Progress in Diverticular Diseases of the Colon

A. A. BROWN, M.B., CH.B., F.R.C.S. (ENG.), *Department of Surgery, University of Cape Town*

SUMMARY

Segmentation is an important factor in the influence of diverticular disease on colon motility.

Psychosomatic factors must be taken into account, and surgery has also contributed to our understanding of diverticular disease. It is important to evaluate the place of surgical intervention in this condition.

S. Afr. Med. J., **45**, 1153 (1971).

COLON MOTILITY AND DIVERTICULAR DISEASE

Disorders of colon motility are being increasingly linked with diverticular disease and points in the physiology of colon motility that are relevant are the following:

Peristalsis, which may be propulsive with onward movement of contents or mixing when segmentation occurs. This segmentation, which results in localized areas being isolated and subjected to increased pressure, is important because if this is excessive it may result in pathologically high pressure. These movements are more important physiologically in the small intestine but occur in both the small intestine and large intestine.

After each meal, or 3 - 4 times a day, a mass reflex occurs and faeces are shunted onwards to the pelvic colon, not normally passing beyond the pelvirectal flexure. There may be a local thickening of the circular muscle at this site, which some think constitutes a sphincter, while others agree that narrowing is demonstrable here radiologically. There is no doubt that most peristalsis occurs physiologically in this area and that faeces do not enter the rectum until just before defaecation. One remembers in confirmation of this how often the rectum is completely empty on sigmoidoscopy.

If morphine is given segmentation increases but propulsive waves disappear, while atropine and other antispasmodics markedly reduce all aspects of motility.

Residue in diet equals residue in faeces. For instance a diet containing coarse brown bread gives three times the weight of faeces of a diet containing fine white bread. Experimentally, low-residue diet causes diverticular disease and our diet tends to be low residue because of the high degree of purification of the food we eat, and because we have been made afraid of eating. Under these conditions diverticular disease is common, and it is rare where the diet is high residue. The suggested pathogenesis is a disorder of segmentation, probably excess segmentation, and occurring most commonly in the pelvic colon. One may well ask whether diverticular disease is then not a deficiency disease caused by lack of roughage in the diet.

PSYCHOSOMATIC FACTORS—SPASTIC BOWEL SYNDROME—DIVERTICULAR DISEASE?

Psychosomatic and environmental differences influence the physiology of the gastro-intestinal tract: for example, the spastic bowel syndrome, or irritable gut syndrome, or spastic colon syndrome affecting both the large and the small bowel and comprising bouts of motor activity comparable to physiological movements but more violent, inco-ordinated, and sometimes excruciatingly painful. The importance of psychosomatic factors in our environment is well shown in the treatment of spastic bowel syndrome where the nature of the symptoms tend to remain constant whatever drugs are used but where their severity lessens proportionately to the degree of care and sympathy in the management. Is there a relation between this syndrome and diverticular disease? Is spastic bowel syndrome a predisposing cause to diverticular disease? If diverticular disease is a complication of disordered colon motility and spastic bowel syndrome does precisely this, surely it may also be associated with diverticular disease. Note, too, how abuse of aperients, i.e. using strong aperients to obtain regular but completely spurious bowel actions in the presence of an inadequate dietary intake, would produce a similar disorder of peristalsis and a basis for development of spastic bowel syndrome and diverticular disease, facts which are well substantiated clinically.

WHAT SURGERY HAS TAUGHT US ABOUT DIVERTICULAR DISEASE

A great deal of information about diverticular disease has been gained by surgery. Some 50 years ago colonic resection carried a high mortality and diverticular disease was a medical problem, surgery being reserved only for the emergencies and complications that threatened life. The treatment was quite empirical and low-residue diet became popular with no evidence that it was beneficial. In the past few decades resection has become safer and resected sigmoid colons, the site of diverticular disease, have become readily available. In a proportion of these specimens, some say over half, there is insufficient histological evidence of inflammation to account for the symptoms that had warranted operation.

What these specimens did show, though, was shortening of the bowel with gross thickening of the circular muscle coat. As the only abnormality present, this must have been the cause of the symptoms and one must attempt to ex-

plain its presence. Is this hypertrophy enlargement of a pelvic colon sphincter? Is it the result of a disorder of peristalsis, i.e. segmentation, or is it the cause of excess segmentation. Either way it must be the exciting cause of the diverticular disease with the increased pressure of abnormal segmentation producing the mucosal herniation. These operations were thus done for painful diverticular disease and not diverticulitis, which is wrong if considered in relation to our standard indications for surgery, i.e. the complications.

Clinically the distinction between diverticulitis and painful diverticular disease is easy, consisting merely of defining the general and local signs of inflammation. Reilly made an interesting application of this knowledge and devised a sigmoid myotomy in which the circular hypertrophied muscle is divided, and he carries this operation out in the management of the complications of the disease, stressing that in diverticulitis the inflammation is on the outside of the colon, i.e. a pericolitis, and that the colon muscle, though hypertrophied, is not affected by the inflammation and can be safely operated on.[1]

IS THERE A PLACE FOR SURGERY IN UNCOMPLICATED DIVERTICULAR DISEASE?

That the gross complications of diverticular disease such as perforation, fistula and obstruction, require surgical treatment is undisputed. We have mentioned that initially surgery was reserved for these complications and being offered late had a high mortality, a higher morbidity, and was required to be done in three stages with a preliminary defunctioning colostomy. As colon surgery became safer with improved pre-operative bowel preparation and better operative technique, one-stage treatment became the aim and the indications for operation were widened to include recurrent attacks of diverticulities, i.e. abdominal pain and fever despite good medical management, episodes suggestive of chronic or subacute intestinal obstruction, recurrent or persistent urinary symptoms caused by diverticulitis, recurrent haemorrhage, and inability to exclude carcinoma with certainty on X-ray.

On occasion today surgery is done for very limited diverticular disease: for instance the patient presenting with a peritonitis which proves at laparotomy to be due to a perforated diverticulum and for which a proximal defunctioning colostomy is done. Subsequent barium enema shows surprisingly little diverticular disease. In this case should you resect the colon as a second stage and finally close the colostomy, or should you merely close the colostomy without resection and hope for the best? Our practice here is always to resect the colon.

Surgery today is also offered for recurrent bleeds in a patient with diverticular disease and in whom no other cause for the bleeding is found. In these cases, apart from the symptom of bleeding and the presence of diverticular and the muscular hypertrophy, there is no other abnormality, certainly not diverticulitis. As this is acceptable should we not consider offering surgery for painful diverticular disease, especially as there is now limited surgery available without resection and anastomosis in the form of myotomy. On the one hand this is the time the disease should be well able to be controlled with antispasmodics although these will presumably have to be taken for ever. On the other hand this is the very time that a myotomy could be expected to work effectively, safely, and with minimal morbidity. The pain these patients have may be very severe—every bit as severe as the pain with true diverticulitis—and if the pathogenesis is gross hypertrophy of the circular muscle is this not irreversible and is myotomy not a rational solution to the problem?

While emphasizing that this suggestion is not practised, is it not a logical extension of the generally accepted trend towards one-stage surgery based on the newer thinking on aetiology of this fascinating disease?