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## Part II The muscular abnormality in the sigmoid colon

Diverticula of the sigmoid colon and associated diverticulitis were described clearly and fully in the early years of this century (Telling and Gruner, 1917). It was noted at this time that thickening of the bowel wall and of the longitudinal muscle in particular was a common finding (Keith, 1910). Since that time, the muscular thickening has often been noted, and shown clearly in illustrations of pathological specimens, but does not seem to have been considered as an element of the disease in its own right. Rather, it has been looked on as a subsidiary and unimportant aspect in comparison with the inflammatory complications. This perhaps stems from the view of Spriggs and Marxer (1925), derived from radiological studies, that the muscle abnormality was more a part of the prediverticular state than part of the established disease. An insight into the true significance of the muscle disorder was first given by Morson, who pointed out that many cases subjected to resection for supposed diverticulitis showed no evidence of inflammatory disease, but that a very considerable degree of muscular thickening was present in all the specimens (Morson, 1963). This paper has stimulated interest in the muscular aspect of diverticular disease, and comprehensive descriptions of radiological appearances (Williams, 1963, 1967b) and of manometric studies (Painter, 1964; Arfwidsson, 1964) have appeared. In these papers, the muscle change often present in the sigmoid colon has been discussed at considerable length, taking as a starting point the advanced stage of the condition which

is characteristic and readily recognized. However, no detailed analysis has been made in that this muscle abnormality has not been defined, and no accurate description has been made of its frequency, extent, and relation to diverticular pathology or clinical symptoms.

An investigation of colons at necropsy has been carried out in an attempt to remedy some of these deficiencies, and in particular, to put forward a tentative basis for further research. The main purpose of the work described in this paper has been to obtain an overall view of this abnormality in the general population, and to assess its relationship to diverticular disease as a whole. Studies on a smaller experimental group have been used to give a more detailed histological picture of the abnormality and its spatial relationship to the sigmoid colon and associated diverticula, and this work is to be reported elsewhere. The introduction of sigmoid myotomy (Reilly, 1966), for the relief of symptoms which have been ascribed to this muscular thickening, has added significance to studies of this type.

### MATERIALS AND METHODS

Two hundred unselected consecutive colons were studied at necropsy as set out previously (Hughes, 1968). The colons were examined carefully in a fixed, undistended state. Details of diverticula, muscular thickening, mucosal folds, and evidence of associated pathology were recorded. Sections were taken for histological study of all colons showing any macroscopic abnormality.

ality, and sections of many of the normal colons were taken for comparison. The specimens were fixed, embedded, and cut carefully to avoid errors due to tangential sections. Sections were examined with a dissecting microscope at 4, 8, and 16 × magnification as well as with a conventional microscope. Muscle thickness was measured at 16 × magnification, when care was again taken to exclude fallacious results from tangential sections.

In order to assess the influence that formalin fixation might have in causing abnormal contraction of the bowel wall, a subsidiary experiment was carried out in which a second group of colons was marked at 5 cm intervals at the time of necropsy, and then measured after 24 and 48 hours' fixation in formalin. Unlike the bowel in surgical specimens, it was found that post-mortem colons showed little or no further contraction when placed in formalin, and in no case did contraction exceed 10% of the original dimension. It was therefore assumed that the effect of formalin fixation was unimportant.

#### OBSERVATIONS AND RESULTS

The results in relation to diverticula and diverticulitis are reported elsewhere Hughes (1969).

**MUSCULAR THICKENING** Muscular thickening was present in two main forms, one in which the thickening took the form of stout circular muscle bands, characteristically extending around only two-thirds of the bowel circumference with successive bands alternating from side to side (Fig. 1), and less commonly a uniform 'thickening' of circular muscle.

The colons were assessed macroscopically, and on this basis divided first into those with and without diverticula, and then grouped again into those with normal muscle, those having evidence of simple spasm (as outlined below), and those showing frank muscle thickening. These groups

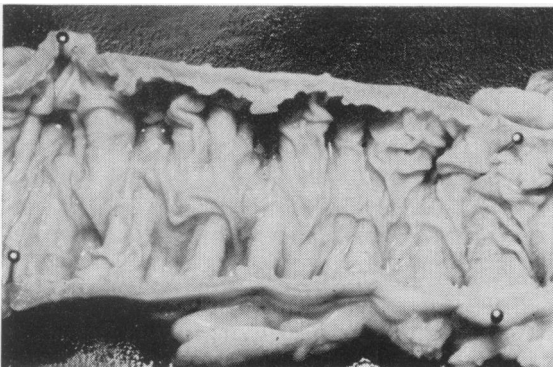


FIG. 1. The characteristic muscle abnormality in diverticular disease of the sigmoid colon.

were then correlated with the thickness of the circular muscle coat measured histologically. The mean circular muscle thickness and the standard deviations, together with the range encountered for each of these groups, is set out in Table I. It can

TABLE I

THICKNESS OF THE CIRCULAR MUSCLE OF THE SIGMOID COLON

Diverticula	Muscle Assessed Macroscopically	Circular Muscle Thickness (mm)	
		Mean ± SD	Range
Nil	Normal	0.9 ± 0.33	0.5 - 1.3
Nil	Spasm only	1.5 ± 0.20	1.2 - 1.8
Nil	Thickened and/or circular bands	2.3 ± 0.21	2.0 - 2.5
Present	Thickened and/or circular bands	2.7 ± 0.31	1.8 - 5.0
Present	Normal	1.2 ± 0.28	0.8 - 1.6

be seen that although the standard deviations indicate wide variations within the groups, there is relatively little overlap in circular muscle thickness between the groups, suggesting that colons can be divided into two groups, with and without muscular abnormality, with good correlation between macroscopic and histological findings. This has led to a definition of diverticular muscle abnormality as a circular muscle thickness in the sigmoid colon of 1.8 mm or more, which is used (as a basis for discussion) in the rest of this paper. A thickening of this degree is designated throughout the rest of this paper as 'the muscle abnormality'.

**MUCOSAL PATTERN AND SPASM OF THE COLON** Spasm of the colon is frequently seen in various forms on barium enema examination (Williams, 1967b) and similar changes were found in postmortem colons. It was found that the mucosa of the normal sigmoid colon was smooth and showed few or no folds. Spasm of either the circular or longitudinal muscle gave rise to mucosal folds which predominated in the opposite direction; thus circular spasm produced longitudinal folds of mucosa and vice versa. Again a combination of longitudinal and circular spasm could occur quite locally, giving excessive mucosal folds in both directions (Fig. 2). The mucosal pattern thus allowed an approximate estimate of the degree of muscle spasm to be made. The same relationship was seen on histological examination. Figure 3 is a longitudinal section which shows this close correlation between excess mucosal folds and increasing circular muscle thickening. Evidence of spasm of this type at necropsy was seen most commonly in the sigmoid colon, but also occurred more proximally. It was found that neither longitudinal nor circular spasm

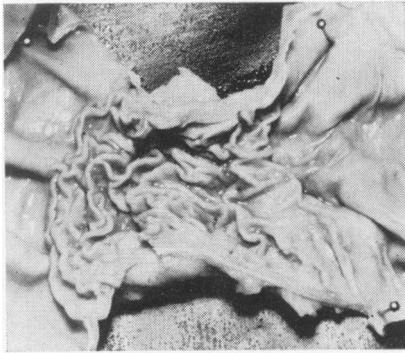


FIG. 2.

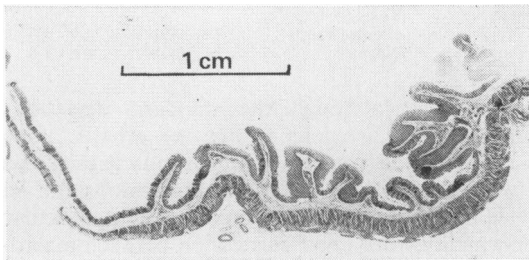


FIG. 3.

FIG. 2. A part of the sigmoid colon showing a localized example of combined longitudinal and circular spasm.

FIG. 3. Longitudinal section from an area of spasm showing correlation between mucosal folds and increased muscle thickening (mm scale).

bore any relation to the presence of diverticula or bowel symptoms. Furthermore, it was found that even gross longitudinal spasm was not associated with any tendency to form distinct circular muscle bands. Cases in this series showed gross longitudinal spasm with circular mucosal crowding and thickening of the taeniae without producing any evidence of the typical thick circular muscle bands of diverticular disease.

**FREQUENCY OF MUSCLE ABNORMALITY** Muscle abnormality was present in 73% of the colons containing diverticula (cases with diverticula confined to the caecum were excluded) and in 8% of those without diverticula. Of five cases with diverticula in the caecum only, muscular abnormality was present in two. The details are given in Table II.

The extent of the muscular change was estimated with some difficulty because of a tendency to merge gradually with the normal bowel above and, to a lesser extent below, although the maximum area of involvement was reasonably well defined.

TABLE II  
INCIDENCE OF MUSCULAR ABNORMALITY IN  
DIVERTICULAR DISEASE

Extent of Diverticula	No. of Cases	Incidence of Muscle Abnormality (%)
Sigmoid colon (less than 5)	13	31
Sigmoid colon (more than 5)	23	64
Left colon	35	72
Total colon	14	86
All cases with diverticula <sup>1</sup>	85	73
Caecum only	5	40
No diverticula	110	8

<sup>1</sup>Solitary caecal diverticula excepted.

A more detailed histological investigation of this aspect is being reported elsewhere. The length varied from 5 to 23 cm, with most cases between 8 and 10 cm.

**THE NATURE OF THE MUSCLE CHANGE** In most cases, the typical thick circular muscle bands alternating from side to side were seen. On cross section, the bands were formed of two elements, an infolding of circular muscle of not greatly increased thickness (Fig. 4) and a true thickening of the circular muscle fibres arranged in regular bands (Fig. 5). Not infrequently both elements were combined (Fig. 6). There was a tendency for smaller, circular muscle bundles to occur proximal to the main area of muscle change (Fig. 7). In most cases the circular muscle bundles were distinct, but in other cases they branched in an irregular fashion with diverticula protruding between the branches (Fig. 8). The circular muscle bands were most numerous at the level of the greatest number of diverticula, but diverticula were regularly seen in small numbers both proximal and well distal to the muscle change. The muscular abnormality was not seen outwith the sigmoid colon.

Circular bands consisting of thickened muscle (rather than simple infolding) were usually seen in the grosser cases of muscular abnormality and in particular in the cases with evidence of diverticulitis. In some cases where the bands were mainly due to infolding, diverticula were often very wide-necked and placed close together, and it appears that the bands were being produced indirectly by compaction of circular muscle fibres around the wide necks of the diverticula (Fig. 8).

In some cases, early but typical circular muscle bundles were found macroscopically in the absence of any spasm and similar changes were frequently seen histologically. It can be seen that the macroscopic and microscopic pictures of spasm (Figs. 2 and 3) and of circular muscle bundles (Figs.

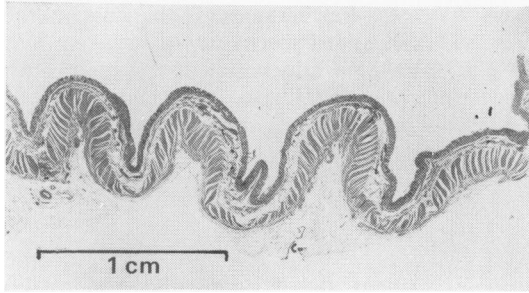


FIG. 4. Section of circular muscle bands due to infolding of wall of only slightly increased thickness (mm scale).

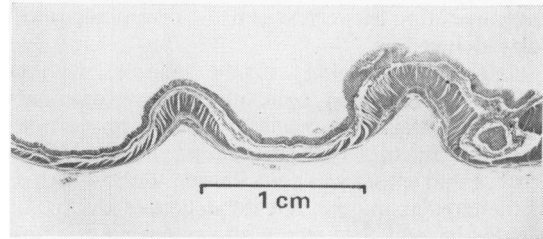


FIG. 7. Section of upper sigmoid colon in diverticular disease showing regularly spaced early circular muscle bands extending proximal to the main area of muscle change (mm scale).

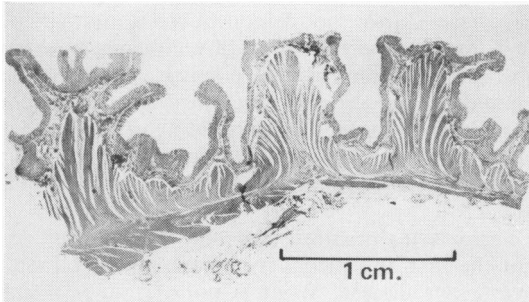


FIG. 5. Diverticular bands due to gross localized thickening of the circular muscle (mm scale).

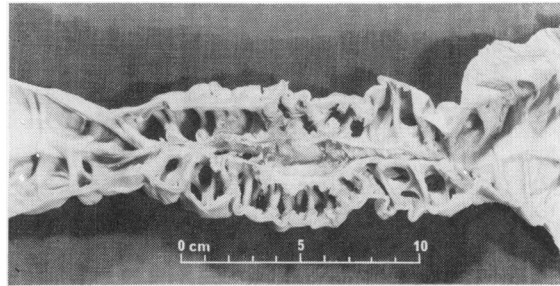


FIG. 8. Specimen of sigmoid colon showing branching of circular muscle bands.

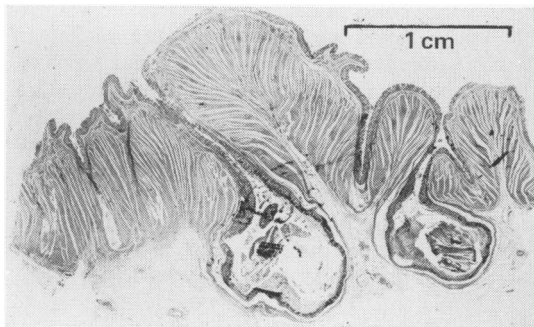


FIG. 6. Muscle bands due to a combination of infolding and local thickening of circular muscle (mm scale).

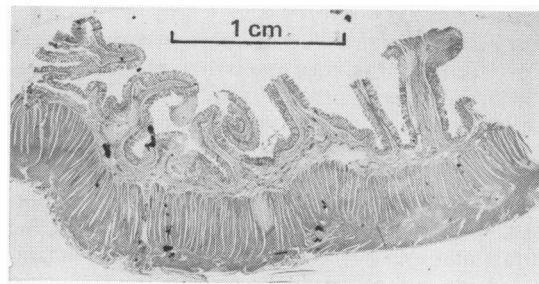


FIG. 9. Section of sigmoid colon in diverticular disease showing uniform thickening of circular muscle.

5, 7, and 8) do not show any similarity, nor is there any correlation between spasm and diverticular muscular change.

Uniform thickening of the muscle, without circular bands, was seen much less commonly but on occasion it reached marked proportions (Fig. 9). In this case the excess mucosal folds showed that spasm was contributing to the thickness of the circular muscle, but the degree of thickening was far beyond that seen with simple spasm alone. On occasion, this uniform thickening occurred in a sharply localized area, giving rise to a 'muscle tumour' superficially resembling a carcinoma. A considerable number of cases in which the thickening appeared quite uniform on macroscopic examination were found to have definite banding in addition when examined histologically.

Muscular thickening in the sigmoid colon of a uniform type was seen in only 10 of 73 colons showing muscle change, the remaining 63 showing typical circular bands.

One case in this series provided the opportunity to study diverticular muscular change in defunctioned bowel. The performance of a high sigmoid colostomy before perineal resection of the lower rectum for malignancy had left a free, defunctioned loop of sigmoid colon involved with diverticular disease. In this case the circular muscle was markedly thickened (to 4 mm) and the cells of Auerbach's plexus were also much more prominent than is usually seen. In this particular case there was obviously no regression of muscular change after a defunctioning colostomy. In fact the changes in this case were the most marked in this series in the absence of frank active inflammatory disease.

Another patient in this series had died from large bowel obstruction due to carcinoma. The bowel proximal to the carcinoma showed muscular hypertrophy and the circular muscle in this case reached a thickness of 2 mm, but was completely regular, with no tendency to form circular muscle bands.

#### MUSCULAR CHANGE IN ASSOCIATION WITH DIVERTICULITIS

Ten cases in this series showed pathological evidence of recent or old inflammatory disease of the sigmoid colon. In all cases definite muscle abnormality was present. One patient had severe diverticulitis with a pericolic abscess which was indirectly responsible for her death, and the muscle thickening and fibrosis were extreme in this case. In fact all cases which were associated with inflammatory disease active at the time of death showed a very marked degree of muscle thickening. However, muscular change did not correlate well with symptoms. Symptoms had been present in only two of the

above 10 cases. Furthermore, 15 patients in this series had complained specifically of recurrent mild left iliac fossa pain and bowel disturbance before their terminal illness (Hughes 1969), and of these 15 muscle abnormality was present in only four, approximately the same ratio as in the whole series.

#### DISCUSSION

Previous writings on the muscular abnormality of diverticular disease (Morson, 1963; Williams, 1963; Slack, 1962; Arfwidsson, 1964; Fleischner, Ming, and Henken, 1964) have dealt predominantly with the advanced stages of the disease. No attempt has yet been made to define the exact nature of the abnormality or the findings in the early stages. This study suggests that there are three types of muscular change, all involving the circular muscle. (It is accepted that the taeniae are frequently shortened and thickened, but otherwise they have shown no demonstrable abnormality.) First, there is the formation of bands due to gross localized thickening of the circular muscle of the sigmoid colon; secondly the presence of circular muscle bands due to simple infolding of the circular muscle (of only a moderately increased thickness); and thirdly a uniform thickening of the circular muscle.

There is no question from the present findings that the first and second types are associated with diverticular disease. What is not certain is which comes first, the muscular change or the diverticula. In this series the two conditions were most frequently associated, but extensive diverticulosis with no muscle change was sometimes seen, and less commonly classical muscle change with no diverticula. It is obvious that neither diverticula nor muscle change appears first in every case, and it does not seem necessary that one must precede the other. Painter (1964) and Arfwidsson (1964) have shown that the segmental pressures developed in the sigmoid colon under certain stimuli are higher in patients with diverticula than in normal subjects. Arfwidsson has also been able to demonstrate these high pressures in patients with radiological evidence of the muscular change of diverticular disease, but in whom no actual diverticula could be demonstrated. It seems likely that the initial change is a functional one in which increased pressures are developed in an anatomically normal colon. This may result in the development of work hypertrophy of segments of the circular muscle leading to muscular thickening, or to the development of diverticula, or most commonly to both. The picture of well localized, early circular muscle bands seen

histologically suggests that these bands are in some way anatomically determined to account for their regular disposition (Fig. 7). It is possible that contraction and/or hypertrophy first occurs in anatomically determined groups of circular muscle fibres (associated with segmenting movements). These may then undergo further simple contraction, 'dragging in' adjacent circular muscle to give the 'infolding' effect, or else develop primarily in the direction of further hypertrophy. Furthermore, the development of large wide-necked diverticula close together also tends to form circular bands by separating the circular muscle fibres into bundles of this type.

Circular muscular hypertrophy is most marked in cases where there is active inflammation, and in all such cases in this series it was very gross. It was less marked in cases showing old inflammatory fibrosis, so it is possible that inflammation may have an irritant effect causing increased hypertrophy, which resolves to some extent in the event of a prolonged remission. It is common clinical experience that acute diverticulitis may resolve so completely that no evidence at all can be found at laparotomy several months after the acute attack.

In the case of the uniform type of circular muscle thickening without ridging, it is more difficult to be dogmatic regarding a relationship with diverticula. However, in eight of the 10 cases of uniform circular muscle thickening in this series, the thickening was associated with diverticula, and circular muscle thickness reached a level not seen in the normal colons or in those with simple spasm. It seems likely that this is a true variant of diverticular muscle abnormality.

The actual nature of the muscular change, particularly where circular bands are seen, is still in doubt. Morson (1963) has suggested that there is no evidence of hyperplasia or hypertrophy, and that all the findings can be accounted for by bunching up of tissues caused by a sustained contraction of the longitudinal muscle. There are several difficulties with this theory, the chief being that spasm of the longitudinal muscle is frequently seen clinically and at necropsy, and as noted above, such spasm bears no resemblance to the appearance of typical circular bands. (It may seem, on first encounter, unusual to speak of postmortem spasm in smooth muscle, because it might be expected that any contracted muscle would relax at the time of death. However, such areas of persisting spasm are common in the postmortem colon. That they are due to simple spasm is shown by an identical appearance with that of spasm as seen on barium enema examination, and the close correlation between increased mucosal folds and muscle thickness seen histologically

(Fig. 3). In fact we have been able to relax such areas of spasm on occasion by perfusing the postmortem colon (within 12 hours of death) with a solution containing propantheline bromide.) Indeed it is difficult to see how a simple longitudinal spasm could give rise to such localized circular muscle changes as are seen with the typical bands. Possibly this could occur where the circular bands are due mainly to infolding, but, where the bands are due to localized muscle thickening, the above theory would require the presence of localized longitudinal muscle spasm, since the longitudinal and circular muscle layers are connected by intermingling fibres (Lineback, 1925). The occurrence of localized longitudinal muscle spasm would seem unlikely. Furthermore, early circular muscle bands can be seen in the complete absence of any evidence of longitudinal spasm as shown by excess mucosal folds. Quite intense spasm of the longitudinal muscle gives rise to a circular muscle thickness of 1.6 to 1.8 mm and it seems unlikely that spasm could increase to a stage where it would produce a thickness of 5 mm. (In operative specimens not included in this series, a muscle thickness of 8 mm has been seen.) On the other hand, Arfwidsson (1964) found evidence of circular muscle hypertrophy from studies of cell nuclear counts, and limited studies of a similar nature in this department have confirmed this work.

Williams (1965) has drawn the interesting analogy between the thickened state of the taeniae and myostatic contracture, a condition of voluntary muscle where fibres held for prolonged periods in a shortened state undergo progressive functional contraction, so that they cannot elongate to their full length, even though there is no anatomical or structural bar to this lengthening. It would seem that this analogy would be even more pertinent if it included the concept of the circular muscle bands being in a state of myostatic contracture as well. Although Williams has also shown (Williams, 1967a) that a mass peristaltic wave can cause considerable relaxation of the muscle bands, in advanced cases this probably occurs only to a limited extent, the circular bands remaining somewhat shortened at all times.

It is interesting to compare the diverticular muscle change with muscle thickening occurring in other colonic diseases, for instance, ulcerative colitis, hypertrophy proximal to an obstructing carcinoma, Hirschsprung's disease, and sigmoid volvulus. In all of these the muscular thickening is considerable but quite uniform. This difference is compatible with the increasing evidence that segmenting and propulsive movements in the colon are quite distinct. Diseases such as ulcerative

colitis, Hirschsprung's disease, and hypertrophy above an obstructing carcinoma represent hypertrophy of the elements involved in propulsive movements, while the circular bands of diverticular disease represent an increase in the elements associated with segmenting movement.

The actual cause of this excess segmenting activity with its subsequent pathological changes is at present conjectural. The marked geographical variations in diverticular disease may give some lead, and two factors could help to explain the high incidence in western countries compared with a low incidence in many of the developing countries. First, emotion and psychological tension can cause increased pressure in the sigmoid colon (Wangel and Deller, 1965). Secondly the low-bulk, constipating diet of western civilization, by failing to provide adequate dilatation, could allow progressive contraction, and the development of a myostatic contracture of the circular muscle bundles. This might also explain the frequency with which closure of a proximal colostomy performed for diverticulitis, without resection of the affected loop, is followed by exacerbation of the disease. Complete faecal diversion would allow contracture of circular muscle bundles to become more extreme, and this, on restoration of bowel continuity, would result in some obstruction to the passage of faeces, and also to the necks of diverticula, which would tend to retain their faecal contents to a greater extent. The single case in this series in which diverticular disease was present distal to a defunctioning colostomy showed gross muscle thickening. The frequency with which left iliac fossa pain and discomfort in patients with diverticular disease is relieved by an air contrast barium enema might also be related to stretching of circular muscle fibres.

**INCIDENCE OF MUSCULAR CHANGE** The incidence of muscle abnormality in this series was 73% of diverticula-bearing colons and 8% of colons without diverticula. The incidence of muscle abnormality was related to the extent of diverticular involvement, since it was present in only 31% of cases of minimal involvement of the sigmoid colon. The maximum incidence of 86% was associated with total colonic involvement with diverticula. The only comparable series is that of Slack (1962), who found muscular hypertrophy in only two of 145 consecutive necropsies, 26 of which had diverticula. It is difficult to reconcile these widely different findings. In part it may be due to the fact that Slack's series was one of particularly mild diverticular involvement, since only 18% of colons had diverticula, and the diverticula were confined to the sigmoid colon in 88%, unusually low and high

figures respectively for western countries. Furthermore, no definition of muscle abnormality was given in this paper, which may have included only the grossest degrees of involvement.

The present figure of 73% would also seem to be high in relation to laparotomy findings, for it is unusual to find significant muscular thickening in palpation of the sigmoid colon at operation, even though the patient may have diverticula. Formalin fixation does make the muscle abnormality more obvious on palpation, although it does not significantly increase its incidence or measured dimensions. The use of atropine-like drugs for premedication may induce relaxation in cases which have not reached an irreversible state, and a thickening of circular muscle to 2 mm, although far in excess of normal, might not be obvious on palpation *in vivo* of the rather soft bowel wall. The finding of Morson (1963) that all surgical specimens showed muscle thickening, even when no inflammation was present, supports the concept of a close association between diverticula and muscle thickening.

There can be no doubt that there is a close association between muscle thickening and inflammation, whether it be due to muscle activity interfering with drainage of diverticula to cause infection, or to inflammation giving a secondary 'irritative' hypertrophy. On the other hand, inflammation does occasionally occur in the absence of any muscle change, especially if the colon is examined after the inflammatory episode has settled. The fact that muscular thickening may be difficult to detect at operation when myotomy or resection is being considered increases the importance of a greater knowledge of the extent and nature of the muscle abnormality.

**RELATION TO SYMPTOMS** There is some evidence that increased sigmoid pressures and radiological evidence of muscle abnormality may be associated with symptoms (particularly left-sided abdominal discomfort and change of bowel habit) and that these symptoms may be relieved by myotomy or sigmoid resection (Reilly, 1966; Arfwidsson, 1964). However, little work has been done on the natural history of these very common symptoms, and clinical experience has shown that many patients are completely relieved when reassured after adequate investigation that no underlying neoplasm is present. Furthermore, it has not been shown that in a large number of cases there is a significant overall relationship between these symptoms and the presence of diverticular disease over and above that which might be expected by chance. In this series, of 15 patients who complained of recurrent symptoms of this type, only four had muscular

change at necropsy, an incidence very similar to that in the series as a whole. Although myotomy seems to be an eminently logical operation in selected cases, further proof is necessary that these symptoms are specifically related to diverticular disease, and that myotomy has a permanent effect on the muscular abnormality, before the operation is used in widespread fashion.

## SUMMARY

A basic knowledge of the muscular abnormality of diverticular disease of the colon is desirable for an understanding of its aetiology and for rational surgical treatment.

The results of an investigation of the muscle of the sigmoid in 200 colons examined at necropsy are presented. These results suggest that a thickness of circular muscle of 1.8 mm is the dividing line between the bowel of diverticular disease and normal bowel or bowel affected by simple spasm. On this basis, diverticular muscle abnormality was present in 36% of all colons and in 73% of colons containing diverticula. The incidence rose with increasing extent of involvement of the colon with diverticula.

The muscular abnormality consisted of three types: circular muscle bands due to localized circular muscle thickening; circular bands due to simple infolding of the circular muscle; and uniform circular muscle thickening. The abnormality was marked in cases with active diverticular inflammatory disease, but on the whole bore little relation to symptoms.

The facts that both diverticula and muscular abnormality may be found without each other, and that the incidence of such abnormality increases with the extent of the diverticula, suggest that the primary change is a functional one of increased

pressures in the sigmoid colon, without associated anatomical change. This increased pressure may subsequently cause either diverticula or muscular change (as a result of work hypertrophy) or in most cases, to both.

I am most grateful to Professor William Burnett for his encouragement and advice throughout this investigation and to the staff of the Pathology Department of the Princess Alexandra Hospital for their cooperation in the collection of colons. This work was much facilitated by the careful preparation of histological sections by Miss N. Carfrae.

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