Pressure changes after balloon distension of the colon wall in diverticular disease

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SUMMARY Balloon distension of the distal colon in diverticular disease does not produce the pressure change in response to increasing volumes that occurs in normal subjects. This phenomenon, though modified at first by resection, is not abolished by resection nor by myotomy or bran. This adaptive quality of the wall of the colon to balloon distension possibly reflects a structural change in its wall, as the phenomenon was present in the colon in diverticular disease *in vitro* as well as *in vivo*.

The muscle of the large intestine is capable of producing a raised intraluminal pressure in diverticular disease.^{1 2} The force created may have a critical role in extruding the diverticulum. Furthermore, the reduction of the pressure appears to be beneficial in that symptoms become less and complications may become fewer.³ A paradox exists, however, in that balloon distension of the colon in diverticular disease⁴ produces a premature relaxation of the colonic wall indicating that it has developed characteristics which suggest reduction in its inherent strength. This phenomenon has been examined in normal subjects and in patients with diverticular disease with and without symptoms. The effects of treatment by cereal fibre and operations such as resection or myotomy which lower the intraluminal pressure⁵ are also assessed.

Methods

Normal subjects and patients with diverticular disease before resection and myotomy were examined by passing an open-ended tube and large balloon system, similar to that of Parks,⁶ as far as the rectosigmoid junction. The balloon was attached to a triple lumen tube used for measuring intraluminal pressure and allowed inflation of the balloon. This was done in 20 ml increments over the range of 20-200 cm H₂O pressure and the change in the balloon pressure was measured

graphically by attachment to a pressure transducer activating a preamplifier and linked pen-writing system. The change in the inserted balloon pressure reflected the tension in the wall of the colon rather than the distensibility of the balloon itself. This could be deduced from pressure/volume curves for balloon distension *in vitro* to the point short of rupture: a very low pressure was established in the balloon at first, which differed from the pressure curve established when the balloon was placed within the colon of normal subjects. The pressure *in vitro* later rose to become similar to those obtained in the colon of normal subjects over the range 120-180 ml H₂O as the distending volume (Fig. 1). Above this figure (at 200 ml) the balloon finally ruptured *in vitro*, though not necessarily *in vivo* because of the support of the colonic wall. These observations suggested that, at the low or mid-part of the curve, the response of the balloon reflected the contribution of the muscle of the colon. As the reactions in diverticular disease show a failure to maintain pressure even in the 'normal range', this could be considered as representing a weak response of the supporting wall of the colon, as was observed by Parks and Connell.⁴ Measurement was made of the point where the graph 'plateaued' and this was considered the critical point of pressure change. Each subject was studied in duplicate and his critical pressure derived from the average of the two tests.

Results

Figure 1 shows in graphical form how a critical point is reached in the balloon pressure in a normal

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Fig. 1 A pressure volume curve after balloon distension in the colon in a normal subject and in one with diverticular disease, relating the tension in the colon wall in cm H₂O to serial distending volumes. The effect of distending the balloon *in vitro* is also shown and was followed by balloon rupture at 200 ml.

Fig. 2 Ten symptomless subjects (I) and a similar number (II) with constipation are compared with 10 symptomless (I) diverticular disease patients and 10 symptomatic (II) cases; the results are expressed as means of the critical pressure $\pm$ SE.

subject at 55 cm H₂O pressure and in diverticular disease at 30 cm H₂O pressure, beyond which on serial distension little or no further increment of pressure takes place, and how in diverticular disease the pressure within the distending balloon may even fall with 'adaptation'. In Fig. 2 the mean critical pressure derived from such tracings is shown for 10 subjects with no symptoms (I) and 10 others (II) with no alimentary disease but with severe recent constipation; there was no significant difference in the critical pressure—that is, in the adaptation levels of the responses. In contrast 10 diverticular disease subjects (I) without symptoms (Fig. 2) and (II) 10 with pain and recent constipation had a significantly lower pressure at which the balloon pressure fell on distension by similar serial 20 ml volumes ($P < 0.001$). Figure 3 shows the critical pressure in seven of the diverticular subjects who were to have resections performed. It will be seen that the critical pressure was the same when the subjects were examined *in vivo* and when their specimens were tested *in vitro*

Fig. 3 Tests repeated in the same subjects; the mean critical pressure in seven subjects with diverticular disease examined *in vivo* compared with the critical pressure determined *in vitro* on resected specimens from the same subjects and again *in vivo* one month after resection. This is also compared with three subjects in whom the colonic circular muscle had been divided by the operation of colomyotomy.

post-operatively ($P < 0.001$). When the same patients were examined a month after the resection, their critical pressure had risen to become similar to that of normal subjects. These responses were in marked contrast with three other subjects with diverticular disease who had had a myotomy performed, in whom the critical pressure remained low. When the same resection patients were tested at one year (Fig. 4) the critical pressure had fallen to the level of diverticular disease; there had been no change in the post-myotomy cases. Ten diverticular disease patients, both symptomatic and asymptomatic, and who had been treated with bran (Fig. 5), showed no significant change; nor did bran affect the properties of the colonic wall in normal subjects.

The responses to the distension at the rectal level were identical though less marked than at the rectosigmoid level.

Fig. 4 The resection and myotomy studies were repeated one year later and are compared with the findings for normal subjects and for diverticular disease.

Fig. 5 The low critical pressure of diverticular disease is seen to be uninfluenced by bran, whether the diverticular disease subjects have (I) or have not (II) got symptoms. The higher critical pressure of normal subjects was uninfluenced by bran.

Discussion

Colonic intraluminal pressure in diverticular disease is raised, but only 'consistently' in patients with low faecal outputs and a delayed transit.⁷ Parks and Connell⁴ and Parks⁶ showed, however, that the wall of the bowel in diverticular disease, though capable of generating the raised pressure, is remarkably adaptive to distension and demonstrated how it yielded prematurely to colonic balloon distension compared with normal subjects. We have confirmed this and have shown that the phenomenon is present *in vitro* as well as *in vivo*, as it is present in tests in excised specimens as well as in subjects *in vivo*.

Resection of the pelvic colon which removes the diverticula does not appear to change the ready tendency of the colonic wall to relax, other than temporarily in the first month after operation. Myotomy, which lowers the intraluminal pressure,⁵ did not change the relaxation of the wall nor did the administration of bran, which also reduced the intraluminal pressure.⁸ It is unlikely, therefore, that bran influences the painful motor action of the pelvic colon in diverticular disease by an easier emptying of the distal bowel through a relaxation of its wall, as this change is already present in diverticular disease.

The fact that the phenomenon of relaxation after distension is present in diverticular disease and is not influenced by *in vivo* or *in vitro* testing or by treatment—be it by division or resection of the

muscle or by bulk additives such as bran—suggests an intrinsic derangement of the arrangement of the collagen or muscle fibres of the colonic wall. Although a continually raised intraluminal pressure may be one factor which causes this change as well as extruding the diverticulum, it would appear that even if the pressure is reduced by treatment, the response to distension is not readily changed.

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