THE PATHOGENESIS OF LARGE BOWEL DIVERTICULA

by

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DIVERTICULA of the alimentary tract may be congenital or acquired. The congenital variety is composed of all coats of the intestinal wall and is said to be a "true" diverticulum. The acquired variety may be either "true" or "false" depending on whether or not protrusion of the muscle coats of the intestinal wall occurs with the mucous membrane. Although the majority of acquired diverticula are "false", an investment from all coats is probably very frequent in the earliest stages of formation (Telling and Gruner, 1917). It is generally agreed that colonic diverticula are of the acquired variety but in the caecum an isolated diverticulum of the congenital type may occur.

The term "diverticulosis" was proposed independently by Case (1914) and de Quervain (1914) to describe the presence of uncomplicated non-inflamed colonic diverticula. The terms "diverticulitis" and "peridiverticulitis" have been used to describe inflammation in and around diverticula. These designations are not entirely satisfactory as the distinction between diverticulosis and diverticulitis is not always clear. Clinical and radiological findings may not coincide and the pathological diagnosis may differ from the clinical and radiological impression (Parks, Connell, Gough and Cole, 1970). In the past it was generally assumed that diverticulosis was a painless and usually symptomless condition. It is now known that troublesome symptoms, including pain, may occur in the absence of inflammation. For these reasons the term "diverticular disease" of the colon which encompasses the complicated as well as the uncomplicated state has been preferred by many workers (Morson, 1963; Williams, 1963; Fleischner, Ming and Henken, 1964; Almy 1965).

INCIDENCE, AGE AND SEX DISTRIBUTION

The incidence of diverticular disease of the colon in the general population is difficult to assess and figures derived from clinical practice do not necessarily give a true picture, for they do not include those subjects who have no complaints and many of those with mild symptoms who do not require hospital care. Most of the estimations of the frequency of the condition have been based either on necropsy reports or data derived from barium enema examinations. The former method often yields a spuriously low figure, since diverticula tend to be elusive unless a meticulous search is made for them. The latter method tends to give a spuriously high estimate, since barium enemata are usually only carried out on

patients with abdominal symptoms and it is to be expected that in this selected group, the incidence would be higher than in the general population.

In earlier autopsy studies the incidence quoted was usually between 3 and 10 per cent (Drummond, 1917; Mailer, 1928; Rankin and Brown, 1930; Ochsner and Bargen, 1935; Morton, 1946). In these series the investigations were of a retrospective nature and it is probable that they underestimated the true incidence at that time. In most of these studies, data were extracted from autopsy records and there is no indication that the authors personally examined the colons of the cadavers.

The Incidence of Diverticular Disease in Northern Ireland

If a worthwhile appraisal of the prevalence of this disease is to be made from post-mortem material, it is essential that a careful and specific prospective investigation is made. A detailed study was carried out by the author on 300 consecutive colons obtained from the Pathology Department, Royal Victoria Hospital, Belfast (Parks, 1968). In order that an adequate examination could be made, each colon was opened from end to end, since minor or even moderate degrees of disease in obese subjects may be undetected, if only the serosal aspect of the bowel is inspected.

The incidence of diverticular disease in this survey is shown for both sexes in Table 1. The colons of 171 males with an average age of 61.3 years at the time of death were examined and colonic diverticula were present in 57, or 33 per cent of the cases. The youngest male noted to have diverticular disease was 44 years, the eldest 86, and the average age of the diseased group was 65.8 years. Of 129 females with an average age at the time of death of 63 years, there were 54 or 41.9 per cent with colonic diverticula. The youngest female with the disease was only 29, the eldest 87 and the average age of those affected was 66.7 years.

TABLE I
Incidence and Sex Distribution of Diverticular Disease of the Colon in 300 Consecutive Autopsies

	Male	Female	Total
Number of colons examined Number of colons with	171	129	300
diverticular disease Affected by diverticular disease	57 33.3%	54 41.9%	111 37%

The frequency of the disorder increased progressively with age, so that in the 6th and 7th decades more than one-third of cases were affected and by the 9th decade one-half of the cases were involved (Fig. 1.).

Other Prospective Autopsy Series

There has been a number of specific investigations in different countries which have attempted to estimate accurately the frequency of the disease. Some of these

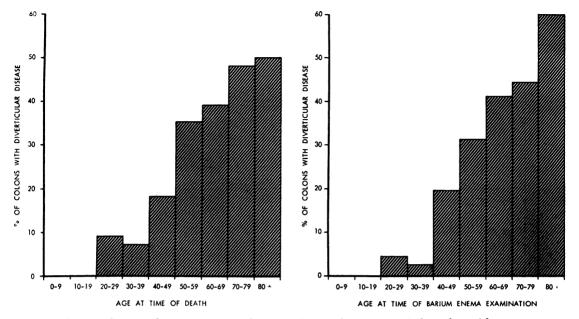


Fig. 1. Showing the increasing incidence of diverticular disease of the colon with age.

(a) in 300 consecutive autopsies

(b) in 500 consecutive barium enemas

are shown in Table 2. In most of these series the authors personally undertook a meticulous examination of each colon and it is likely that the most accurate figures of the frequency of the condition to-day come from this data.

TABLE II

Specific Prospective Studies of the Incidence of Diverticular Disease of the Colon in Autopsy Material

Author	Country	Number of Autopsy colons examined	Percentage of colons with Diverticular Disease	Remarks
Mourgues (1913)	France	100	30%	Consecutive autopsies on aged subjects
Bumm (1933)	Germany	920	33%	Necropsies on subjects over 50 years of age
Bevan (1961)	England	116	12%	Consecutive autopsies on subjects over 40 years of age
Watt and	England	76	550/	Composition
Marcus (1962) Slack (1962)	England England	76 141	55% 18%	Consecutive autopsies Consecutive autopsies
Parks (1968)	N. Ireland	300	37%	Consecutive autopsies
Hughes (1969	Australia	200	45%	Two series of consecutive autopsies

Hughes (1969) reported a remarkably high figure of 45 per cent among 200 unselected autopsies. The incidence of diverticular disease was 36 per cent among cases in the 6th and 7th decades and 56 per cent among those who were over the age of 80 at the time of death.

Radiological Series in Northern Ireland

An assessment was made of the findings on 500 consecutive barium enema examinations performed at the Royal Victoria Hospital, Belfast, during a six month period. Colonic diverticula were demonstrated in 55 (28.9 per cent) of 190 men and in 96 (31 per cent) of 310 women (Table 3). Although the ages of patients ranged from eleven years upwards, the youngest male with colonic diverticula was aged 40 and the youngest female was 29 years. Fig. 1 shows that the incidence of the disease increased with age and the condition was demonstrable in about half of the patients over 70 years of age. It should be remembered that in this series most of the patients had abdominal symptoms, so that the circumstances are weighed in favour of revealing pathology of one kind or another.

The average age of the 500 patients undergoing barium enema examinations was 51.6 years but the average age of patients affected by diverticular disease was 63.7 years.

TABLE III

Incidence and Sex Distribution of Diverticular Disease of the Colon in 500

Consecutive Barium Enema Examinations

	Male	Female	Total	
Number of barium enemas	190	310	500	
Number with diverticular disease	55	96	151	
% Affected	29%	31%	30.2%	

Other Radiological Series

The incidence of diverticular disease in barium enema series has increased from an average figure of around 10 per cent in the 1920's to a figure in the region of 25 or 30 per cent in the last decade. Welch, Allen and Donaldson (1953) found that one-third of barium enemas at the Massachusetts General Hospital showed diverticula. These workers noted an increasing incidence in the second half of life until, at the age of 85, two-thirds of their patients were affected.

In order to ascertain the prevalence of colonic diverticula in the general population in the Oxford area, Manousos, Truelove and Lumsden (1967) studied 109 healthy volunteers by a modified barium meal and follow-through. More than one third of subjects over the age of 60 had diverticula.

Changing Sex Incidence

The early series reported a higher incidence of diverticular disease of the colon

in males (Telling and Gruner, 1917, M: F 2: 1; Spriggs and Marxer, 1927, M: F 5: 2; Fraser, 1933, M: F 3: 1). In recent surveys male preponderance has not been a feature and in the majority of reports the incidence was higher in females (Smith, 1951; Greene, 1957; Horner, 1958; Boles and Jordan, 1958; Manousos, Truelove and Lumsden, 1967; Parks, 1969a).

Geographical Distribution

Wells (1949) pointed out that in Western Africa, the native who eats a bulky diet does not develop diverticula whereas volvulus of his excessive large colon is relatively common. On the other hand, colonic diverticula are not infrequent in the negro of North America. Vague (1951) stated that diverticular disease was more common in Anglo-Saxons than in the French population and Nordic races were predisposed to diverticular formation more than the Southern races. The rarity of the disease among Italian people and those of Latin extraction is exemplified by Piccinni's (1952) report of a single case of diverticula of the sigmoid among approximately 18,000 surgical cases observed over a 12 year period at Bologna Surgical Clinic. Kohler (1963) reported an incidence of 15.8 per cent in industrialised Sweden, in contrast to an incidence of 5.1 per cent in neighbouring Finland. An extensive epidemiological study has been carried out by Painter and Burkitt (1971). They confirm a low incidence of the disease in developing areas, such as rural Africa and Asia, in contrast to the high frequency in Western civilisation and North America. Avery Jones (1969) was of the opinion that the disease was rare in China and Reilly (1969) came to the same conclusion after visiting Russia.

DIET AND ITS POSSIBLE RELEVANCE

In his Harveian Lecture, Sir Berkerley Moynihan (1927) stated: "A diet leaving little residue is the one generally advised but I am not sure that a diet leaving a bulky residue is not better, provided that the bowels act once per day". Almost half a century later, after millions of low residue meals, there has been a swing towards a high residue regime in the management of the disease. Cleave, Campbell and Painter (1969) have shown that the death rate for diverticular disease has increased progressively since the early 1920's except during the war and immediate post-war years when white bread and refined sugar were restricted. Wells (1949) commented on the possible beneficial effects of a bulky diet in protecting the West African against the disease.

There is some experimental evidence in support of the hypothesis that a low residue diet predisposes to diverticular formation. Carlson and Hoelzel (1949) carried out dietary experiments on rats and found that those fed on a low residue diet, developed diverticula more readily than those fed on a similar diet with hemi-cellulose added as a bulk-former. Rats initially fed on a high residue diet and subsequently changed to a low residue diet were affected most of all. On the other hand, Naunton Morgan and Ellis (1969) were unable to produce diverticula in the colons of rats maintained on a low roughage diet for up to 70 weeks. The effects of low-residue diet on the canine colonic wall has been studied

by Havia (1971). No hypertrophy in the circular or longitudinal muscle was observed and there was no alteration in intracolonic pressure measurements. Hodgson (1972) has produced a form of "diverticulosis" in rabbits by feeding them on a refined diet of white bread, butter, sugar and milk with vitamin supplements. After four months on this diet the animals had increased considerably in weight, were constipated and had contracted colons. When these colons were stimulated to contract vigorously by prostigmine injected parenterally, broadbased blue-domed bulgings were produced.

Obesity

The relationship of obesity to diverticular disease of the colon has attracted much attention since Klebs (1869) postulated that fat in the region where a blood vessel passes through the colonic muscle might weaken the wall. Mayo, Wilson and Griffen (1907) believed that obesity was a frequent concominant of weakened colonic musculature. Bland-Sutton (1903) found obesity in 77 per cent of cases of diverticula of the colon, while Babcock (1941) considered that obesity was a factor in 17 per cent.

On the other hand it must be admitted that diverticular disease of the colon can, and does, occur in lean and wasted individuals. Ochsner and Bargen (1935) concluded that patients with diverticula of the colon were no fatter than normal subjects. Their view was supported by Morton (1946) and Horner (1958).

In the Northern Ireland clinical survey, it was found that the weights of patients with diverticular disease were marginally higher than those of the general population. In the autopsy specimens examined locally it was estimated that excessive fat was associated with one-half of the diverticular colons compared with one-quarter of the non-diverticular colons. It is probable that in some cases of diverticular disease the obesity is apparent rather than real and is due to shortening of the muscle wall resulting in bunching of the fat.

ASSOCIATED PATHOLOGICAL LESIONS

Table 4 gives the frequency of some pathological conditions which occurred in 521 patients with diverticular disease surveyed clinically (Parks, 1969b). Since many patients did not have any radiological investigations of the upper gastro-intestinal tract or gall bladder, it is likely that the true incidence of associated or coincidential lesions is rather higher than that recorded.

The association of hiatus hernia, cholecystitis and colonic diverticula, known as Saint's triad, has been generally accepted. The tendency to hernia formation and diverticular formation in other regions of the body is of particular interest and raises the question of a more widespread muscular or connective tissue defect. Foster and Knutson (1958) studied patients who had had previous cholecystectomies and found that in 19 per cent of cases both hiatus herniae and colonic diverticula could be demonstrated. Horner (1958) made a specific examination for hiatus herniae in 292 patients with diverticular disease and noted a frequency of 15.3

TABLE IV

Associated or Co-incidental Pathological Lesions in 521 Cases of Diverticular

Disease

λ	Number of Patients		
	Affected	% of the Total Cases	
Cholecystitis + gallstones	72	13.8%	
Hiatus hernia	38	7.3%	
Abdominal hernia (excluding excisional)	65	12.5%	
Incisional hernia	18	3.5%	
Peptic ulcer	76	14.6%	
Duodenal diverticulosis	8	1.5%	
Small bowel diverticulosis	9	1.7%	
"Appendicitis"	76	14.6%	
Carcinoma of colon	16	3.1%	

per cent, which contrasted with a 3 per cent incidence of hiatus hernia in non-diverticular controls. De la Vega, Naves and Ponce de Leona (1964) considered that there was a predisposition to inguinal and femoral herniae in patients with colonic diverticula and reported an incidence of 20.5 per cent of groin herniae among 190 diverticular patients.

Colonic diverticula may be associated with diverticulosis of other viscera. Acording to Fifield (1927) this may be as high as 11 per cent. Conversely, it is claimed that patients with diverticula of the duodenum or jujeno-ileum are more liable to diverticula of the colon than controls. Lee and Finby (1958) studied 45 patients with diverticula of the jujenum and/or ileum and noted associated colonic diverticula in 25 cases, an incidence of 53 per cent. Other workers found that the frequency of large bowel involvement in cases with the small bowel disorder ranged from 30 to 50 per cent (Benson, Dixon and Waugh, 1949, Baskin and Mayo, 1952).

The association of visceral diverticulosis and Marfan's syndrome has been reported by Clunie and Mason (1962) and Miekle, Becker and Gross (1965). These workers suggested that there may be a qualitative deficiency or alteration in the supporting connective tissue of the colon in diverticular disease.

MORPHOLOGIC DERANGEMENTS Muscular Abnormality

Several workers have drawn attention to the muscle abnormality associated with colonic diverticula (Keith, 1910, Edwards, 1939, Morson, 1963, Arfwidsson, 1964). Whether this abnormality is primary or secondary to the development of diverticula is less certain. It is present in a high proportion of autopsy colons bearing diverticula and in virtually every surgical specimen removed on account of symptomatic diverticular disease. There is accumulating evidence that the

muscular abnormality may be a primary phenomenon and precede the formation of diverticula (Morson, 1963; Arfwidsson, 1964; Williams, 1967; and Parks 1970). The early radiological features of the muscular abnormality of diverticular disease is shown in Fig. 2: no diverticula as yet demonstrable.



FIG. 2. Barium enema of the sigmoid colon demonstrating the intraluminal projection of muscular folds in early diverticular disease. There are no diverticula present.

One hundred and thirty consecutive colons were carefully examined for the muscular abnormality so frequently seen in association with colonic diverticula (Parks, 1968). Forty-five specimens were found to have diverticula and of these 14 (31 per cent) had a marked degree of muscle thickening. A further 19 (42 per cent) were judged to have a minor to moderate degree of thickening, although the distinction between normal and abnormal was not always easy to make. The thickened segments averaged 40 cm. in length. The circular coat of the sigmoid was especially involved and thick muscle bands which extend around one-half to twothirds of the bowel circumference encroached markedly on the lumen. Heaping up of redundant mucosa, suggestive of bowel shortening, was a characteristic feature.

A similar incidence has been recorded in Australia by

Hughes (1969 who found that 73 per cent of autopsy colons bearing diverticula had muscle thickening. An even higher incidence of muscle abnormality in surgically resected specimens has been reported by Morson (1963) who found that thickening was the most consistent and striking feature; the abnormality was present in all of the 173 cases treated by resection over a five year period at St. Mark's hospital.

Macroscopically and microscopically the muscle shows thickening and usually both the circular and the longitudinal coats are involved, sometimes to a marked degree. It may be that this thickening is produced by sustained contraction of the muscle bundles rather than either hypertrophy or hyperplasia of muscle cells (Morson, 1963). It has been suggested that shortening of the longitudinal muscle causes the circular muscle to be thrown in corrugations resulting in the formation

of prominent crescentic folds which project into the lumen. The mucosa is also made redundant and this further encroaches upon the lumen.

Williams (1965) considered that contraction and shortening of the colon are the predominant features in the pathogenesis of diverticular disease. Following a microscopic study Arfwidsson (1964) concluded that the increased thickness is due to hypertrophy of the muscle cells. As a result of detailed biochemical investigation of muscle cells Slack (1966) considered that the thickening is not due to hypertrophy, but he could not ascertain whether hyperplasia of the muscle cells or longitudinal contraction of the bowel wall is the more likely cause. It is thus true to say that the aetiology of the muscle thickening is still uncertain.

Distribution of Colonic Diverticula Lengthwise in the Bowel

Diverticula may occur in any or all of the segments of the colon, but there is a marked predilection for the sigmoid region. The descending colon is also commonly involved. Table 5 reveals the sites where diverticula were distributed along the large bowel in three separate studies by the author. The sigmoid was by far the commonest site affected. It was involved alone in 45 per cent to 65 per cent of the patients. In addition, the sigmoid was often also involved in combination

Table V			
Distribution of Colonic Diverticular			

Site of Colonic Diverticula	Autopsy Series (111)	Clinical Survey (461)	Radiological Series (151)
Sigmoid	50	302	77
Descending	2	14	4
Transverse	1	4	7
Ascending	3		1
Ascending and caecum			1
Sigmoid and descending colon	30	83	33
Sigmoid and transverse colon	4	5	5
Sigmoid and ascending colon	3	3	
Sigmoid and caecum	2		
Sigmoid, descending and transverse colon	6	19	7
Sigmoid, descending and ascending colon	1	-7	·
Sigmoid, transverse and ascending colon			2
Sigmoid, descending, transverse and caecum	2		
Sigmoid, descending, ascending colon			
and caecum	2		
Whole colon	5	31	14

with other regions so that 93 per cent of those with diverticular disease in the three series had this segment affected.

There has been much speculation as to the reason why diverticular disease affects the sigmoid colon more often than any other part of the bowel. Undoubtedly, the left half of the colon and particularly the sigmoid, differs both anatomically and physiologically from the right half of the colon. It has been postulated that pressure/tension relationships in the narrow segments of the colon may be a factor in the formation of the mucosal herniation. According to the law of Laplace, the tension in the wall of a hollow viscus is proportional to the radius multiplied by the pressure within the cylinder. If it is assumed that contraction of all the circular muscle fibres in the whole colon is equally strong and produces the same tension, then the intracolonic pressure would be greater in the narrow lumen of the sigmoid.

Position of Diverticula in relation to the Circumference of the Colon

The anatomy of the colon is such that concentration of the longitudinal muscle fibres into three bands or taeniae leads to relative weakness of the intervening areas. Between the taeniae, the muscle coat consists chiefly of circular fibres with a few longitudinal ones, and it is here that diverticula are apt to occur. Fig. 3 is a diagrammatic representation of the colon on cross section and demonstrates that the usual sites of herniation of the mucosa are in the lateral intertaenial areas.

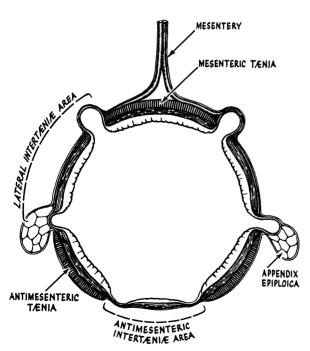


FIG. 3. Diagrammatic representation of the relationship of diverticula and blood vessels to the taeniae coli.

There is a tendency for diverticula to develop in two longitudinal rows on each side of the colon. Fig. 4 shows a pelvic colon which has been opened up along its mesenteric taenia. The central portion consists of the two antimesenteric taeniae with a narrow strip between them. On each side of this, the lateral intertaenial areas are shown and in each of these there are basically two rows of diverticula. The strip of colon between the two antimesenteric taeniae is narrow and diverticula in this area are rare. When they occur they are always small and globular, but again they tend to form in two rows (Fig. 5).

Klebs, in 1869, drew attention to the relationship of

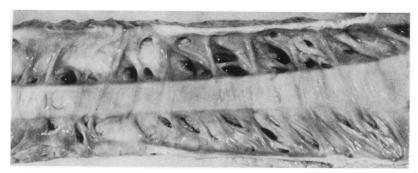


Fig. 4.

The mucosal aspect of a colon opened along its mesenteric taenia. Two rows of diverticula are seen in each lateral intraenial area.

diverticula to the blood vessels of the intestinal wall and suggested that the pull on the bowel wall by the mesenteric vessels produces areas of diminished resistance. The proximity of diverticula to the site of the passage of blood vessels was also noted bv early workers (Edel. 1894, Hansemann,

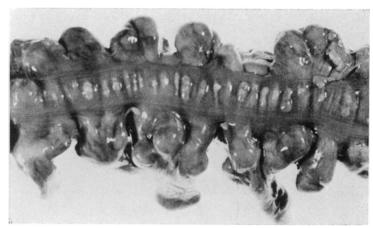


Fig. 5. Colon showing early diverticular formation between the two antimesenteric taeniae.

1896, Graser, 1899, Beer, 1904). Drummond (1917) believed that the blood vessels predispose to the formation of diverticula in much the same way as the spermatic cord predisposes to the development of an inguinal hernia. However, this is not a true analogy as there is no pre-formed sac adjacent to the blood vessels which penetrate the colonic wall.

The important role of the canal through which blood vessels pass has been re-emphasised recently (Slack, 1960, Tagliocozzo and Virno, 1961, Arfwidsson, 1964). Other workers do not subscribe to the view that the colon is less resistant at these points (Noer, 1955, Deelman, 1957, Laumonier and Martin, 1957, Fleischner, Ming and Henken, 1964). The relationship of diverticula to the blood vessels which penetrate the wall was also studied by the author following intravascular injection of dye. Sizeable vessels were frequently found adjacent to the necks of the diverticula (Fig. 6). The common symptom of bleeding is readily explained by the proximity of significant sized blood vessels but whether their presence is related to the aetiology of the diverticular disease is still debated.



Fig. 6.

Showing a blood vessel penetrating the colonic wall close to a diverticulum.

DISTURBANCES OF COLONIC MUSCLE FUNCTION

Theoretically diverticula of the colon might develop (a) if the intraluminal pressure is raised but there is no weakness of the colonic wall or (b) if the intraluminal pressure is normal but weakness of the colonic wall exists.

There is no evidence that colonic diverticula are more frequent in conditions such as Hirschsprung's disease or benign stricture of the lower bowel in which raised intracolonic pressure may be present for a long time. The importance of muscular contraction rather than passive distension as a cause of mucosal herniation was stressed by Stout (1923). He stimulated the resected appendix of the dog, having cut gaps in the muscular wall, and was able to produce protrusion of the mucosa through the defects. Wilson (1950) in similar experiments on human appendices, found that diverticula only appeared as a result of muscular contraction. If the results of these studies can be applied to the colon, the importance of muscular activity is apparent.

Intraluminal Pressures

There is now a considerable amount of evidence that abnormalities of physiological function accompany the morphologic derangements which are an integral part of diverticular disease. The advent of optical manometers and electro-manometers has led to more accurate and meaningful measurements of intraluminal

pressures in the large intestine. Using small bore open-ended water-filled polythene tubes or very fine air filled tubes with miniature balloons attached at their tips, resting intracolonic pressures and the responses to physiological and pharmacological stimuli are readily measured.

While reports differ as to whether or not there is an increase in intraluminal pressure under resting conditions (Painter and Truelove, 1964, Arfwidsson, 1964, Parks and Connell, 1969, Attisha and Smith, 1970), there is a general agreement that the colon responds excessively to certain pharmacological stimuli such as morphine and prostigmine. As pharmacological stimuli are uncommon in real life, it is important to consider the possibility of recurring elevation of intraluminal pressure in response to physiological stimuli. There is some support from motility studies for an exaggerated response to the ingestion of food (Arfwidsson, 1964; Parks and Connell, 1969; Attisha and Smith, 1969).

Following experiments using simultaneous pressure manometry and cine-radio-graphy, Painter et al (1965) postulated that contractions of the interhaustral rings, which can effectively occlude the lumen, result in the formation of multiple short isolated segments of the colon. It is evident that in a colon with prominent crescentic folds of circular muscle and redundant mucosa, it is relatively easy for segmentation to occur. Contraction of the wall of a closed segment may lead to a considerable rise in intraluminal pressure.

Reilly (1964) introduced the operation of colomyotomy in which the circular muscle fibres of the colon are divided by a longitudinal incision along one of the antimesenteric taenia – a procedure corresponding to Heller's operation at the cardia or Rammstedt's operation on the pyloric canal. Attisha and Smith (1969) measured the intraluminal pressures before and after sigmoid myotomy in man. Pre-operatively the resting pressures were not elevated, but there was an excessive response to prostigmine. This was significantly reduced by myotomy but the effect was of a temporary nature and returned to the pre-operative values within two or three years (Smith – personal communication).

Recently Balfour, Giannakos and Smith (1972) measured the intracolonic pressures manometrically in rabbits before and after colomyotomy. For a time following myotomy the colon was less responsive to prostigmine stimulation than pre-operatively, but there was a complete return to pre-operative values within four months of operation. These workers concluded that the effects of myotomy in the rabbit, and by analogy in man, are of a temporary nature. From the limited number of studies in man and animals it would appear that there is little experimental support for the operation of sigmoid myotomy.

Hodgson (1972) has used adult rabbits in an attempt to reproduce an animal model for the study of diverticular disease. After preliminary intracolonic pressure measurements the rabbits were given a refined low-residue diet for four months. Repeat pressure measurements at this stage revealed an increased basal motility and a markedly exaggerated response to prostigmine which contrasted with the pre-diet values.

Compliance of Colon Wall

In addition to the question of raised intracolonic pressure the other major factor to be considered is the possibility of weakness of the colonic wall which

might yield to normal intraluminal pressures. In order to elucidate this point, Parks and Connell (1969) measured the resistance of the colonic wall to distension. By incremental distension of a balloon in the lumen of the sigmoid, the ability of the muscle to resist an opposing force was assessed. It was shown that in spite of its thickness, the colonic muscle was deficient, in terms of its capability to resist pressure. It may be that the pathogenesis of diverticular formation should not be explained simply on the basis of raised intraluminal pressure alone but cognizance should also be taken of another phenomenon, i.e. weakness or loss of compliance of the wall which tends to increase with advancing age.

Possible Relationship to Irritable Bowel Syndrome

In view of the fact that some features of diverticular disease are similar to those of irritable bowel syndrome, interest has been aroused concerning the possibility of a common underlying aetiological factor. Lumsden, Chaudhary and Truelove (1963) stated that the radiological appearance of the sigmoid in irritable colon syndrome closely resembles the findings in early diverticular disease. Fleischner et al (1964) believed that a relationship exists and contended that contractions of the irritable colon leads to heaping up of muscle folds with narrowing of the lumen, which may facilitate the development of diverticula. It is true that there are some similarities in the motility responses to parasympatheticomimetic stimuli in the two conditions but important differences do exist (Parks and Connell) 1972. Observations during the clinical survey of diverticular disease in Northern Ireland did not suggest that irritable colon was a precursor of diverticular formation (Parks, 1969a). The latter finding was in agreement with Edwards (1965) who failed to find any casual relationship on follow-up of patients with irritable bowel.

CONCLUSIONS

A number of factors which may be relevant to the formation of colonic diverticula have been discussed. Much new information has come to light on the pathogenesis of the disease and further approaches are currently being made. However, it is evident that the aetiology of diverticular disease is still not fully understood.

REFERENCES

ALMY, T. P. (1965). Gastroenterology, 49, 109.

ARFWIDSSON, S. (1964). Acta chir. scand. Suppl. 342.

ATTISHA, R. P., and SMITH, A. N. (1969). Brit. J. Surg., 56, 891.

AVERY, JONES, F. (1969). Recent Advances in Surgery ed. S. Taylor. London, Churchill.

BABCOCK, W. W. (1941). Rev. Gastroent. 8, 77.

BALFOUR, G., GIANNAKOS, V., and SMITH, A. N. (1972). In preparation.

BASKIN, R. H. Jr., and MAYO, C. W. (1952). Surg. Clin. N. Amer., 32, 1185.

BEER, E. (1904). Amer. J. med. Sci., 128, 135.

BENSON, R. E., DIXON, C. F., and WAUGH, J. M. (1943). Ann. Surg., 118, 337.

BEVAN, P. G. (1961). Brit. med. J., 1, 400.

BLAND-SUTTON, J. (1903). Lancet, 2, 1148.

BOLES, R. S. Jr., and JORDON, S. M. (1958). Gastroenterology, 35, 579.

BUMM, R. (1963). Langenbecks Arch. klin. Chir. 174, 14.

CARLSON, A. J., and HOELZEL, F. (1949). Gastroenterology, 12, 108.

CASE J. T. (1914). Arch. Roentg. Ray., 19, 375.

CLEAVE, T. L., CAMPBELL, G. D., and PAINTER, N. S. (1969). Diabetes, Coronary Thrombosis, and the Saccharine Disease. 2nd. ed. Bristol, Wright.

CLUNIE, G., and MASON, J. (1962). Brit. J. Surg, 50, 51.

DEELMAN, H. (1957). Arch. Anat. path., 33, 161.

DE LA VEGA, J. M., NAVES, J., and PONCE DE LEONA, A. L. (1964). In Gastroenterology, Bochus, H. L. (Ed.). vol 2. 2nd ed. Philadelphia, Saunders.

DE QUERVAIN, F. (1914). Dtsch. Z. chir, 128, 67.

DRUMMOND, H. (1917). Brit. J. Surg., 4, 407.

EDEL, M. (1894). Virchows Arch. path. anat., 138, 347.

EDWARDS, D. A. W. (1965). Ann. roy. coll. Surg. Engl., 37, 275.

EDWARDS, H. C. (1939). Diverticula and Diverticulitis of the Intestine. Bristol, Wright.

FIFIELD, L. R. (1927). Lancet, 1, 277.

FLEISCHNER, F. G., MING, S., and HENKEN, E. M. (1964). Radiology, 83, 859.

FOSTER, J. J. and KNUTSON, D. L. (1958). J. Amer. med. Ass., 168, 257

FRASER, I. (1933). Causation, Pathology and Treatment of Diverticula of the Large and Small Intestine. M.D. Thesis, Belfast

GRASER, E. (1899). Munch. Med. Wschr. 46, 721.

GREENE, W. W. (1957). Amer. J. Surg. 94, 282.

HANSEMANN, D. V. (1896). Virchows Arch. path. anat., 144, 400.

HAVIA, T. (1971). Acta chir. Scand. Suppl. 415.

HODGSON, J. (1972). In preparation.

HORNER, J. L. (1958). Amer. J. dig. Dis., N.S., 3, 343.

HUGHES, L. E. (1969). Gut, 10, 336.

KEITH, A. (1910). Brit. med. J. 1, 376.

KLEBS, E. (1869). Handbuch der Pathologischen Anatomie, Bd., 1, 271, Berlin, Hirschwald.

KOHLER, R. (1963). Acta chir. Scand., 126, 148.

LAUMONIER, R., and MARTIN, E. (1957). Arch. Path., 33, 403.

LEE, R. E., and FINBY N. (1958). A.M.A. Arch. Intern. Med. 102, 97.

LUMSDEN, K., CHAUDHARY, N. A., and TRUELOVE, S. C. (1965). Clin. Radiol. 14, 54.

MAILER, R. (1928). Lancet, 2, 51. MANOUSOS, O. N., TRUELOVE, S. C., and LUMSDEN, K. (1967). Brit. med. J., 3, 762.

MAYO, W. J., WILSON, L. B., and GRIFFEN, H. Z. (1907). Surg. gynaec. Obstet., 5, 8,

MIELKE, J. E., BECKER, K. L., and GROSS, J. B. (1965). Gastroenterology 48, 379.

Morson, B. C. (1963). Brit. J. Radiol. 36, 385.

MORTON, J. J. Jr. (1946). Ann. Surg., 124, 725.

Mourgues, P. de (1913). Des Diverticules acquis du gros intestin. Thesis, Lyons.

MOYNIHAN, B. G. A. (1927). Lancet, 1, 1061.

NAUNTON MORGAN, M., and ELLIS, H. (1969). Brit. med. J., 2, 53.

NOER, R. J. (1955). Ann. Surg., 141, 674.

OCHSNER, H. C., and BARGEN, J. A. (1935). Ann. intern. Med., 9, 282.

PAINTER, N. S., and BURKITT, D. P. (1971). Brit. med. J., 2, 450.

PAINTER, N. S., and TRUELOVE, S. C. (1964). Gut. 5, 201 and 365.

PAINTER, N. S., TRUELOVE, S. C., ARDRAN, G. M., and TUCKEY, M. (1965). Gut, 6, 57.

PARKS, T. G. (1968). Proc. roy. soc. Med., 61, 932.

PARKS, T. G. (1969a). Brit. med. J., 4, 639.

PARKS, T. G. (1969b). Brit. med. J., 4, 642.

PARKS, T. G. (1970). Gut. 11, 121.

PARKS, T. G., and CONNELL, A. M., (1969). Gut, 10, 534 and 538.

PARKS, T. G., CONNELL, A. M., GOUGH, A. D., and COLE, J. O. Y. (1970). Brit. med. J., 2, 136.

PARKS, T. G., and CONNELL, A. M. (1972). In Press.

PICCINNI, L. (1952). Ann. Ital. chir., 29, 829.

RANKIN, F. W., and Brown, P. W., (1930). Surg. gynaec. Obstet. 50, 836.

REILLY, M. (1964). Proc. roy. soc. Med., 57, 556.

REILLY, M. In Recent Advances in Surgery p. 504, Ed. S. Taylor, Churchill, London, 1969.

SLACK, W. W. (1960). Gastroenterology, 39, 708.

SLACK, W. W. (1962). Brit. J. Surg., 50, 185.

SLACK, W. W. (1966). Gut, 7, 668.

SMITH, A. N. Personal communication.

SMITH, N. D. (1951). Amer. J. Surg., 82, 583.

SPRIGGS, E. I., and MARXER, O. A. (1927). Lancet, 1, 1067.

STOUT, A. P. (1923). Arch. Surg., 6, 793.

TAGLICOZZO, S., and VIRNO, F. (1961). Ann. Ital. chir., 38, 301.

TELLING, W. H. M., and GRUNER, O. C. (1917). Brit. J. Surg., 4, 468.

VAGUE, J. (1951). Arch. Mal. Appar. dig., 40, 1387.

WATT, J., and MARCUS, R. (1964). J. Path. Bact., 88, 97.

WELCH, C. E., ALLEN, A. W., and DONALDSON, G. A. (1953). Ann. Surg., 138, 332.

WELLS, C. (1949). Brit. J. Radiol. 22, 449.

WILLIAMS, I. (1963). Brit. J. Radiol. 36, 393.

WILLIAMS, I. (1965). Brit. J. Radiol., 38, 473.

WILLIAMS, I. (1967). Radiology, 89, 401.

WILSON, R. R. (1950). Brit. J. Surg., 38, 65.