indicate that not all patients with diverticulosis have increased motility indices and symptoms referable to diverticular disease (52).

Two theories have been suggested to explain the relation between the motor abnormalities and the mucosal herniations. Painter proposed that the sigmoid colon normally functions to halt the fecal stream, thereby preventing constant distention of the rectum (47,48). Excessive segmentation in the sigmoid colon could isolate segments and occlude outflow both proximally and distally. Consequently "little bladders" or

chambers would be formed in which much higher luminal pressure and increased tension in the colonic wall would develop since the pressure could not be distributed proximally or distally along the colonic lumen.

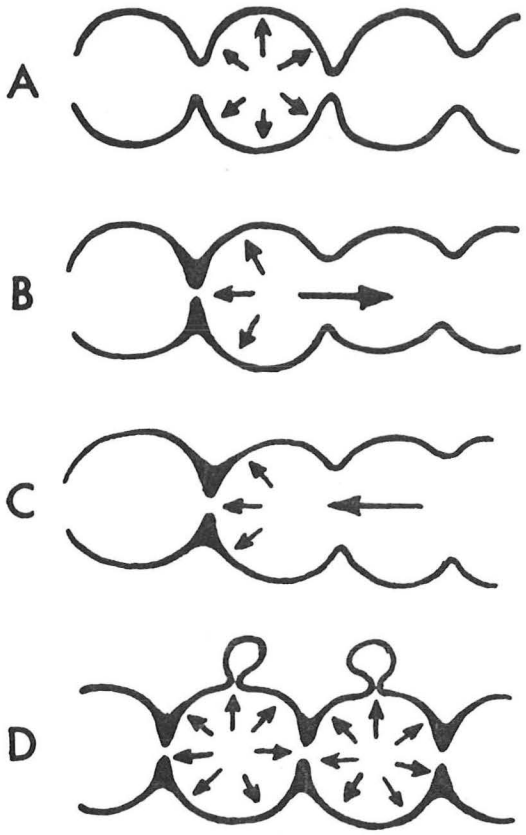



Figure 10. Complete segmentation could occlude the proximal and distal lumen in segments of the colon, creating "little bladders" with herniation of mucosa. From Painter (25).

Almy suggested that the sigmoid colon develop diverticula because of its smaller luminal diameter. This would cause an increase in the intraluminal pressure in accordance with the law of Laplace (1). With a given tension (T) in the wall of a cylinder, the pressure (P) would be greater in a cylinder with a small radius (R) than in one with a large radius, P=T/R. However, it is the increased tension on the weak foci in the colon wall which leads to diverticula formation. Therefore, with a given pressure, a smaller diameter would produce a smaller tension in the wall than a larger colonic diameter. In a way, therefore, contrary to Almy's suggestion, a smaller diameter itself in the colon may provide

$P = T/R$

some protection against diverticula formation. However, a primary muscle abnormality causing excessive increase in wall tension may be responsible for diverticula formation with smaller luminal diameter and increase pressure as associated consequences. Moreover, once a narrowed lumen is achieved, it is more likely to lead to "chamber formation" as segmentation could more completely occlude the lumen (25).

It is reasonable to assume that the primary defect in a substantial group of patients with diverticular disease is increased muscle activity manifested by excessive segmental contractions. This produces the muscular thickening, excessive intraluminal pressures, and mucosal herniations. The factor(s) which initiate(s) the increased motor activity are not known. Theoretically, a number of factors could be involved such as diet or emotional stress.

↑ muscle activity
1° Defect
Diet

3. Diet. Epidemiological and experimental data suggest that an abnormal diet is the factor responsible for the development of diverticular disease (11,25,53,54). For the last 100 years in industrialized countries of the world, natural fiber has been removed from foodstuffs as a result of technological advances in food processing (4,25). This is most noticeable in food made from sugar and wheat in which most of the fiber is removed (24,55). Consequently, there has been a decrease in the consumption of food containing fiber and an increase in the consumption of refined sugar and meat. However, the definition and measurement of fiber in the diet is complex (4,6,11,56,57). Dietary fiber is that part of plant material which is resistant to digestion by the human gastrointestinal tract as listed below.

Diet Changes
1. ↓ fiber?
2. ↑ sucrose fat etc. → ↑ bacteria

COMPONENTS OF DIETARY FIBER

| Structural Polysaccharides | Primary Unit |
|---|---|
| 1. Cellulose | Glucose |
| 2. Hemicellulose | |
| A | Xylose, Galactose, Mannose, Arabnose, Glucose |
| B | "Acid" (Uronic Acids in Addition to Other Sugars) |
| 3. Pectin | Mainly Galacturonic Acid |
| **Structural Nonpolysaccharides** | |
| 1. Lignin | Phenyl-Propane |
| **Nonstructural Polysaccharides** | |
| 1. Gums, Mucilages, Algal, Miscellaneous | |

Fiber is also associated with unavailable lipids such as waxes and cutins, unavailable nitrogen, trace elements, enzymes, and mineral salts.

I. The term crude fiber, which is often included on food tables, is derived from acid, alkali, alcohol, and ether extraction of food substances and underestimates the fiber component by greater than 50 percent.

II. The term dietary fiber is used for measurements of residue after in vitro digestion with intestinal enzymes.

The digestion of fiber-which occurs in the colon as a result of the action of bacteria is not taken into consideration. A high residue substance increases the bulk in the colon as a result of the undigested fiber and its ability to absorb water, and probably because of the net colonic secretion of water and electrolytes produced by the products of bacterial degradation of fiber and bile salts (58). A highly refined diet is absorbed in large part in the small intestine and results in a decrease in fecal bulk. It is postulated that increased segmentation is required to halt and mix this "abnormal fecal stream" (25). The increasing incidence of diverticular disease in industrialized societies appears to parallel the degree of refinement of food. The low incidence of diverticulosis in underdeveloped areas of the world, particularly in Africa, could be explained by the finding that diets in these areas have a high fiber content (11,59). By a local mechanical effect, the large bulk which enters the colon might prevent excessive segmentation. It is of interest that some of these people have very large colons which sometimes develop volvulus rather than diverticula (25). The increasing incidence in urban Africa also appears to parallel dietary changes (11).

However, not all studies confirm the relation between low residue diet and diverticular disease. Manouses et al reported that in Greece a higher incidence of diverticulosis is found in residents of urban areas than among the rural population even though no differences could be detected in the quantity of residue consumed between the two populations (60). The notion that residue in the diet has decreased in the past 100 years has also been challenged (27,61). Rather, it is suggested that diet contains more vegetable fiber and less grain fiber. It is possible that the content of fat, protein, or carbohydrate in the diet (especially increased sucrose) is the detrimental factor rather than the absence of dietary fiber (62). There is evidence to indicate that changes in diet produce alterations in fecal bacterial flora and bile salts and their metabolities which in turn might alter colonic motility (4,63). Therefore, the putative effect of diet may not be merely a local mechanical one. Diet may also alter colonic motility via neurogenic and/or hormonal mediators (46,64). The segmental colonic contractions seen immediately after a meal certainly appear to be the

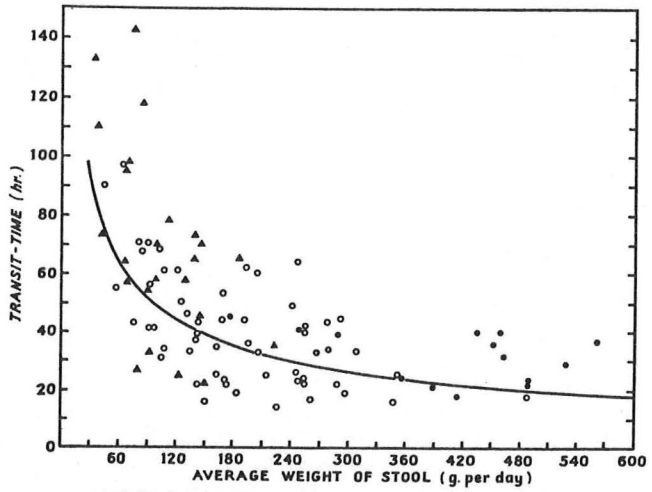
alt. bacterial flora.

result of neurogenic or hormonal factors (65). For example, the intravenous injection of pharmacological doses of cholecystokinin can cause an increase in the sigmoid motility index and cause abdominal pain (64). The development of marked bowel wall thickening and even acute diverticulitis in bypassed or defunctionalized colons indicates that local stimuli in the colonic lumen may not be necessary for segmentation and progression of diverticular disease (66).

Animal studies. The relation between diet and diverticular disease was derived in part serendipidously from experiments in rats in which low residue diets produced contracted colons and cecal diverticula proximal to an acute angulation in the colon (67). Since rats have colons with longitudinal muscles that encompass the colon without localized taeniae formation, studies have been performed in rabbits who have colonic musculature more like that of man (68). Controlled, but unpaired studies, reveal that low residue diets produce abnormal segmental pressure responses with contracted and thickened colons in these animals. Transient diverticula were demonstrated only in vivo in response to neostigmine stimulation.

Human studies. Studies in man have been hampered by firm conviction of some investigators that the theory is fact and that administration of low residue diets is unethical (69). Uncontrolled studies using diets supplemented with agents that increase fecal bulk such as bran reveal a marked decrease in subjective symptoms attributed to diverticular disease, and even some symptoms unrelated to diverticular disease (69,70). Additional uncontrolled studies by other investigators in small numbers of patients with diverticular disease reveal that the addition of fiber to the diet results in increased fecal weight, decrease in intestinal transit time, and/or a decrease in the colonic motility index (50,71-78).

Bran ✓SX




**Relation between fibre intake, transit-time, and stool weight.**
○ = Vegetarians, vegans, and African boarding-school (mixed diet).
● = African villagers (high-residue diet).
▲ = English boarding-school and British Navy (low-residue diet).
The curve (which is based on more data than the points shown here) is:
log (time) = 2·81633–0·56057 log (weight).

Figure 11

Diverticular disease. Pre- and post bran. Nine patients. Transit time (hrs).

| | Pre-bran | Post-bran | Change |
|---|---|---|---|
| Normals | 66.3±18.1 | 50.0±11.5 | -14.7±10.9 |
| Diverticular Disease | 93.4±13.8 | 57.9± 8.0 | -36.9± 7.0 (P<1%) |

Diverticular disease. Pre- and post bran. Nine patients. Motility indices.

| | Pre-bran | Post-bran | Change |
|---|---|---|---|
| Basal | 892±196 | 648±339 | -244±370 |
| Food | 1513±456 | 446±130 | -1067±383 (P<1%) |
| Prostigmine | 2127±405 | 1216±398 | -910±347 (P<1%) |

Figure 12. From Smith, AN, et al (10).

Controlled trials. In a double-blind, controlled, and randomized study reported by Soltoft et al in 1976 in 52 outpatients with the irritable colon syndrome (including some patients with diverticulosis), no beneficial effect was demonstrated with the administration of 30 grams of bran per day for six weeks compared with a placebo (79).

PATIENT'S ASSESSMENT OF TREATMENT RESULT

| ASSESSMENT | NO. PATIENTS TREATMENT GROUP | PLACEBO GROUP |
|---|---|---|
| Much improved | 7 | 11 |
| Slightly improved | 8 | 4 |
| Unchanged | 13 | 8 |
| Slightly worse | 1 | 0 |
| Worse | 0 | 0 |
| Total | 29 | 23 |

Bran trial & placebo effect

This amount of bran is said to double the fiber content of the patient's diet. Bowel frequency, stool consistency, abdominal distention, pain, and laxative use were assessed. However, 52 percent of patients in the treatment group and 65 percent of patients in the placebo group reported improvement in their symptoms.

In a more recent study, Brodribb studied 18 patients with chronic symptomatic diverticular disease in a double-blind trial carried out over 12 weeks (80).

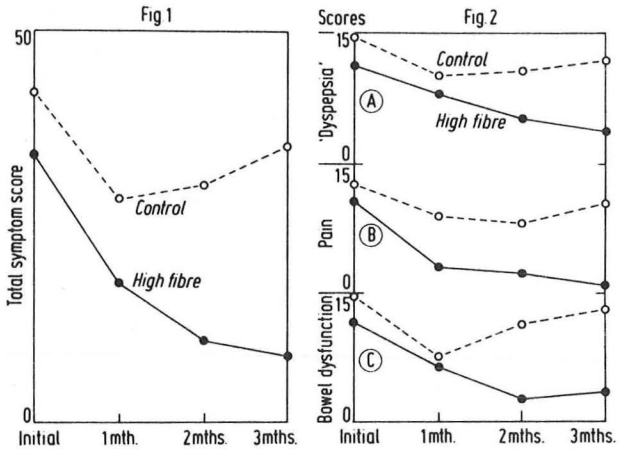



**Fig. 1—Changes in mean symptom score over 3 months for high fibre and control groups, for total symptoms.**

Mean reduction in symptom score after 3 months: high fibre, 26·2; control, 6·9. P<0·002, U=3·5.

**Fig. 2—Changes in mean symptom score over 3 months for high fibre and control groups, for dyspepsia (A), pain (B), and bowel-dysfunction symptoms (C).**

Mean reduction in symptom score after 3 months: (A) high fibre 7·7, control 3·1 (not significant, U=24); (B) high fibre 10·0, control 2·5 (P<0·02, U=14); (C) high fibre 8·4, control 1·6 (0·05< P<0·1, U=17·5).

Figure 13. From (80).

Although improvement in (A) dyspepsia symptoms (abdominal distention, eructation, etc.) and (C) bowel dysfunction (passage of gas, straining at stool, stool consistency, and use of laxatives) did not reach statistical significance, (B) abdominal pain and the total symptom score was statistically reduced at the end of the twelve week study in those nine patients on the high fiber diet. Brodribb emphasized the need for several months of treatment with the high residue diet to distinguish its effectiveness from the improvement produced by the placebo alone. Although Brodribb initially planned to include more patients in the trial, he felt the therapeutic value of increasing fiber intake was clearly demonstrated, and it was unethical to deprive patients of such treatment.

4. Psychological stress. Another factor which might explain the excessive segmental contractions in the colon in some patients with diverticulosis is psychological stress. Increased sigmoid contractions reflected by increased segmental pressure can be produced in response to stress interviews (81). Certainly modern society may provoke excessive psychological stress which might explain the differences in the geographical distribution of diverticular disease (60,82). It is even possible that the relatively inaccessible modern commode and/or the social constraints against passing of flatus in public are factors in the etiology of diverticular disease (83A).

C. Relation to irritable colon. The identification of patients with prediverticular disease provides indirect evidence (10) that motor abnormalities precede the development of mucosal herniations (13). In the small number of such patients studied, the motility index was elevated in response to neostigmine and food ingestion. Subsequent pathological examination of the sigmoid colon revealed muscular thickening without diverticula. The finding of reducible diverticula by barium studies and the demonstration in pathological specimens of gaps between thickened circular muscle where herniations might have occurred support the notion of a prediverticular state.

However, the finding of a normal motility index and no specific symptoms in a substantial group of patients with diverticulosis suggest that there are at least two forms of diverticulosis (51,52).

Fleischner proposed a classification of diverticular disease based on radiographic, manometric, and pathologic criteria and related this classification to irritable colon disease (3,33). Chaundhary and Truelove previously had postulated there are two forms of irritable colon disease (83).

1. The spastic colon form of irritable colon includes patients with abdominal pain and alternating diarrhea and constipation.
2. The painless diarrhea variety of irritable colon includes patients with diarrhea without other symptoms (sometimes called nervous diarrhea variety).

According to Fleischner, spastic colon diverticulosis (myochosis coli) is that category in which there is muscular thickening and abnormal colonic pressure responses. In addition to diverticula, irregularity, narrowing, and even a sawtoothed appearance of the colonic lumen are sometimes demonstrated with a barium enema examination (33,83). In some patients with the spastic colon form of irritable colon, motor abnormalities, radiographic, and clinical features (see page 17) are similar to those described in patients with spastic colon diverticulosis (3,33,84). This form of irritable colon disease may represent the prediverticular phase of myochosis.

Fleischner distinguishes a second form of diverticulosis called simple massed diverticulosis in which the colon is narrowed and shortened, haustral folds are crowded or absent, and multiple grape-like diverticula of the colon may be found. It is suggested that the colonic musculature is not thickened in simple massed diverticulosis. Fleischner proposed without good evidence that simple massed diverticulosis may be a late sequalae of the painless diarrhea variety of irritable colon in which segmental colonic pressures are decreased (3). Radiographically, simple massed diverticulosis is difficult to distinguish as a distinct entity, and sigmoidal pressure responses and clinical symptoms have not been defined in this type of diverticulosis.

Havia et al found a marked increase in the development of diverticulosis in patients followed for irritable colon disease compared with the expected incidence of diverticulosis in their population of patients (85). Certainly about two-thirds of patients with irritable colon disease continue to have symptoms when followed for long periods of time (83,85). However, to substantiate a relation between diverticular disease and the irritable colon syndrome, longitudinal studies involving radiographic, manometric, clinical, and even pathological descriptions are required.

D. Relation to other disease. Painter, Burkett, and others have claimed that diverticular disease is related to a large number of other diseases including varicose veins, appendicitis, inguinal hernia, hiatal hernia, colonic cancer and polyps, and ischemic heart disease (4,11,24,53,86). It is proposed that the common factor in these diseases of Western civilization is the straining at stool and/or low residue diet. In addition it has been suggested that the development of dental caries, obesity, and diabetes are related at least in part to the ingestion of highly refined diets (8). This is a provocative theory, and confirmation will require a large number of carefully performed studies.

E. Clinical features of diverticulosis. Although many patients with diverticulosis have no symptoms, a spectrum of symptoms can occur in patients with diverticula which are similar to those of irritable colon syndrome. (Figure 14)

CLINICAL MANIFESTATIONS IN CASES OF UNCOMPLICATED DIVERTICULOSIS AND IN IRRITABLE COLON SYNDROME

| | Uncomplicated Diverticulosis (150 cases) (%) | Irritable Colon Syndrome (100 cases) (%) |
|---|---|---|
| Constipation | 44 | 44 |
| Diarrhea | 50 | 39 |
| Flatulence | 52 | 53 |
| Abdominal Pain | 30 | 32 |
| Colicky Pain | 50 | 44 |
| Mucus in the Stools | 26 | 18 |
| Tenemus | 25 | 15 |

Figure 14. From Bockus, HL (87).

The degree of pain and altered bowel habits appears to relate to the intensity of segmentation and the presence of colonic distention proximal to partial colonic obstruction rather than the diverticula per se. The results of motility studies suggest that the pain often occurs simultaneously with excessive segmental pressure, and excessive segmentation appears to occur primarily in those patients with abdominal colic (51). When severe symptoms are associated with diverticulosis, it has been termed painful diverticular disease.

Physical examination may be normal or reveal abdominal tenderness or distention. A loop of sigmoid colon filled with firm stool may be palpable in the left lower quadrant. Evidence of localized or generalized peritonitis must be absent. Fever and leukocytosis should not be attributed to uncomplicated diverticulosis.

Diverticulosis as a cause of symptoms is a diagnosis of exclusion. A barium enema must be performed and may reveal varying numbers of diverticula and spasm or muscular thickening (3,33,84). Occasionally diverticula are present only on the post-evacuation radiograph. Sigmoidoscopy is essential and sometimes a colonoscopic examination is helpful in excluding other disease. A potential but uncommon complication of colonoscopy is perforation of the colon since high pressures can be produced during air insufflation (88). Colonoscopy is especially hazardous if the tip of the colonoscope is inadvertently wedged in a diverticulum. As with the irritable colon syndrome, lactose intolerance should be excluded as a cause of the patient's symptoms.

F. Treatment. In the past, diverticulosis discovered on a barium enema examination was often thought to be an insignificant finding except for the potential of bleeding or the development of diverticulitis. No therapy was initiated if the patient was asymptomatic. When treatment was instituted, it was often directed toward symptoms such as constipation, intermittent diarrhea, abdominal cramps or distention which were not necessarily thought to be related to diverticular disease. If the patient was thought to have symptoms of diverticular disease (usually ascribed to diverticulitis), the classic approach was to prescribe a low roughage diet (87,89,90). The presumed rationale for this approach was to avoid obstructing the diverticula or irritating the colon with harsh residue.

Presently, there is no theoretical or experimental data to support the use of low residue diets in patients with uncomplicated diverticular disease. Instead, our current understanding of the pathogenesis indicates that treatment should be directed toward increasing the bulk in the diet so that more residue reaches the distal colon. In this manner, it is hoped that further narrowing of the colon and excessive segmentation can be prevented. However, as previously noted, the efficacy of a high residue diet in treatment of diverticular disease is based on anecdotal reports, animal studies, clinical observations, and inconclusive controlled trials (67-70,79,80,91,92).

Although high fiber diets are likely to be of benefit, it is also possible that such therapy may produce nutritional deficiencies (62,92). There is evidence to indicate that increased loss of nitrogen and fat in the stool occurs as the dietary fiber content is increased. Glucose tolerance, serum lipids, and serum folate may also be affected. Of greater concern are the results of preliminary studies which reveal that urinary calcium excretion is slightly decreased in patients on a high residue diet, suggesting that fiber binds calcium in the intestine. These nutritional effects of high fiber diets are most likely inconsequential or even beneficial in well-nourished patients, but may be harmful in patients with malnutrition. Therefore, long-term studies are needed to determine if patients with diverticular disease with or without associated disorders develop deficiency states. In addition, carefully performed controlled studies are necessary to determine if dietary alterations effect the symptoms of diverticular disease, prevent or reverse diverticula, or prevent the development of diverticulitis.

Thus despite the widespread belief in the benefits of high-residue diets and bulk-forming agents for treatment of diverticular disease, it is difficult at the present time to be dogmatic about their use, especially in the asymptomatic patient. Certainly purgatives, medications such as mineral oil, bile salt derivatives, and other colonic irritants should be avoided. The patient should be reassured that he does not have cancer or colitis. There is no reason to prescribe a low residue diet. It is reasonable to encourage the ingestion

of fresh fruits, vegetables, whole-wheat bread, and breakfast cereals (69), provided milk is not used in patients with lactose intolerance.

With the ingestion of a well-balanced diet, the use of bulk agents derived from semi-synthetic polysaccharides (methyl, carboxymethyl, or ethylhydroxyethyl cellulose), mucilaginous seeds (Metamucil, L.A. Formula), seed coats (bran), and mucilaginous gums (sterculia) are unlikely to be harmful. In patients with symptomatic diverticulosis, many authorities recommend the administration of one of these agents. Crude fiber intake for each person in the United States is estimated to be about 8 to 11 grams a day, and dietary fiber intake is about 24 to 33 grams a day (4). A normal helping (two ounces) of a high-fiber breakfast cereal like Kellog's All-Bran increases dietary fiber intake about 10 grams. The ingestion of unprocessed bran is an inexpensive means to increase stool bulk. Since unprocessed bran is similar in appearance and taste to brown sawdust, it is recommended that it be added to cereal, soup, milk, or fruit juice (69). Initially, about two teaspoonfuls three times a day (six grams a day) should be taken, and, after two weeks, the dose can be gradually increased until the stool becomes soft and easily passed. Patients may have a transient feeling of bloating or flatulence, but this usually disappears in three to eight weeks. Other semisynthetic and natural bulking agents may be used but they should be given in divided doses with meals and/or with liberal fluid intake to reduce the small risk of producing bowel obstruction (53). Indeed, there are some patients who cannot tolerate bran or other bulk-producing agents (93).

In patients with painful diverticular disease or severe diarrhea, anticholinergic agents may also be helpful in reducing cramping pain since anticholinergics inhibit segmental contractions in the sigmoid colon. However, it should be emphasized that there is a paucity of adequately controlled studies reported which confirm the efficacy of anticholinergics in either irritable colon or diverticular disease (76). Antidiarrheal agents such as codeine, Lomotil, or Loperamide have not been shown to provide any benefit in patients with intermittent diarrhea and constipation. The administration of small doses of phenobarbital, diazepam, or chlordiazepoxide for a few days may be helpful in the anxious patient, although these agents appear to have no specific pharmacological action on the bowel itself. Morphine should be avoided even when the patient complains of severe pain because it increases segmental pressures in the colon and might aggravate the problem.

Surgical treatment of painful diverticular disease. Sigmoid colectomy has been utilized for treatment of intractable painful diverticular disease with satisfactory results (13,94). Reilly introduced the colomyotomy for poor risk patients in which a longitudinal myotomy of the circular muscle of the affected colon was performed (95). Transverse taeniomyotomy which is reported to lengthen and widen the

colon has also been advocated and appears to be a safer and simpler procedure without the risk of bowel perforation (96,97). In the selected patients studied, it appears that myotomy reduces the muscular spasm and elevated motility index only temporarily. Treatment with bran will apparently prevent the return of an abnormal motility index. (Figure 15)

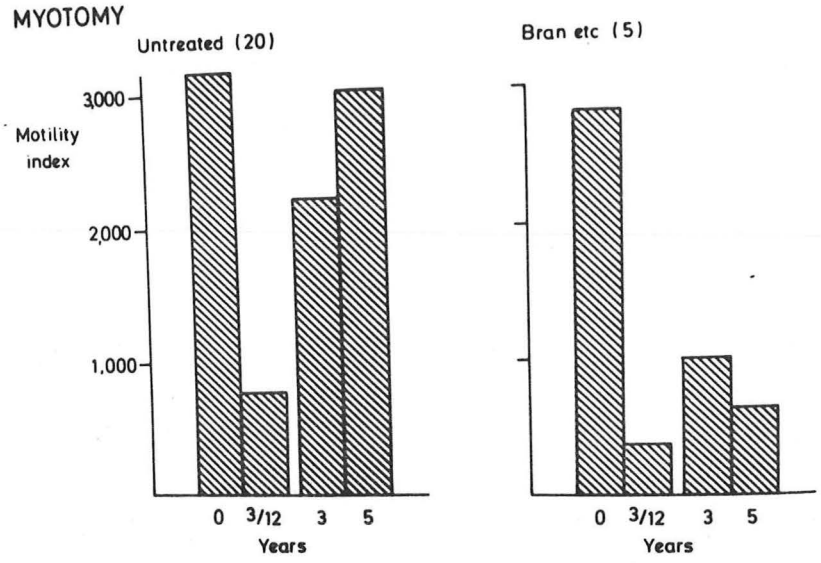



Figure 15. From Reference 45.

Adequate studies are not yet available to permit evaluation of the effectiveness of surgical treatment of painful diverticular disease. Furthermore, intractable symptoms resistant to medical therapy occur uncommonly, and the surgical treatment of a non-inflamed colon should be considered experimental. Certainly before resorting to surgery a prolonged trial with a high residue diet supplemented with natural or synthetic bulk preparations should be instituted.

## IV. Diverticulitis

A. Pathology. Clinically, diverticulitis is said to develop in 20 to 25 percent of patients with diverticulosis (16,29), but this is almost certainly an overestimation. In pathological studies, diverticulitis was found in only about 12 percent of patients with diverticulosis; moreover, in many cases the diverticulitis was clinically

asymptomatic (19,29). Conversely, in one-third of patients with prior clinical diagnosis of diverticulitis, no pathological evidence of inflammation can be demonstrated (15,31). This discrepancy is in part due to the diagnosis of diverticulitis in patients who have no evidence of abdominal inflammation. The frequency of diverticulitis is proportional to the extent of colonic involvement with diverticula. In one study 50 percent of patients with diverticulosis of the entire colon had pathological evidence of diverticulitis (29). Pathological studies almost always reveal suppuration outside the diverticulum (peridiverticulitis) in patients with clinical diverticulitis (14,15,98). Inflammation often spreads outside the serosa and produces local peritonitis which is confined by the pericolic fat, mesentery or adjacent organs. The residue may be a focus of granulation tissue, inflammatory cells, or fibrotic tissue indicating that a microperforation has occurred which remained localized and resolved. In other cases a pericolic abscess may localize in the wall of the colon causing an intramural mass or there may be dissection or inflammation around the colon which produces segmental narrowing of the lumen. Chronic phlegmonous diverticulitis can develop when a chronic fibroblastic inflammation surrounds the colon (15,99). Inflammation can also dissect subserosally and form a paracolic fistula or intramural abscess which may communicate with several diverticula (100,101). (Figure 16)

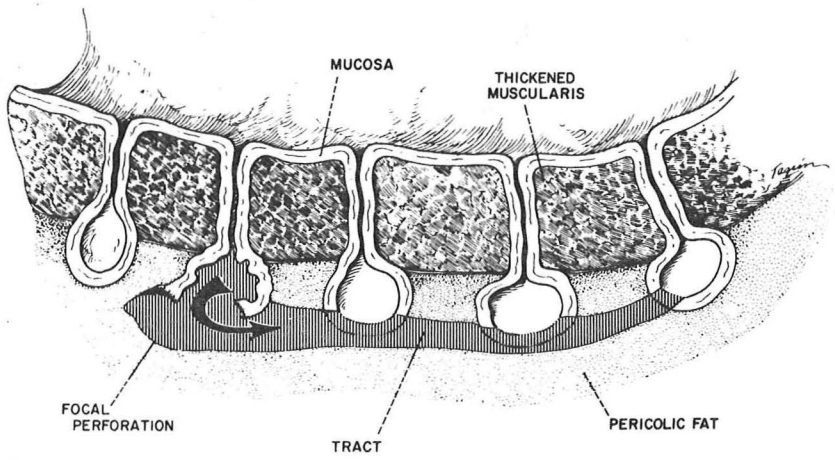



Figure 16. Presumed mechanism for multiple sites of tract-lumen communication in dissecting peridiverticulitis. A diverticular abscess dissects longitudinally within pericolic fat serially involving adjacent diverticula producing several points of fistulous communication with bowel lumen. From Reference 101.

Inflammation can burrow into adjacent organs forming a fistula between the colon and the bladder, ureter, vagina, small intestine, colon, or even the abdominal wall and cutaneous tissue. Since most diverticula are orientated obliquely toward the mesentery, free perforation occurs infrequently. However, generalized peritonitis and/or noncontiguous intraperitoneal abscesses may develop when adjacent viscera, omentum, mesentery, or the peritoneum fail to contain the inflammation.

B. Pathogenesis. It has long been assumed that diverticulitis, like appendicitis, is inflammation within the lumen of the diverticulum which results from narrowing or obstruction of the neck of a diverticulum. Subsequently, inspissation of retained fecal material results in inflammation, formation of a retention abscess, and mucosal ulceration. However, radiological and/or pathological studies performed on patients with previous clinical evidence of diverticulitis (localized abdominal tenderness, fever, and leukocytosis) suggest perforation of a diverticulum (14,33,84). The inciting factor(s) for diverticular perforation is not known. Rupture of the diverticulum itself due to increased tension is possible.

The finding of muscular thickening and/or abnormal segmental pressure responses in most patients with previous attacks of diverticulitis might suggest that perforation of diverticula occurs predominantly in those patients with the spastic colon diverticulosis. However, clinical studies indicate that about one-half of patients have no symptoms suggestive of irritable colon disease prior to an attack of diverticulitis (7,102).

C. Clinical features. The predominant clinical symptoms of diverticulitis are pain and fever (103). Pain is usually prominent and is located in the lower abdomen, particularly on the left; so-called left-sided appendicitis. Pain can also be present in the lower part of the back (104). Fever is usually present and may be the only obvious clinical manifestation. Pericolitis or a pericolic or intramural abscess may cause partial or complete colonic obstruction with constipation, obstipation, abdominal distention, anorexia and nausea. If the bladder or ureter is adjacent to the inflammation, the patient may have symptoms which simulate a urinary tract infection.

On physical examination, the patient usually has localized left lower quadrant tenderness and fever. Since the process may involve the serosal surface of the colon and its peritoneal cover, patients are often found to have marked tenderness, both direct and rebound, because of local peritonitis. Rectal examination may reveal a tender mass if the inflamed sigmoid or a pelvic abscess is reached.

The results of laboratory examination almost always reveal a blood leukocytosis with an increase in polymorphonuclear forms. Contiguous involvement of the bladder or left ureter by a pericolic

abscess may result in red cells and/or white cells in the urine. In older patients and those receiving corticosteroids, many of the symptoms, signs, and laboratory values may be normal.

The diagnosis of diverticulitis is often based entirely on clinical assessment of the patient and exclusion of other disease. Despite discomfort to the patient, sigmoidoscopy should be performed, using no air, to rule out other conditions. Usually a mass completely or partially obstructing the lumen may be encountered. Plain abdominal radiographs may be normal or show paralytic ileus due to peritonitis or complete sigmoid obstruction. Free air in the abdominal cavity indicates rupture of the diverticulum into the peritoneal cavity.

Many clinicians advise that a barium enema examination is contraindicated in the acute phase of diverticulitis. It is proposed that the increased intraluminal pressure exerted by the barium column may disrupt a previously localized perforation and soil the peritoneal cavity with barium and feces. However, other investigators point to the rarity of significant complications from barium enema examinations in patients with diverticulitis and emphasize the usefulness of a precise diagnosis afforded by a careful examination (5,105,106). A barium enema should be performed at some time in the course of the illness. If there is any doubt concerning the diagnosis by which therapy will be altered, it should be performed promptly, providing the patient is stable and there is no evidence of a free perforation. It is not known if the timing of a barium enema influences the likelihood of demonstrating the site of perforation.

Radiographic features such as irregularity, thickening, and even a saw-toothed appearance of the bowel contour are typical of the spastic type of diverticulosis and are not manifestations of diverticulitis (3,84). Roentgenographic evidence of diverticulitis include the presence of barium outside a diverticulum, the delineation of a paracolic or intramural mass, or the demonstration of a fistula or sinus tract originating in the colon. The latter two abnormalities can be seen with other entities, such as carcinoma or granulomatous colitis.

D. Complications of diverticulitis. The most common complications of diverticulitis are colonic fistula, colonic obstruction, intra-abdominal abscess, and peritonitis. It should be noted that many authors state that perforation is a complication of diverticulitis. This is inaccurate since in virtually all cases diverticulitis results from a perforation.

Fistula. Fistula formation is a frequent complication of diverticular disease. Colocutaneous, colo-ureteric, and multiple fistulae usually occur only after operative intervention. Colo-vesical fistula

occurs primarily in men, because there is no intervening vagina and uterus. Recurrent urinary tract infections, chronic cystitis, pneumaturia or fecaluria are the hallmarks of this complication (108). A plain abdominal radiograph may show gas in the bladder. Intravenous pyelography, cystoscopy, or a barium enema may demonstrate the presence of a colo-vesical fistula. A fistula may dissect along the wall of the colon producing "double tracking" of the barium column (100,101). Multiple sites of reentry into the colon lumen may be present. Sigmoidoscopy or colonoscopy are useful in detecting the presence of granulomatous colitis or carcinoma as a cause of a colonic fistula. A colo-vaginal fistula may result in the passage of feces or air through the vagina. Diarrhea or steatorrhea may occur with a colo-intestinal fistula if significant absorbing surface of the small bowel is bypassed.

Obstruction. Signs and symptoms of partial or complete large bowel obstruction may occur during the initial attack of acute diverticulitis or with recurrent diverticulitis and may even become the predominant clinical manifestation (98). An intramural abscess or widespread pericolic inflammation can produce partial or complete colonic obstruction. Contiguous involvement of the small intestine or peritonitis may produce a clinical picture or paralytic ileus or small intestinal obstruction. Partial colonic obstruction often subsides with medical management if the obstruction is caused by acute inflammation. However, when acute obstruction is superimposed on chronic muscular thickening and fibrosis of the colon, it is often irreversible, and surgical treatment is necessary (9).

Intra-abdominal abscess. Large collections of pus may develop adjacent to the bowel, in the pelvis, or under the diaphragm. In the acute phase it may be difficult to distinguish a large peridiverticular abscess from a phlegmon. Persistent or recurrent temperature elevation or the persistance or recurrence of a tender mass should suggest an unresolved intra-abdominal abscess. A mass may be felt on abdominal palpation, or by rectal or vaginal examination. Barium enema examination may identify a paracolic abscess. Abdominal sonography and computerized axial tomography should prove to be very useful in identifying and following the course of an intra-abdominal abscess. Portal septicemia and hepatic abscesses may also develop in patients with diverticulitis and should be suspected in patients with associated liver tenderness or abnormal liver function tests.

Generalized peritonitis. Although diverticula usually are directed obliquely toward the mesentery and close promptly after perforation, occasionally a perforation occurs which is not promptly sealed off and generalized bacterial peritonitis may result. With diffuse peritoneal spread of bacteria and other fecal material the symptoms are often dramatic--severe abdominal pain, distention, and fever. The onset of symptoms may be sudden, and in some cases appears to occur during defecation (109).

E. Medical treatment. There is virtually no data available to determine the proper medical treatment of diverticulitis. Therapeutic maneuvers for acute diverticulitis can only be derived from anecdotal experiences and are directed toward "resting" the colon and combating infection so that inflammation, edema, and spasm will subside and obstruction, if present, will be relieved. The patient in whom the diagnosis of diverticulitis has been made or is suspected should be hospitalized and given nothing by mouth. Nasogastric suction should be instituted especially if nausea, vomiting, or abdominal distention are present. Intravenous replacement therapy should be given to maintain intravascular volume and screening blood tests, urine analysis, and blood cultures should be obtained. One would anticipate in cases of a perforated diverticulum, that the normal colonic bacterial flora (enterococci, escherichia coli, and anaerobic bacteroides) would be found in the peridiverticular abscess or phlegmon. Consequently a combination of antibiotics with a range of activity against all these organisms should be used. This would include an antibiotic program such as penicillin for enteroccus, kanamycin or gentamycin for gram negative rods, and chloramphenicol or clindomycin for the anaerobic bacteroides. However, in cases with minimal clinical symptoms when the infection appears to be localized, ampicillin, cephalothin, or penicillin and tetracycline have been used successfully.

The patient should be observed carefully with frequent abdominal examinations and appropriate roentgenograms of the abdomen so that complications such as enlarging abscess or peritonitis can be detected. In all instances, surgical consultation should be obtained at the onset of therapy, because surgical intervention may be required at any time, and the surgeon's evaluation will be vital in future decisions of therapy.

If the decision is made not to operate, long term therapy is identical to that outlined for diverticulosis. In this situation, perhaps an even stronger case can be made for use of diets and supplements containing high residue, although as previously noted, there is no evidence as yet that this will prevent recurrent diverticulitis. However, there is no reason to believe that various nonabsorbable substances irritate or obstruct diverticula and result in inflammation. It should be emphasized that a barium enema should be performed if it has not been performed earlier in the disease course. About two-thirds of patients treated successfully with medical therapy for a first attack of diverticulitis do not have recurrent attacks (20,29,102,110).

F. Surgical treatment. Urgent surgical intervention will be necessary if there are findings of generalized peritonitis or if in the course of treatment there is evidence of failure of medical therapy (7,9). Failure of medical therapy is signaled by the development of a

tender mass, the enlargement of a previously noted mass, increasing or persistent intestinal obstruction, or generalized peritonitis. Since the most dreaded complication is septic peritonitis caused by rupture of a pericolic abscess, medical management is justified only as long as there is prompt and progressive resolution. If peritonitis is localized, many patients can be managed with medical therapy for 24 to 48 hours and if definite evidence of resolution occurs, emergency surgery can be avoided. If an intra-abdominal abscess progresses rapidly to generalized peritonitis, surgical intervention should be delayed only by rapid attempts to stabilize the patient's condition.

## INDICATIONS FOR SURGERY

A. Urgent Surgical Intervention
   1. Generalized peritonitis
   2. Failure of medical therapy for acute episode
      a. Evidence of increasing inflammation
      b. Enlarging mass
      c. Persistent obstruction

B. Elective Surgery
   1. Recurrent diverticulitis
   2. Fistula
   3. Partial colonic obstruction
   4. Inability to exclude carcinoma
   5. Urinary tract involvement
   6. Patients less than 50 years of age

Indications for elective surgery include recurrent attacks of diverticulitis, the presence of a fistula, partial colon obstruction, and the inability to exclude a carcinoma as a cause of narrowing or deformity of the colon. Some authorities suggest that elective surgery be performed in any patient with urinary tract involvement or in patients younger than 50 years of age with one attack of diverticulitis (7,9). Although a high percentage of younger patients with diverticular disease are treated surgically, there is inadequate data available to evaluate this approach (21).

A variety of surgical approaches have been advocated for the management of diverticulitis (7,104,110). The results of each of these is difficult to evaluate because of the absence of control studies, differences in surgeon's experience and skills, differences in patient populations, and the great variation in the stage and severity of the patient's disease. In general, attempts should be made to suppress or eradicate the inflammation by medical therapy so elective surgery can be performed. An increasing number of surgeons advocate a one-stage

procedure with primary resection of the involved colon and an end-to-end anastomosis without proximal colostomy (9,11,112,113). When active inflammation, an abscess, fistula, or obstruction are present, the safest approach is a two or three stage procedure:

(1) proximal colostomy, drainage of abscesses and/or resection of diseased bowel with a distal mucus fistula;
(2) at a later date resection of the diseased colon if not previously performed;
(3) closure of the colostomy (7,9,104).

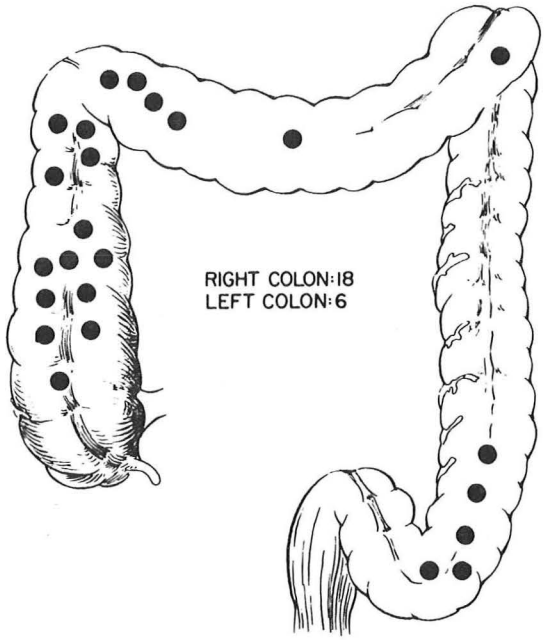
The advantage of primary resection of an inflamed or even freely perforated colon is that it could reduce hospitalization and avoid the need for several operations. However, the reports of a high rate of significant complications, including leakage at the anastomotic site and intra-abdominal infection appear to limit the application of this approach to patients in whom there is no significant obstruction and the inflammatory process is confined (113).

The mortality rate after surgical intervention for diverticulitis varies according to the age group, associated illnesses, and most important, the time in the course of the disease at which surgery is performed. The overall surgical mortality is reported to be 4 to 8 percent (7,9,104). Seventy-five percent of those who die during or after surgery are over the age of 60. Elective surgery is attended by a mortality rate no higher than for most other major abdominal procedures, and is, therefore, advised for most patients with recurrent attacks or persistent symptoms of diverticulitis. About two-thirds of patients treated surgically for diverticulitis become asymptomatic (102). This obviously varies with the stage and extent of the disease and length of follow-up.

## V. Diverticular bleeding

Bleeding from colonic diverticula is said to be the most common cause of lower gastrointestinal tract hemorrhage (114-117). From 10 to 30 percent of patients with diverticulosis have rectal bleeding attributed to the diverticula. Severe life-threatening hemorrhage is said to occur in from three to five percent of patients with diverticular disease (118-120). However, these estimates are derived from surveys in which the site of bleeding was usually not identified, the diagnosis being made by exclusion (118). With the wider application of arteriography in patients with massive bleeding and the greater use of the air-contrast barium enema and colonoscopy in patients with chronic or intermittent lower gastrointestinal bleeding, it is likely that other causes for rectal bleeding will be found in many patients, and the incidence of bleeding from diverticular will be less than in the currently published estimates.

A. Pathology of diverticular bleeding. In patients with documented massive bleeding from colonic diverticula, rupture of a vas rectum at the dome or along the antimesenteric margin near the neck of the diverticulum has been demonstrated (37,121). In careful pathological studies, Meyers et al found striking eccentric fibromuscular internal thickening, thinning of the media, and duplication and fragmentation of the internal elastic lamina of the vas rectum on the side of the vessel adjacent to the lumen of the diverticula. The artery which often is occluded by a thrombus is usually ruptured asymmetrically beneath focally eroded mucosa. There is minimal or no inflammation in the mucosa and in the wall of the vessel. It is of interest that in over 70 percent of the cases in which the bleeding site is identified at surgery or by arteriography, the bleeding diverticulum is in the right colon (116,121-126). (Figure 17)




***Site of diverticular hemorrhage in twenty-four patients as shown by angiography.***

Figure 17. From Athansoulis et al (125).

Massive bleeding in patients with diverticular disease uncommonly comes from two sites simultaneously or from an inflamed diverticulum (118,119, 123). In patients with chronic or intermittent bleeding attributed to diverticulosis and diverticulitis, ulcers or inflammatory granulation tissue are sometimes found in the base or neck of diverticula (32,114,127).

B. Pathogenesis of diverticular bleeding. The studies by Meyers et al and others indicate that the vasa recta which are intimately associated with the dome and border of the diverticula and the highly vascular plexus around the neck of the diverticula are potential bleeding sites (37,121). Major bleeding appears to result from rupture of a vas rectum. Meyers et al suggest that injurious factors in the lumen of the diverticulum produce eccentric damage to the underlying vessel wall which leads to rupture of the vessel and secondary mucosa erosion (121).

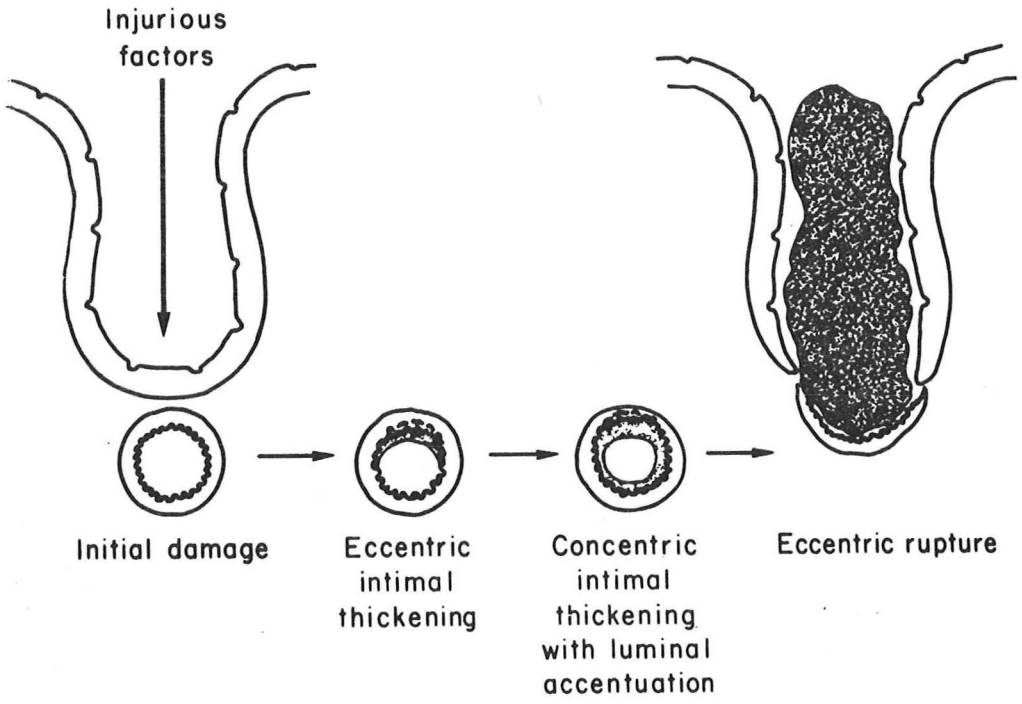



Figure 18. Proposed pathogenesis of bleeding colonic diverticulosis. From Meyers (121).

The failure to find significant inflammation in the mucosa and in the wall of the vas rectum supports the proposition that focal diverticulitis or an ulcer in the diverticulum is not the primary event causing major bleeding. The eccentric fibromuscular intimal thickening, medial thinning, and fragmentation of the internal elastic lamina of the artery are similar to the vascular changes produced experimentally in response to injury.

The apparent predisposition for right-sided diverticula to bleed massively may be related to the wider necks of these diverticula. This might allow greater exposure to injury from within the colonic lumen (121). The putative injurious factors have not been identified. In distinction to massive hemorrhage, it is possible that the minimal bleeding that is said to occur in diverticulosis and diverticulitis is the result of disruption of the submucosal capillary plexus by focal inflammation in the wall of a diverticulum.

C. Diagnosis and treatment. In patients with minimal chronic or intermittent rectal bleeding, one should be extremely hesitant in attributing the bleeding to diverticular disease (118,127). A systematic evaluation with barium studies, endoscopy, and in some cases arteriography, should be performed before the diagnosis of diverticular bleeding is made in patients with minimal rectal bleeding. Detecting blood in the gastric or small bowel aspirate or finding negative guaiac stool proximal to friable hemorrhoids during proctoscopic examination largely excludes diverticula as a cause of minimal rectal bleeding.

Diverticular bleeding is almost always recognized as massive rectal bleeding in patients without diverticulitis (115-121,127). The patients are usually elderly, and often have other associated serious disease (117). When confronted with a patient with massive rectal bleeding, the initial therapeutic step is to maintain adequate circulating vascular volume. Bleeding subsides spontaneously in about 80 percent of the cases. Proctoscopy should be performed promptly to rule out distal colon and rectal lesions.

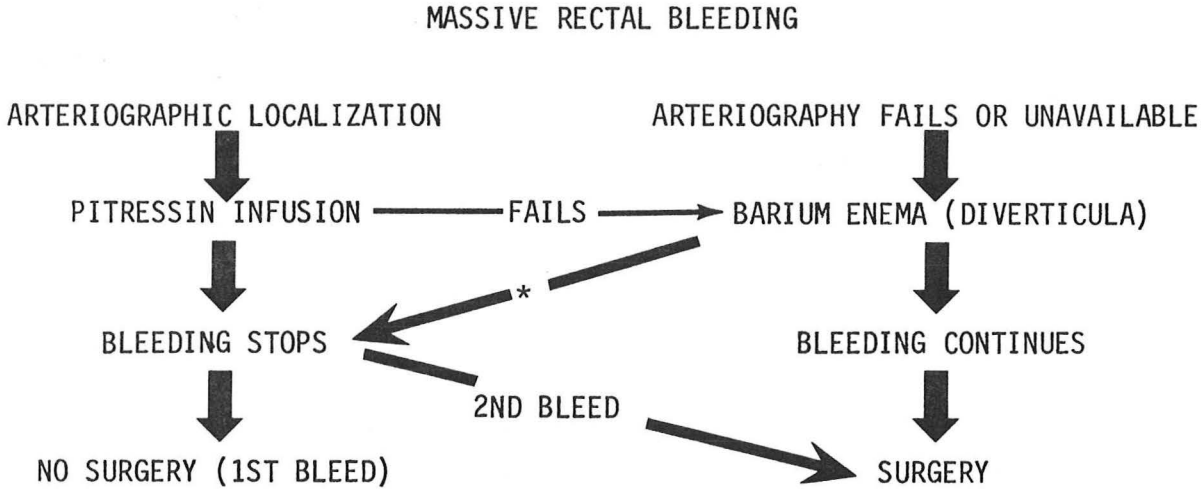
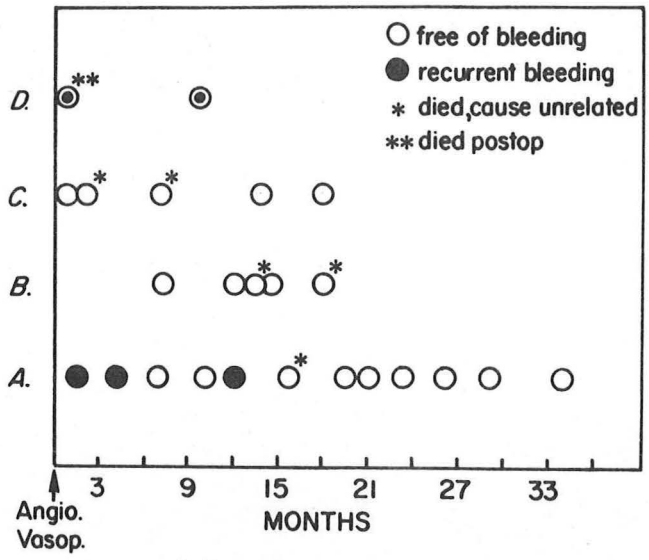



Figure 19

Selective visceral angiography appears to be the most accurate and successful method for localizing the bleeding site in patients with diverticulosis (116,121-125). Angiography must be performed before a barium enema examination, since barium may be retained in the colon for several days and interfere with the interpretation of an arteriographic study. It has been estimated that a bleeding rate of 0.5 ml per minute is necessary in order for contrast material to be visualized in the lumen of the gastrointestinal tract (130). Preliminary studies by experienced angiographers reveal that the bleeding site can be identified in 60 to 90 percent of the cases (121-125). About one-third of patients with rectal bleeding are found to have other lesions such as arteriovenous malformations, tumors, or cecal ulcers as the cause of hemorrhage. Once the arteriogram has been successfully completed, and/or bleeding has stopped, it is advisable to perform a barium enema to evaluate the colon for other lesions. There is some evidence to suggest that the barium enema will result in cessation of diverticular bleeding (131). However, barium contrast studies cannot prove that diverticula are the source of bleeding and may fail to disclose some lesions which might cause rectal bleeding such as arteriovenous malformations.

Pitressin infusion. If the bleeding site has been identified by visceral angiography, in many cases the hemorrhage can be stopped by infusion of pitressin through the arteriographic catheter (116,121,124, 125). It should be emphasized that during arteriography and while pitressin is infused, the patient must be monitored closely. About thirty minutes after the pitressin infusion has been started a repeat angiogram should be performed to determine the extent of vasoconstriction and to determine if extravasation of contrast material still occurs. Uncommon complications associated with pitressin infusion include water retention and hyponatremia, transient hypertension, and hematoma at the site of insertion of the catheter in the groin. Recurrent bleeding occurs in about one-fourth of patients followed for short periods of time.




A. Vasopressin only
B. Vasopressin Elective Part. Col.*
C. Vasopressin- Rebleed, Part. Col.
D. Failure of Vasopressin-Part. Col.

Figure 20. Follow-up data in twenty-four patients with diverticular hemorrhage. (Part. Col.=partial colectomy) From Reference 120.

Emergency surgery. Current information from a small number of studies suggests that a resection of the bleeding colonic segment should be performed if pitressin infusion fails to control the hemorrhage or if bleeding recurs (116,123-125). Other investigators suggest that a subtotal colectomy be carried out since bleeding from other diverticula could occur (132). The rationale for a local resection of the segment of colon containing the bleeding diverticulum is that recurrent bleeding after local resection appears to be unusual and the likelihood of bleeding from other diverticula is no greater than it would be in a patient with diverticulosis who has never bled. In a patient with persistent massive bleeding in whom angiography is unsuccessful or has not been performed, a barium enema examination would be useful in excluding some other lesions as the cause of rectal bleeding as well as perhaps stopping the bleeding. However, if bleeding persists, a subtotal colectomy will be necessary (116,119,120,127,133). Attempts can be made at surgery by a variety of techniques to localize the site of bleeding, but this is often unsuccessful, prolongs the surgery, and may allow further bleeding. The failure of a limited sigmoid colectomy to prevent continued or recurrent bleeding in a large percentage of patients must be attributed to the observations that in the majority of the patients, the bleeding site is in the more proximal colon.

If bleeding ceases spontaneously or in response to pitressin, a decision must be made concerning elective surgery. If the patient has had documented recurrent hemorrhage from diverticular disease, surgery should be performed because bleeding after a recurrence appears to occur in half of these patients (119). Current data indicate that only about 20 to 25 percent of patients rebleed after a single documented bleeding episode (117,121,124,125). Thus, it is not unreasonable to defer surgery in those patients who have bled once from diverticular disease especially if the bleeding site is not localized.

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