Diverticular Disease of the Colon—

A DEFICIENCY DISEASE OF WESTERN CIVILIZATION

by

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To
JOY, RICHARD and MARK

Preface

The purpose of this book is to describe some of the recent advances that have been made in our understanding of colonic physiology and their relation to the causation and to the symptoms of diverticular disease of the colon. In particular, the traditional and current views as to the aetiology and to the pathogenesis of the disease are discussed at length as they form the basis of the modern management of diverticular disease. The conservative treatment of the condition by dietary means is dealt with more fully than are the complications of true inflammatory diverticulitis because the latter have been described many times elsewhere and are dealt with in Bentley Colcock's excellent monograph, 'Diverticular Disease of the Colon'.

Colonoscopy has been mentioned but briefly because, at the present time, it is available only to the few and its advantages and indications have yet to be determined.

The name, 'Diverticular Disease of the Colon' is now used to embrace the time-honoured terms 'diverticulosis' and 'diverticulitis' which date from the days when it was believed that the diverticula were the primary abnormality. It is now known that changes in the behaviour and the structure of the colonic muscle occur before the mucosa herniates and that diverticula are not responsible for symptoms except when they become infected and true inflammatory diverticulitis is present. This modern name for the condition has the advantage of shifting our attention away from the diverticula and directing it towards the changes in the colonic muscle that are so prominent a feature of the disease.

However, I believe that these muscular changes are merely the equivalent of the trabeculation which is seen in the urinary bladder that has had to struggle for years to propel its contents and, consequently, that the thickening and distortion of the muscle is secondary to extra-colonic factors. The historical

emergence of this 'new disease' on the clinical scene when taken together with its geographical prevalence leads me to conclude that diverticula are only the outward visible signs of an acquired abnormality caused by our fibre-deficient Western diet.

In the industrialized countries, most foodstuffs have been processed to improve their keeping qualities and to facilitate their transport and many have been refined to make them more palatable so that they can be sold at a greater profit. Consequently, the citizens of these countries eat much more refined sugar and white flour than did their forebears with the result that their intake of plant, and in particular cereal, fibre has diminished dramatically. One object of this book is to show that diverticular disease is a deficiency disease that is due to a diet depleted of fibre. The symptoms of the uncomplicated disease can be relieved or abolished by the addition of cereal fibre in the form of bran to the diet and so there is every hope that it will be possible to prevent the appearance of this disease in succeeding generations by a simple change in our eating habits.

It is hoped that the recognition of diverticulosis coli as a deficiency disease will stimulate research into the causation of the other diseases which also have appeared in the Western world in this century and which I believe are due, at least in part, to the refining of carbohydrates.

February 1975 N.S.P.

Acknowledgements

This book would never have been written had not I benefited from the expert tuition, encouragement and kindness of Dr S. C. Truelove. The pressure studies combined with simultaneous cineradiography which led to the understanding of the pathogenesis of diverticulosis were made in 1961 during the tenure of a research grant which was held while under his supervision at Oxford. This grant was obtained by the efforts of the late Professor P. R. Allison. As a result of this, I received a basic training in clinical research that I would otherwise have missed. The cine-radiographic films were taken by the late Mr Maurice Tuckey, Chief Technician to the Nuffield Institute for Medical Research, Oxford. Dr Kenneth Lumsden, Radiologist to the United Oxford Hospitals, spent many hours assisting me to interpret these films and Professor L. J. Witts gave me facilities in his Nuffield Department of Medicine.

Since then, I have been helped by many people including Dr Basil Morson, Pathologist to St Mark's Hospital and Dr John Madden of St Clare's Hospital, New York, regarding the pathology and the surgical treatment of the disease.

Although the study of the intracolonic pressures had convinced me that the 'low residue diet' is contra-indicated in the treatment of the disease, especially as it is absent in Africans who eat a bulk-forming diet, it was Surgeon Captain T. L. Cleave, R.N., (retired), who convinced me that diverticulosis is only one of many 'modern' diseases that are caused by our over-refined diet. Later, he and Mr Harold Dodd told me of their experiences with millers' bran in the treatment of constipation, with the result that a high-fibre diet has now been used for over seven years at Manor House Hospital where it has been shown that the simple addition of bran will relieve the symptoms of diverticulosis and lessen the need for surgery in the treatment of the disease. Mr Denis Burkitt, Dr Hugh Trowell and Dr Alec

Walker have given their help freely regarding the epidemiology of the disease and its relation to other diseases of civilization. Dr Kenneth Heaton has helped me to design a diet sheet advising patients how to take cereal fibre. Mr Edmund Godding and Mr Harold Godfrey have advised me regarding the properties and dangers of laxatives and bulk-formers.

My thanks are also due to Mr. Norman Tanner for writing the Foreword, Mr Michael Reilly for the chapter on his operation of sigmoid myotomy and Dr Louis Kreel for describing the radiology of the disease. Miss Margaret McLarty and Mr Frank Price drew the diagrams, some of which are published by the kind permission of the editors of the journals in which they first appeared.

Finally, it is a pleasure to thank my colleagues at Manor House Hospital, Mr Anthony Almeida, Dr Kenneth Colebourne, Dr Stewart Reynolds, Dr Florence Telfer, Mr John Barnfather of the Pathological Laboratory and Mrs Sheila Garrod, Superintendent Radiographer, who helped with the transit time studies, together with the pharmacists, the nursing staff and Records Department.

Mrs Joan Rhodes and Mrs Vivienne Dimant assisted my personal secretary, Miss Joan Inglis, who gave invaluable help with the follow-up of patients and typed this manuscript.

I wish to thank Mr Owen R. Evans and Dr Raymond Greene of William Heinemann Medical Books Ltd for their advice and patience in the preparation of this book.

To these and many others, I offer my sincere thanks.

Foreword

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Diverticular disease of the colon remains one of the enigmas of pathology. It is of interest to the physician because of its symptoms, but of paramount concern to the surgeon who has to deal with its complications.

Mr Painter has written perhaps the most outstanding study of colonic diverticulosis and diverticulitis since the classic work of Harold Edwards, and has brought our knowledge of it up to date.

This book will hold the interest of all who read it, family doctors, physicians, surgeons, epidemiologists and even laymen.

In the review of the disease in different countries, emphasis has been rightly placed on diet. This was an emphasis brought very much to the fore in the teaching of the late Professor A. Rendle-Short of Bristol in his studies of the rise of appendicitis and reflected in the writings of Surgeon Captain Cleave (who like myself was one of Rendle-Short's students).

Nevertheless, *every* variation in diet and habit between the diverticulous and nondiverticulous races must be debated. In the Middle East, diverticulosis is almost unknown and yet in some areas a low residue diet is used and constipation is a common complaint. The squatting position is commonly adopted in everyday rest and conversation. Does the squatting position reduce the intra-colonic pressure or even more important increase the ease of gas release through the anus? We look forward to further studies on this subject.

In the meantime, if we wish to study the up-to-date history, the anatomy, the pathology and complications of diverticulosis there can be no better preparation than to read Mr Painter's book, one which has involved years of arduous, honest and unbiased research.

Chapter 1

Definitions

A Colonic Diverticulum is, for all practical purposes, a herniation of the colonic mucosa through the muscular wall of the colon and hence diverticular disease is an acquired condition, (Fischer, 1900–1901). True diverticula of the colon containing all coats of the colonic wall have been described, but they may well have been exaggerated haustra (Edel, 1894). Fifield (1927) found no true diverticula in 10,167 consecutive autopsies at the London Hospital (Fig. 1).

The term 'diverticulum' is derived from the Latin 'diverto'

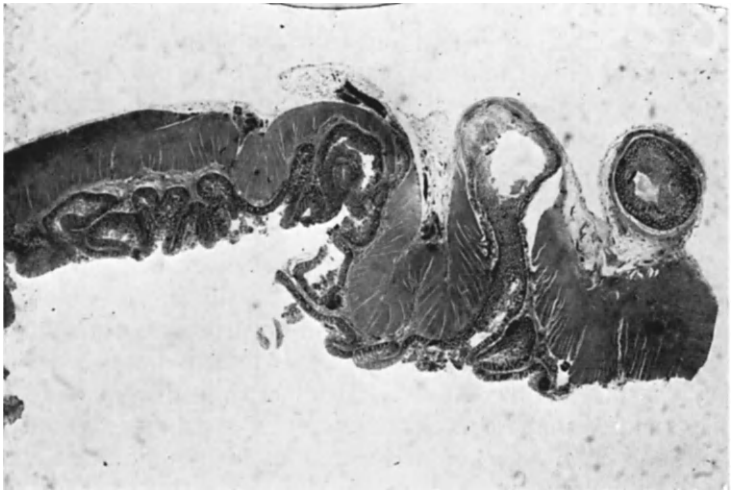

FIG. 1. Longitudinal section of colon bearing diverticula. The colonic wall on the left side of the illustration is apparently normal but it abruptly changed to bowel that is obviously abnormal on the right. The muscle coat on the right is thrown into folds of reduplicated muscle between which the mucosa has herniated to form two diverticula, only one of which has been sectioned so as to show its neck.

The change of structure from the normal to the diseased bowel is sudden and it is not difficult to see why the apparently normal bowel in diverticulosis produces a different pattern of pressures from that part of the bowel that is narrowed and is beset with diverticula (from Painter, 1964).

which means 'I turn aside'. The *-culum* is a diminutive which corresponds to the English—*icle*, as in *follicle* or *cubicle*. So the English equivalent to 'divericulum' is 'divertick' meaning a 'small turning aside' that is a pouch of limited size (Edwards, 1939). The former name is hallowed by official usage although the English 'diverticle' is used colloquially.

Diverticulosis implies that diverticula are present but that they are not inflamed. It was once thought that they did not cause any symptoms unless they were inflamed. This is still probably true but diverticulosis may be *associated* with symptoms as these are caused by some abnormal action of the colonic muscle of which diverticula are only the outward visible sign. Patients with diverticula may complain of symptoms even in the absence of true inflammatory diverticulitis and, furthermore, these symptoms may be cured by changing the diet or by Reilly's operation, and neither of these procedures remove the diverticula. Consequently, it is safer to say that symptoms are associated with diverticulosis as it may well be that they owe their origin to that part of the intestine that is proximal to the colon (Painter 1968 and 1972; Painter, Almeida and Colebourne, 1972).

The term 'diverticulosis' came into being when contrast radiology revealed that diverticula were common and, as the name diverticulitis was already in common use, the word 'diverticulosis' was coined independently by the German-speaking de Quervain, the American J. T. Case and by Sir Ernest Spriggs in England.

Diverticulitis obviously means that diverticula are both present and inflamed. In practice, it usually means that pericolitis is also present as the inflammatory process is not confined to one or more of the mucosal pouches once patients complain of symptoms of sufficient severity to merit this diagnosis. It was once assumed that colonic pain and other abdominal symptoms in patients with diverticulosis were caused by inflammation of the diverticula but it is now realized that excessive segmentation of the colon may cause recurrent pain or colic that is so severe that it may merit sigmoid colectomy. However, when resected sigmoid colons are examined, histological evidence of true inflammatory diverticulitis is often lacking (Morson, 1963). This condition is called *Painful Diverticular Disease*. It causes pain that is usually colicky in character as it is due to intermittent functional obstruction of the colon brought about by excessive

segmentation, but it may be so severe that it may be mistaken for left renal colic and morphine or pethidine given for its relief (Painter, 1964 and 1968).

The term *Low Residue Diet* has been used since the nineteen-twenties and, although it may mean different things to those in differing disciplines, to doctors it means that such a diet is, in the main, absorbed by the bowel and consequently it leads to a small quantity of faeces being passed. Most doctors also understand it to mean that pips, seeds, skins and stalks and so-called 'roughage' have been removed from any fruit and vegetables that form part of this diet.

The term *High Residue Diet* implies that the diet contains all those constituents which are not absorbed by the gut so that the stools are large and usually soft, whereas it has a different meaning to some dieticians. In this book, a High Residue Diet is synonymous with a *High Fibre Diet*. This latter term obviously means that the diet contains plenty of fibre. Dietary fibre is difficult to define but it will be seen, when the connection between diet and disease is under consideration, that cereal fibre probably has a special part to play in the physiology of the gastro-intestinal tract.

Chapter 2

The History of Diverticular Disease of the Colon

The diagnosis of diverticulosis depends on the demonstration of diverticula. Until radiological methods of examining the colon became available, the prevalence of diverticula was not realized and only the complications of diverticulitis had attracted attention. The profession became aware of the common occurrence of diverticula only when the radiological demonstration of diverticula had become a routine matter, and only then could the disease be classified and the symptoms and the complications of the condition be differentiated from those caused by other disorders. The changing pattern of the disease and the progress of medicine have divided the story of diverticular disease into six parts.

PART I

COLONIC DIVERTICULA AS A PATHOLOGICAL CURIOSITY

Voigtel (1804) first described these acquired hernia of the large bowel. Fleishman (1815) used the term 'divertikel' to refer to duodenal diverticula found at autopsy and also described single and multiple diverticula of the colon which he found mostly at the mesocolic border. He suggested that they were caused by distension of the gut by food, drink and air. Some have credited Matthew Baillie (1797) with the first description of diverticula but it appears that he described recto-vesical and rectovaginal fistula due to abscesses of the bowel.

Samuel Gross (1845) of Philadelphia described colonic diverticula and illustrated those of the small intestine. 'Sac like tumours . . . are sometimes found in the bowel, caused by protrusion of the mucous and cellular tunics across the muscular fibres, in the same manner as pouches are occasionally formed

in the urinary bladder . . . Their number ranges from one to several dozens . . . Professor J. B. S. Jackson of Boston . . . has met with them most frequently in the large bowel, in aged and corpulent persons. Their development seems to depend on some mechanical obstacle to the passage of faecal matter by which the muscular fibres are separated from each other so as to permit the mucous . . . membranes to protrude through the resulting intervals.'

Thus the earliest writers put forward the two theories of the pathogenesis of diverticula. The former believed that passive distension was to blame for their appearance, but the latter was proved right more than a century later when functional obstruction of the colon was shown to cause diverticulosis (Painter, 1962 and 1964).

Rokitansky (1849) described colonic diverticula and their contained faecoliths. Cruveilhier (1849) blamed the hardening of the faeces and straining at stool for their appearance and realized that faecal matter in the mucous pouches might lead to inflammation and to perforation. This complication probably occurred without being recognized at this time as Virchow (1853) described 'chronic adhesive peritonitis' usually with fibrosis that usually affected the sigmoid colon and might be accompanied by abscess formation that was not tubercular in origin.

Bristowe (1854) exhibited a typical case of sigmoid diverticulitis to the Pathological Society of London and described the tendency of the mucosal herniations to enter the appendices epiploica. On microscopic examination, he failed to find muscle fibres covering the pouches and concluded that they were herniations of the mucous membrane. He looked for, but failed to find, any mechanical obstruction of the bowel distal to the diverticula and concluded 'that the cause producing the abnormal condition must have resembled that operating in the case of the sacculated bladder; very likely habitual costiveness may have brought about some of the ill effects which might be expected to follow on actual obstruction'.

Haberschon (1857), physician to Guys' Hospital, described diverticula very accurately in a chapter on constipation contained in what is probably the first textbook of gastro-enterology published in the English language (Lloyd Davies, 1953). However, he did not believe that they produced symptoms or were in any way dangerous. By contrast, Sidney Jones (1859) described

vesico-colic fistula due to diverticulitis in a man of sixty-four who noticed faeces in his urine. He complained of abdominal pain and suffered from extravasation of urine to the scrotum, penis and lower abdomen 'from which he rapidly sank'. Autopsy revealed the sigmoid musculature thrown into transverse folds and a diverticulum that had ulcerated into the bladder which contained a calculus. This had caused urinary obstruction and death.

Generalized peritonitis due to diverticulitis was reported by Loomis (1870) according to Dunn and Woolley (1911); he believed that 'infection traversed the wall of the diverticulum without rupture'.

Arbuthnot Lane (1885) found a loop of colon in the sac of a left inguinal hernia at autopsy. This loop had been obstructed by adhesions and Lane noticed that there were no diverticula on the distended part of the gut but that 'the proximal end of the loop was very narrow and presented a double row of sacculi separated by . . . the band of longitudinal muscle fibre'. Dissection of this specimen convinced him that colonic diverticula resembled those seen in the bladder. He noticed that they were to be found only where the muscle wall was thickened and thrown into ridges. He concluded that diverticula were formed by 'vigorous contraction' of the colon on to its contents (Figure 2). He anticipated the views of Morson (1963) and Painter (1962 and 1964) when he realized that diverticulosis was caused by the action of the colonic muscle.

Harrison Cripps (1888) believed that the majority of vesico-colic fistula were more commonly caused by inflammatory mischief than by cancer but, in describing sixty-three cases, he mentioned diverticula only when quoting the example given by Jones (1859).

Thus, diverticula and the complications of diverticulitis were described accurately in the nineteenth century but they remained objects of curiosity. The muscle changes that precede their appearance were recognized and speculation as to the mechanism of their causation was surprisingly prophetic. Nevertheless, it is obvious that diverticulitis, which so commonly causes death, must have been a rare disease at that time or it would have been recognized more frequently at autopsy. The writings of our predecessors have been quoted at length to show that these men, who over a century ago could foretell the findings of modern

research, would have recognized the complications of diverticulitis had the disease been common in their day.

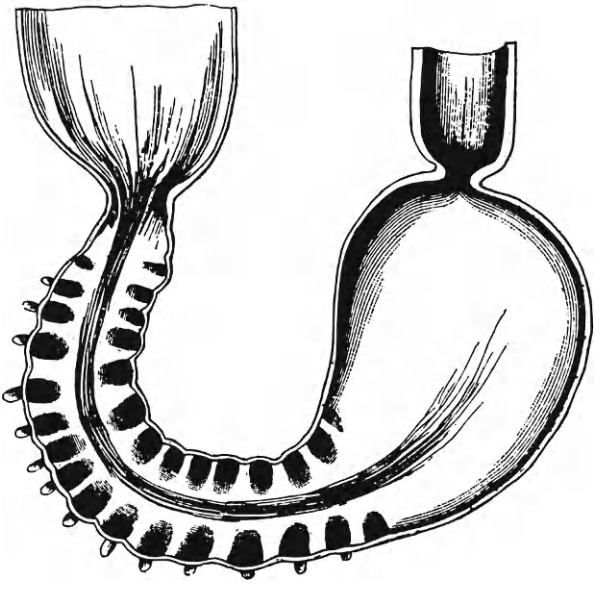

FIG. 2. Arbuthnot Lane's diagram of a sigmoid colon bearing diverticula. He observed that the diverticula were found on the contracted part of the colon and not on the thin distended loops which had resulted from the colon's incarceration in an inguinal hernia. Lane postulated that diverticula were produced by vigorous contraction of the colonic muscle. (Reproduced from Guys' Hospital Reports, by kind permission of the editors.)

PART II

THE RECOGNITION OF DIVERTICULITIS AS A SURGICAL PROBLEM

In 1899, Graser pointed out the potential danger of diverticula in that they became inflamed and gave rise to perisigmoiditis, stenosis and fibrosis of the colon. He warned that peritonitis would follow their perforation. In the next decade, he was proved to be right.

As laparotomy became more commonly practicable, surgeons were surprised by the damage wrought by diverticulitis. In 1903, Bland-Sutton found a piece of straw near to an epiploic appendage which he thought had perforated the colon. He believed

that in fat people these appendages caused diverticula by traction and that foreign bodies might penetrate this weak point. He had not been aware of this complication of diverticulitis previously despite being so competent a craftsman that, even in those early days, he could save the life of his patient who had faecal peritonitis. Even Beer (1904) who studied the literature relating to false diverticula, and who collected nineteen reports of complicated diverticulitis, still believed that diverticula seldom caused trouble.

Sir d'Arcy Power (1906) believed in 'pericolic inflammation' which originated in the connective tissue adjoining the sigmoid colon, even though he had the opportunity of seeing four cases of diverticulitis in only a year.

This attitude soon changed. Diverticulitis had entered the clinical arena and was being recognized with increasing certainty. In the same year, Gordinier and Sampson (1906) stated that diverticula caused symptoms more often than had been thought and in the next year Brewer (1907) described abscess formation and peritonitis due to diverticulitis; the latter still thought the condition was rare but caused trouble more often than the literature of the time suggested. Moynihan (1907a and b) described the ability of diverticulitis to cause obstruction and to mimic carcinoma. It is obvious that, at this time, this was still newsworthy as Mayo *et al.* (1907) reported five cases in which diverticulitis with tumour formation had been diagnosed during life; two out of the five patients died of peritonitis following resection, a measure of the hazards attending colonic surgery at that time.

Nevertheless, such diagnoses as 'pericolitis sinistra' and 'torsion of appendices epiploica' lingered on and were still considered respectable. Donaldson (1907) reported abscess formation due to pericolitis in a man of forty-four and believed it was due to some rupture of the mucous membrane due to an unknown cause. Lloyd Roberts (1908) believed that constipation had caused ulceration of the mucosa and abscess formation in the left iliac fossa in one patient, but he did point out that the colon was narrowed and spastic and that diverticula were present. Even as late as 1910, Gordon Taylor and Lakin were at first reluctant to attribute peritonitis to diverticulitis but in retrospect realized that they had dealt with three examples of this problem and suggested that this condition was not so un-

common as was generally thought. In 1908, Dr Maxwell Telling of Leeds described in detail the secondary pathological processes and the symptoms of sigmoid diverticulitis while pointing out that the subject had received 'but scant attention in the literature, especially in England'. It is significant that he said the whole subject was only a few years old and that French, German and American writers also were ascribing increasing importance to the role of diverticula in the causation of disease. There seems no doubt that the condition was a newcomer to the clinical scene and that Telling's paper in the Lancet established diverticulitis as a definite entity in this country.

Patel (1911) collected twenty-eight cases and discredited the 'torsion of appendices epiploica' theory and agreed with Telling that perisigmoiditis was caused by inflamed diverticula. Giffin (1911) reported abscess formation due to a rectal diverticulum and stated that between 1902 and 1910 fifteen operations for diverticulitis had been performed at the Mayo clinic but by 1912 he could review twenty-seven such cases and discuss the differential diagnosis between diverticulitis and carcinoma of the colon.

In 1917, two papers appeared in the British Journal of Surgery which summarized what was known of colonic diverticula and of diverticulitis at that time. Hamilton Drummond described the anatomy of the colon and of the mucosal herniations and their tendency to follow the course of the blood vessels (Fig. 3). Unlike many others, he did not claim that the weakness caused by these vessels was the cause of diverticulosis but concluded that, 'the blood vessels of the normal colon may be said to predispose to sacculi to the same extent as the spermatic cord does to an inguinal hernia'.

In the same year, and in the same journal, Telling and Gruner (1917) published their classic paper on 'Acquired Diverticula, Diverticulitis and Peridiverticulitis of the Large Intestine'. Encouraged by Lord Moynihan, they enlarged on Telling's paper of 1908 and discussed the theories of the causation of the disease as they were surprised to find that only 21% of their patients were constipated. They said that the disease should never be diagnosed under the age of thirty-five. They thought that it was acquired and due to high intra-colonic pressures as did Sir Arthur Keith (1910) who blamed muscle contraction for these postulated pressures and for the hernia. They were

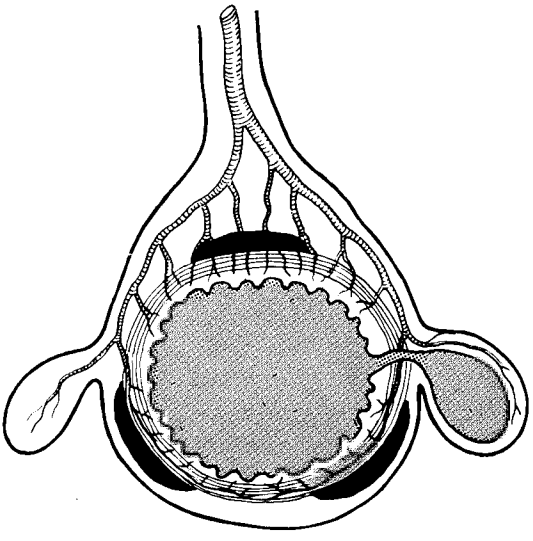

FIG. 3. Diagram of cross section of human colon in diverticulosis. The colonic lumen is surrounded by a layer of circular muscle that is complete outside which the longitudinal muscle is gathered into three taenia, one of which is at the mesocolic border. The colonic wall is obviously weaker between the taenia. The blood vessels that supply the colon pierce the circular muscle and weaken it further by forming tunnels. It is through these points of least resistance that diverticula in the main emerge. This is the classical diagram of Hamilton Drummond (1917) but Slack (1962 and 1966) has shown that there are two rows of diverticula on each side of the colon.

able to collect no less than 324 examples of diverticulitis from the literature of which 14% had led to stenosis, 28·4% had led to perforation and 19·8% to fistula formation. Not only did they suggest that diet might alter the flora and the speed of the faecal stream but they prophesied that barium x-rays of the sigmoid might well make diagnosis easier. They also advocated resection of the diverticula-bearing gut to avoid future complications.

Their contribution contained all that was known of the disease before radiology became widely used. The disease had changed from a rarity to a common complaint in only twenty years. Dr Maxwell Telling said that he saw his first case in 1899 when he could find nothing about it in the medical writings or anyone who was familiar with it but, by 1908, he could describe the pattern of the disease in detail. Diverticulitis surprised even

surgeons of high repute early in the century but by 1920 Sir John Bland-Sutton remarked, 'that in the last ten years acute diverticulitis . . . is recognized with the same certainty as appendicitis. Thus, diverticulitis is a newly-discovered bane of elders.'

PART III

THE DISEASE BECOMES A MEDICAL PROBLEM OF INCREASING MAGNITUDE

Between the two world wars, diverticulosis was found to be a common disorder whose incidence was steadily increasing. The radiological appearances of this new condition were described and its pathological anatomy were investigated in detail. Not surprisingly, there was much speculation as to its aetiology but almost no research was done on this subject. Consequently, the medical management of the disease was founded on theory which was later proved to be incorrect. It was believed at that time that 'roughage' irritated the gut, so a low residue diet became the accepted basis of conservative treatment despite the absence of any evidence that it was of benefit. Similarly, the surgeons of the day were hampered by the technical limitations of colonic surgery. Although they realized that resection afforded the only chance of cure, they were reluctant to operate when anastomosis of the large bowel carried a mortality of up to 25%, and so surgical intervention was reserved for complications that threatened life.

The changing incidence of diverticulosis is discussed in Chapter 11. By 1923, diverticulitis was sufficiently common to be listed as a cause of death in the Registrar General's reports for England and Wales. This was due in part to the advent of radiology. Haenisch (1912) demonstrated diverticula in the descending colon but it was de Quervain and Spriggs in Europe together with Carman and Case in America who made the barium enema popular. De Quervain (1914) recorded that his assistants diagnosed diverticula first by oral barium and then gave a barium enema to a second patient with acute diverticulitis and this was followed by perforation and peritonitis. Le Wald of New York outlined diverticula by means of a combined opaque meal and enema; his diagnosis was confirmed by subsequent surgery (Abbé, 1914). By 1915, Case had found

diverticula thirteen times by x-rays but Carman (1915a and b), who had demonstrated them as early as January 1914, still thought they were uncommon as did Spriggs (1920) who saw diverticula sixteen times in one thousand x-rays.

The term 'diverticulosis', meaning that diverticula were present but were not inflamed or causing symptoms, was proposed by the German-speaking de Quervain (1927) and used independently by Case (1928) and by Sir Ernest Spriggs (Spriggs and Marxer, 1925). By 1924, Judd and Pollock realized that the current statistics were wrong and clinical experience showed that the disease was not rare as one third of positive x-rays at the Mayo Clinic showed diverticula.

In 1925 Sir Ernest Spriggs and his radiographer, Marxer, described the radiological appearances of the disease and discussed its aetiology and treatment. These acute observers recognized that changes occurred in the colon before the actual herniation of the mucosa took place. They called this the 'prediverticular stage' of the disease. It is now thought that this 'stage' represents changes in the colonic musculature which are akin to trabeculation of the urinary bladder (Painter and Burkitt, 1971), but they interpreted it as evidence of 'sigmoiditis' due to the stagnation of faeces in the colon. Consequently, it seemed logical at the time to give liquid paraffin to cleanse the colon and sufficient fruit and vegetables to keep the bowels open easily in the hope of stopping concretions forming in diverticula that might lead to inflammation. They did not advocate a low residue diet any more than did Case (1928) or Mayo (1930) both of whom believed that the colon had to struggle with a low residue diet.

However, by 1930, Slesinger of Guy's Hospital was prohibiting the intake, not only of roughage, pips and seeds, but also of green vegetables. Thus, the low residue diet became the basis of medical treatment (Oschner and Bargen (1935), Willard and Bockus (1936), Brown and Marcley (1937), Edwards (1939)).

Harold Edwards (1934a and b) reported that between 1925 and 1931, 10·8% of barium enemas at Kings College Hospital showed diverticula. He studied the histology of this 'new' disease and demonstrated that before the diverticula appeared the circular muscle was thrown into ridges, when seen on longitudinal section, due to the muscle fibres being gathered into bundles, between which the muscle was extremely thin and

the serosa and mucosa came into close relationship. This work was of the greatest importance as it provided a material basis for the changes that had been reported radiologically and, also, for the appearance of diverticula. Edwards suggested that the sigmoid might harbour the highest pressures as it contracted against solid faeces, echoes of Arbuthnot Lane (1888), but as apparatus capable of measuring intra-colonic pressures accurately was not then available, his conclusions were not confirmed until Painter (1962 and 1964) and Arfwiddson (1964) measured these pressures.

Likewise, the surgeons were handicapped by the technology of the time. They knew that successful resection of the colon offered the only hope of cure but colonic anastomosis was still so dangerous that a relieving colostomy was often the only intervention that could be justified (Rankin, 1930; Lockhart-Mummery, 1938). It must be remembered that gastric suction, intravenous therapy and blood transfusion were almost unknown before the last war, and anaesthesia was primitive by modern standards. The contemporary attitude of the profession was summed up by Eggers (1941) who, after describing his experience of eighty-two patients suffering from symptomatic diverticulitis, said that surgery should be reserved for serious complications. Consequently, most patients were given a low residue diet with liquid paraffin in an attempt to prevent the onset of diverticulitis and so the rate of cure for the condition remained extremely low.

PART IV

THE SURGICAL ATTACK ON DIVERTICULITIS

In 1942, Reginald Smithwick published a paper that showed that the dangers attending colonic surgery could be greatly reduced by careful planning. He concluded that defensive surgery achieved little and that a more aggressive attitude would enable the diseased colon to be attacked successfully, and as a result, after the second World War 'pre-emptive' surgery became routine in the treatment of diverticulitis.

He estimated that only between 10 and 20% of patients being treated for the disease ever required some form of surgery and, as the condition was still comparatively rare at that time, no

one surgeon's experience would be very great. Therefore, he collected several published series and combined them with the surgical experience of the Massachusetts General Hospital over the previous fifteen years. The following facts emerged from this study.

The complications of acute perforation, abscess and fistula accounted for about 40% of all operations. The mortality from all procedures, including simple colostomy, varied from 9 to 24%, average 17·1%, while no less than one quarter of those treated by primary resection and anastomosis died. Among the fortunate survivors, only 60% were well and about another 10% died of the disease later. Procedures that were lesser than resection were not only ineffective in relieving symptoms but did not even reduce the eventual mortality from diverticulitis. He found, however, that if resection was successful, only 3·7% died later due to further episodes of diverticulitis and over three quarters remained well and symptom free.

He concluded that, 'the outlook of patients with severe forms of diverticulitis is not very bright'. To say the least, this was an understatement.

He argued that, as diverticulitis was localized to only one defined part of the sigmoid colon, resection was feasible.

Attempts to exteriorize the affected bowel, with or without colostomy, gave poor results, and although the Mickulicz type of resection was safe, it was not always technically possible. He noticed that a more extensive resection gave better results than did a more limited excision. Leakage from the anastomosis was a major cause of death and neither an accompanying or a preliminary caecostomy lessened the incidence of this complication. However, if a preliminary colostomy had been performed and the inflammatory process allowed to settle for between three and six months, the subsequent resection and anastomosis were relatively safe.

His plea for planned resection before serious complications occurred is a milestone in the story of the disease. It came at a time when few doctors would subject patients to colonic surgery for a benign disease because of the risks involved. At the same time, many realized that the quality of life of patients with recurrent diverticulitis or with a permanent colostomy was far from satisfactory and so many surgeons were sympathetic to the view that a planned resection of the sigmoid was preferable

to lethal complications at some future date—always providing that the operation could be made safe.

Fortunately, Smithwick's advocacy of more aggressive surgery coincided with the advent of the antibiotics, advances in anaesthesia and blood transfusion and in supportive therapy generally. As a result, elective resection became a standard procedure in the next decade. The credit for this is mainly due to American surgeons as most of the reports of this era came from the U.S.A. The author believes that there may have been a good reason for this. Diverticulitis had increased in prevalence in England until 1939 when the death rate from the disease became static. This may well have been due to the less refined wartime diet forced on the British people, which provided them with extra fibre and helped to prevent the disease. No such change occurred in America and there the disease continued to cause an increasing problem which had to be tackled.

There is no doubt that it was tackled. Smithwick's claim that staged resection was the safest procedure was echoed by Morton (1946). By 1947, Pemberton, Black and Maino examined and restated the role of surgery in diverticulitis. They found that at the Mayo Clinic between the years 1908 and 1940 even simple colostomy killed one in twenty patients, but after this date its mortality had dropped to only 1%. The death rate from resection in the corresponding period had fallen from 17% to 5%. They stressed that if a proximal colostomy had been fashioned it should never be closed unless the distal diseased bowel had been resected as in twenty-nine cases where closure preceded resection no less than twenty patients had suffered subsequently from recurrent diverticulitis. Having shown that both colostomy and resection now could be carried out with an acceptable risk, they argued that the indications for resection should be broadened so as to prevent the possibility of lethal complications at some future date. They attributed the improved results of surgery to the sulpha drugs and antibiotics, in the main, while admitting that both surgical technique and after-care had also advanced with the setting up of a special unit for colon surgery.

Boyden (1950) reported that Dr Thomas Joyce of Portland, Oregon, had attacked the disease by resection between 1929 and 1944 in twenty-seven cases and almost always without a preliminary colostomy. This had lowered morbidity and so Boyden favoured early resection if only because of the difficulty in

distinguishing between diverticulitis and carcinoma. He believed that a colostomy should always be established in the presence of obstruction and that resection should follow after only two to nine weeks. He also reported nine successful primary resections performed without a colostomy.

It was soon realized that this more radical surgery cured symptoms and lowered morbidity. This led to the adoption of elective resection, preferably as the procedure of choice. By 1950, Mayo and Blunt could report that resection used in the correction of vesico-colic fistula only carried a 6·5% mortality. Lloyd-Davies (1953) favoured early resection especially when obstruction had occurred because in about one half of such cases it was impossible to exclude the presence of cancer.

The good results of staged resection almost inevitably encouraged others to perform a one-stage resection without colostomy. When successful, this lessened the time in hospital and the risks attending more than one operation. Judd and Mears (1953) treated recurrent diverticulitis, fistula, abscess and even obstruction at one operation with only one death due to concurrent disease; while others suited the type of operation to the problem that confronted them using procedures involving one, two or three stages. The changes that occurred in the attitude of surgeons can be gauged by the report of Waugh and Walt (1957), namely that 30% of 320 patients with diverticulitis had been treated by a one-stage resection from 1945 to 1954; primary resection was safe as only one of ninety-three patients had died. McCune and Iovine (1957) favoured one stage resection when the disease was in remission but stressed that they would add a colostomy if the extent of the disease prevented them obtaining normal bowel for anastomosis; this accords with the recommendation of Welch, Allen and Donaldson (1953) that over 20 cm. of bowel should be resected. The virtues of primary resection were also extolled by Strenger (1957) and Waugh and Walt (1959), while in England, Lloyd-Davies (1953) advocated earlier resection and pointed out that even the most complicated cases, that necessitated long and difficult operations, could be cured by more aggressive surgery. Todd (1955) remarked that resection was safe and that surgeons were still too reluctant to advise radical surgery.

Thus the treatment of diverticulitis changed dramatically after the second World War. Previously, surgeons had only

operated for complications when life was threatened. Often this meant that they did too little and too late. Once resection was shown to be safe, especially if all the thickened bowel was removed, elective surgery became commoner. At first advanced cases with complications were tackled successfully and then, as confidence increased, recurrent pain in the left iliac fossa became an indication for sigmoid colectomy. This was performed in the belief that the patient had recurrent diverticulitis and as less diseased colons were resected, more and more one-stage operations were performed. This represented a great advance and by 1958 Barborka concluded that elective resection was not only the treatment of choice but should be performed even earlier. One-stage sigmoid colectomy had become so commonplace that he felt it necessary to say that when operating for perforation or obstruction, preliminary colostomy was mandatory.

This sudden swing to primary anastomosis was not without danger. Disruption of a colonic anastomosis is a serious complication that may be fatal. The distal colon is the most treacherous part of the gut to anastomose and most surgeons have experienced or seen the serious complications that may follow leakage from an anastomosis in the pelvic colon. Such disasters are seldom published and it is certain that a number of patients have died due to disruption of an anastomosis, following one-stage resection, which had been performed because the operation had become fashionable in the surgical literature. These patients would have survived for many years if a simple colostomy had been fashioned, whether or not a curative operation had been performed later. While colonic surgeons can keep the mortality of sigmoid resection low, this is not the case with those who are less experienced or less interested in this branch of surgery. Consequently, Colcock (1951 and 1971) has warned quite rightly against being over-confident when dealing with a disease which so often calls for the greatest skill and judgement. There is no disgrace in performing a colostomy or in staging procedures, to suit the patient or the extent of the disease process. Discretion is the better part of valour when the patient's life is at stake. There is no doubt that the surgery of diverticulitis is theoretically simple but often technically difficult (Thompson, 1959).

By 1960, elective resection, with or without colostomy, had

become the procedure of choice in diverticulitis. The methods employed are still a matter for discussion. In the preceding twenty years the disease had become one of the major problems of surgery, but the majority of patients were still looked after by their physicians who gave them laxatives and a low residue diet. The fourth phase of the story of the disease ended when the physiology of the colon became understood better and the pathology of the disease was examined by Morson (1963). This led to further improvements in the medical and surgical management of what was soon to be called 'diverticular disease of the colon'.

PART V

THE RECOGNITION OF THE ROLE OF THE COLONIC MUSCLE IN THE PATHOGENESIS OF DIVERTICULA

The abnormality of the muscle coat that had been blamed for the disease as long ago as 1845 by Gross and later by Harold Edwards was shown to be responsible for the herniation of the mucosa by Painter (1962 and 1964) in England and by Arfwidsson (1964) in Sweden. This led to the recognition of the part played by segmentation in colonic physiology in the production of the localized pressures that cause diverticulosis. These advances ushered in the fifth phase of our understanding of the disease process.

At the same time Morson (1963) studied the pathological changes in 155 specimens resected at St Mark's Hospital with a pre-operative diagnosis of 'diverticulitis'. He found that only in one third of them was there histological evidence of sufficient inflammation to justify this diagnosis or to account for the severe pain that had led to major surgery. He showed that the thickening of the colonic muscle was a constant feature in his specimens and he also reported that he could not demonstrate the presence of diverticula in some specimens even though the sigmoid colon had been resected for 'diverticulitis'. Likewise Arfwidsson studied the typical changes in the muscle and confirmed that in the 'pre-diverticular state' the thickening of the circular and longitudinal muscle occurred before the diverticula appeared and that these changes were accompanied by the

ability of the altered colon to produce higher pressures than the normal colon. He came to the same conclusion as Morson, namely that the muscle changes preceded the actual herniation of the mucosa while Painter and his colleagues (1965) recorded abnormally high pressures in the immediate vicinity of a single diverticulum in the sigmoid, thus showing that the abnormal pressure response was present in a very early stage of the disease.

The muscle thickening, and its radiological appearances, were described by Williams (1963) who showed that the classical radiological signs of 'diverticulitis' cannot be explained on the basis of inflammation but only by a primary fault in the action of the colonic muscle which could be present in the absence of diverticula.

Painter (1964) suggested that the pain of so-called 'acute diverticulitis' was often the colic of intermittent functional obstruction brought about by excessive segmentation of the colon and was not due to the inflammation of diverticula. He pointed out that the urinary bladder produces severe pain, even in the absence of infection, when its outflow is obstructed, and that a similar state of affairs might apply to the segments of the colon whose outflow is occluded on each side by excessive segmentation. The term 'Painful Diverticular Disease' was used by Painter (1968) to describe this colic which waxes and wanes with a rapidity that is inconsistent with the onset and resolution of an inflammatory process.

These observations led to a reappraisal of the term 'diverticulitis'; the term means that an inflammatory process is present and should no longer be used to describe pain of colonic origin.

Thus the attention of the profession shifted from the actual diverticula towards the colonic muscle. The present author believes that the changes seen in the muscle of the colon in diverticulosis represent 'trabeculation' of the colon and that this corresponds to that seen in the urinary bladder whose outflow has been partially obstructed for many years.

The thickening of the sigmoid muscle is often very localized and Reilly (1964, 1966) realized that this pathological change was often the only abnormality present and so he questioned whether it was necessary to resect an otherwise normal sigmoid colon just because its lumen was narrowed. He devised the operation of sigmoid myotomy in which the muscle is divided

longitudinally down to the sub-mucosa as an alternative to resection. The operation is similar to a Ramstedt's or Heller's procedure and in selected cases is as effective as resection but entails less risk. The division of the circular muscle fibres restores the lumen of the bowel, and reduces the intracolonic pressures. The fact that Reilly's operation relieves symptoms shows that they are due to muscular activity and not to the diverticula which remain *in situ* at the end of the operation.

Thus in the last few years modern technology has made it possible to show that abnormal activity of the colonic muscle is responsible, not only for the production of diverticula, but also for the symptoms that had been attributed previously to 'diverticulitis'. This knowledge was applied by Reilly who devised his 'physiological' operation of sigmoid myotomy. The term 'diverticular disease of the colon' came into use once it was realized that the diverticula were secondary to some disorder of colonic motility. The pathogenesis of the condition was now understood but the aetiology of diverticulosis still remained a mystery.

PART VI

DIVERTICULOSIS AS A DEFICIENCY DISEASE

The appearance of the disease and its emergence as a major medical problem, which have been described, leave little doubt that it is a 'new' disease. Evidence is accumulating that it is the result of the changes which occurred in our diet towards the end of the last century and which altered the environment of the colon. The geographical distribution of the disease reveals that it is rare or unknown to this very day in the rural African and Asian and in all countries where traditional eating habits have not changed. The disease is unknown where the diet contains plenty of cereal fibre but it has become the commonest disorder of the colon in those countries whose food is processed and refined (Cleave, Campbell and Painter, 1969; Painter, 1970; Painter and Burkitt, 1971).

The connection between dietary fibre and bowel behaviour has been demonstrated and discussed by Burkitt, Walker and Painter (1972) and by others. Where the diet is fibre deficient the intestinal transit time is prolonged and the daily weight of

stool passed is low. Conversely, the inclusion of plenty of cereal fibre in food produces large soft stools that traverse the bowel rapidly. The replacement of the bran fibre which is missing from our modern diet has been shown to relieve 85% of the symptoms of uncomplicated diverticular disease (Painter, Almeida and Colebourne, 1972).

At the present time, it appears that the aetiology of diverticular disease is linked to fibre-deficiency. Consequently, like scurvy, the disease should be preventible. One purpose of the book is to draw attention to the fact that if the citizens of this country would return to a less refined diet containing the natural amount of fibre, there is every reason to believe that our children might not suffer from the disease or from its companions, appendicitis and cancer of the colon.