

## Diverticular disease of the colon

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### **Aetiological considerations**

During the present decade new light has been thrown on the aetiology and pathogenesis of diverticular disease of the colon by several workers, yet it is true to say that the exact cause of the mucosal herniation is still uncertain. Many factors at one time or another have been incriminated but the main emphasis recently has been on the muscle abnormality which exists in diverticular disease. Of course, earlier workers drew attention to the pathological and functional disorder of the musculature in this condition.

Keith in 1910 contended that excessive contractions of the taeniae in diverticula-bearing segments throw the circular muscle and mucosa into folds. He postulated that heaped-up mucosa tends to occlude the bowel lumen and irregular spasms of the circular muscle cause raised intracolonic pressure which forces mucosa through weak spots in the wall where the blood vessels enter. It was postulated by Edwards (1939) that irregular spasms of colonic muscle occurring over a long period of time probably cause the interluminal pressure to rise resulting in mucosal herniation.

The cause of the thickening of the bowel musculature in diverticular disease remains obscure and while it has generally been assumed to be secondary to the development of diverticula, there is now a strong current of opinion that the muscle abnormality may be the primary lesion and precede the appearance of diverticula. Morson (1963) emphasized that the muscle thickening was the most consistent abnormality in 173 resected specimens of so-called 'diverticulitis' examined pathologically at St Mark's Hospital. The taeniae felt thick and cut almost like cartilage. There was marked thickening and corrugation of the circular muscle and the mucosa was redundant. He also found that in one third of the resected specimens inflammation was entirely absent and approximately 3% of specimens had no diverticula, yet all manifested the muscle abnormality. He postulated that there is a great increase in the muscle tone of both longitudinal and circular muscle, particularly the former, and spasm of the taeniae results in thickening and

corrugation of the circular layer. He pointed out that this thickening is not a true hypertrophy and that there is no evidence of hyperplasia of individual fibres. Shortening of the colon was also considered by Williams (1965) to be the predominant feature in the pathogenesis of diverticular disease.

Arfwidsson (1964) examined and measured microscopically the size of individual smooth muscle cells and their nuclei in diverticular disease and in controls. He claimed that cell hypertrophy exists but Morson (1963) and Williams (1965) contended that the distinction between a hypertrophied and a contracted smooth muscle cell is a difficult problem.

The recent biochemical studies of Slack (1966) support the view that the muscular thickening is not a true hypertrophy but could result from either cell hyperplasia or longitudinal contraction of the colonic wall.

Both the incidence of diverticula and the occurrence of the muscle abnormality predominate in the sigmoid region and this portion differs anatomically and physiologically from the rest of the large bowel. Almy (1965) pointed out that according to the law of Laplace the tension in the wall of a hollow cylinder is proportional to the radius multiplied by the pressure within the cylinder. If it is assumed that contraction of all the circular fibres in the whole colon are equally strong and produce the same tension, then the intracolonic pressure would be greatest in the narrow lumen of the sigmoid. Almy suggested that this mechanism may have an influence on the site of election of the disease.

### **Motility in diverticular disease**

Intraluminal pressure studies of the colon in diverticular disease have shown that under certain conditions variations from normal patterns occur. Painter & Truelove (1964a, b) found no evidence of any major difference in diverticular disease and in health during basal recordings but they detected an exaggerated response to a therapeutic dose of morphine in those segments bearing diverticula while normal segments in the same patients gave a normal response. The

increase in frequency and amplitude of the pressure waves following prostigmine was also more marked in segments bearing diverticula than in normal segments. Pethidine, on the other hand, did not generate high intracolonic pressure in patients with diverticular disease.

Arfwidsson (1964) found that the overall pressure activity was significantly higher in diverticular disease than in control subjects under resting conditions. He claimed that in diverticular disease the response to eating was five times normal and after prostigmine intravenously the colonic activity was three times normal.

Pressure studies undertaken by the author (Parks, 1966) revealed that the basal and post prandial colonic activity detected by the open tube method was higher in diverticular disease than in control subjects but when miniature balloons were used the pressure activity detected in diverticular patients resembled the control subjects. Distension studies on the colon *in vivo* indicated that in spite of its thickness the colonic musculature in diverticular disease has a diminished resistance to stretch.

## Management of diverticular disease

### Medical treatment

For many years doctors have usually insisted on a low-residue diet for patients with diverticula of the colon but evidence is accumulating that this practice may be undesirable. Carlson & Hoelzel (1949) observed that rats fed on a low-residue diet frequently developed colonic diverticula while those fed on a bulk-forming diet usually had normal colons. Rats initially fed on a bulky diet and subsequently changed to a low-residue diet developed diverticula most readily of all. Wells (1949) pointed out that colonic diverticula are rarely seen in the West African native who eats a bulk-forming diet. On the basis of motility studies, Painter (1964) suggested that segmentation occurs more readily in the narrowed colon of diverticular disease and that this narrowing may be accentuated by persistent adherence to a low-residue diet, thus predisposing to further diverticula formation.

The time has come to abandon the practice of low-residue diet except perhaps in elderly or unfit patients with obstructive-type symptoms whose complaints are less troublesome on this regime. In fact, a bulk-forming agent such as isogel may at times be advantageous.

According to Painter (1964) pethidine as an analgesic is preferable to morphine in acute cases of diverticular disease as it tends to reduce intracolonic pressure, whereas morphine may lead to

considerable intraluminal tension and thus predispose to perforation.

During an acute inflammatory episode, if an antibiotic is being given, the drug prescribed should be one that is carried in the blood stream since non-absorbable chemotherapeutic agents cannot be expected to influence significantly the peri-diverticular inflammation.

### Surgical treatment

Resection and restoration of bowel continuity in one, two or three stages still remains the mainstay of treatment for the more seriously affected cases. In the past decade several authors have advocated more elective resections as a one-stage procedure in an effort to reduce the incidence of serious complications.

Reilly (1964) introduced the operation of sigmoid myotomy, a procedure similar to Rammstedt's operation for hypertrophic pyloric stenosis. He has claimed good success with this method but some other workers have had a considerable morbidity following sigmoid myotomy.

A fact which must be faced is that many of the complicated cases of diverticular disease present at hospital with a short history and a high proportion of the fatalities occur in this group. The question therefore arises whether emergency resections as advocated by Ryan (1958), Bevan (1961) and others should be carried out more often in the presence of acute complications especially perforation. This is a reasonable policy in well-selected cases in experienced hands but it must be stressed that anastomosis under such circumstances is not without risk. In some cases it is preferable to resect the diseased bowel and bring out the proximal end as a colostomy. The distal end may be over-sewn or brought out as a mucous colostomy and the anastomosis delayed until later.

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