some degree of progressive unco-ordinated growth. It has nothing to do with suppressed twins or duplication phenomena. A teratoma contains multiple tissues, dermal, neural, skeletal, dental, respiratory or alimentary and Willis is at great pains to emphasize that with one exception multiple tissues can always be demonstrated if the specimen is studied in sufficient detail; the exception is the rare but highly malignant growth which contains only one kind of tissue. The usual sites for a teratoma are the gonads, the mediastinum, the retroperitoneal and presacral regions and the base of the skull.

Most presacral teratomata are benign and cystic, are present at birth and are rare in adults; they are commoner in females. They usually project externally but clearly originate from the presacral tissues. They often contain loops of bowel and parts of a limb. Calcified areas are seen on X-ray in 50% and this is a useful diagnostic aid. The incidence of malignancy is thought to be 10–20%, commonly an adenocarcinoma or less frequently a squamous cell carcinoma arising in part of the tumour.

The 2 cases were malignant teratomata, both men. One, aged 54 years, died of a coronary thrombosis shortly after operation and the other, aged 45, had a huge malignant teratoma involving the whole of the left buttock. A colostomy was performed and deep X-ray therapy given, but he died a few months later.

The only *meningocele* presented in a female patient aged 44 years with a three-month history of a feeling of rectal fullness. After a presacral cystic swelling measuring 8 × 5 cm had been excised with some difficulty, a leak of cerebrospinal fluid occurred through the perineal wound and necessitated a further exploration to repair the defect in the dura mater.

No cases of *pelvic kidney* were noted in the records of St Mark's Hospital. The post-mortem incidence of this condition is approximately 1 in 2,000 cases (Forbes 1945) and sometimes the kidney may be felt *per rectum*.

There were 5 presacral cystic lesions of unknown ætiology similar to those described as *cyst-hamartomata* by Edwards (1961). Their ages ranged from 43 to 52 years. The average diameter was 5 cm and they contained multiple cysts embedded in smooth muscle. This part of the investigation is as yet incomplete.

Bone Tumours

The common lesion in this group is a chordoma which is a malignant tumour with a poor prognosis. The histology and situation of this tumour leave little doubt that it is derived from the notochord. There were 5 chordomata in the records, 3 males and 2 females whose ages ranged from 50 to 75 years. They presented with a swelling or severe sacral pain. The swelling presented on the anterior and posterior aspects of the sacrum as a smooth multilocular cyst expanding and destroying the bone. All 5 lesions were biopsied, but considered inoperable. The results of deep X-ray therapy were disappointing.

Neurogenic Tumours

There were two *neurofibromata;* one was diagnosed clinically on the basis of its hard consistency, smooth surface and fixity to the sacrum. The presenting symptoms were sacral pain and frequency of micturition in one case and frequency of micturition in the other case. Both tumours were excised completely through the abdomen.

Miscellaneous Tumours

There were 2 metastatic presacral tumours, one from a carcinoma of the bladder and the other from a carcinoma of the pelvic colon. The fibrosarcoma, measuring 2·5 in. in diameter, presented in a middle-aged woman with a sensation of rectal fullness. The hæmangio-endothelioma occurred in a boy of 17 years; the prognosis of this tumour is very poor.

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REFERENCES

Edwards M (1961) *Dis. Colon Rectum* **4, 103**
Forbes G (1945) *Brit. J. Surg.* **33, 139**
Lovelady S B & Dockerty M B (1949) *Amer. J. Obstet. Gynec.* **58, 215**
Nicholson G W de P (1950) Studies on Tumour Formation. London; p 247
Willis R A (1960) Pathology of Tumours. London; p 944

The Muscle Abnormality in Diverticular Disease of the Colon

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During the past five years 173 surgical specimens removed for 'diverticulitis' have been examined in the laboratory at St Mark's Hospital. All were studied for evidence of inflammation, however slight, and for the presence of diverticula. Observations were made on the state of the smooth muscle layers of the bowel wall, for it is not generally realized that there is a striking abnormality of the smooth muscle in diverticular

disease (Lane 1885, Keith 1910, Williams 1963, Morson 1963). The results are given in Table 1. Five specimens showed the muscle abnormality only. It should be remembered that these surgical specimens were removed from patients who had been labelled on clinical and radiological evidence as 'diverticulitis'. In my opinion the muscle abnormality must have accounted for their symptoms.

Table 1
Surgical pathology in 173 cases of 'diverticulitis' at St Mark's Hospital, 1958–62

	No. of cases
Muscle abnormality + diverticulosis + diverticulitis (extensive 67 cases, focal 45 cases)	112 (64·7%)
Muscle abnormality + diverticulosis	56 (32·4%)
Muscle abnormality only (muscular strictures)	5 (2·9%)

The muscle abnormality is the most consistent and striking abnormality in diverticular disease. The tæniæ coli appear thick, assuming an almost cartilaginous consistency. The circular muscle is thick and has a corrugated or concertina-like appearance. In between these muscular corrugations the mouths of the diverticula are found as they penetrate the bowel wall to lie in the pericolic fat. These corrugations are interdigitating processes of circular muscle. They are not continuous around the circumference of the bowel wall, being only semicircular arcs of muscle confined to the two zones between the mesenteric and antimesenteric tæniæ. Each arc consists of a double layer of circular muscle and the thin investing layer of longitudinal muscle. This appearance is always found in specimens without inflammatory changes and in those with a relatively slight amount of inflammation. But when there is extensive pericolic inflammation the accompanying extramural fibrosis seems to have a restricting effect on the normal freedom of movement of the sigmoid colon, leading to the most bizarre and irregular shapes and in particular to the formation of a fibromuscular tumour.

The muscle abnormality of diverticular disease could be explained by excessive shortening of the bowel due to spasm of the tæniæ coli. But an increase in the number of segmental circular muscle contractions of the affected segment of bowel is probable. In other words the appearances are the result of a gross exaggeration of the normal physiological action of the sigmoid muscle. This view is supported by the absence of any detectable histological abnormality of the muscle in diverticular disease. The thickening seems to be due to shortening rather than a true hypertrophy or hyperplasia of muscle cells. On the other hand there is, in some specimens, a curious 'ribbing' of the circular muscle which is reminiscent of the muscle abnormality of the bladder in chronic prostatic obstruction.

Other evidence which incriminates shortening as the explanation for the muscle thickening is the distinctive redundancy of the mucous membrane seen in diverticular disease. Also the excessive amount of fat around the sigmoid colon can be explained by bunching of the pericolic and mesenteric tissues consequent upon shortening of the bowel by muscular contraction.

There is uncertainty about the actual mechanism of diverticulum formation. The simple distension theory of origin is still influential despite the evidence of Stout (1923), Edwards (1939) and Wilson (1950). These authors provide evidence in support of muscular activity as an important factor in the ætiology of diverticulosis. The well-established view that diverticula are found at the points of greatest weakness in the bowel wall, namely where the blood supply penetrates the muscularis (Slack 1962), seems to support distension as an ætiological factor. However, Wilson states that distension alone cannot produce diverticula and that muscular activity is inhibited by simple distension. He resolves these apparently conflicting factors by postulating that any distending force is the product of the power of muscular contraction.

Applying this to the sigmoid colon, it could be postulated that sustained muscular contraction causes distension, the effect of which would be maximal in the pouches or sacculations between the muscular clefts. It is perhaps significant that diverticula are only found in the bowel wall between these clefts. Moreover, close examination of the mucosal surface of the pouches in specimens with the muscle abnormality only or in early diverticulosis sometimes shows little pits where the mucosa is beginning to push between the circular muscle fasciculi. It would appear that the sacculation of the bowel wall causes separation of the circular muscle fasciculi and this, in turn, could cause widening of the gaps through which the blood supply penetrates. The widening of these gaps allows mucosa to be pushed through the bowel wall.

If diverticulosis is basically a disorder of muscle function then what is the cause of the muscle abnormality? Grace *et al.* (1951) describe experiments from which it would appear that emotional states can have a profound effect on the muscular activity of the colon. They observed contraction of the longitudinal muscle together with shortening of the bowel as well as an increase in circular muscle activity.

It is probable that there are other factors which may influence colonic muscle function. Diverticular disease is not the only colonic condition in which a muscle abnormality can be detected by conventional methods of examination of surgical

specimens. In ulcerative colitis, for example, there is a pronounced thickening of the muscle layers of the colon with loss of the haustral pattern. In Hirschsprung's disease the muscle layers proximal to the aganglionic segment hypertrophy, whereas in many surgical specimens of idiopathic megacolon there is a pronounced atrophy of the muscle layers. However, the ætiological significance of the muscle abnormality of diverticular disease and other colonic conditions is unlikely to be solved until our present ignorance of the normal anatomy and physiology of the colonic muscle has been remedied.

REFERENCES
Edwards H C (1939) Diverticula and Diverticulosis of the Large Intestine. Bristol
Grace W J, Wolf S & Wolff H G (1951) The Human Colon. London
Keith A (1910) *Brit. med. J.* i, 376
Lane A (1885) *Guy's Hosp. Rep.* **43**, 48
Morson B C (1963) *Brit. J. Radiol.* **36**, 385
Slack W W (1962) *Brit. J. Surg.* **50**, 185
Stout A P (1923) *Arch. Surg., Chicago* **6**, 793
Wilson R R (1950) *Brit. J. Surg.* **38**, 65
Williams I P (1963) *Brit. J. Radiol.* **36**, 393

The Effect of Morphine in Diverticulosis of the Colon [*Summary*]

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It has been suggested that diverticulosis of the colon may be the result of abnormal intracolonic pressures and this hypothesis was tested by measuring the pressures in the human sigmoid colon both in health and in diverticulosis. The effects of certain drugs were also studied. Simultaneous cine-radiography was employed to determine the mechanism responsible for pressure production.

The intrasigmoid pressures were recorded with open-ended water-filled polythene tubes which were connected to a Cambridge multi-channel pressure recorder. Recordings were made for a period of one hour under basal conditions and for a further hour after the administration of a drug.

Results
(1) Under basal conditions the pattern of pressure in the normal sigmoid colon was essentially the same as the pattern obtained from the sigmoid affected by diverticulosis.
(2) After morphine had been given, the normal colon produced more waves of pressure; these seldom exceeded 20 mm Hg in height. In the colon with diverticula, non-affected parts of the sigmoid behaved like the normal colon but those segments that bore diverticula responded to morphine by producing an excessive number of waves of high intracolonic pressure.
(3) The high pressures that followed the use of morphine were seen on occasions to distend diverticula to a great extent.
(4) Pethidine did not increase either the number of pressure waves or their dimensions.
(5) Cine-radiography combined with intraluminal pressure recording revealed that when high pressures were being produced, the colon was divided into segments by contraction rings. When these segments contracted, their contents were not free to escape and high pressures developed in the segments. Morphine caused the colon to segment and to generate high pressures.

Conclusions
(1) Segmentation converts the colon into a series of segments or 'bladders' whose outflow is obstructed so that high localized pressures develop. This mechanism appears to be the final common path whereby high localized pressures are produced regardless of the stimuli which evoke them. The similar structure of vesical and colonic diverticula is thus explained.
(2) Morphine evokes high intracolonic pressures which render the drug potentially dangerous in acute diverticulitis and which would tend to disrupt anastomoses in the colon. Pethidine does not have this effect and would appear to be the analgesic of choice in acute diverticulitis.

REFERENCE
Painter N S (1962) MS Thesis. University of London

The following papers were read and will be published in a later issue of *Proceedings:*

Juvenile Polyps
Mr Ian Todd (*St Mark's Hospital, London*)

Wound Infection in Surgery of the Colon and Rectum
Mr P F Jones, Dr H G Smylie & Mr L W Baker (*Aberdeen Royal Infirmary*)

The following papers were also read:

Malignant Melanoma of Anal Canal
Dr P G H Volkstädt (*St Mark's Hospital, London, and University School of Erlangen, West Germany*)

REFERENCES
Morson B C & Volkstädt P G H (1963) *J. clin. Path.* **16**, 126
Volkstädt P G H & Morson B C (1963) *Arch. klin. Chir.* **302**, 194

***In vitro* Study of Human Colonic Muscle**
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(*Meeting to be continued*)