

# DIVERTICULAR DISEASE OF THE LARGE INTESTINE

D. E. BOLT F.R.C.S.

Consultant Surgeon, West Middlesex Hospital, Isleworth; Royal College of Surgeons Tutor, West Middlesex Region

## Summary

THE PATHOLOGY, AETIOLOGY, and natural history of diverticular disease are reviewed, with particular reference to the generally benign nature of the condition and of its inflammatory complications and to the characteristics of the unfavourable case in which radical measures are indicated.

The management of the acute case of diverticulitis is discussed and the importance of differentiating between the case with rupture of a peridiverticular abscess and the case with true colonic perforation and faecal contamination is emphasized. Particular reference is made to the importance of avoiding colostomy in all but a minority of patients and to the absence of any indication for immediate or delayed resection in the great majority of cases.

The indications for surgical management of elective cases are also reviewed, with emphasis upon the value of high-residue regimens and with suggestions for the appropriate use of sigmoid myotomy. The problem of the case of doubtful malignancy is discussed and a brief reference made to the management of fistulae.

## Introduction

WILLIAM OSLER ONCE said, 'As is our pathology, so is our practice', and provided that in the word 'pathology' we include our concept of the cause and natural history of the disease we are considering, this quotation contains a fundamental truth. I make no apology, therefore, for beginning this discussion of diverticular disease of the colon with some comments on its pathology.

## Morbid anatomy

There are three elements in the morbid anatomy of diverticular disease.

**1. The diverticula.** These are projecting pouches of mucosa through the muscle wall of the colon, classically in two rows between the taeniae at points where entering vessels create foramina. Commonly in the sigmoid colon, stopping at the rectosigmoid junction where the taeniae are replaced by a continuous longitudinal muscle coat, they extend proximally over varying lengths of the colon, but in only 2% of cases are they present proximal to the splenic flexure<sup>1</sup>. More importantly, resected bowel from patients with the clinical and operative picture of diverticular disease may contain no diverticula on the most detailed dissection<sup>2, 3</sup>. In diverticular disease as we understand it diverticula are a frequent but not invariable feature.

**2. Inflammation.** Faecal retention in diverticula with ulceration and

inflammation leading to an inflammatory mass, a pericolic abscess, peritonitis, fistula, or large-bowel obstruction has been the subject of surgical attention for years. Important as these complications are, the inflammatory aspects of the disease have in the past occupied too large a place in our thoughts, confusing our policy in treatment. One-third of specimens from cases sufficiently impressive to warrant resection show negligible evidence of inflammatory disease<sup>2, 3</sup>. Follow-up of patients diagnosed radiologically for periods of between 10 and 30 years shows that 60% have no evidence of inflammatory disease at any time<sup>4, 5</sup>. Significant inflammation is an even less constant feature of diverticular disease than are the diverticula themselves.

**3. Muscle thickening.** This is the one constant feature of the disease<sup>2, 3</sup> and affects the circular muscle, giving rise to the 'prediverticular state' visualized radiologically in 1926<sup>6</sup>. Counts of muscle cells suggest that the thickening is due to spasm, not hypertrophy<sup>2, 3</sup>, while detailed studies of affected colons show that bands of spastic circular muscle create intraluminal projections, typically on alternate sides of the bowel, with diverticula arising at the apices of the intervening mucosal trenches<sup>7</sup>. The constancy of the muscle thickening strongly suggests that when discussing diverticular disease we are in fact talking about a disease of the colonic musculature, with which pouches and inflammatory disease may be incidentally associated.

### **Aetiology**

The classic explanation of the condition has been that a rise in intraluminal pressure causes the protrusion of mucosal pouches through the apertures in the muscle coat traversed by blood vessels<sup>8</sup>. Modern investigations using balloons or open-ended catheters to study intraluminal pressures have not greatly elucidated the situation, although it has been suggested that in the diverticular state pressures in the sigmoid, especially after eating, are higher than in the rectum but are comparable in the two viscera in normal subjects<sup>9-14</sup>. A relationship has been suggested by radiological investigations with that other obscure condition, 'spastic' or 'irritable' colon<sup>15-16</sup>, but the increasing incidence of diverticular disease in Western countries compared with the low incidence in Afro-Asian peoples at home does suggest a relationship to low-residue Western diets, a theory supported by experimental work in rats<sup>17-20</sup>. Hodgson<sup>21</sup> reports that the difference in intraluminal pressures between sigmoid and rectum in diverticular subjects can be eliminated by a 6-month period of medication with methylcellulose, thus giving experimental support to the view that there is no place for the continued use of low-residue diets in patients with diverticular disease.

### **Natural history**

In 1957 McLaren<sup>22</sup> said 'There are two surprising features of the

## DIVERTICULAR DISEASE OF THE LARGE INTESTINE

British and American literature on perforated diverticulitis. The first is the lack of statistical information regarding the incidence and mortality of the condition. The second is that there has not been any investigation of the factors determining death or survival.'

Since this statement was made there have been a number of studies aimed at clarifying these points. In a series of 503 cases diagnosed radiologically and followed up for 18 years, 36.7% showed some evidence of inflammatory disease, but only 2 patients required surgery<sup>4</sup>, while a similar series of 249 cases followed up for 30 years showed a 40% incidence of inflammation, with 16 patients requiring surgery<sup>5</sup>. In a series of 455 patients with symptoms from their diverticular disease sufficiently severe to require hospital admission, 317 were treated medically in the acute attack and 43% of these continued to have symptoms, 5% severely. The remaining 138 were treated surgically, symptoms persisting in 32% of those who did not undergo resection and in 28% of those who did. In these large series other relevant facts become apparent. The distribution of diverticula is determined early in the disease and there is little tendency for the pouches to spread along the colon, being confined to the sigmoid in 65% of cases. The prognosis with respect to inflammatory disease is determined by the sigmoid diverticula, and multiple diverticula elsewhere in the colon do not significantly increase the risk of complications. Moreover, major inflammatory complications tend to occur within a short time of the onset of symptoms, 80% of the patients who die of the disease having histories of a month or less<sup>23-25</sup>. This is a very important observation in relation to prophylactic resection for diverticular disease, which is widely advocated in the American literature. At the Lahey Clinic 70% of patients diagnosed as having diverticulitis undergo resection to anticipate serious complications<sup>26</sup>.

The surprising similarity in the prognosis among patients suffering the inflammatory complications of diverticular disease treated with or without resection was confirmed in a series of 100 consecutive patients with diverticulitis who underwent laparotomy and were reviewed 5-15 years after their initial operation<sup>27</sup>. Of those who underwent resection, 59% remained symptom-free for the period of follow-up, compared with 58% of those not treated by resection. However, while the majority of recurrences in both groups were of a minor nature, among those who had not undergone resection there was a group of about 15% who exhibited severe progressive disease culminating either in resection or in serious disability. Even in those with major disability or fistula the disease tended eventually to burn itself out, with only one late death attributable to the disease, but it is clearly important to identify this group of cases at an early stage if prophylactic resection is to be applied usefully to the patients actually needing it and avoided in cases

with a good prognosis. The characteristic feature of cases with potentially progressive disease is a history of weeks or months of increasing symptoms culminating in the acute episode requiring admission, in contrast to favourable cases, in which the acute episode occurs almost or completely without warning<sup>25, 27</sup>. It should be emphasized that a history of the progressive type is particularly unfavourable in the young patient<sup>28</sup>. It is clear, however, that the patient suffering from the isolated acute attack of perforated diverticulitis is unlikely to become the subject of major complications subsequently, and that prophylactic resection in such cases will not diminish the mortality or morbidity of the disease<sup>29</sup>.

This, then, is our concept of the pathology of diverticular disease. Let us now consider its application to our surgical practice in various types of case.

### **The acute case**

#### **1. The acute inflammatory episode without major peritonitis.**

These patients present as emergencies with evident perisigmoid inflammatory disease, possibly associated with minor lower abdominal peritonitis but without serious diagnostic uncertainty. The majority will resolve on conservative management with drip, suction, and antibiotics, allowing mature assessment and a rational selection of a very few for resection. The few requiring radical surgery will be those showing incomplete resolution, which identifies them as belonging to the 15% of cases with severe progressive disease carrying a bad prognosis without resection.

#### **2. The acute inflammatory episode with major peritonitis.**

These are patients in whom the degree of peritonitis or uncertainty regarding the source of the peritonitis make surgical management imperative. Within this group there are two important subgroups: (a) those with turbid or purulent peritonitis, and (b) those with faecal peritonitis.

It is imperative to distinguish between these subgroups, which are entirely distinct in clinical picture, operative findings, surgical management, and prognosis, as failure to do so generates an illogical demand for routine primary resection<sup>30-32</sup>. If the two groups are separately analysed, it will be seen that the prognosis is related to the type of case rather than to the method of treatment employed<sup>33</sup>.

Patients with turbid or purulent peritonitis show the picture of a toxic illness with fever and tachycardia, lower abdominal or diffuse tenderness, guarding and peritonism, and diminished or absent bowel sounds, but are in a reasonable general state with no evidence of circulatory collapse. At laparotomy the abdomen contains turbid or

purulent fluid, reddened and oedematous peritoneal surfaces, and a thick, oedematous sigmoid colon, commonly adherent in the pelvis, without an evident site of perforation. Clearly a pericolic collection has ruptured into the peritoneum, but as the neck of the relevant diverticulum has been closed by the inflammatory process, communication with the bowel lumen has not been established and faecal contamination of the peritoneum will not occur. Consequently handling of the inflamed sigmoid should be minimal to avoid the risk of creating a faecal leak, and no attempt should be made to find and suture the perforation. The contaminating fluid should be aspirated, free drainage from the pelvis established, and the abdomen closed.

Patients with faecal peritonitis exhibit a different order of clinical disturbance. They have clear evidence of diffuse peritonitis, but the degree of associated circulatory disturbance is almost always profound, with marked tachycardia and hypotension. Free gas is commonly to be seen in a plain X-ray of the abdomen, and at laparotomy the peritoneum contains evident faecal fluid from a freely communicating perforation into the lumen of the sigmoid colon. Despite the anomalous findings in the Australian survey of perforated diverticulitis<sup>34</sup>, suture of the perforation is quite ineffective in controlling the faecal leak and the only hope for the patient lies in exteriorization of the perforation or resection of the affected segment, with creation of a terminal colostomy, leaving the possibility of anastomosis at a later date<sup>35</sup>. Neither patient nor peritoneum is fit for the more complex manoeuvre of primary anastomosis under these conditions.

The management I have suggested for the acute case with inflammatory complications includes neither of the two measures most commonly recommended in these circumstances—namely, transverse colostomy and primary resection with anastomosis, each of which deserves some individual comment.

*Transverse colostomy.* There is little place for this device in the treatment of acute perforated diverticulitis. In patients with turbid or purulent peritonitis there is no faecal leak and consequently little advantage in diversion of the faecal stream, since pericolic inflammation commonly continues unabated in the presence of a colostomy<sup>27</sup>. Moreover, it pre-judges the need for resection. In diverticulitis closure of colostomy without resection is commonly followed by severe progress of the disease, while permanent retention of the colostomy does not prevent major complications developing in half the patients concerned<sup>23, 27, 30</sup>. Consequently establishment of a colostomy in cases of diverticulitis must be regarded as a preliminary to resection, and it is clearly undesirable to create a situation in which resection is inevitable unless a logical indication for resection already exists.

In patients with faecal peritonitis transverse colostomy represents wholly inadequate treatment. It will not arrest continuing faecal contamination from the colon distal to the colostomy sufficiently quickly to save the patient's life. Only exteriorization or resection of the perforation will do this. Occasionally, transverse colostomy and pelvic drainage may be all the intervention that the patient will stand, but it should not be regarded as representing adequate treatment.

*Primary resection with anastomosis.* This form of management for all cases of diverticulitis coming to emergency surgery is widely advocated in the United States<sup>26, 36, 37</sup>, because it is erroneously believed that the majority of such patients will ultimately require resection. More recently a trend towards primary resection with delayed anastomosis, in the interests of a lower mortality, has become apparent<sup>38-40</sup>, while some authors have begun to question the need for routine resection in these patients<sup>41, 42</sup>, suggesting a changing attitude to resection for diverticulitis in North America.

The objections to primary resection with anastomosis are: (1) In patients with purulent peritonitis it is unnecessary to save life and may be unnecessary at any time. (2) Under emergency conditions it is difficult to make the kind of assessment which will enable those cases to be selected in which resection will ultimately be necessary. (3) In faecal peritonitis resection is imperative, but neither patient nor peritoneum is fit for safe anastomosis. (4) The technical difficulties of colonic resection and anastomosis are very great under emergency conditions and the mortality will be needlessly high in any but very skilled hands.

**3. Cases with intestinal obstruction.** With few exceptions<sup>43</sup>, most authors report a similar experience to my own—that large-bowel obstruction is an uncommon complication of diverticular disease. Commonly obstruction in this condition is due either to adhesion of the ileum to an inflammatory mass, a situation important to recognize if a disastrous error in management is to be avoided, or to a functional obstruction of the colon enveloped in an inflammatory mass, a condition capable of resolution as the inflammation subsides. When a true fibrous stricture of the colon does occur resection will be inevitable and preliminary transverse colostomy, with subsequent staged resection, the management of choice. If there is a place for primary resection and anastomosis in diverticular disease, this is it, and I have used it on a number of occasions with a satisfactory outcome; but in general I am sure that a staged programme carries a lower mortality.

**4. Cases with haemorrhage.** Haemorrhage is the one acute complication of diverticular disease unrelated to inflammation. It is episodic, commonly profuse, and often occurs in elderly hypertensive patients with very multiple diverticula. The source is probably ulceration at the neck of a diverticulum eroding one of the major vessels closely related to the diverticulum at this site. While 60% of cases require transfusion, only 20% are sufficiently persistent to need surgery<sup>44</sup>. When laparotomy is unavoidable it is unsafe to assume that any inflamed segment of colon discovered is the source of the bleeding, and the only safe course is total colectomy with ileorectal anastomosis<sup>45</sup>. Happily, the great majority can be managed without surgery.

### The elective case

There are four groups of patients with diverticular disease in whom elective surgery is justified.

**1. Severe progressive inflammatory disease.** These patients may present with an acute episode managed medically or by conservative surgery but give a history of weeks or months of increasing symptoms suggesting progressive inflammatory disease; or, following such an acute episode, they may have persisting symptoms suggesting continuing inflammatory disease. Alternatively, without an acute episode requiring admission to hospital, they may have a similar history of progressive trouble, implying continuing active inflammation. All of these belong to the group of 15% of cases with a bad prognosis and, after due assessment, should be offered resection. However, it should be emphasized that a second or third acute attack of diverticulitis with freedom from symptoms between the episodes does not imply a worse prognosis than the first attack and carries no specific indication for radical surgery.

**2. Continuing disturbance of colonic muscle function.** In the past we have been unduly preoccupied with the inflammatory complications of diverticular disease and too ready to assume that all symptoms were attributable to diverticulitis. The frequent absence of histological evidence of inflammatory disease in resected colons has drawn attention to the fact that significant pain and tenderness in the left iliac fossa, with frequent loose, mucus-containing stools, may result from disturbed muscle function in the uninflamed colon, allowing a more rational approach to management. For many such patients their problems can be solved by the use of bulk aperients such as methylcellulose<sup>17, 20</sup> or bran, sometimes combined with antispasmodics, eliminating the need for surgery. The few failing to respond to this treatment are probably best dealt with by the technique of colomyotomy described by

Reilly<sup>46-49</sup>, but it must be remembered that even this very conservative operation carries a mortality<sup>50</sup> and that in patients with the most satisfactory symptomatic relief the initial reduction in intraluminal pressure is not maintained<sup>51</sup>.

**3. Doubtful malignancy.** In a significant number of patients with proven or suspected diverticular disease it may be impossible to establish by clinical, radiological, and endoscopic means that the colonic lesion is not a carcinoma in itself or, if clearly diverticular, that it does not conceal a small carcinoma. In such cases laparotomy is clearly unavoidable but unfortunately does not always solve the problem<sup>52</sup>. Among my own cases coming to laparotomy for this reason, 15% required resection because the diagnosis remained uncertain, while Lloyd-Davies<sup>53</sup> records 2 cases in which abdominoperineal resection of the rectum was performed for diverticulitis because, at operation, the lesion was still regarded as carcinomatous.

In assessing the situation at operation, the important fact to be remembered is that diverticulitis is a pericolic lesion, while carcinoma is a mucosal one. If the sigmoid mucosa can be adequately palpated errors can be avoided and, in difficult cases, a small proximal colotomy to allow finger exploration of the mucosa directly may be worth while<sup>8</sup>. When a routine anterior resection of the rectum is possible the difference in scale between resection for carcinoma and for diverticulitis will not be fundamental, but if effective treatment of carcinoma would involve abdominoperineal resection of the rectum or widespread removal of adjacent viscera, a firm diagnosis must be established to avoid unjustified risk.

**4. Fistulae.** Time permits only a brief reference to the most disabling complication of diverticular disease. It would evidently be preferable to anticipate the appearance of a fistula and prevent it by timely surgery, but unfortunately in up to 85% of cases the symptoms are insufficient before appearance of the fistula for the patient to seek advice<sup>27</sup>. Nevertheless, patients with persistent *Escherichia coli* infections of the urinary tract in whom investigation reveals no cause frequently merit a barium enema.

Management of the established fistula involves resection of the affected colon, as attempts to separate and close fistulae are foredoomed to failure. However, in frail patients who are judged unfit for radical surgery it should be remembered that, eventually, most such fistulae will undergo spontaneous cure<sup>27</sup> and that the effect of them on the urinary tract is less disastrous than might be anticipated.



## DIVERTICULAR DISEASE OF THE LARGE INTESTINE

### REFERENCES

1. HUGHES, L. E. (1969) *Gut*, **10**, 336.
2. MORSON, B. C. (1963) *Proceedings of the Royal Society of Medicine*, **56**, 798.
3. MORSON, B. C. (1963) *British Journal of Radiology*, **36**, 385.
4. HORNER, J. L. (1958) *American Journal of Digestive Diseases*, **3**, 343.
5. BOLES, R. S., and JORDAN, S. M. (1958) *Gastroenterology*, **35**, 579.
6. SPRIGGS, E. I., and MARXER, O. H. (1926) *British Medical Journal*, **1**, 130.
7. HUGHES, L. E. (1970) *Gut*, **11**, 111.
8. ELLIS, H. (1970) *British Medical Journal*, **3**, 565.
9. PAINTER, N. S. (1964) *Annals of the Royal College of Surgeons of England*, **34**, 98.
10. PAINTER, N. S. (1964) *Gut*, **5**, 201.
11. PAINTER, N. S. (1964) *Gut*, **5**, 365.
12. PAINTER, N. S. (1965) *Gastroenterology*, **49**, 169.
13. PAINTER, N. S. (1968) *British Medical Journal*, **3**, 475.
14. PARKS, T. N., and CONNELL, A. M. (1969) *Gut*, **10**, 534.
15. FLESCHNER, G. F. G. (1971) *Gastroenterology*, **60**, 316.
16. SAWYER, K. C., SAWYER, R. B., and WAGGENER, H. U. (1969) *Diseases of the Colon and Rectum*, **12**, 49.
17. PAINTER, N. S. (1969) *British Medical Journal*, **2**, 764.
18. CALDER, J. F. (1971) *British Medical Journal*, **2**, 654.
19. GARY, R. C. (1971) *British Medical Journal*, **2**, 773.
20. PAINTER, N. S., and BURKITT, D. P. (1971) *British Medical Journal*, **2**, 450.
21. HODGSON, J. (1972) *British Medical Journal*, **3**, 729.
22. McLAREN, J. F. (1957) *Journal of the Royal College of Surgeons of Edinburgh*, **3**, 129.
23. PARKS, T. G., and CONNELL, A. M. (1970) *British Journal of Surgery*, **57**, 775.
24. PARKS, T. G. (1969) *British Medical Journal*, **4**, 639 and 642.
25. PARKS, T. G. (1970) *Proceedings of the Royal Society of Medicine*, **63**, 1262.
26. COLCOCK, B. P. (1971) *Surgical Clinics of North America*, **51**, 791.
27. BOLT, D. E., and HUGHES, L. E. (1966) *British Medical Journal*, **1**, 1205.
28. COLCOCK, B. P. (1963) *Postgraduate Medicine*, **32**, 217.
29. MACGREGOR, A. B., ABERNETHY, B. C., and THOMSON, J. W. (1970) *Journal of the Royal College of Surgeons of Edinburgh*, **15**, 137.
30. BYRNE, J. J., and GORICK, E. J. (1971) *American Journal of Surgery*, **21**, 379.
31. ZOLLINGER, R. W., and ZOLLINGER, R. M. (1971) *Advances in Surgery*, **5**, 255.
32. MADDEN, J., and TAN, Y. T. (1961) *Surgery, Gynecology and Obstetrics*, **113**, 646.
33. ROXBURG, R. A., DAWSON, J. L., and YEO, R. (1968) *British Medical Journal*, **3**, 465.
34. KILLINGBACK, M. J. (1970) *Diseases of the Colon and Rectum*, **13**, 444.
35. SAMES, C. P. (1961) *Proceedings of the Royal Society of Medicine*, **54**, 725.
36. BYRNE, R. V. (1966) *American Journal of Surgery*, **112**, 273.
37. DANDEKAR, N. V., and McCANN, W. J. (1969) *Diseases of the Colon and Rectum*, **12**, 172.
38. BATSFORD, T. W., ZOLLINGER, R. M., and HICKS, R. (1971) *American Journal of Surgery*, **121**, 702.
39. MILLER, D. W., and WICHERN, W. A. (1971) *American Journal of Surgery*, **121**, 536.
40. WHELAN, C. S., FURCINITTI, J. F., and LAVARREDA, C. (1971) *American Journal of Surgery*, **121**, 374.
41. JACOBS, E. (1971) *Diseases of the Colon and Rectum*, **14**, 29.
42. BARABAS, A. P. (1970) *British Medical Journal*, **3**, 767.
43. RAMSDAHL, M. M., and COLE, W. (1962) *Archives of Surgery*, **86**, 751.
44. RAMONATH, H. K., and HIRSHAW, J. R. (1971) *Archives of Surgery*, **103**, 311.
45. HEALD, R. J., and RAY, J. E. (1972) *Proceedings of the Royal Society of Medicine*, **65**, 779.
46. REILLY, M. C. T. (1964) *Proceedings of the Royal Society of Medicine*, **57**, 556.
47. REILLY, M. C. T. (1965) *Diseases of the Colon and Rectum*, **8**, 42.
48. REILLY, M. C. T. (1966) *British Journal of Surgery*, **53**, 859.
49. REILLY, M. C. T. (1970) *British Medical Journal*, **3**, 570.
50. DICK, E. T. (1971) *Diseases of the Colon and Rectum*, **14**, 341.
51. PRASAD, J. K., and DANIEL, O. (1971) *British Journal of Surgery*, **58**, 304.
52. WAUGH, J. M., and WALT, A. J. (1957) *Surgery, Gynecology and Obstetrics*, **104**, 690.
53. LLOYD-DAVIES, O. V. (1953) *Proceedings of the Royal Society of Medicine*, **46**, 407.