# Current Practice

## Diverticular Disease of the Colon

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The title of this paper may seem strange to those who are used to the terms diverticulosis and diverticulitis. Once it was thought that colonic diverticula caused symptoms only when they were inflamed. Recent advances in colonic physiology and pathology have shown that this is not the case. The time has come to examine these terms and to attempt to relate the symptoms of the disease to the physiological mechanisms and pathological processes that cause them.

A classification of diverticular disease is given in Fig. 1, but this must remain provisional until the natural history of the disease is fully understood. However, it already seems certain that some abnormal activity of the colonic musculature is responsible not only for the appearance of diverticula but also for some of the symptoms which used to be attributed to diverticulitis.

The term "diverticular disease of the colon" has gained acceptance because it shifts attention away from the assumption that diverticula must be infected before they cause symptoms, and it infers that the primary abnormality lies in the colon. The term "diverticulitis" is now reserved for the manifestations of the disease, which are secondary to infection and for which treatment is mainly surgical.

### Historical Background

Colonic diverticula were regarded merely as pathological curiosities until the early years of this century, when their lethal complications were recognized at laparotomy. Telling and Gruner[1] and Spriggs and Marxer[2] classified the disease process, and—probably because they thought that undigested roughage might perforate diverticula—they recommended that a low residue diet and mild aperients should form the basis of conservative treatment. Between the wars surgery was reserved for very ill patients, as resection of the colon carried a mortality rate of at least 10%. A permanent colostomy or a persistent fistula was often the fate of patients with complicated diverticulitis, even though it was known that resection could cure the disease and its complications. Fortunately, Smithwick[3] showed that resection of the sigmoid could be performed with an acceptable mortality and that this cured the disease. Since then surgery has become safer owing to improved anaesthesia and supportive therapy, so that today elective resection of the diseased colon is the standard treatment of diverticular disease with complications.

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### Pathological Anatomy

Colonic diverticula are herniations of the mucous membrane through the colonic wall. The colonic musculature consists of an inner layer of circular muscle, which is complete, and an outer longitudinal layer, which is gathered into three bands or taenia. Between these taenia the single layer of circular muscle is weakened by the segmental blood vessels which pierce it. Just as the spermatic cord favours the occurrence of oblique inguinal hernia, the tunnels formed by these blood vessels constitute points of less resistance along which the mucosa herniates. At first the diverticula are reducible, so consecutive barium enemas performed on the same patient may show different numbers of them.

As diverticula enlarge their muscle covering atrophies and disappears until they finally consist of sacs of mucous membrane covered by peritoneum. They can then no longer contract and cannot expel faeces, which may become dried into the hard pellets that are felt at operation. Diverticula are found in two rows on each side of the colon and less often on the antimesenteric border where the perforating vessels are smaller. This regular arrangement may be lost if diverticula coalesce or burrow intramurally. Diverticula which follow the blood vessels track into the appendices epiploicae and are thus difficult to see at operation—especially in fat subjects—but palpation may reveal faecaliths.

The most striking pathological change is the thickening of the muscle of the sigmoid colon. Often the structure of the

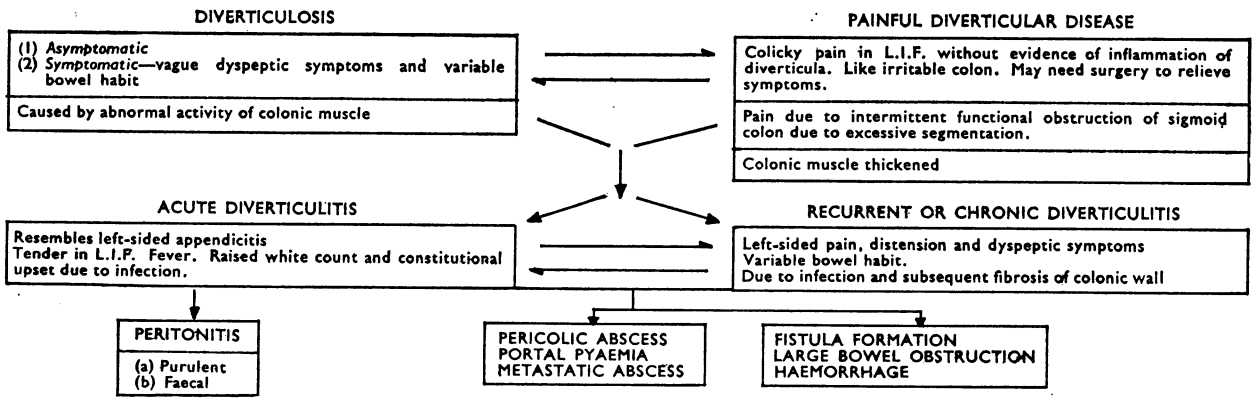


FIG. 1.—Diagram of manifestations of diverticular disease of the colon.

bowel changes abruptly from apparently normal colon to a rigid tube. Both the longitudinal and circular muscles are thickened. The circular muscle and mucosa are thrown into ridges which surround and narrow the lumen like incomplete sphincters, between which the diverticula are found. This structural change may be present without any evidence of inflammation and even in the absence of diverticula in sigmoid colons resected for "diverticulitis." The reason for this thickening is as yet unknown, but it has been suggested that it is caused by the prolonged ingestion of a diet that is not natural and to which the human colon is not yet adapted.[4]

Usually diverticula occur first in the sigmoid region, where the lumen of the colon is narrowest. As more diverticula appear the disease spreads proximally until sometimes the entire large gut and even the rectum may be affected. Solitary or multiple diverticula may occur in the caecum and ascending colon, where they may give rise to symptoms that mimic appendicitis. Occasionally the right half of the colon may be beset with diverticula while its left half is normal.

## Incidence

Diverticula appear in the latter half of life and are rarely seen in patients under the age of 35. It is said that 20% of the population of Western countries over the age of 40 have the disease, and its incidence increases with age until 60% of barium enemas performed on 80-year-old patients show diverticula. Men were said to be more prone to the disease than women, but this has been reversed in the last 30 years. The incidence is increasing in Western society, and this is true not only of those of European stock but also of those of African descent. The American Negro was once less apt to be affected by the condition than his white compatriots, but now complicated diverticulitis is found as commonly in the Negro as the white American.

## Pathogenesis

In the past it was thought that old age, obesity, constipation, and straining at stool were of aetiological significance in diverticulosis, but no convincing evidence has been produced to support this view. The little that was known about the causation of the disease was based on the study of dead tissues until cine-radiography and apparatus that was capable of recording the intracolonic pressure became available. Recently knowledge of colonic physiology and pathology has been widened and recent advances[4–7] may be summarized as follows:

(1) The pressures in the human sigmoid colon have been measured both in health and in diverticulosis. These pressures are usually less than 10 mm./Hg in amplitude, but when higher pressures are generated cineradiography shows that the sigmoid becomes demarcated into segments by contraction rings between adjacent segments. Each of these segments then behaves like a "little bladder" whose outflow is obstructed at each end by the contraction rings, which narrow and which may at time occlude the colonic lumen. Very high pressures in excess of 90 mm./Hg may be localized in such a segment. These pressures distend diverticula arising from the segment and have been shown by cineradiography to cause the extrusion of diverticula. Segmentation of the sigmoid into "bladders" causes the pulsion force responsible for diverticulosis; so not only the structure but also the pathogenesis of colonic and vesical diverticula have been shown to be essentially similar.

Segmentation occurs frequently, as it plays an important part in the transport and halting of faeces (Fig. 2). It is increased by morphine, prostigmine, eating, and emotion, which also increase the number and amplitude of the pressure waves. Pethidine lessens both segmentation and the intrasigmoid pressures, while propantheline bromide, which paralyses the colonic muscle so that it relaxes into an open tube, simultaneously abolishes the production of pressure. These observations show that the colonic muscle is responsible for the intraluminal pressure that leads to diverticulosis.

(2) Sigmoid colons resected for "diverticulitis" show the thickened muscle coat referred to above, but in only one-third of such specimens is sufficient inflammation found to account for the preoperative symptoms. In some specimens no diverticula are found, although the pain of "diverticulitis" was cured by operation. Thus the muscle abnormality not only precedes the appearance of diverticula but is capable of causing symptoms of sufficient severity to warrant surgery even when no diverticula are present.[8]

(3) Segments of the colon that are beset with diverticula produce more pressure than segments of the normal sigmoid or segments that do not bear diverticula in patients with diverticulosis. This differential pressure response of the diseased segments was found at an early stage in the disease when only one diverticulum was demonstrated. This also suggests that abnormal intraluminal pressures due to some muscle abnormality precede the appearance of diverticula.

(4) Diverticular disease is most prevalent in communities that eat a "civilized" diet which contains refined sugar and white flour but lacks natural fibre. Thirty years ago the American Negro was less prone to the disease than Americans of Anglo-Saxon stock, but he is equally afflicted by the disease today; concurrently, the sale of maize in the U.S.A. has decreased. Africans, who eat a bulky diet, have colons of large diameter which do not bear diverticula, but if they change to a Western diet for a sufficient time they develop diverticula.

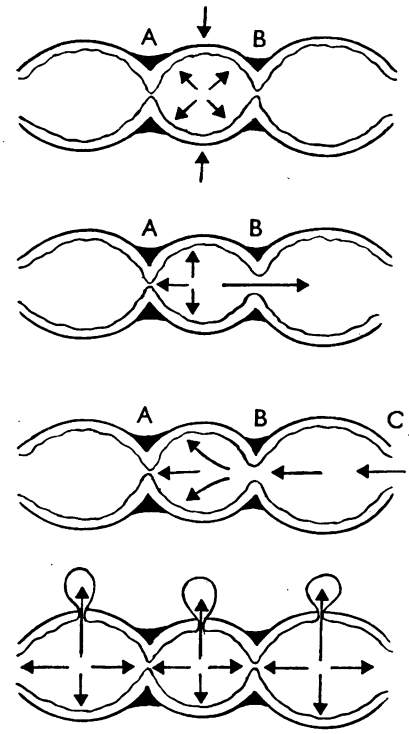


FIG. 2.—The role of segmentation in the physiology of the sigmoid colon and its relationship to the pathogenesis of diverticula. Diagram of longitudinal sections of human colon: *Top section* shows segment bounded by contraction rings, A and B, on each side of it; these rings obstruct its outflow at both ends. If the segment contracts its contents will resist the movement of the walls of the segment, as they cannot move into adjoining segments easily. Pressure will be produced in this segment and this pressure will be localized to this segment and will not affect its neighbours, which harbour different pressures. *Second section* shows ring A remaining contracted while ring B relaxes. The segment bounded by A and B harbours a higher pressure than its neighbours so that relaxation of ring B allows the segment's contents to move to the right in the direction of the arrow. This mechanism is usually responsible for the transportation of the colonic contents and it involves segmentation. *Third section* shows how segmentation may halt materials passing through the colon. Material moving from C towards B and A is slowed by the narrowing of the colonic lumen by the contraction of ring B and finally halted by ring A which has occluded the lumen. Pressure results from this segmentation and the intersegmental contraction rings act as a series of baffles. When inflammation of the colon leads to loss of function, these rings do not contract efficiently enough to slow the faecal stream and diarrhoea results. This is seen in active ulcerative colitis when the colon remains open like a drain. *Bottom section* shows how segmentation causes diverticulosis. The segmented colon acts as a series of "little bladders" whose outflow is obstructed at both ends by contraction rings. Between these rings the segment's wall is weaker and it is here that the mucosa is forced through the colonic wall by the pulsion force that is generated by segmentation.

(5) Rats fed on a low residue diet develop diverticula while those on a bulk-forming diet do not. Rats fed a high residue diet followed by a low residue diet are the most likely to have diverticula.[9]

To sum up, it appears that diverticula are caused by pressures generated by excessive segmentation resulting from abnormal activity of the colonic muscle. One of the factors that cause this abnormal activity is probably lack of natural fibre in the diet.[10]

The disease may be preventable when its aetiology is known, but meanwhile medical treatment should aim at combating the underlying muscle abnormality which causes diverticulosis and its symptoms. Diverticula become the prime cause of symptoms only when they are inflamed and true diverticulitis is present.

## Uncomplicated Diverticular Disease

The disorder of the sigmoid muscle which precedes the appearance of diverticula is almost certainly responsible for the vague dyspeptic symptoms of which patients with diverticula commonly complain—though anorexia, flatulence, and a feeling of abdominal distension are symptoms common to many other disorders of the gut. Even severe pain may be unaccompanied by inflammation of diverticula, and it is probably caused by excessive segmentation of the colonic muscle. The resultant functional obstruction leads to colic, which may wax and wane with a rapidity that is incompatible with the onset and resolution of an underlying inflammatory process. In the uncomplicated diverticular disease the bowel habit is variable. Most patients are not constipated but pass more than one soft motion daily. Mucus may be passed per rectum, but this suggests that another colonic condition is also present.

These symptoms should always suggest the diagnosis in patients over 40 years of age. Examination may reveal a contracted pelvic colon. Sigmoidoscopy is essential, but it is rarely helpful; an acute angle at about 15 cm. from the anus is consistent with the condition. The diagnosis must be made by barium enema. Even when diverticula are revealed by a barium meal followed through the gut, a barium enema and sigmoidoscopy are still essential if the extent of the disease is to be assessed and the presence of a neoplasm is to be excluded. Any change in the bowel habit, such as diarrhoea alternating with constipation, must not be attributed to diverticulosis in a patient who is known to have the disease until another barium enema and sigmoidoscopy have excluded a co-existent neoplasm. Diverticula must never be blamed for symptoms until the presence of other diseases has been excluded.

## Treatment

It has been suggested that patients in whom symptoms are absent or mild should not be told of the presence of diverticula lest they worry unduly. This attitude is usually wrong. Patients co-operate better when the nature of their condition has been explained to them, preferably with the aid of a simple diagram. They are asked to report any urinary symptoms, the passage of blood or mucus per rectum, or any change in the bowel habit. This leads to the earlier discovery of any superimposed carcinoma. Urinary symptoms must be investigated fully without delay, as they may herald the formation of a vesico-colic fistula.

There is no evidence that the hallowed regimen of a low-residue diet coupled with liquid paraffin or some other aperient is in any way beneficial. The prolonged use of paraffin may cause vitamin deficiency. The habitual use of aperients is dangerous and a low-residue diet is probably harmful. Treatment should ensure that the bowels are opened easily. This is best achieved by giving a bulk-former such as Normacol in a dosage of a teaspoonful daily with plenty of fluids. Alternatively a teaspoonful of bran may be taken with each meal and its quantity increased till the stools are formed but soft. Neither of these substances is useful in episodes of acute constipation, as they must be taken regularly to exert their effect. If a laxative is required, Senokot is the preparation of choice. Strong purgatives, enemas, and colonic lavage are dangerous and should be forbidden.

## Painful Diverticular Disease

Severe pain referred to the left iliac fossa or lower abdomen may be felt in the absence of any evidence of inflammation of diverticula. This pain may be recurrent, and is probably due to functional obstruction owing to segmentation of the colon. It is usually described as "cramping" or "colicky," and was once attributed to diverticulitis. The pain may cause the patient to sit down or to go to bed. It may be worse after a meal, probably owing to the gastro-colic reflex. Pain may be felt in the right iliac fossa if the sigmoid curves across the mid-line, and this may lead to a diagnosis of appendicitis being made. This pain and a feeling of distension are often relieved by passing wind or faeces. The bowel habit may vary, constipation or diarrhoea being experienced in acute attacks. Palpation usually reveals a tender contracted sigmoid colon. Subacute intestinal obstruction and left renal colic must be considered in the differential diagnosis. The diagnosis must not be made unless the temperature and white blood cell count are normal. It is confirmed later by barium enema, which must not be performed until the symptoms have subsided, as perforation may follow any enema given during an acute episode of pain.

The patient should rest and in severe attacks must be confined to bed. Diverticulitis, pericolitis, and peritonitis must be excluded by repeated examination and by the absence of fever or raised white cell count. Any doubt about the diagnosis, deterioration of the patient's general condition, or any increase in the severity of the pain warrants immediate admission to hospital. A light diet with plenty of fluids together with a bulk-former to soften the stools should be given. Antispasmodics may be helpful; intravenous or intramuscular propantheline will relax the colon, but its side-effects are unpleasant. Morphine should not be given, as it may predispose to perforation by raising the intrasigmoid pressures. Pethidine, which lowers these pressures, is the analgesic of choice. In some patients milk exacerbates symptoms, particularly when taken in its natural state. Purgatives and enemas must be withheld, as they may lead to perforation. After the acute attack has subsided a sigmoidoscopy and a barium enema are required to assess the extent of the disease and to exclude a colonic carcinoma. The regimen outlined for diverticular disease should then be followed after the acute attack has subsided.

Some patients notice that episodes of pain are precipitated by emotional stress. A small dose of phenobarbitone or a tranquillizer may lessen the frequency and severity of their symptoms. The majority of patients will never require surgery, but a few will have pain so frequently that life becomes intolerable, and they should be cured by sigmoid colectomy.

## Diverticulitis and its Complications

Inflammation of diverticula and the adjacent colon may occur at any time and give rise to acute, subacute, or chronic diverticulitis, with or without complications. Recurrent episodes of infection may occur frequently, or diverticulitis may resolve and be followed by years of freedom from ill-health. Diverticulitis and its complications are shown diagrammatically in Fig. 1 (page 475).

## Acute Diverticulitis, Pericolitis, and Pericolic Abscess

Inflammation that remains limited to one or more diverticula may give rise to a minimum of symptoms and then resolve spontaneously. These symptoms may be so slight as to make

it impossible to distinguish the condition from painful diverticular disease, and the two conditions may exist together and each contribute to the symptomatology.

If several segments of the sigmoid and its mesocolon become inflamed the symptoms are more marked and less intermittent than in painful diverticular disease, as the underlying inflammatory process takes longer to resolve than does intermittent obstruction due to muscular contraction. The infected colon, which usually lies in the left iliac fossa, gives rise to pain and tenderness due to local peritonitis. Fever, malaise, anorexia, and nausea together with a constitutional upset serve to distinguish diverticulitis from painful diverticular disease. Local peritonitis leads to tenderness and guarding in the lower abdomen maximal over the affected bowel. The diagnosis is usually obvious, but the signs may be mistaken for appendicitis if the colon curves into the right iliac fossa. Rectal examination gives rise to pain if the tip of the finger palpates inflamed bowel or a pelvic abscess. The small bowel may be involved and then ileus causes vomiting, abdominal distension, and constipation, although sometimes the motions are loose initially. Bowel sounds are decreased or absent, depending on the degree of ileus, and an erect radiograph of the abdomen may show fluid levels. The temperature and white count are raised.

Acute diverticulitis has been likened to a "left-sided appendicitis." The infection may resolve or progress to abscess formation or generalized peritonitis.

*Treatment.*—The patient should be nursed in hospital. No solid food should be allowed. Fluids should be given orally, or intravenously if ileus is present, when gastric suction must be instituted. Sulphonamides may be given by mouth, but if the patient is vomiting a broad-spectrum antibiotic should be given by another route. Pethidine may be given to relieve pain.

This conservative regimen may be followed by the resolution of diverticulitis and the gradual disappearance of the symptoms and signs, the return of appetite, and improvement in the patient's general condition. After recovery the state of the colon must be assessed by barium enema and sigmoidoscopy. Once carcinoma or organic obstruction has been excluded the medical regimen for diverticulosis should be instituted. Any urinary infection should be treated vigorously and a vesico-colic fistula excluded by cystoscopy. An episode of diverticulitis which resolves may be followed by little in the way of symptoms, but if recurrent episodes of infection or pain make life intolerable resection of the sigmoid should be performed, as only surgery will cure the condition.

### Abscess Formation

An abscess presents as a tender mass in the left iliac fossa or pelvis. The white count is raised, and peritonitis causes local ileus and some degree of obstruction in two-thirds of patients. A localized abscess may discharge into the bowel or decrease in size, but there is the ever-present danger of it enlarging and pointing through the overlying skin or bursting into the bladder or vagina or another part of the bowel. The most dangerous complication is rapidly spreading peritonitis following a rupture of the wall of the abscess. Conservative treatment of an abscess always carries the very real danger of sudden spreading peritonitis, and this is particularly so when both symptoms and signs are masked by gastric suction, intravenous therapy, antibiotics, and analgesics. Repeated examinations are necessary, and if there is the slightest suspicion that the abscess is enlarging or failing to localize laparotomy should be performed as a matter of urgency. Drainage of an abscess may be followed by fistula formation, which will be cured only by later resection of the affected sigmoid followed by closure of the colostomy.

### Peritonitis

Acute diverticulitis may lead to general peritonitis, which may be purulent or faecal in type.

*Purulent peritonitis* may present as a "left-sided appendix" that has failed to localize or as pelvic peritonitis secondary to appendicitis. In the early stages of peritonitis tenderness and guarding are maximal over the affected bowel, and as this is usually in the left iliac fossa the diagnosis presents no difficulty. Once peritonitis has become generalized its cause may be obscure, but diverticulitis should figure in the differential diagnosis of peritonitis occurring in patients over the age of 40.

*Faecal peritonitis* may follow the use of purgatives or enemas, and is caused by perforation of a diverticulum, allowing faeces to escape into the peritoneal cavity. The onset of symptoms is frequently catastrophic. Board-like rigidity may occur so quickly that it is impossible to be sure that the pain began in the left iliac fossa, and a diagnosis of a ruptured viscus requiring urgent laparotomy is the only diagnosis that can be made. Shock is more marked than in purulent peritonitis.

Choice of procedure is still a vexed question. The mortality of purulent peritonitis has dropped from over 30% to about 12% as a result of the advent of antibiotics and advances in supportive therapy, though faecal peritonitis remains as lethal as it was before the war.

Purulent peritonitis may respond to mere drainage of the abdomen. This was once thought to be adequate, but the addition of a proximal colostomy does not increase the mortality, and besides resting the distal bowel it constitutes the first part of a three-stage curative resection. Drainage and colostomy has been the most commonly used emergency procedure for many years, but still it is followed by a high mortality and a very great morbidity. The colostomy does not prevent faeces leaking from any subsequent perforation of the sigmoid that may occur in the immediate postoperative phase. For this reason exteriorization of the inflamed sigmoid and the perforation, coupled with drainage of the abdomen, is considered to be a better procedure by many surgeons. The exteriorized sigmoid is opened as a colostomy. This procedure reduces likelihood of further soiling of the peritoneum postoperatively. After about three weeks the appendices epiploicae have disappeared, leaving granulating healthy colon which may then be resected and an intra-abdominal anastomosis performed. The patient is thus cured by two operations, provided that all the thickened sigmoid is resected and not merely that portion which is outside the abdomen.

Exteriorization has one serious disadvantage. It does not remove the suppurating colon, which continues to discharge its toxic products into the patient's circulation. Perforated diverticulitis is a desperate emergency, with such a high mortality that it has been argued that it is the disease and not the operation that kills the patient, and that more drastic surgery is not only justified but essential if more patients are to be saved. For this reason emergency resection of the suppurating sigmoid has been practised by an increasing number of surgeons. An adequate sigmoid colectomy can only be performed intraperitoneally in the great majority of cases. A primary end-to-end anastomosis is theoretically the ideal, and in the hands of experts resection and immediate anastomosis has lowered the mortality to about 7%. It must be pointed out that these excellent results have only been obtained by very experienced surgeons who are specifically interested in this problem. For this reason I believe that resection followed by closure of the lower sigmoid and the conversion of the open end of the proximal colon into a terminal colostomy should become the standard procedure. This technique removes the toxic sigmoid, allows peritoneal toilet and drainage to be performed, and does not involve an emergency intraperitoneal anastomosis, which prolongs the operation and which is in itself not without risk.

### Fistula Formation

Recurrent inflammation may cause the sigmoid to adhere to neighbouring structures, and if a pericolic abscess then forms

it may rupture into the bladder, vagina, or small or large bowel. Drainage of an abscess may lead to a fistula connecting the sigmoid to the skin surface. These fistulae rarely heal without the prior resection of the diseased colon.

*Vesico-Colic Fistula.*—This complication is more common in men than in women, as the uterus tends to keep the bladder and colon apart. The fistula allows the intestinal flora to infect the urine, and it cannot be too strongly emphasized that any urinary infection in a patient with diverticulosis must be investigated to exclude the presence of a fistula. The symptom of pneumaturia or the passage of faeces in the urine makes the diagnosis obvious. Vesico-colic fistula may be caused also by carcinoma of the colon. Visual differentiation of benign and malignant fistulae at cystoscopy may be difficult. Large fistulae tend to be malignant. Transurethral biopsy may give proof of neoplastic infiltration of the bladder, but a negative biopsy does not exclude malignancy. Sigmoidoscopy will rule out carcinoma of the lower bowel and a barium enema may outline the fistula. An intravenous pyelogram must be performed, not only to assess the renal function and to show that the ureters are patent, but also to exclude a nephro-colic fistula.

Vesico-colic fistula can be cured only by surgery. A one-stage resection of the affected bowel coupled with closure of the bladder is the ideal operation. Dense adhesions and granulation tissue may make it necessary to use a three-stage procedure; a defunctioning colostomy allows the inflammation to resolve so that sigmoidectomy and closure of the bladder can be performed safely. The colostomy is closed at a third operation. If the presence of a carcinoma is suspected, the resection must take place as early as possible.

Very old and frail patients with these fistulae may be spared major surgery, as modern chemotherapy will control the inevitable recurrent urinary infection. This conservative approach will allow the patients to finish their lives without a permanent colostomy, which would otherwise be the fate of those who are not fit to withstand resection.

*Colo-vaginal Fistula* results in the passage of air, faeces, or pus per vaginam. Such fistulae may be caused not only by diverticulitis but by carcinoma of the bowel or cervix or following irradiation. The treatment of this complication of diverticulitis is again surgical.

Entero-colic fistulae and colo-cutaneous fistulae require similar investigation. The former may only be demonstrated with the aid of cineradiography. Resection of the diseased colon must precede closure of the fistula.

## Large Bowel Obstruction

Intestinal obstruction due to temporary ileus is seen in two-thirds of the patients with acute diverticulitis. Organic obstruction, on the other hand, is due to stenosis of the sigmoid colon following recurrent attacks of diverticulitis, which cause fibrosis of the colonic wall and narrowing of its lumen. This leads to subacute obstruction, which presents as an alteration in the bowel habit, constipation being interspersed with attacks of diarrhoea, which are often brought on by the use of purgatives. The patient feels distended, and this sensation is sometimes relieved by passing wind or faeces. These symptoms are common to both diverticular disease and carcinoma of the colon. As these common diseases may exist together, it is essential that a full investigation of any change in the bowel habit is carried out urgently even in patients who are known to have diverticular disease, otherwise a superimposed cancer may be missed. Sigmoidoscopy, barium enema, and the finding of malignant cells in rectal washings help to make the diagnosis, but sometimes the true diagnosis is not clear even at operation.

The relief of symptoms can be achieved only by sigmoid colectomy. When possible, this should be performed in one stage, but it must be remembered that diverticulosis is a benign condition with a good prognosis, and so its surgical treatment must be doubly safe: so if the operation is technically difficult a relieving colostomy or a staged procedure must be used.

## Haemorrhage in Diverticular Disease

The passage of dark, undigested blood per rectum in elderly patients is almost diagnostic of bleeding due to diverticulosis. The haemorrhage almost always stops spontaneously with rest in bed. The cause of the bleeding can then be investigated and any anaemia corrected. Rarely bleeding may be so severe that an emergency laparotomy is necessary.

## Sigmoid Myotomy

Reilly[11] introduced sigmoid myotomy as an alternative to resection of the sigmoid for diverticular disease. His claim that this procedure is less hazardous in sick patients has yet to be substantiated. The operation consists of incising the muscle of the sigmoid colon longitudinally in the manner of Ramstedt's or Heller's operations; the incision is made through a taenia so as to avoid perforating diverticula. Postoperative barium enemas show that this widens the colonic lumen, and Reilly and others have reported excellent results with a restoration of a normal bowel habit. Theoretically the operation is attractive, as the contraction rings that cause segmentation are destroyed, but it is too early to say whether the operation yields results that are permanent or whether scar tissue leads to a recurrence of stenosis and symptoms. Sigmoid myotomy is unlikely to replace colectomy as the procedure of choice for recurrent diverticulitis, but it may well find a place in the management of painful diverticular disease.

## Diverticular Disease of the Caecum and Ascending Colon

Diverticula are found in the caecum and ascending colon:

(1) As part of a generalized diverticulosis.

(2) As solitary diverticula, especially in the caecum.

(3) In the right half of the colon when the left colon is normal.

It appears that the incidence of diverticulosis of the right half of the colon has been underestimated in the past, and recently it has been found in a large series that nearly one-third of those with diverticula of the right colon have no diverticula on the left side. Clinically, acute diverticulitis occurring in the right half of the colon and caecum presents as, and is diagnosed as, acute appendicitis unless a mass is palpable, when the differential diagnosis includes Crohn's disease, carcinoma of the caecum, and appendix abscess. The so-called "solitary ulcer of the caecum" may be the result of inflammation of a solitary diverticulum. The treatment of diverticulitis of the right colon is surgical. An abscess should be drained, but if there is any suspicion that a neoplasm is present an immediate right hemicolectomy should be performed. Solitary diverticula should be invaginated with a purse-string suture.

What symptoms may be caused by diverticula in the right colon which are not inflamed is still a matter of conjecture, as little has been written on this subject. Diverticula in this site are, however, prone to bleed, and in one series nearly half of those patients with diverticula in the right half of the colon passed blood per rectum.

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