

## ACQUIRED DIVERTICULA, DIVERTICULITIS, AND PERIDIVERTICULITIS OF THE LARGE INTESTINE.

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LEEDS.

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### I.—HISTORICAL AND INTRODUCTORY.

DIVERTICULITIS is a condition which has now passed out of the realm of doubt and uncertainty into that of proved and accepted fact. It has an important place in medical literature, and in the experience of every operating surgeon of large practice. This last statement is not as yet true of all clinicians, some of whom are still apparently unaware of the condition, and not a few of its frequency and clinical importance. Not until a morbid condition is described in all the ordinary student's text-books of medicine and surgery can it be said to have attained complete recognition, and this is not as yet the case with diverticulitis.

Diverticula of the intestinal tract have been noted by various observers for more than a century, but for the most part as isolated cases, often obscurely 'buried' in the literature. Some of these have presented features of considerable clinical interest, but the condition was looked upon as a rare pathological 'curiosity' of no real clinical importance. This aspect of diverticulum-formation in the large bowel (particularly in the sigmoid flexure) was

first given prominence by Graser\* in 1898, who showed that such cases are not very uncommon, and are of great importance. In particular he described the hyperplastic stenosing type known as peridiverticulitis, and its simulation of carcinoma of the sigmoid flexure. Shortly after this the subject was extensively studied in America, where Beer, Fischer, and others made contributions to the etiology, and the Mayos, Wilson, Brewer, Giffin, and others to the clinical aspects; also Patel in France (especially in regard to sigmoiditis and the acute types of the condition), and Moynihan in England. The last observer published the first case (1907) in this country of peridiverticulitis causing a 'mimicry of carcinoma,' and moreover laid very special stress on the rôle of the diverticulum in causing non-malignant vesico-sigmoid fistulæ.

In 1908, for the first time, the recorded cases were collected and analyzed, and the pathological changes and clinical results collated and classified. Prior to this, observers had for the most part dealt with special and isolated aspects and results of the condition. This classification has furnished the basis for most articles of importance written since that date. In the main it has been adhered to in the present contribution, which is but an amplification of the subject as then outlined.† A much larger number of cases—324 in all—have been studied, and an attempt has been made to present a conspectus of the whole subject.

Though we have sought to lay special emphasis on the clinical aspects, the histology and bacteriology have been presented in what we believe to be a more accurate relationship to the naked-eye anatomy and symptomatology generally. In this way it seems to us that the clinical facts emerge, so to speak, naturally and logically from the pathological facts which precede their study. It may be added that the subject appears to have received far less attention from observers in this country than in America, France, Germany, and elsewhere, judging from the comparative paucity of recorded cases and references to it in the more important periodicals.

One does not wish to overdraw the picture of a condition which is not of everyday occurrence, but diverticulum-formation in the intestinal tract, especially in the large bowel, and very particularly in the sigmoid flexure, besides having a complex symptomatology of its own, enters into the consideration of many other pathological processes and syndromes. We have no hesitation in saying that it can only usefully be studied as a separate and special condition, and not, as has been very generally done in the past, merged into and confused with a variety of other lesions and disguised under a variety of terms. These points we hope to establish conclusively in the succeeding pages.

**Definition.**—Diverticula of the gut are divided into *congenital* (of which Meckel's is the type), and *acquired*, the chief anatomical difference being that

\* The bibliography is arranged alphabetically at the end of this paper. The numbers in brackets throughout the article refer to the list of selected case-abstracts given as an appendix.

† The present study has been undertaken at the request of Sir Berkeley Moynihan, whose clinical experience of the frequency of the condition has suggested the desirability of reviewing all the facts known at the present time.

the former are composed of all three coats of the bowel, while the latter have been described as hernial protrusions of mucosa and serosa only (von Rokitsansky, Cruveilhier, Leichtenstern, and others). This is not strictly true, however, as very many examples of acquired diverticula have now been described into the structure of which all three coats of the intestine enter, and this is probably very frequent in the earliest stages of their formation. Diverticula are also classified as *true* or *false*, on the same basis of differentiation, and Edel has used the terms 'congenital' and 'true' as synonymous; likewise 'acquired' and 'false'. But whereas congenital diverticula are always true, *acquired diverticula may be true or false*, though more usually the latter in the stages in which they are mostly studied. The first description of false diverticula of the intestine is usually attributed to Sömmerring, in his translation of Baillie's *Morbid Anatomy* (1794), but reference to the original shows that polypi and not diverticula were described. Moreover, Voigtel quotes earlier references.

**Distribution.**—The consensus of modern opinion leans to the view that acquired diverticula of the whole intestinal tract, from œsophagus to anus, owe their origin to similar tendencies, and are but local variations of the same morbid process or processes. The results of the presence of diverticula vary greatly with their site of origin. In the small intestine, their occurrence is practically devoid of secondary changes or clinical symptoms (Gordinier and Sampson record an exception to this), and the reason is undoubtedly to be found in the fact that in this region of the bowel they rarely have permanent fœcal contents. This tendency to be filled with and to retain fœces is the leading characteristic of the diverticula of the lower bowel, and from this fact almost all their secondary pathological and clinical importance is derived.



FIG. 268.—Sketch of an appendix with multiple diverticula. (After Lejars, 1904.)

In this study we limit ourselves to the consideration of diverticula in the lower bowel. Here they occur in the appendix, cæcum, all sections of the colon, with overwhelming frequency in the sigmoid flexure, in the rectum, and even at the anal margin. In the sigmoid it is the distal rather than the proximal portion which is affected, often it is the last few inches. According to Patel, the more mobile part of the loop is the site of election. Diverticula of the appendix have already a literature of their own, Lejars and Ménétrier, Edel, Mertens, Upcott and others have described and figured them very

fully. In this structure the diverticulum-formation is essentially the same process as elsewhere, and tends, on the whole, to occur at an earlier age than in the rest of the colon. Perhaps some of these appendix diverticula owe their origin to preceding inflammatory changes in the wall of the gut—the sequence is difficult to establish, if it occurs. It would be in sharp contrast to the diverticula of the rest of the large bowel, where inflammatory change invariably *follows* the formation of the diverticulum. Inasmuch as the whole clinical importance of the appendical diverticula is merged into that of

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appendicitis generally, almost without special feature, we do not propose to consider them at length.

The site of election for these evaginations is the sigmoid flexure, especially in its distal portion down to the junction with the rectum, where they often stop abruptly, however numerous they may have been above it. Various reasons for this have been given; the absence of appendices epiploicæ, and a stronger musculature (Schreiber), are among the most convincing. At

the same time some cases do occur in the rectum (30, 33, 36, 42, 44, 50, 57, 79); and one case has been described in the anal margin. We shall describe the anatomy and arrangement of the diverticula as they occur

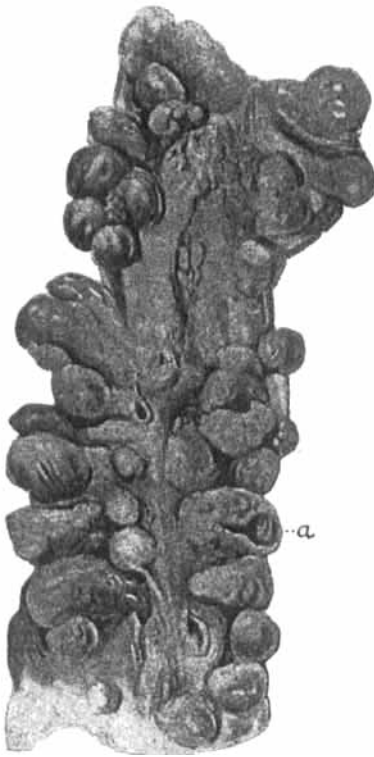


FIG. 269.—Diverticula of sigmoid. The fat has been dissected from the outer aspect of the bowel. The pouches are for the most part into the appendices epiploicæ. *a*, A sac which has been laid open. (Case 82.)



FIG. 270.—Inner surface of the bowel of the specimen shown in Fig. 269. *b*, Concretion presenting at the orifice of one of the diverticula, *c*, Lipped orifice of a diverticulum. (Case 82.)

most typically in the sigmoid flexure, for—vermiform appendix apart—diverticulitis of the *large bowel* may almost be written diverticulitis of the *sigmoid flexure*. They do occur in the cæcum, and may even be confined to this region; with less frequency are they encountered in the ascending and transverse colon, at the splenic flexure they are more frequently noted, in the descending colon again less numerous (though more so than in the proximal reaches of the colon), until the sigmoid flexure is reached. Sometimes the whole colon is studded with them, as many as 400 having been described (Hansemann, 80), or noted

as "in hundreds" (17). Not infrequently diverticula occur in both small and large bowel in the same subject (84), in some cases even in the œsophagus as well, thereby emphasizing their essential etiology.

In the small intestine they almost invariably occur at the mesenteric attachment; this is in rather sharp contrast to the colonic diverticula, which are relatively seldom on the concavity of the gut. Some authors state that in the sigmoid flexure they are usually *close to* the mesentery, but most observers, including ourselves, find them most frequently nearer the *convexity* of the gut. They are usually multiple, and tend to occur in two rows. Very frequently, indeed, they occur as hernial protrusions of the mucosa into the appendices epiploicæ, and may be confined to this situation. The probable explanation of this is that offered by Bland-Sutton, that the appendices are frequently filled with fat, and so are areas of lessened resistance (*Fig. 271*).

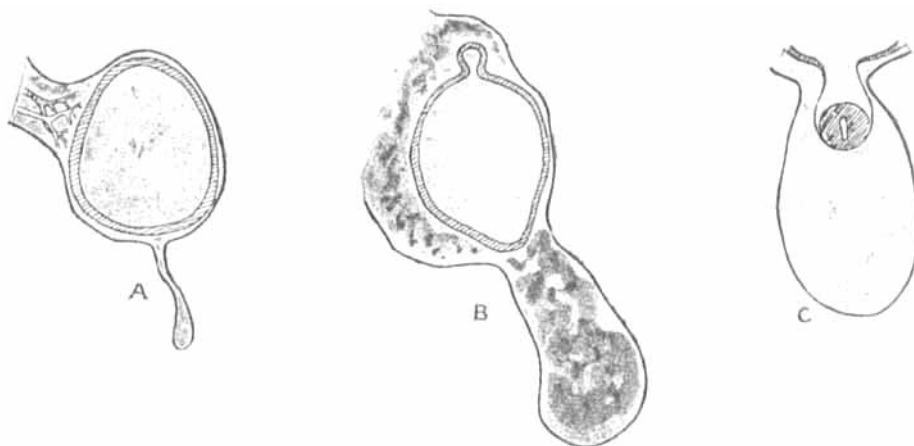


FIG. 271.—To show the manner in which richness of the appendices epiploicæ in fat predisposes to diverticulum formation.

A. Diagram of a transverse section of the colon from a thin individual, to show the relationship of an epiploic appendix to the subserous fat.

B. Diagram of a transverse section of the colon from a very obese male, age 55, to show the disposition of the subserous fat and its relationship to the epiploic appendage. A diverticulum is in process of formation.

C. Diagram showing a diverticulum pouching into the epiploic appendage, and containing a fecal concretion, in the interior of which is a foreign body (*Case 67*). (After Bland-Sutton.)

The diverticulum starts as a hernial outpushing of the mucosa, either through or accompanied by the muscular coat (one or both layers), and of course covered externally by the serosa. It very early acquires a *flask shape*, the neck being narrower than the distal 'body,' and there is frequently lipping of the oval or circular orifice when viewed from within the bowel. The orifices are often quite small, and are frequently largely or entirely concealed by the circular folds of the lumen (*Fig. 284*), indeed, a feature of diverticulum-bearing gut is an unusual tendency to rugosity. No great size is as a rule attained, it varies from mere microscopic visibility to the size of a plum-stone, a very usual size is that of a currant, or less (*Fig. 269*). They may cause trouble when only microscopic (4).

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These small diverticula, which are potential for mischief out of all proportion to their size or number, are still further concealed by their frequent occurrence in fat-laden gut, particularly so when they enter the appendices epiploicæ. *Fig. 272* shows this clearly, for here one-half of the gut is seen with its fatty covering (a single diverticulum only having been dissected out), and the other half has had all the fat removed, leaving the diverticula to reveal their true proportions. *Fig. 269*, in which the diverticula have been dissected out, is a typical example of the condition, they are so numerous as to resemble bunches of currants.

As has been emphasized, they mostly contain fæces, of varying degrees of hardness and inspissation, often actual calculi. Sometimes these fæcal masses can be seen through or slightly projecting from the lipped openings: this is well seen in *Fig. 270*, and again in *Fig. 273*.

Their naturally concealed positions help us to understand the infrequency with which the condition has been noted in the past, and we shall see, when the secondary pathological processes to which they are liable are described, that the diverticula are frequently modified in such ways as to defy recognition by any but the most careful search, and that prompted by fore-knowledge. These facts make it difficult at present to express any final opinion as to the absolute frequency of these diverticula. Until quite recently many morbid anatomists of experience have been quite unaware of their occurrence, this explains the great difference between the statistics furnished from various hospitals, and impairs their value. If looked for they will be found not infrequently by anyone making a considerable number of post-mortem



FIG. 272.—Diverticula of sigmoid, showing enteroliths.<sup>1</sup> The abundant fat has been dissected from one-half of the bowel, showing the pouches which entered the appendices epiploicæ. *a*, A single pouch which has been dissected out, showing well how they are buried in fat, and liable to pass unrecognized unless specially sought for, *b*, Longitudinal muscular band; *c*, A concretion.

examinations. This agrees with our own experience. But in the majority of autopsies the sigmoid flexure is a neglected and little explored region, the rectum even more so. As bearing upon this point, Graser found diverticula on *microscopic* examination in 10 out of 28 subjects, and Sudsuki in 15 out of 40. More microscopic research on this point is required, for it is of course the most certain evidence of the *tendency* to diverticulum-formation. In 100 consecutive aged subjects, de Mourges found naked-eye diverticula 30 times.

In 1908 one of us collected 105 cases (macroscopic, and mostly giving rise to symptoms), adding among them 22 to the literature. We base the present

analysis on a series of 324 collected cases (excluding those in the vermiform appendix); but this gives no idea at all of absolute frequency. For instance, in our first series of 105 cases, 33 were examples of diverticulum-formation only, without secondary changes. Since then such specimens have for the most part not been considered worthy of putting on record (save perhaps in small provincial journals and in reports of medical societies), most cases published since that date are examples of diverticula which have acquired special pathological (and therefore clinical) interest. It is to the experience of individual surgeons and morbid anatomists that we must turn for some true estimate of frequency. for instance, in the Mayo Clinic 27 cases were operated on

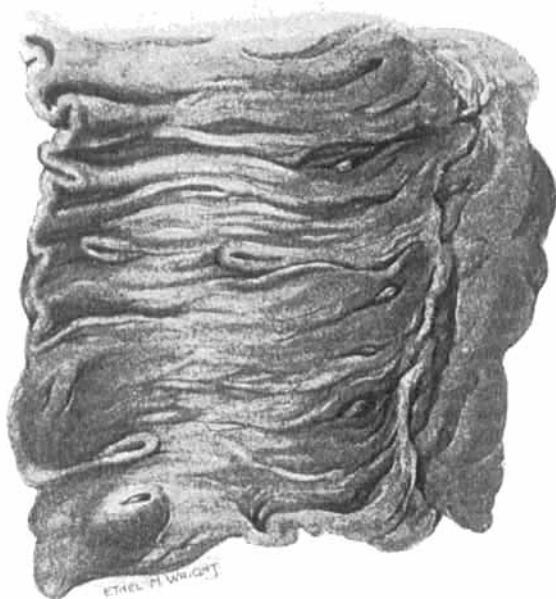


FIG. 273.—Sigmoid showing diverticula inner aspect of gut. Normal but rugose mucosa. The orifices show well-marked lipping, and concretions protrude partially from some of the pouches.

between 1902 and 1912 (Giffin), and surgeons of large practice meet the condition with some regularity. They are mostly able to refer to their own series of cases, and our own experience in the post-mortem room is some indication of the number of cases that can be discovered if regular search is made.

## II.—ETIOLOGY.

What is the cause of the diverticulum-formation? Much controversy has raged round this question, and we do not propose to do more than briefly summarize the arguments and conclusions. Two main views at present hold the field, and they are by no means mutually exclusive. No observer is of

opinion that the diverticula are entirely of congenital origin, though some facts cited below lend support to the view that there may be in some bodies a congenital predisposition to their formation. The view that is most widely held is that the diverticula are essentially *pulsion diverticula*—that is, are the result of increased pressure within the bowel; this may be due to the accumulation of faeces or the presence of gas. The site of election in the lower sigmoid is alone a great fact in support of this view, for here the faecal stagnation tends to be greatest. It is the chief 'delay point' in the intestines, the stools are becoming firmer and less moist, and the formation of gas is frequent and often considerable. It seems to us impossible to dissociate the frequent implication of the sigmoid flexure from the physiological rôle of that structure. In support of the 'increased pressure' theory the frequency of constipation in the case-histories is adduced. We find it in 21 per cent. But it is absent not infrequently, and in some cases (3·5 per cent) actual looseness of the bowels has been the rule. Moreover, constipation is exceedingly common and diverticula relatively very rare, therefore some other factor must be sought. Constipation is commoner in women than in men, yet 68 per cent of our cases are in males. In regard to the internal pressure as a causal factor, we think the importance of gas-formation has been overlooked. Many persons normally produce much gas in the lower bowel, and have perfectly regular defæcation, as Sir Lauder Brunton long ago pointed out, in his 'pop-gun' simile. One has the impression that the gas factor may be more important in the male sigmoid.

It is easy to insist unduly upon the increased-pressure view, there must always be a definite pressure from within outwards in the bowel, it is obvious that the formation of acquired diverticula must be determined by the *ratio* between the resisting power of the intestinal walls and such internal pressure. Anything, e.g., constipation, tending to increase the pressure, will tend in the direction of diverticulum-formation. They have been noted in great abundance in the sigmoid when the bowel below has been for a long time compressed by a large pelvic fibroma (de Mourgès). We have found them above a polypus (85).

Much attention has therefore been devoted to studying the condition and tone of the bowel wall in these cases. Certain general causes of weakness have been alleged. De Mourgès comments on the frequency with which the condition is found in association with various visceral ptoses resulting from a generally weak and relaxed abdominal musculature. Obesity is

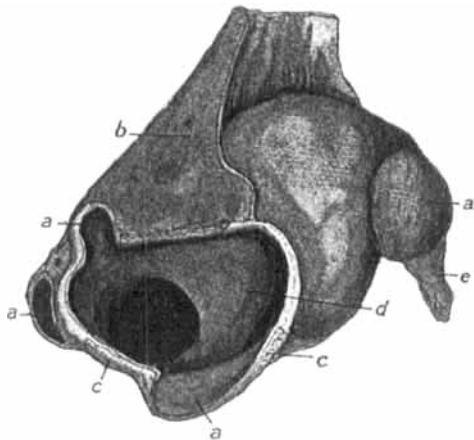


FIG. 274.—Diagram showing the relationship of the diverticula (a) to the tæna (c), and the manner in which the lumen is occluded by semilunar folds (d). b, Mesocolon, e, Appendix epiploica. (After Keith, *Brit. Med. Jour.*, 1910, 1, 379.)



another such cause of weakness. It is noted as being present in 60 per cent of cases in which the point is mentioned. Undoubtedly more diverticula occur in fat-laden gut than in that possessing a poor fatty covering; the fat

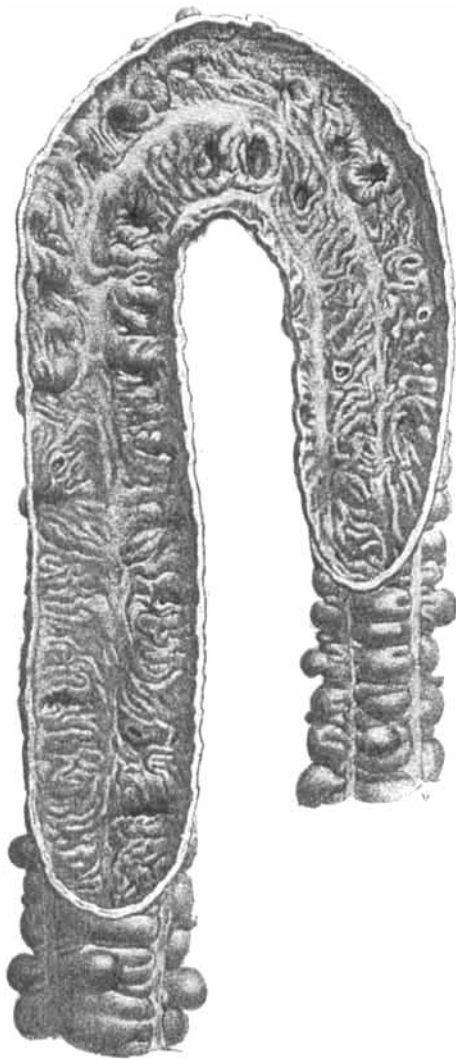


FIG. 275.—Portion of descending colon from a male subject, age 72. The specimens laid open to show the internal orifices of diverticula, and the characteristic appearance of the mucosa. Some of the cavities contained solid faeces. At each extremity is shown the characteristic appearance of the exterior of the colon. (Case 60.)

may weaken the musculature by causing gaps between the fibres, and *Fig. 271* furnishes a fairly conclusive explanation of the great tendency of the diverticula to invade the appendices epiploicæ. But Hanseemann has commented on the cachectic condition of many subjects, with emaciation, or absence of anything like a great amount of fat. Some subjects have undoubtedly been fat, and have lost it, perhaps acutely, from marasmic diseases. But in both obese, cachectic, and emaciated subjects the point in common is probably a *weakened musculature*, this helps us to see the chief cause in the *ratio* between pressure from within and strength of the gut wall, rather than in a great increase in the one or a serious defect in the other. Both conditions must frequently occur without diverticulum-formation resulting.

Keith, who regards the diverticula as true evaginations of the mucous membrane through the muscular coat, the result of high intracolonic pressure, has drawn attention to the (tonic) contraction of the tænia in the segment of the gut where the diverticula occur (*Fig. 274*). As a result of this, he says, the colon is thrown into circular concertina-like folds, which so deeply invade the lumen as seriously to obstruct the forward movement of the faeces. This condition is present in some specimens (85), but not by any means in all (we have observed it several times), but the observation is of great interest. From this

it might appear that the tonic muscular contraction is primary, and constipation the result of it. The point is worth further study. He finds also irregular contraction of the circular coat,

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and concludes that the diverticula are produced at weak spots (among which are those where the vessels perforate) in the musculature, as the result of the irregular and sustained contraction above noted.

It is not impossible that a congenital predisposition to diverticula may occur. The suggestion was long ago made (Hale White, Lafite-Dupont) that the condition is a representation of that found in some of the lower animals. The researches of Keibel on the so-called epithelial 'pearls' in human and other embryos are of interest in this direction. Hedinger thinks they stand in causal relation to some, if not all, diverticula; but much more investigation is required before any certain conclusions can be drawn.

A great argument against the congenital view has always been the fact that most cases occur at an advanced age. Up to 1908 the youngest recorded age was twenty-two (Fiedler (59), *Figs. 275 and 276*), and many writers have even denied the possibility of a much earlier occurrence. Senility certainly favours the condition, and Hansemann showed that diverticula could be produced experimentally in senile intestines, but not in those of young subjects under similar conditions. *Fig. 277* gives the age-incidence diagrammatically. But if this incidence is corrected to show the proportion to the population surviving at the various ages, it shows a curve steadily rising with advancing age (*Fig. 278*). We think the frequency of the condition in advanced age is

such that it may be used with some confidence in differential diagnosis. The existence of the condition is open to great doubt under thirty-five, and under thirty no specimen should be labelled diverticular unless diverticula can be certainly demonstrated. *Fig. 279* is from a patient six years old. It may

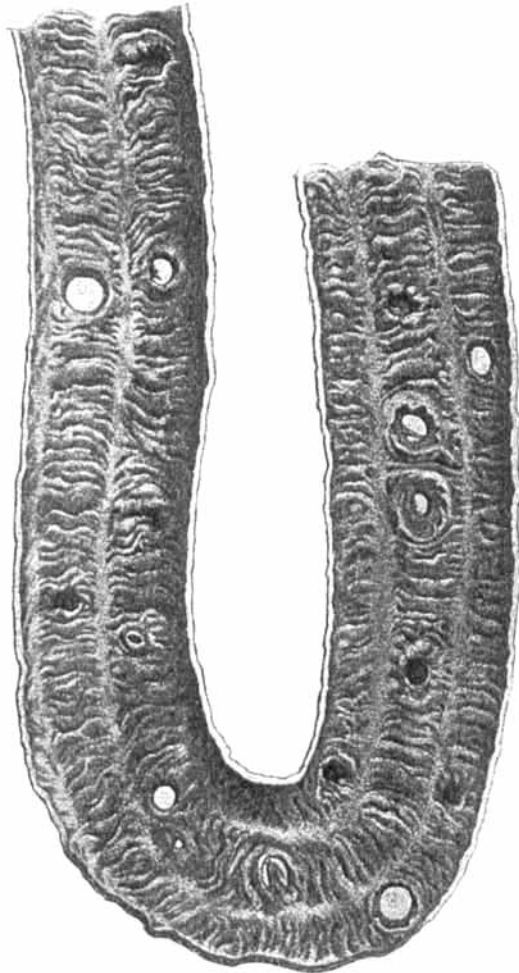


FIG. 276.—Portion of the sigmoid in a case of sigmoid diverticulitis in which multiple perforations had taken place. The orifices are circular, and show the relics of the attenuated mucosa around their margins. Some of the diverticular orifices are seen to be plugged with faecal material. (*Case 60.*)

now be taken as certain that cases in early life, though extremely rare, do occur.\* But even here the bearing of the *ratio* above defined cannot be lost sight of.

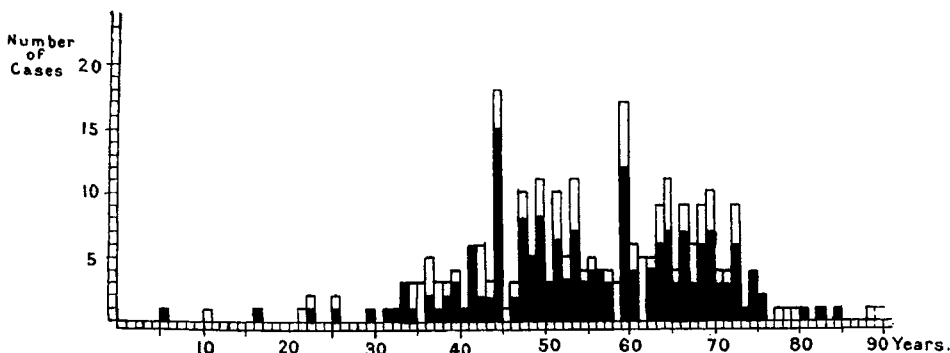


FIG. 277.—Diagram to illustrate the number of cases of diverticula which have been met with at successive ages. The black areas represent male cases, the white areas indicate female cases. The chart shows the greater incidence of the disease between the ages of 45 and 60. The greatest number of cases met with at 45, 50, and 60 is of interest.

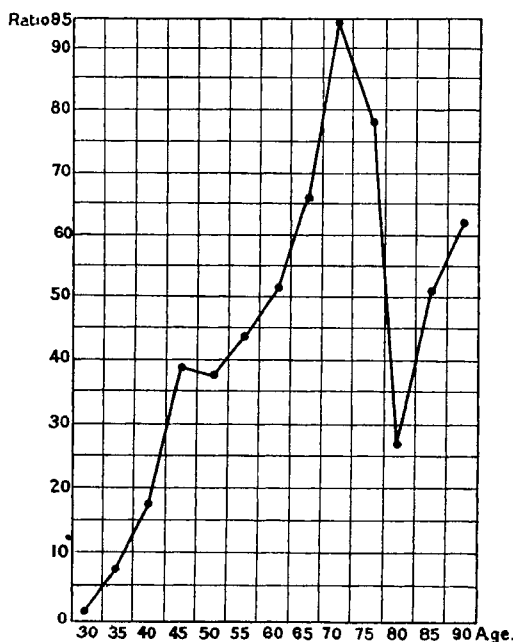


FIG. 278.—Diagram constructed from the same figures as the preceding, making the necessary corrections to bring out a nearer approximation to the absolute frequency of the occurrence of diverticula in relation to the population living at each successive age.

\* We think it worth noting that a number of cases recorded in early life are unconvincing, in that their diverticular nature is not proved. Particularly so is this in Ashhurst's and Ransohoff's series, though the latter observer does not himself claim them as more than examples of *sigmoiditis*, for this reason. Incidentally, this observer says our 1908 series gave only 1 case under forty. This is incorrect, it included 5 cases under forty.

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The determination of the site of the diverticula in the circumference of the gut wall has been the subject of much research. The contrast between the site of election in the small intestine (at the mesenteric attachment) and in the large intestine (away from it, often into the appendices epiploicæ) has been stated. Many 'distention' experiments have been resorted to (Heschl, Good, Hansemann), but the results are not very clear or convincing. As Patel says, the results thus obtained are of very little value, because the dead intestine behaves differently from the living, and would be expected to react quite differently to an acutely distending pressure than to a pressure operating gradually over a long period of time.



FIG. 279.—Drawing of a piece of colon obtained from a boy, age 6. In one of the haustra are seen two diverticula, one on either side of the anterior taenia. (Hartwell and Cecil's case.)

Graser emphasized the tendency for the evagination to occur at the 'vessel-holes' in the gut wall, i.e., the points at which the vessels perforate the gut. He also found a causal factor in chronic venous engorgement, stating that patients suffering from diseases causing this (especially a *variable stasis*, e.g., in cardiac conditions) were more liable to diverticula. But quite contradictory, and, to our view, convincing, statistics have been published (Sudsuki), and we do not think there is more in the point than that the 'vessel-holes' are *loci minoris resistentiæ*; any such locus, from whatever cause arising (e.g., fat appendices epiploicæ), may determine the site of diverticulum-formation.

### III.—SECONDARY PATHOLOGICAL PROCESSES.

Once the anatomy of the diverticulum has been appreciated, together with the tendency to faecal retention, the secondary changes may almost be anticipated on theoretical grounds. When these pathological processes are in their turn appreciated and understood, the varying clinical types of case may be deduced and predicted from a consideration of such anatomical and pathological details.

This sequence of events we have endeavoured to summarize in the schema Section IX (q. v.). This, which may be elucidated fully by the text, aims at giving a conspectus of the whole series of pathological accidents and the

clinical consequences which follow, in many cases, from the presence of these diverticula. The table conveys at a glance a better impression of the number, complexity and inter-relationships of these various secondary results than many pages of description would do. It further makes it easier to insist upon, not only the desirability, but the absolute necessity of classifying diverticulitis as a primary pathological entity, with varying clinical syndromes. Only in this way will progress be made in separating this condition from, say, other causes of sigmoiditis and pericolicitis, etc. It is certain that the diverticulum is a primary condition, but lack of appreciation of this has led authors to regard the formation of diverticula as secondary to the (e.g., hyperplastic) inflammatory process.

Once a diverticulum has formed, it tends to undergo *progressive enlargement*. This is clearly inevitable as a result of even normal pressure within the bowel. It has been pointed out that as a rule no great size is attained, the chief reason probably being that one or more of the secondary processes about to be described supervenes and interferes with what would otherwise be the normal development of the diverticulum. Another reason is that in many instances the diverticula form so late in life that there is time neither for the attainment of considerable size nor the development of any serious degree of secondary inflammation. It is some confirmation of this that the average size attained by diverticula of the small intestine is greater than that of the large bowel. As the diverticulum enlarges the muscular coat tends to disappear, if it was present in the earliest stage of evagination.

Owing to the usual shape of the diverticulum—a sac with a narrow neck—the faecal contents tend to be retained, and this retention occurs usually in that portion of the bowel where the fluid content tends to be lessened. Consequently they frequently become hard, often forming concretions of stony hardness. This process leads to certain mechanical results, the main one of which is perforation. The tip of a concretion, often projecting slightly into the lumen of the bowel (*Figs. 270, 272*), is of course frequently in contact with the main faecal mass. It is not surprising, therefore, that several cases of sudden ('pistol-shot') perforation have occurred, while straining at stool (20, 55), or as a result of any sudden trauma (8, 24, 32).

But the most frequent secondary change is an inflammatory process, of varying type and intensity. This is always due to the mechanical irritation of the faecal mass, combined with the presence of micro-organisms, the rich and variable flora of the intestine providing an abundant supply for the nidus formed by such faecal contents. The sacs are very imperfectly drained, not only from the usually narrow neck, but sometimes also owing to an inflammatory or toxic oedema of the lipped orifice. In this way is determined either an acute or gangrenous inflammation of the diverticulum, or a latent or chronic one. Sometimes the inflammation is of the slightest, yet is enough, in a thinned diverticulum, to permit the passage of micro-organisms to the peritoneum, setting up an acute or general peritonitis.

In addition to an inflammation of the sac wall, there is very liable to occur a low-grade, chronic, hyperplastic inflammation *around* the diverticulum. This process may become very marked, and may surround a section of the gut with a constricting band of fibrous tissue, leading to tumour formation by its

hyperplasia and stenosis of the gut by its contraction. To this the term *peridiverticulitis* has been applied. It is due largely to the low inflammatory reaction induced by a leaking into the tissues of bacterial toxins through the damaged sac walls; possibly, in addition, to the passage of a certain number of the organisms themselves, of a low virulence. Precisely comparable to this process is that which occurs in the hyperplastic tuberculosis of the cæcum. So close is the resemblance, that certain observers (e.g., Kidd) have described cases of chronic perisigmoiditis of this type in the sigmoid flexure (where this form of tuberculosis very rarely occurs) without anything like adequate evidence of the presence of tuberculosis. In discussing the differential diagnosis of the condition at length, he fails even to mention the possibility of diverticula in this section of the bowel. We think it not too much to say that all cases of so-called hyperplastic tuberculosis of the sigmoid flexure are probably examples of peridiverticulitis unless the tuberculous nature of the lesion is reasonably proved. Put another way: an uncertain peridiverticulitis is much more probable than an uncertain hyperplastic tuberculosis of the sigmoid flexure.

#### **Histology and Bacteriology.**—

The microscopic structure of the wall of the sigmoid diverticulum would call for little comment, were it not for the association of inflammatory lesions of noteworthy type in the peridiverticular tissues. The structure of an uninflamed diverticulum is most conveniently observed in a diverticulated appendix, owing to the fact that the whole lesion can be observed in a single microscopic section. Specially full investigations in material of this kind are to be found recorded and illustrated in Lejars and Ménétrier.

*Fig. 280* shows the essential features in an actual specimen prepared with iron hæmatoxylin and van Gieson. The increased size of the appendix lumen is produced by a yielding of the mucosa, and is associated with a loss of muscular tissue. The lymphoid follicular tissue (which comes out a greyish black) is present in the evaginated portion, and does not appear to be the seat of undue formative activity such as might indicate bacterial infection of the tissue. *Figs. 281 and 282*, which are taken from Hartwell and Cecil's study, show the absence of muscular tissue at the fundus of diverticula very clearly. The sigmoid diverticula, as compared with



FIG. 280.—Low-power view of a transverse section of a diverticulum of the appendix. Iron hæmatoxylin and van Gieson preparation. The specimen shows a pouching of the mucosa through the weakened outer coats. (By kind permission of Dr. M. J. Stewart.)

the appendix specimens, have a much attenuated wall, or apex, and the lining mucosa is not only stretched out, but the glands or crypts tend to disappear. Nor is there any thickening of the serosa to be observed. These authors lay special stress upon the existence of a vein and artery of conspicuous size in the wall of the diverticulum (*Fig. 282*).

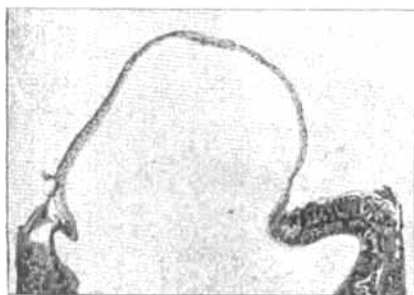


FIG. 281.—Microscopic section through a diverticulum, to show the muscularis terminating suddenly at the neck of the sac. The other coats are much attenuated. (After Hartwell and Cecil.)

Whereas the controversy appears to have been focussed on the question of the 'true' or 'false' character of these diverticula, with the result that the microscope has been used solely for the decision of this matter, yet the more profitable purpose of histological study lies in ascertaining the *relation between tissue structure and bacterial invasion*. The extreme thinning of the wall suggests that micro-organisms can readily pass through and affect the peritoneal coat. On the other hand, many cases are recorded in which the adjacent bowel wall is extremely thickened, the

thickening overshadowing the atrophic diverticular cupola and concealing it from view. While this phenomenon may be passed off as a safeguard of nature, it is necessary to realize that the difference in structure in these two extreme types of case is due to a *difference in the bacteriological process at work*.

We are at once reminded of the varying histological pictures which characterize appendicitis, where careful study proves conclusively that certain types of histological change are related to the existence of certain bacteriological conditions—it is a case not merely of type of organism, but it is a question of the chemical composition of the contents of the appendix at a given

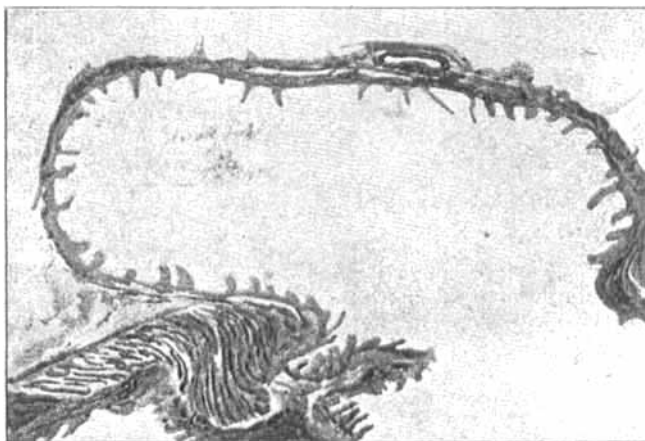


FIG. 282.—Microscopic section of a diverticulum, to show the artery and vein in the wall of the sac. (After Hartwell and Cecil.)

moment, as well as of the existence of aerobic or anaerobic media. In some sense the medium is always anaerobic, but, in a particular sense, anaerobiosis may be secured by mechanical obstruction to the outflow of the contents,

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aided by a preponderance of venous blood in the walls of the tissue against which the organisms are constantly encamped.

It will be evident that a histological study of diverticulitis should ignore none of the factors which play so potent a part in appendicitis itself, and that, as a fact, all the complications met with in the appendix are equally feasible, and are equally realized, in the diverticulum. The question is, therefore, not one of the presence or absence of muscularis in the wall, but of the physiological potency of the lining glands, the existence or not of lymphoid tissue, the ease with which protective cell-proliferation can take place in the walls, and the nature of the cell-complexes that arise in those cases in which the sigmoid walls are massively thickened.

Schreiber's descriptions of the structure of the diverticula may be referred to, inasmuch as the necessary data appear therein, serial sections having been carried out. From these it appears that the lining glandular epithelium is apt to be in a state of catarrh, thus accounting for blockage of the lumen by a mucoid plug in some cases. Lymphoid tissue is not described, and, as is well known, it is not conspicuous in this portion of the intestine. There is frequently undue dilatation of the vessels in the atrophic mucosa, especially of the veins—a condition which is favoured by the pressure of retained faecal matter, besides providing a link in the vicious circle of this lesion, in that the venous blood favours further atrophy of the tissue-elements, and provides favourable conditions of relative alkalinity for the development of micro-organisms.

The question of permeability of the intestinal wall to the bacterial inhabitants has received careful attention at the hands of Helmberger and Martina, who show that immunity from bacterial invasion is ensured by, but is not dependent on, the presence of a perfect mucosal barrier. Extensive necroses may occur, and yet the deeper tissues resist microbic multiplication. The character of the lymph lying in the cell-clefts of the submucosa has therefore some importance, and in some unexplained way the muscular fibres exert a protective influence. From the fact of a mention of persistence of muscular irritability being a condition for such resistance, the suggestion arises that the electrical potential of the juices round the muscular fibres has something to do with the resisting power of the tissues in question. The fact that the muscular fibres become rapidly attenuated in the diverticular wall indicates that we have here a loss of one of the natural barriers. The presence of bacteria amongst catarrhally necrotic epithelial cells of the crypts does not indicate a dangerous invasion; it is only when there are necrotic foci in the submucosa, and when the remaining coats are reduced to mere areolar tissue, that one may expect bacterial multiplication of importance. A change of reaction in the lymphatic clefts here plays its part.

The important factor of the character of the contents of the intestine requires to be borne in mind, inasmuch as some observers note that abundance of cellulose-residues in the faecal material is associated with bacterial flora which differ in type from those met with where the faecal material is flowing by with suitable rapidity. Herein lies a means of therapeutically modifying the danger from bacterial perforation of the diverticula, since certain Gram-positive flora are associated with the most serious tissue-changes.



There remains the question of the histology of those cases in which the intestinal wall is greatly thickened. There can be no doubt that the intense hyperplasia which has been noted by many authors is the result of permeation of the tissues with toxins rather than with the organisms themselves. The limitation of the hyperplastic process to the deeper parts of the submucosa, and in some instances (45) to the subserosa itself, and the remarkable development of a dense felt-work of young fibroblasts, which appear to draw together even widely separated tissues (see *Table I*, showing the multiformity of adhesion-formation), strongly call to mind the parallel picture which is met with in *linitis plastica*. In each case we have a histological picture in which delicate connective-tissue fibrillæ run through the bowel-wall in all directions, leaving minute spaces occupied by medium-sized cells, round or polygonal in shape, and having a regularly large oval nucleus with scanty nuclear markings (very amblychromatic and with a very clearly defined nuclear membrane). In other places we should meet with ovoid and feebly-staining young fibroblasts, whose nuclei are relatively large and finely granular and feebly-staining. Intermingled, therefore, would be lymphoid cells of the size of mesolymphocytes, and gradational forms towards the fibroblastic type would be expected.

This picture is that of a slowly formative process akin to that seen in granulomatous hyperplasia, and dependent on a noxa which stimulates abnormal proliferation of potentially hæmopoietic cells to a slight extent, but irritates the purely stromatic cells to a very marked degree, thus inducing an entire loss of balance in the ultimate histological picture. We therefore consider these changes indicative of some chronic infective agent whose nature should be determinable, and detect in these considerations a reason for the close mimicry, to the naked eye, of the process regarded as scirrhus carcinoma in annular constrictions of the sigmoid.

The granulomatous nature of the thickening is emphasized by the occurrence in the tissue of multinucleated cells, whose appearance recalls that of the giant cell of tubercle. Whereas some authors lay stress on the appearance of such cells as evidence of the tuberculous nature of a colonic thickening (Kidd), it would be easy to show that giant cells in the midst of a granulomatous mass, without the natural histological architecture characteristic of tubercle, are to be interpreted in an entirely different sense. This is well illustrated by the fact that such cells occur in *linitis plastica*, in which no suspicion of tubercle has been advanced by any author; and it is even more strikingly illustrated by the fact that, in lymphogranulomatosis, not only do we have giant cells, but we have even acid-fast organisms present, capable of producing similar lesions by inoculation—and yet the disease is not tuberculous.\*

The opportunity may be used for emphasizing the fact that a histological picture is not to be interpreted according to the nomenclature of a disease, but that the cellular composition of an inflammatory mass is merely the

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\* The investigations of Wree point to lymphogranulomatosis being related to mycobacteria of the diphtheroid group. It must not be forgotten that bacterial symbioses may lead to incorrect deductions as to the precise nature of a given histological lesion.

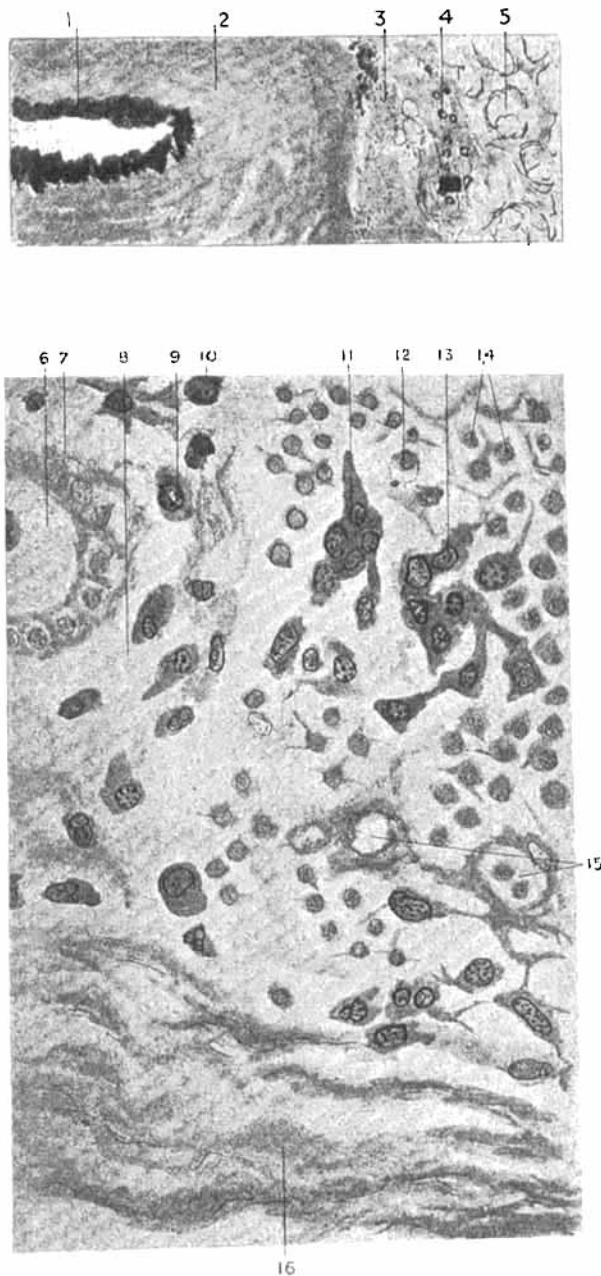


FIG. 283.—MICROSCOPIC CHARACTERS OF PERIDIVERTICULITIS.

a. Schematic representation of the whole section. 1, Position of the mucosa of the diverticulum, 2, Surrounding laminated fibrous tissue of great density (very few nuclei present) 3, Peripheral tissue containing numerous transverse sections of arterioles, 4, Arterioles, surrounded by inflammatory cell-infiltration, 5, Areolar tissue of the subserosa, which also shows some inflammatory cell-infiltration. The small oblong marked area shows the portion represented under high magnification in b.

b. Cellular details of a typical area in the infiltrated perisigmoid tissue. 6, Lumen of capillary, 7, Walls of capillary, formed of a single layer of cuboidal cells 8, Perivascular lymphatic clefts, 9, Perithelial connective-tissue cell, 10, Mast cell (parental type) 11, A clump of cells with dusky-staining cytoplasm, suggesting the appearance of a giant cell under the  $\frac{1}{2}$  lens 12, Vacuolated lymphocytoid cell, 13, Dusky (plasmoid) connective-tissue cells, 14, Lymphocytoid cells such as constitute a large part of the inflammatory cell-infiltration, 15, Two young capillaries, 16, Fibrous strands among which nuclei are absent.

The drawing shows a portion of the scene in which the phenomena of peridiverticulitis take place, and indicates some of the actors in the processes of which the bulk of this paper is the theme.

(From a preparation in the Museum of the Medical School, Leeds. Oil-immersion lens, ocular 4.)

reflection of the existence of certain chemical substances gathered together, or in process of formation, within the confines of the affected area. If there are insoluble substances of a certain type present, the adventitial connective-tissue cells will certainly gather together round them, and may, in consequence, provide a giant-celled lesion. On the other hand, this type of substance may not be present, or it may be adequately dealt with by smaller wandering cells, or by the catalytic oxidative processes of which eosinophile cells are an occasional result. There can be no doubt that the complexity of the picture, be it in the submucosa or in the subserosa, is largely dependent on the type of organism present—which is another way of saying that it is dependent on the chemical groups to which the bacterial excreta belong. Those cases in which the mucosa itself is intact, and yet organisms have penetrated into the recesses of the tissue, possibly *via* the gland-crypts, are such as are enabled to manifest a local development of organisms in the submucosa, at a slow rate, and with the production of conditions which bring about a marked hyperplastic process in the tissue-elements of the region.

The question of the superaddition of carcinoma now arises. In the consideration of this question it is advisable to bring out clearly the possibility of two entirely distinct types of new growth. The recorded cases are concerned with adenocarcinoma, but the symptoms and naked-eye appearances of stenosed sigmoids belong rather to scirrhus carcinoma, of whose presence there is no record. In one of Kidd's cases there was a polypoidal formation at the point of the constriction of the sigmoid, which had led to a diagnosis of carcinoma by a cursory observer, and the absence of carcinoma was only established by the special care which the author (Kidd) expended upon the case. This would be quite in conformity with our own experience. It is not until all questionable tissues are microscopied as a routine procedure, that one realizes that all strictures of the colon are not malignant. Even a polypoidal condition of the gut may be only the expression of increased irritability at the edge of a diseased area, causing the glandular elements to overgrow in much the same way as the epidermal papillæ may hypertrophy into cancer-simulants at the edge of a chronic ulcer. However, the development of an adenocarcinoma at one extremity of a diverticulated gut may be met with, and would be favoured by the existence of the same noxious agent as at other times incites a hyperplastic process in the bowel wall.

There remains the question of the development of a scirrroid constriction of the bowel. Where this occurs, the stricture is usually annular, and is very often remarkably limited in extent, producing a constriction exactly as if a piece of stout string had been tied round the bowel. The diagnosis of such a form of carcinoma from the blood-cells proves to be impossible, the changes described as occurring in the nuclei of the leucocytes, as well as in the cell-bodies, and the occurrence of parasitiform structures in the large mononuclears, fail completely. This is not the case with adenocarcinomata, in which the blood changes provide a sure means of diagnosis.\* Nor is the histological structure any more typical. The amount of fibrous tissue is extreme, and in many respects such a tissue bears a singular resemblance to what might be

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\* BRITISH JOURNAL OF SURGERY, 1916, Jan., p. 506.

called an end-stage of the hyperplastic process already discussed. The conclusion is that there must be a very close relation between perisigmoiditis and scirrroid stenosis of the sigmoid, and we should feel a strong incentive to investigate these cases histologically with very great care. The absence of blood-changes itself suggests that this form of carcinoma is of different genesis, and the histological structure, when interpreted (as advocated elsewhere\*) in terms of the fundamental (chemical, structurally specific) agencies at work in the tissue, may be expected to furnish information of more than academic interest.

#### CLASSIFICATION.

We can usefully divide these secondary pathological processes into two groups (a) *Mechanical* (mainly), and (b) *Inflammatory* (mainly). Both processes may go on in the same case, just as there may be present together varying types of inflammatory reaction.

##### A. MECHANICAL.

1. Formation of *faecal concretions* in the diverticula.
2. *Torsion* of the diverticulum.
3. Lodgement of *foreign bodies* within the diverticular sacs.

##### B. INFLAMMATORY.

1. *Diverticulitis*: (i) Gangrenous; (ii) Acute, (iii) Subacute; (iv) Chronic; (v) Latent.
2. *Passage of organisms* without perforation.
3. *Peridiverticulitis*—chronic proliferative inflammation with tendency to stenosis of the bowel.
4. *Perforation* of the diverticula, giving rise in particular to. (i) General peritonitis; (ii) Local abscess, (iii) Fistula, especially into the bladder, (iv) Suppuration in a hernial sac.
5. *The formation of adhesions*, especially to. (i) The small intestine; (ii) The bladder; (iii) The female genitalia.
6. *Chronic peritonitis*, local.
7. *Chronic mesenteritis* of the sigmoid loop.
8. *Metastatic suppuration*.
9. The development of *carcinoma*.

These various processes may now be considered somewhat more in detail.

##### A.—MECHANICAL.

1. **Formation of Faecal Concretions.**—The tendency to this is the rule: the explanations have been given. There is no doubt that the presence of a more or less rough-surfaced concretion in a thin-walled sac is liable to set up a mechanical irritation, easily passing into inflammation as a result of ever-present organisms. This irritation is undoubtedly increased, and its results enhanced, by the mobility of the sigmoid loop and the movements of other coils of intestines about it. Such concretions constitute *points d'appui* for the action of force from within, readily leading to perforation.

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\* *Biology of the Blood-Cells*, chap. vii. (1913, Wright & Sons, Bristol.)

**2. Torsion of Diverticulum.**—This again is the result of mobility acting on a more or less pedunculated sac. The phenomenon occurs usually in the case of a diverticulum into an epiploon, particularly when this latter structure is large and gravid with fat, as is so often the case. This torsion may lead to an inflammation, often acute, of the diverticulum (a true epiploic appendicitis), or it may lead to gradual obliteration of the lumen at the neck of the sac. In this way the neck of the sac or epiploic appendage may become

elongated as a result of continual movement and traction, and may be finally separated from the gut wall and form a *loose body in the peritoneum*. Fig. 284 is a striking example of this process, and shows a diverticulum containing a stony-hard concretion; the neck of the sac is no longer permeable, and is lengthening. In this case a loose body, similar to the one in the process of becoming separated, was actually found lying in the pelvis.



FIG. 284.—Diverticula of sigmoid. The fat has been partly dissected from the outer surface of the gut, showing several pouches. *a*, Large pouch containing a calcareous concretion, with a thin fibrous pedicle in the process of separation to form a loose peritoneal body. A similar concretion the size of a bean was free in the pelvis. (Case 64.)

### 3. Lodgement of Foreign Bodies.

Bland-Sutton has laid special stress on this complication, and Fig. 271 is reproduced from his drawing. He has observed and recorded a number of cases of foreign bodies (straw, metal, etc.) being found in the sac, and being the source of mischief; causing either an acute diverticulitis or a perforative peritonitis (66, 67). Possibly sharp foreign bodies may enter a fat appendix without the aid of a pre-existing diverticulum, but the latter is usually the predisposing cause of the lodgement.

## B.—INFLAMMATORY.

### 1. Inflammation of the Wall of the Diverticulum: Diverticulitis.

—This may occur in every degree of acuteness, but in the *acute* and *gangrenous* forms it leads to one of the most characteristic clinical syndromes. All the familiar phenomena of

acute appendicitis on the right side of the abdomen are reproduced in the left lower quadrant as a result of this diverticular inflammation. It would be expected that such an inflammation might either subside completely, cause an acute general peritonitis or a localized abscess, or merely leave adhesions. All these results are in fact common, the first, of course, is established mainly inferentially.

*Chronic inflammation*, apart from merely causing local chronic peritonitis or leading to adhesions, frequently occurs in association with the hyperplastic

peridiverticulitis, and is a predisposing cause to rupture, especially of the sudden and unexpected type, resulting from any form of trauma.

*Latent ulceration* of the diverticula is not very infrequent; it is so called because the inflammation is of so low a type as not to give rise to any symptoms. Examples are usually found unexpectedly in autopsies on old and debilitated subjects.

**2. Passage of Organism without Perforation.**—A few cases of acute peritonitis have been recorded (Loomis, Rixford, Cameron and Rippmann) in which the source was undoubtedly an inflamed diverticulum through the damaged walls of which organisms had passed into the general peritoneal cavity. This happening has been proved in connection with the vermiform appendix, and might therefore be expected to occur occasionally in diverticulitis. All recent investigators have come to the conclusion that organisms do not so pass unless the intestinal wall be damaged or inflamed. In the case of a diverticulum, not only is there usually a damaged and inflamed wall, constantly irritated by faecal friction and pressure, but the wall itself is greatly thinned. The point is a practical one, for, like that of the existence of diverticula in general, it must be borne in mind by the operating surgeon when faced by a general peritonitis of unknown origin.

**3. Peridiverticulitis.**—This is a chronic proliferative inflammation, tending to thickening and subsequently to stenosis of the bowel. It is one of the most characteristic results of chronic inflammation of a number of diverticula more or less closely aggregated. Its method of production has already been described (see under **Histology**, p. 481). The result is that there is formed an annular band of greater or less length, which leads in the first place to thickening and palpable tumour formation, and later, by inevitable contraction of the newly-formed fibrous tissue, to actual stenosis of the bowel—which may be almost cartilaginous in its density (45, 47). McGrath



FIG. 285.—Peridiverticulitis, with great thickening of the gut wall, causing stenosis and simulating carcinoma, for which it was mistaken when resected at operation. *a*, Thickening due to fibrosis. (Case 72.)

in 1907 emphasized the fact that this chronic peridiverticulitis is the most frequent and important initial change following infection through the diverticulum, and that it may be present with little or no discernible inflammation in the mucosa (73), in 19 cases quoted by him such inflammation varied from a mild to a moderate degree, and in 2 cases the mucosa appeared intact. *Figs.* 285, 286, and 287 are good examples of this chronic hyperplasia. But side by side with this inflammatory change around and outside the diverticular sac there is usually some degree of chronic ulceration of its walls, and this may,

and often does lead to perforation and the formation of one or many abscesses. Being formed under such chronic conditions, these abscesses do not so often give rise to sudden rupture into the general peritoneal cavity, being mostly shut off by adhesions, but they form pockets of pus which still further add to the density of the inflammatory mass, by increasing the hyperplasia and adhesions. These abscesses may of course tunnel and rupture in various directions, sometimes the intestinal wall is riddled with submucous fistulae (8, 57, 58).

It is not difficult to premise that the discovery of such a matted, hopeless-looking mass, following upon symptoms of chronic or subacute intestinal

obstruction, has again and again led to the incorrect diagnosis of malignant disease (68, 70). It is safe to assume that prior to the knowledge of the occurrence and potentialities of the diverticula, almost every one of such tumours was regarded as malignant. In consequence, surprising 'cures' were noted from time to time, and to-day many specimens repose on museum shelves wrongly labelled.

One important result of this peridiverticulitis is the way in which it modifies the anatomy of the diverticulum itself. The hyperplastic thickening may be extreme, as much as two inches having been recorded, and a half to one inch is not uncommon.

As a result, the neck of the diverticulum is greatly elongated and narrowed, and its direction made more or less oblique. The sac itself is usually found near the outer layer of the inflammatory mass, and *Figs. 285 and 287* are typical examples of this. It is this feature in particular which has so long delayed the recognition of the diverticular origin of these cases. The small orifices in the intestinal mucosa are concealed beneath the folds of the rugose mucous membrane, and unless careful sections are made through the fibrous mass no evidence of the diverticular nature will be forthcoming. They should be examined very carefully in the way described by Wilson (see under **Carcinoma**, p. 494).

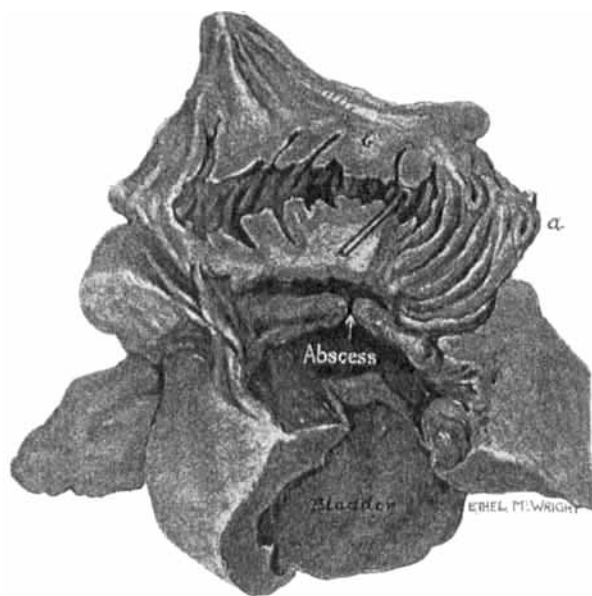


FIG. 286.—Chronic peridiverticulitis, with thickening and stenosis of the gut wall, simulating carcinoma. There was adhesion to the small intestine, causing chronic obstruction, and to the bladder. There were several small chronic abscesses in the neighbourhood, the largest between the bowel and the bladder. *a*, Lipped orifice of a diverticulum; *b*, Thickening of the gut. (Case 70.)

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We have found thickening in 24 per cent of our series of 324 cases, and actual stenosis of the lumen is noted, or is to be inferred from the symptoms, in 14 per cent. We shall return to the point later in considering the clinical features.

**4. Perforation of the Diverticulum** (28.4 per cent), giving rise to general peritonitis, local abscess, fistula or suppuration in a hernial sac.—From what has been said it follows that perforation of the diverticulum must be a relatively common occurrence. The first case was noted by Jaboulay in 1898.

The results of perforation differ according as the diverticulum so perforated is shut off from the general peritoneal cavity, or not. If not, a *general peritonitis* is extremely probable, unless, as happens in the case of the appendix, the peritonitis is localized by newly-formed adhesions. The perforation may be absolutely sudden and due to mechanical causes, of which we later quote examples. Many cases of diverticula first reveal their presence by the supervention of an acute general peritonitis.

But owing to the frequently chronic nature of the preceding diverticulitis or peridiverticulitis, the sac which is about to perforate is frequently shut off by adhesions, and only a *local abscess*, often small and latent, is formed. This may persist, adding to the connective-tissue proliferation and consequent tumour formation, and giving signs such as fever and leucocytosis. It may be cured spontaneously by discharge through the bowel, externally, or into the bladder.

One very important difference between the carcinoma-simulating mass of peridiverticulitis and true carcinoma-formation is the absence in the former of ulceration or involvement of the mucous membrane. Seldom is any exception to this noted, and when present, it may occur as the result of re-entry of fistulæ and abscesses into the bowel; but it practically never leads to such an appearance of the mucosa as would deceive an experienced morbid anatomist. This makes it the more remarkable that so many specimens have been wrongly labelled and put into museums in the past. Many recent observers (Moynihan, Keith and others) have commented on this.

Often a *number of small abscesses* may exist, and the possibility of such 'pocketing' has to be taken seriously into account in an operation that might otherwise be performed aseptically.

**Fistula Formation** (19.8 per cent).—This is a not uncommon result of perforation of a diverticulum which has previously become adherent to an adjacent structure. Apart from the submucous fistulæ which sometimes

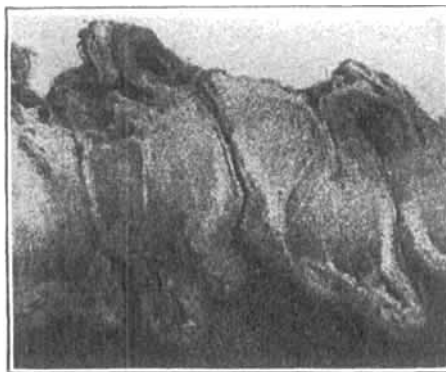


FIG. 287.—Great peridiverticular thickening, with high-grade stenosis. An elongated and tortuous diverticulum is well seen. (Case 59.) (After W. J. Mayo.)



occur in the intestinal wall, there have been noted. (a) Sigmoidovesical fistula; (b) Rectovesical fistula, (c) Sigmoido-uterine fistula (16); (d) Anal fistula, (e) External fistula.

Of these by far the more important clinically are the bladder fistulæ, and these will be fully described later.

Anal fistula has only been recorded once, by C. Mayo, as the result of a rectal diverticulum, which had opened on the skin near the anus. He says such cases would fail to become cured after repeated operation because the high internal opening of the fistula preserves the mucous lining of a portion of its wall.

A fistula between the sigmoid and the Fallopian tube has been noted by le Jemtel (16), and is an example of the extent to which the pelvic viscera can be implicated secondarily to diverticulitis.

*Perforation into a hernial sac* is an interesting though rare happening (61), cases have been recorded by Klebs and Stierlin.

**5. The Formation of Adhesions.**—At first sight it may seem a little unnecessary to make a special enumeration of this tendency, which, it is obvious enough, will be frequently encountered. But the matter is worthy of a little careful study, for some of the results of the adhesion tendency are striking and important. In a series of 280 cases of diverticula the following adhesions were noted —

*Table I.*—DISTRIBUTION OF ADHESIONS RESULTING FROM DIVERTICULA (280 CASES).

<i>To Alimentary Tract</i>		Douglas's pouch	3
Small intestine	13	General	6
Appendix	2	Uterus	5
Cæcum	1	Left adnexa.	6
Pelvic colon	1	Right adnexa	1
Omentulæ	3	Broad ligament	1
Mesentery	2	<i>Left Iliac Fossa</i>	8
Omentum	2	<i>To Inguinal Region</i>	3
Pericolic	7	<i>To Parietes</i>	
Rectum	4	Anterior	5
Pendiverticular	3	Posterior	5
		Not stated	1
<i>To Bladder</i>	33	<i>Forming Bands</i>	5
<i>In Pelvis</i>		<i>General</i>	14
Posterior pelvic wall	5	<i>Site not specified</i>	9
Pelvic brim (left)	2		
Pelvic brim (right)	1		
Sacrum	2		

*Adhesion to the small intestine* has caused acute intestinal obstruction, in one case a long band, extending from the tip of a diverticulum-containing epiploic appendage to the cæcum, was the cause of the strangling of a loop of bowel eight feet in length (84).

*Adhesions to the bladder*, apart from the important complication of fistula, are frequent, and may give rise to symptoms referable to that viscus.

*Adhesions to the pelvic viscera* are being increasingly noted, and their importance is such that we have now included a 'pelvic syndrome' as one of the clinical presentations of diverticulum formation. There is no doubt of

the pathological relationship of the sigmoid flexure to the pelvic viscera, and future research in this branch of the subject, which is comparatively new to the literature, will help to determine with what frequency the diverticula play a part in pelvic lesions.

**6. Local Chronic Peritonitis.**—This, a pericolitis (*sinistra*), may be an important and the principal result of the presence of diverticula. It may be due to the leaking of toxins through the damaged sac-walls, and will tend naturally to the formation of adhesions, one result of which may be the tethering of the sigmoid loop in abnormal places, e.g., on the right side, where this abnormal localization may give rise to trouble in diagnosis.

Chronic peritonitis of the sigmoid region is exceedingly common ; Byron Robinson found it in 80 per cent of males and in 85 per cent of females in 800 consecutive adult autopsies. Of course, only a small proportion of these cases are due to demonstrable diverticula, but a point that has so far been insufficiently investigated is whether small diverticula, such as would ordinarily escape recognition, may not be responsible. Many observers explain the frequency of adhesions here to irritation from contained faeces, especially in constipated persons. There is no doubt that the subject is one which merits further study. Pennington has particularly drawn attention to these peritonitic formations, and his article is well illustrated. The clinical importance lies in the fact that thereby the free movement of the sigmoid may be interfered with and sometimes kinkings and angulation may result, causing volvulus or other forms of obstruction, more or less complete.

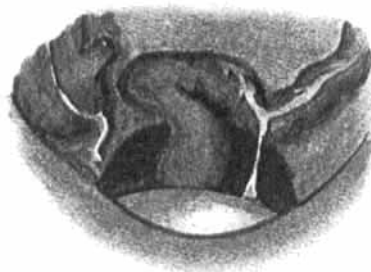


FIG. 288.—Diagram of the brim of the pelvis, to show an adhesion between the sigmoid and the pelvic floor, dragging the intestine down and allowing the production of a kink. A portion of the cæcum and appendix is shown on the left. The anterior portion of the diagram (unshaded) indicates the position of the bladder. (After Pennington.)

**7. Chronic Mesenteritis of the Sigmoid Loop.**—The occurrence of this condition has been noted for a considerable time ; Ries, in particular, has written about it, emphasizing its effects on the sigmoid loop. Other observers (Brehm, Riedel, Cignozzi) support his statements as to the various forms of twist, kink or volvulus resulting from the contraction of the mesentery, with the consequent distortion of the loop. This leads to partial, complete or recurrent forms of intestinal obstruction, and acute or chronic symptoms of various kinds.

The authors quoted above have all emphasized the importance of diverticulitis in setting up this mesenteritis, and Ries in 1896 wrote . “ It is safe to prophesy that this association of diverticular ulceration and mesosigmoiditis will be frequently discovered in the near future.” This has been verified by experience to a certain extent. A number of the diverticular cases have been complicated by various types of mesenteritis, and in some (27) the mesenteric lesion has been the more important one. But what has not been observed with any frequency is the type of case in which the mesenteritis, producing

the mechanical results described, has been proved to be of diverticular origin. This, again, opens up an avenue of research, as it is clear that many specimens of mesenteric kinking have not been examined specially as to the presence of diverticula. The observed cases of diverticular mesenteritis lead us to expect this origin more frequently than it has already been noted. Cignozzi has divided mesosigmoiditis into two varieties (1) Inflammation localized to the attachment line of the mesosigmoid, causing sclerosis and retraction of the colon in an angular position, (2) A circumscribed adhesive fibrosis of considerable extent, giving a special character to the lesion as (a) retracted mesosigmoiditis, or (b) fibrous adhesive perisigmoiditis. It is the last two varieties which are most usually encountered as the result of diverticulitis between and near to the sigmoid mesentery.

**8. Metastatic Suppuration.**—So far only one example of this has been recorded, by Whyte multiple abscesses in the liver resulted from a portal pyæmia originating in the septic diverticula (62).

**9. The Development of Carcinoma.**—The association of carcinoma with diverticula is a highly interesting and important one. To Hochenegg belongs the credit of recording the first case (63); in 1908 only three cases were recorded, Stierlin furnishing one (58), and ourselves a third. To-day we collect 11 cases, and much of interest has been noted in connection with them. It is suggested that the irritation and inflammation of the diverticula furnishes a special predisposition to the development of carcinoma, which would not have developed but for the presence of the diverticula. Wilson has devoted special attention to this, and Delore and Lambert have noted the association of cancer and sigmoiditis.

It has been demonstrated that carcinoma develops from the isolated epithelial areas in the bases of gastric ulcers; the same causation is highly probable in the case of primary carcinoma of the tip of the appendix. Undoubtedly segregation of epithelium occurs with frequency in inflamed diverticula. When the blood-supply of such islets is interfered with, the epithelium dies, but if it has an adequate blood-supply, proliferation is the rule, and an adenocarcinoma might result. Scirrhus carcinoma of the sigmoid, colon and rectum is frequently ring-like in character, and we suggest that this may in some cases be connected with a diverticular origin. This aspect of the subject has already been fully discussed (p. 486). We can only record the fact that the association of diverticula with carcinoma has been noted 11 times at least, and within a comparatively short period during which attention has been specially directed to it.

It must be pointed out that the detection of diverticula in a specimen of cancer is a very delicate business, and can only be satisfactorily carried out in the recent specimen. Wilson advises the use of a glass probe with a bulbous extremity of not more than one millimetre in diameter. The fresh surface of the carcinoma should be systematically and very gently searched with this. When the probe slips in to any depth, a diverticulum is probable, and if present may be demonstrated by slicing the mass in thin layers parallel with the lumen of the bowel, proceeding from without inwards. How often is this done? We agree with Wilson and others that it is certain that many cases have been overlooked, what proportion, a much more systematic series of researches must be left to determine.

## IV.—CLINICAL ASPECTS.

Enough has been written up to this point to emphasize the fact that a knowledge of diverticulum formation must be indispensable to a clinician, and the variety and complexity of the secondary pathological processes make it certain that the symptoms and clinical phenomena generally will be protean and confusing. At the same time the majority of cases conform more or less exactly to one or other of the clinical types which we now propose to describe.

In the schema we have drawn up, these clinical types are set out in the right-hand column. The way in which the various pathological conditions lead up to and determine them is indicated thus diagrammatically, and it seems to us that the more such a table of processes and their results is studied, the more will the clinical perspective be sharpened and cleared in regard to this condition.

Speaking generally, diverticulum-formation was discovered, rediscovered, and noted from time to time, by morbid anatomists, isolated examples of secondary processes were similarly noted. But the full significance of the diverticula was not realized and measured, and certainly the subject was not grasped as a complex but complete whole. It was not until clinicians, in this instance almost exclusively operating surgeons, found themselves confronted with lesions of an unusual type and abdominal happenings of a kind difficult to explain, that specimens were investigated with special care and the results recorded. Such records it is our task to-day, as in 1908, to collate and link up, and we feel that no excuse is needed for an attempt to present the clinical facts as completely as possible, and to harmonize them with the morbid anatomy which has already been described. In the preceding pages we have indicated the *lacunæ* still existing in our knowledge, and the consequent need for further research. Some of this can only be supplied by morbid anatomists, with their special opportunities, and we would once more enter a plea for an absolutely systematic examination of the whole intestinal tract in every autopsy. Physicians will be able to do much to solve the difficulties presented by cases of localized inflammatory processes in the colon (so-called sigmoiditis), presently to be discussed in some detail. But it is to operating surgeons that we must continue to look for most of the new material and fresh facts. In particular, the meticulous examination of all resected specimens is urgently called for, to discover the mere presence of diverticula, and to establish their causal relationships (if any) to the development of carcinoma.

The following types of case comprise the majority of diverticular lesions (1) Inflammatory trouble in (a) The left lower quadrant, (b) The right lower quadrant, (2) General peritonitis, (3) Vesicocolic fistula, (4) Pelvic syndromes, (5) Intestinal obstruction, (6) Mimicry of carcinoma.

The frequency of tumour or abscess formation in association with any or all of these will be described, and one may say in advance that *any* case from the age of 30 onwards, presenting these symptoms, calls for a mental reservation as to the possibility of a diverticular origin, and in some instances the diagnosis can be and has been made with certainty and accuracy.

We have based our conclusions on the records of cases in which clinical symptoms resulted from or were found in association with the diverticula, or

in which the presence of diverticula, though not categorically stated (in many cases the possibility obviously not being known to the observer), is practically certain. In making this selection we have rejected a very much larger number of cases, the vast majority of which are undoubtedly diverticular in nature, but which, from the single-case point of view, are not so certain as those from which we quote. The impression we derive ourselves from an exhaustive study of recorded cases of diverticulitis, sigmoiditis, pericolitis, and other variously-named lesions, is that we should not overstate facts nor vitiate conclusions did we multiply our series several times over. This impression appears to be shared by all observers who have devoted any close attention to the subject.

Table II.—ANALYSIS OF LEADING CLINICAL FEATURES.

	Per cent			Per cent
Acute intestinal obstruction	6·7	} 15·20	Arteriosclerosis noted	2·50
Chronic intestinal obstruction	8·5		Local peritonitis	5·30
State of nutrition	Obese	60·10	Chronic mesenteritis	5·80
	Thin	18·70	Stenosis of bowel	14·00
	Losing weight	21·20	Thickening of bowel without stenosis	24·00
	Unspecified	66·00	Adhesions	37·90
State of bowels	Constipation	21·00	Acute diverticulitis (non-perforating)	5·14
	No constipation	2·50	Perforating cases	28·40
	Diarrhœa	3·50	Gangrene of diverticulum	6·25
Left-sided tumour		30·00	Suppurative peritonitis	18·00
Left-sided pain		11·40	Abscess	28·80
Right-sided cases		6·25	Fistula formation	19·80

**GENERAL DIAGNOSTIC POINTS.—Age.**—This is a most important point, as *Fig. 277* proves. Below the age of 35 the diagnosis is open to considerable doubt. As *Fig. 278* proves, most of the 'clinical' cases occur between the ages of 40 and 65.

**Sex.**—There is a preponderance of males over females (*Fig. 277*). This is an interesting fact, as constipation is undoubtedly much commoner in women, and in them one would suppose the musculature of the bowel also tended earlier to be worked out. On the other hand, we are inclined to think that gas formation may be greater in the male sigmoid, and this could equally cause pulsion diverticula.

**Nutrition.**—Most patients are in good or obese nutrition, though this point is not noted in the great majority of cases (for figures see *Table II*).

**Constipation.**—The importance of this in etiology has already been dealt with. The question of how far its presence or absence aids the diagnosis is what now concerns us (see *Table II*.) Murray comments upon the value of a history of 'rabbit's-dropping' stools over a long period, supervening upon a previously normal action of the bowels.

An alternating constipation and diarrhœa, with left-sided colicky pain, is very suggestive (Dunn), as it is also in the case of suspected malignant disease. But in carcinoma this history is almost invariably a comparatively recent one, while in diverticulitis it tends to be of much longer standing, in which case it is undoubtedly a valuable aid to diagnosis. One's impression from the case-histories is that the most infrequent preceding bowel action is

that of many years' looseness. In such a case, therefore, the diagnosis of diverticulitis is improbable.

**1a.—Inflammatory Trouble, more or less Acute, in the Left Lower Quadrant of the Abdomen.**—Cases of this type constitute by far the largest group, and the other clinical types (obstructive, peritonitic, fistulous, pelvic) are not infrequently mere sub-groups of this main type, in that they represent the 'end-results' of a case that for months or years has exhibited symptoms about to be described, and on that account necessitating inclusion in this inflammatory group. Erdmann has gone so far as to say that the occurrence of left-sided pain, with mass or not, but with tenderness and rigidity, is practically pathognomonic of diverticulitis. We shall see that this represents the extreme view, favoured by the many supporters (e.g., Patel) of the almost exclusive rôle of diverticula in inflammatory troubles in this quarter. Fitz in 1912 said that the cases comprised in this 'inflammatory' group made a fairly complete picture of what must be recognized as a new disease of the lower abdomen—diverticulitis of the left quadrant, analogous to appendicitis of the right quadrant.

Inflammation here has been variously described under the following terms: Diverticulitis and peridiverticulitis, sigmoiditis and perisigmoiditis (including infiltrating colitis and hyperplastic colitis), sacculitis, pericolitis (*sinistra*), left-sided appendicitis, epiploic appendicitis; pelvic peritonitis. We must here digress a little to consider the usefulness of these terms—as to which may be permanently discarded, or which should be provisionally retained until our knowledge is more complete.

*Sacculitis* is entirely a bad term. Its employment is urged mainly by Rutherford Morison, who misconceives the views of one of us in his letter to the *Lancet* on the point. Sacculation of the colon is one thing, and diverticulum-formation another, they are quite distinct, though they may co-exist (14, 75, 81). Sacculation is generally understood to mean an exaggeration of the normal haustra, in the great proportion of cases no marked exaggeration of the normal haustra occurs, though Keith has drawn attention to a tonic contraction of the *tænia* which may have some influence in this direction. Lockhart Mummery also says that the diverticula are a kind of exaggeration of the normal sacculi of the colon occurring between the longitudinal muscular bands. We think the comparison with sacculi of the bladder has something to be said for it, but under any circumstances the term diverticulitis has come to stay, and we must therefore make its connotation as precise as possible.

*Pericolitis* is a term of more subtle utility. Lockhart Mummery adopts it as the main and inclusive title of a large mixed group of cases, including cancer, syphilis, and tubercle, which, with diverticula, form a sub-group, albeit he ascribes to the diverticula an importance and frequency which is overwhelming save in comparison with cancer. We think this classification is not the most useful, and we would urge that the various inflammatory lesions met with in this region should not be grouped under any such vaguely inclusive term, but should at present be separated as sharply as is possible the one from the other. We urge that diverticulum formation be given a separate and special place, to mark as completely as possible this separation from the

other morbid processes with which it has been and still is being confused. In this way will our knowledge most rapidly progress.

*Left-sided appendicitis* is a term which should always stand for a true vermiform appendicitis with left-sided lesions or manifestations. Such cases are rare, and in the past have certainly been confused with those now under consideration. The instance recorded by Bland-Sutton (67), in which the suggestion of an appendicitis in transposed viscera was seriously advanced as an explanation of diverticular disease in the left lower quadrant, occurred at a time when diverticulitis was still practically unknown.

*Epiploic appendicitis* is, of course, a true title for *some* of the cases, and a little more will be said about it later, but it is only limited in its application, and therefore is not a synonym for diverticulitis.

*Sigmoiditis*.—In regard to this (and its sub-group perisigmoiditis), however, there is a very great deal more to be said. The present question is this: Are diverticulitis and sigmoiditis different terms for the same condition, which is always primarily a diverticulitis? Or are there two distinct pathological conditions, a diverticulitis and a non-diverticular sigmoiditis? Graves, for instance, says that these inflammations of the sigmoid are not yet thoroughly understood, and that the inflammatory tumours (sigmoiditis) described by various observers, and with which occurs general fusiform and local swelling about the gut somewhat resembling diverticulitis, appear to have a mysterious origin. He is inclined to connect them with the very frequent occurrence of adhesions about the sigmoid.

In regard to diverticulitis, we are certain (1) of its reality, and (2) of its very considerable frequency, a frequency which becomes the more apparent the more closely the subject is studied. Some clinicians would have us believe that all such cases are diverticular—reaching in this direction, Patel said in 1907 “Peut-être un jour sera-t-on en droit de dire que, dans presque tous ces cas décrits sous le nom de sigmoidites, il s’agit de véritables *diverticulites*, absolument comme l’appendicite a été substituée pen a peu à la typhlite.” This observer, while obviously a firm believer in the importance and frequency of the rôle of the diverticulum in sigmoiditis, has made most of his valuable contributions—principally on the acuter cases—under the title of sigmoiditis. In 1911 he collected a number of cases of *perforating sigmoiditis*, causing general peritonitis and abscess. Under this title one is certainly able to include all such lesions apparently originating in inflammatory processes in this section of the bowel, and the grouping has a certain clinical usefulness. But it does not help to define and limit the exact influence and frequency of the diverticulum formation.

As to the reality of sigmoiditis, there is not complete unanimity of opinion. Older observers such as Nothnagel and Ewald have failed to meet with these sharply localized inflammatory processes in the colon, but on the other hand there is now a considerable volume of literature (except perhaps in England) describing and classifying cases of sigmoiditis. The cases exist, it is stated, in all grades of severity, from transient infiltration of the mucosa to severe involvement of the whole gut, with, in some cases, suppuration and abscess formation. The general tendency is to a more or less defined tumour formation, particularly the ‘sausage-shaped’ tumour. The condition is generally

ascribed to bacterial inflammation of the gut wall—some authors say almost invariably as *dysenteric* inflammation, of greater or less virulence, constipation, particularly sigmoid stasis, is assigned a leading rôle in etiology. The terms infiltrating colitis (Leube) and hyperplastic colitis (Schutz) are used for varieties of sigmoiditis.

In the study of these cases, one is struck by the fact that nearly all the symptoms and physical signs of the condition are identical with those encountered in the vast majority of the *proved* cases of diverticulitis. Indeed, it seems to us that on broad lines the main differences may be summarized by saying that in the (non-diverticular) sigmoiditis cases (i) A relatively considerable number occur at an earlier age than is the rule for proved cases of diverticulitis (Ransohoff, Ashurst), (ii) Symptoms pointing to involvement of the intestinal mucosa are relatively much more frequent, and (iii) Subsidence under treatment *without* operation is almost the rule. This last result has been proved to be possible in cases of diverticulitis, indeed, Giffin says the previous appearance of a mass, and its *subsequent disappearance*, is strongly confirmatory of other signs and symptoms pointing to diverticulitis.

We confess to an unfamiliarity with these cases of non-diverticular sigmoiditis and perisigmoiditis, and have been much impressed with the fact that a large number of writers (e.g., Rosenheim, Matthews, Mayor, Durant) on the subject have failed to reveal (in their articles) any knowledge of the diverticular process. This is evidenced by the omission to consider it in differential diagnosis, and is a serious flaw in the value of such contributions. Such a series of cases as that, for instance, given by Stern, leaves us quite unconvinced. He enlarges upon chronic sigmoiditis, diagnosed almost invariably by the use of the sigmoidoscope, but we feel that where his cases have not a diverticular origin, they are probably nothing more than examples of chronic constipation. Dahl, also, makes a severe, but we think justifiable, criticism of Rosenheim's case of sigmoiditis of mucosal origin. While we feel that there *are* cases of sigmoiditis, to be sharply differentiated from diverticulitis, we are inclined to the view that they are infrequent, and our inclination is also to support the generalization of Patel quoted above. Our excuse for discussing the point somewhat at length is that it must be obvious to anyone who urges a wider recognition and acceptance of diverticulitis as an entity, that there is need for much more careful work to be done in regard to exact definition and weeding out of the non-diverticular cases. In the bibliography at the end of this article we give a number of references which are essential to a full study of the subject of diverticulitis and its relation to sigmoiditis.

**SYMPTOMS OF THE INFLAMMATORY LESION.**—In the first place it is important to note that symptoms from the intestinal lumen are not to be expected in the early stages of the process. Stenosis takes a long time to develop into symptomatic obstruction, and the great infrequency of involvement of the mucosa has already been noted.

*Pain.*—This, of one kind or another, is very frequent. It is referred in most cases to the left iliac fossa, very suggestive is its colicky and *recurrent* character. There may be only a single attack of pain, as in the gangrenous or acute cases, but there are frequently many attacks, spread over periods of



months or years (twelve years—Giffin). The pain is seldom accompanied by attacks of vomiting.

*Tenderness.*—This is an extremely frequent symptom elicited by palpation, and reference to *Fig. 289* will give at a glance its site of election. It is usually accompanied by more or less definite *muscular rigidity*.

*Tumour.*—This has been noted in 30 per cent of the cases. Its localization is also clearly indicated in *Fig. 289*, which is constructed from a number of typical cases. A specially characteristic tumour is an elongated or 'sausage-

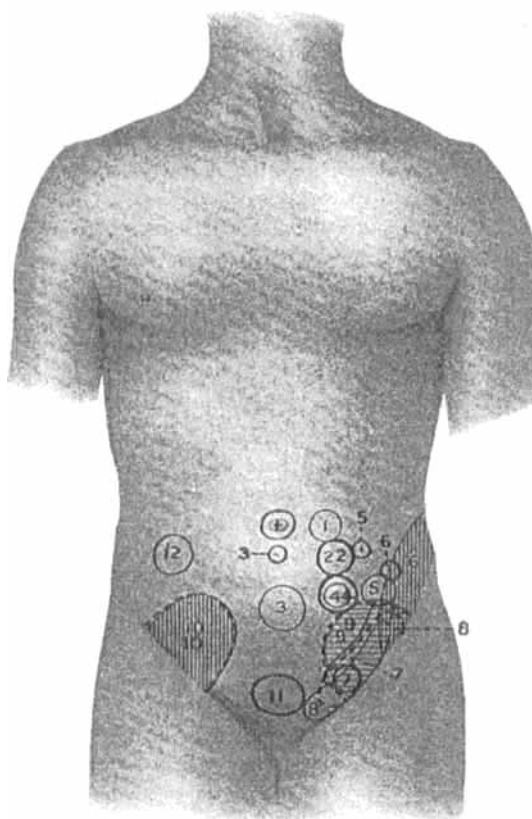


FIG. 289.—Diagram to show the position of a palpable mass (black), and of tenderness (red), in cases of diverticulitis, as found recorded in the literature. The numbered areas refer to cases in the list of selected cases given at the end of the article. Some of the areas marked are more frequently illustrative than are others. The sharp outline is adopted as a matter of convenience in drawing attention to the variety of sites which can be met with in different cases, in only a few instances is there a record of precise sharpness and limitation of outline.

**BLACK AREAS.**—Palpable mass in area 1, *Case 87*, 2, *Cases 38, 88*, 3, *Case 27*, 4, *Case 38*, 5, *Case 11*, 6, *Case 3*, 7, *Case 7*, 8, *Case 32*; 9, *Cases 2, 9, 18*, 10, *Cases 8, 17, 35*.

**RED AREAS.**—Tenderness in area 1, *Case 90*; 2, *Case 88*, 3, *Case 86*, 4, *Cases 23, 38, 90*, 5, *Case 91*, 6, *Case 92*; 7, *Case 10*; 8, *Case 32*, 9, *Case 18*, 10, *Case 17*, 11, *Case 58*, 12, *Case 89*.

shaped' one lying just above and more or less parallel with Poupart's ligament. In size it varies greatly, having been noted as large as a child's head. It may be fixed in the pelvis and mistaken for an ovarian or other pelvic tumour. It is usually more or less tender, and is not as a rule mobile.

A feature of this tumour is its variability (cf. sigmoiditis). It may appear and disappear, even several times, and such a *recurrent tumour*, with the usual concomitants of inflammatory trouble in this area, is a specially diagnostic feature (as it is also of sigmoiditis).

## DIVERTICULA OF THE LARGE INTESTINE 501

*Abscess.*—This is of very frequent occurrence (28·8 per cent), and frequently leads to tumour formation. It is responsible for the pyrexia and leucocytosis, and also for the sudden and spontaneous disappearance of both tumour and symptoms, by spontaneous discharge through bowel or other viscus or externally, as has been insisted upon by Stengel.

*Fever.*—This is usually moderate and irregular; it may be manifested as irregular attacks over a number of years (12, 43). Rigors are sometimes present, in one case simulating malaria (7).

*Leucocytosis.*—This is frequently met with, and, with fever, is a valuable point in the differential diagnosis from cancer, though, as emphasized, cancer itself in this region is liable to be accompanied by inflammatory phenomena, making the diagnosis one of great difficulty or impossibility.

*Stools.*—Constipation and diarrhoea have been discussed; the important point otherwise is the absence of visible blood, over prolonged intervals. Though blood has been noted in some cases (40, 65), yet its presence is so exceptional that it should give rise to the greatest reluctance in diagnosing diverticulitis as opposed to carcinoma. Its absence is of course due to the fact that the mucosa of the bowel is so seldom involved.

*Bladder Symptoms.*—Apart from actual fistulous communication with the bladder, which constitutes a separate clinical group, there are frequently symptoms of bladder irritability, probably due to the nearness of inflammatory foci or to actual adhesion.

*Pelvic Symptoms.*—These are of some frequency, and sometimes make the clinical feature. These cases are discussed more in detail later.

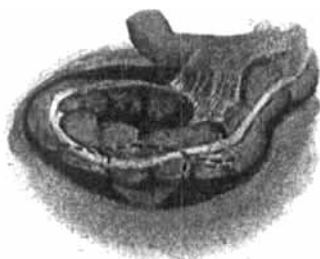
Summarizing the foregoing symptoms, the inflammatory cases may be roughly subdivided into the following sub-groups —

- i. *Acute and Fulminant* (gangrenous).—Symptoms practically identical with acute right-sided appendicitis.
- ii. *Recurrent.*—Acute or subacute attacks over a long period.
- iii. *Cases ending in Spontaneous Recovery.*
- iv. *Protracted Cases.*—Those which continue till the clinical picture is changed by one or other of the following developments (a) Simulation of carcinoma; (b) Intestinal obstruction (acute or chronic); (c) Bladder perforation; (d) General peritonitis; (e) Incidence of pelvic symptoms.

**TRAUMA.**—The mode of onset of many of the acute cases is of interest as showing the great part which trauma of various kinds may play. It would seem to have a definite diagnostic value when diverticulitis is under suspicion. An analysis of our cases revealed the following varieties of trauma, apparently acting as exciting causes of, at any rate, the acute manifestations. while straining at stool (20, 55), during an administration of an enema (5), following a dose of castor oil (23, 33); jolting in a motor-car (24); following a heavy meal (9, 24); while lifting a weight (8), while jumping (32); while at work; during an abdominal operation.

**1b. Cases with Symptoms in the Right Lower Quadrant** (1, 11, 15, 35, 56, 60).—Just as true appendicitis may occur with left-sided localization or symptoms, so, with much greater relative frequency, may diverticulitis occur with right-sided symptoms. This is due mainly to the relative mobility

of the sigmoid loop (see *Fig. 290*). These cases are, when acute, practically indistinguishable from appendicitis. A diverticular origin should perhaps occur as a possibility if such a characteristic happening as bladder fistula resulted; but, as with other less usual localizations of diverticulitis in the transverse and upper descending colon, correct diagnosis is not to be expected. Right-sided manifestations may result from diverticula of the



**FIG. 290.**—Diagram of the brim of the pelvis, to show a long sigmoid loop drawn over to the right side, and enveloping the uterus (indicated by dotted lines). A displacement of this kind explains the occurrence of right-sided pain in diverticulitis of the sigmoid. (After Pennington.)

cæcum, or from a true *diverticulitis of the vermiform appendix*. Lejars and Ménétrier have suggested that these latter cases sometimes lead to special localizations of abscess, owing to the tendency of the diverticula to form between the appendicular mesentery. This point does not seem to have been very fully worked out, and is of course of but minor importance.

**Epiploic Appendicitis.**—There is an interesting group of cases, a small number of which are scattered through the literature, of inflammatory changes, acute or chronic, confined to an epiploic appendage. Many of these—perhaps most—are due to the appendage being more or less hollowed by a diverticulum. This may lodge a foreign body (*Fig. 271 C*), which may inflame or per-

forate, and set up a general peritonitis or local abscess. A more chronic type of inflammation may cause adhesions, and later an obstruction by band formation (84) or kinking. Tuttle records a case of chronic obstruction of the sigmoid flexure due to two epiploic appendices being adherent to each other.

While not *all* cases of epiploic appendicitis are of diverticular origin, the lesion is one which is very closely related in practice, and must be considered differentially in a certain number of cases.

**2. Acute General Peritonitis.**—This is not infrequent, and is usually a lethal complication of a diverticular perforation. We have noted it in 18 per cent of our cases. It may be mainly mechanical in origin (the sudden ‘pistol-shot’ perforation, as a result of trauma), or the result of the gangrenous sloughing of a diverticulum or a diverticulum-containing appendage. This latter possibility may be kept well in mind, for only the hole in the bowel may be visible at operation, and no doubt many cases of assumed perforation of a stercoral or ‘solitary’ ulcer have had this origin. The practical point is that any such finding at operation should lead to a search for the presence of other diverticula, which may be found to be on the verge of perforation or in an inflamed condition (3, 30).

As many of these cases occur in aged or debilitated subjects, the peritonitis may be of the asthenic or latent type, and may be discovered only at a post-mortem examination.

**3. Vesicocolic Fistula** (and bladder symptoms generally).—Reference to the table of adhesions noted as the result of diverticulitis (p. 492) shows that in a large number of cases (33 in 280) adhesion takes place between the bladder

and the sigmoid (but occasionally between the rectum and bladder). The adhesion may be direct. i.e., the viscera themselves are in contact, or through a more or less dense fibrous mass, or an intervening chronic abscess cavity. The mass may be tunnelled by diverticula, as is well shown in *Case 69*. These adhesions may cause no bladder symptoms, but in a number of diverticulitis cases vesical irritability, which may be transient, has been noted. Sometimes, however, the adhesion goes on to perforation, so that by the diverticular channel a fistulous communication between the gut and the bladder may occur, with consequent passage of air and fæces per urethram. Some of these cases undergo spontaneous recovery (13).

Like all else connected with the pathology of diverticula, the fact that they were not only a cause, but a relatively frequent cause, of vesicocolic fistula was overlooked for long enough. Sidney Jones described a case (53) very clearly in 1858, but Cripps in 1888, when specially investigating the subject and noting the case, makes no comment on its unusual pathology. To Heine, in 1904, belongs the special credit of emphasizing the comparative frequency of diverticulitis as a cause of this form of fistula. he collected 7 certain and 9 extremely probable cases at that date. In the present series we note 38 cases of bladder fistula, of which 25 are of certain and 13 of very probable diverticular origin. Moynihan was the first observer in England to emphasize the diverticular origin of vesicosigmoid fistulæ; he is worth quoting exactly. He wrote in 1907: "The formation of a vesico-intestinal fistula seems to be one of the tendencies of a perforated false diverticulum, a search through the literature has shown that it is far more common than was supposed. In cases where a hard growth in the intestine is accompanied by the passage of flatus and fæces by the urethra, a diagnosis of carcinoma seems irresistible, yet the *probability* is that 'the growth' would be simple, and that the cause of the fistula would be a false diverticulum which had burrowed its way through all the coats of the bowel, and thence through the wall of the bladder which had become adherent." Experience has confirmed the truth of this.

The italics in the above quotation are ours: they serve to emphasize the fact that the common belief is that most vesicocolic fistulæ are malignant in origin. Cripp's series refutes this; he gives, for 63 cases: traumatic, 2; cancerous, 9; inflammatory, 45; unascertained, 7. Moreover, he gives reasons for thinking that malignant cases would tend to bulk less largely in the records, because they would be regarded as of the more common type. Chavannaz, writing later, puts the proportion of malignant cases at less than 20 per cent, and comments on the customary exaggeration of the frequency of fistulæ by direct extension from a malignant focus. Indeed, Kelly and McCallum say that in malignant cases the fistula is more often *above* the growth than resulting directly from it. this latter class of case forms less than 10 per cent of the whole.

Cripp's table of localization gives. rectum, 25 cases; colon (sigmoid flexure 11, not stated 4), 15; small intestine, 12; colon and small intestine, 5; unascertained, 6. Chavannaz (95 cases) gives 24 per cent between bladder and sigmoid.

The subject has been recently (1916) reviewed by R. C. Bryan, and he

has collected and analyzed 42 cases. Males are at least twice as frequently affected as females. He gives the following age table : not stated, 6 ; twenty to thirty, 3 ; thirty-one to forty, 7 ; forty-one to fifty, 5 ; fifty-one to sixty, 14 ; sixty-one to seventy, 4 ; seventy-one to eighty, 2 ; eighty-one to ninety, 1.

These statistics prove (1) that vesicocolic fistula is not so frequently due to malignant disease ; and (2) that it is very commonly situated between the bladder and the sigmoid flexure. But this does not prove the *diverticular* origin of such fistulæ. In this direction Heine's series is strong evidence, and Bryan's later series is still more cogent. The latter gives an etiological table of his 42 cases as follows sigmoid diverticulitis (certain), 15, sigmoid diverticulitis (probable), 6, inflammation (?), 4, surgical traumatism, 8 ; carcinoma of sigmoid, 3, carcinoma of bladder, 2 ; carcinoma, not specified, 2, ovarian abscess, 2, amœbic sigmoiditis, 1 ; carcinoma or gumma of sigmoid, 1 ; tuberculosis, 1 ; stricture, 1 ; ulceration, 1. Naturally, he says, "it appears well substantiated that diverticulitis is the most frequent cause of sigmoid-vesical fistulæ."

A diverticular origin is more particularly to be suspected when the symptoms of fistula follow long-standing evidences of inflammatory or bowel trouble in the left lower quadrant of the abdomen, as has been described. When, on the other hand, the preceding history is essentially 'vesical,' a diverticular origin is improbable.

These facts have a practical bearing, and point to greater prospects of relief from surgical interference. Two cases (21, 22) recently recorded by Littlewood strikingly bear out this, the possibility of attaining brilliant results by operation. On the other hand, Chute, commenting on the failure of his method in his two cases, says. "Beside the technical difficulty of doing a plastic operation in the depths of the pelvis, there are two other things that militate against success in closing these diverticular openings in the sigmoid. The first is the frequency with which there is a narrowing of the sigmoid below the opening of the diverticulum, which increases the pressure on the suture line, and if this narrowing is marked, it makes success practically impossible. Such a narrowing may be due either to an infiltration of the bowel wall with secondary contraction of the lumen, or to the pressure of a mass of inflammatory exudate against the bowel from the outside. The other factor that renders success difficult in these cases is the poor condition of the bowel for suture. It is thickened, making folding-in of the wall practically impossible ; it is friable, making it difficult to have sutures hold ; and finally, its nourishment is interfered with to a degree that makes union slow and uncertain."

**4. Pelvic Syndromes.**—In the last few years much attention has been drawn to the relationship between sigmoidal and pelvic diseases. The anatomy and mobility of the sigmoid loop will make it probable that inflammatory processes originating therein may cause adhesion with, or spread to, or otherwise involve the pelvic viscera. Poulain has analyzed the literature and cases bearing on this relationship, and separates out three types of case : (i) Inflammation primary in sigmoid loop, and involving adnexa and uterus, (ii) Inflammation of genitalia, involving sigmoid ; (iii) A sort of sigmoido-salpingitis, in which inflammation starting in either augments or doubles the lesions (1).

Probably the most widely held view has been that these adhesions generally resulted from pelvic inflammation of one sort or another. Bernitz and Goupil in 1857 showed that pelvic cellulitis did not tend to produce these adhesions to the sigmoid flexure, but this view is not shared by all observers. Since then, however, one observer after another has given prominence to the sigmoidal factor. Poulain comments on the occurrence of diverticulitis as a cause, and records a case. Bodkin states that *sigmoiditis* may produce left-sided phlebitis and inflammation of the broad ligament. Albrecht gives a very remarkable case (1), in which the symptoms were on the right side of the pelvis, and a diagnosis of pelvic carcinoma was made at operation, and also comments on the mimicry of pyosalpinx by sigmoiditis. Shoemaker also quotes some illustrative cases (36, 37), in which the symptoms were left-sided, and in one of them pelvic carcinoma was suspected. Many observers have commented on the frequency of adhesions about the sigmoid. Pennington has drawn attention to them as producing symptoms of pelvic disease in not a small number of cases. Such symptoms include almost all of those described under the various uterine and pelvic inflammations. he says that in any suspected case of pelvic or utero-ovarian disease, a routine and thorough examination of the recto-sigmoidal bowel and its functions is just as important as a vaginal and uterine examination. Byron Robinson's series of figures for sigmoid adhesions has already been quoted (p. 493).

It is not suggested, of course, that these frequent sigmoidal adhesions are in anything but a small proportion of cases connected with the presence of diverticula. But advance is being made, in that the pathology of the sigmoid flexure and that of the pelvic viscera are being linked together in many respects. We only seek to emphasize the connection in respect of the presence of diverticula.

Giffin, in a series of 39 cases of diverticulitis, mentions 4 (all women) in

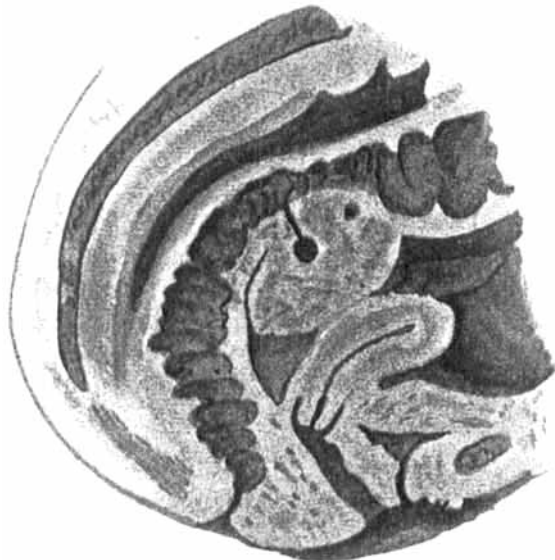


FIG. 201.—Diagram of a vertical section of the pelvis in a case of perforating diverticulitis. The bowel wall is seen to be stenosed for some distance, and a narrow channel leads from the lumen of this portion into a mass of inflamed tissue placed between the intestine and the uterus. The round black mark on the right of this channel is the cross-section of a second diverticulum. Infection of the cellular tissue had taken place from the larger diverticulum, and, spreading in all directions, had led to the formation of the dense mass indicated in the drawing. The posterior cul-de-sac is occupied by fluid—inflammatory exudate. (Case 44.)

whom there existed pelvic tumours *without* any bowel symptoms. In our present series, pelvic symptoms, sometimes completely filling the clinical picture, are noted in twenty-three cases, or 7 per cent (3, 13, 26, 36, 37, 44 (*Fig. 291*), 59, 60), and reference to our table of adhesions is interesting in this connection. Seeing that a pelvic tumour may be entirely sigmoidal in origin, but without bowel symptoms, and that pelvic cellulitis may be completely simulated clinically, we agree with Giffin that diverticulitis should be considered in differential diagnosis in all cases of 'left lower quadrant' and pelvic inflammations and tumours. Yet how short of this standpoint are most of the works of gynæcology—even the lengthiest treatises! Here, again, it is safe to predict that in future, gynæcologists, mindful of diverticulum formation, will add greatly to recorded cases of this type, and consequently to our exact knowledge of the condition.

**5. Intestinal Obstruction.**—Many cases present themselves clinically in this guise, with obstruction either as the first important symptom, or consecutive to a varying period of 'inflammatory' symptoms. The obstruction may be acute, and may then be due to obstruction of the small intestine by bands, as in the interesting example in *Case 84*, or to involvement in adhesions (83). Acute obstruction may suddenly supervene in a sigmoid which has been developing stenosis for years past. Angulation or kinking of the sigmoid flexure may result from contraction of the sigmoid mesentery due to a chronic (diverticular) mesenteritis, as pointed out by Ries and others. More often the obstruction is recurrent, partial, or subacute, the final stage of repeated threatenings (41, 72).

**6. Mimicry of Carcinoma.**—This is discussed in the next section.

## V.—DIAGNOSIS.

**Carcinoma.**—The stenosis which causes obstruction is frequently palpable as a tumour, and it is this clinical feature which leads so frequently to a mimicry of carcinoma, as Graser, Moynihan, and indeed most writers on the subject have pointed out. The differential diagnosis is frequently impossible; but with a full appreciation of the facts given in these pages, a correct diagnosis can not infrequently be adumbrated, if not made positively. Correct diagnoses prior to operation have now been made by a number of surgeons. Examination by the procto-sigmoidoscope, *x* rays, and cystoscopy furnishes aid to diagnosis in some cases. A few recent observations in regard to these methods, as applied to diverticular cases, may be useful.

**SIGMOIDOSCOPE.**—The use of this instrument, which is not entirely devoid of risk, has been extremely disappointing as regards affording positive data for the diagnosis of diverticula. Such was the conclusion arrived at by Beer in 1911. He has been keenly interested in the subject of sigmoid diverticula, and has made valuable contributions to the literature thereon, but up to 1911 had not been rewarded by making a positive sigmoidoscopic observation, though he quotes Zweig as having done so. Tuttle says he was once able to insert a probe into an abscess cavity in the mesosigmoid through the sigmoidoscope. The diverticular nature of the cavity was not stated. Friedel (quoted by de Quervain in 1914) said at the Surgical Congress in Paris (1915) that one

could *sometimes* recognize the opening of a diverticulum by its red edges and the fact that it was oozing pus. This statement is to us particularly unconvincing, as these diverticula seldom or never 'ooze pus.' As Beer says (*loc. cit.*), and as we have throughout insisted, it is difficult enough to recognize these orifices, concealed as they are by the folds of mucosa, when one has the specimen in one's hand, so that observation by sigmoidoscope is unlikely to yield valuable results, except negatively a negative finding is on the whole *against* carcinoma, and therefore in favour of diverticulitis.

**X RAYS** (2, 39).—That an *x*-ray examination may be a valuable method of confirming the diagnosis is clearly demonstrated by *Fig. 292*. By its aid

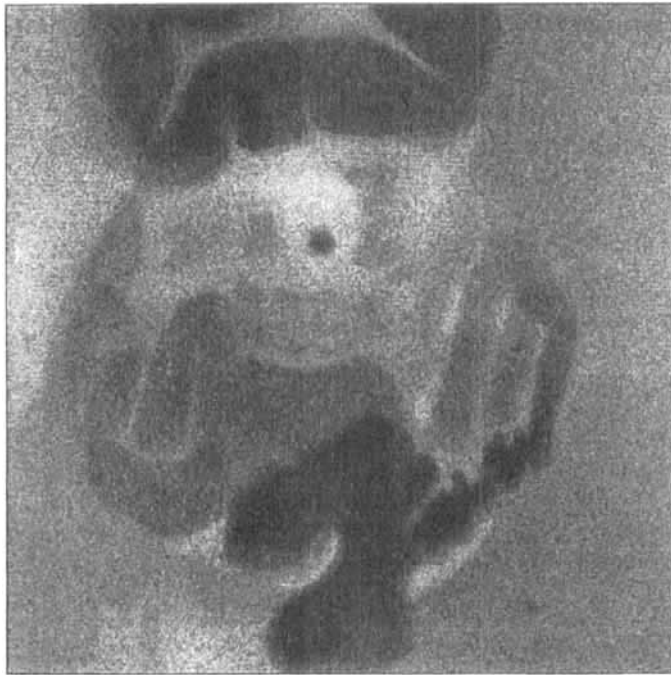


FIG. 292.—X-ray photograph to show several shadows thrown by diverticula of the sigmoid after barium injection. (*Case 31.*)

certain cases of stenosis of the sigmoid flexure have been demonstrated, but this does not help greatly differentially. Bismuth may be given by the mouth or by enemata (the latter with care, as a rupture of a diverticulum has occurred after enemata on more than one occasion). De Quervain quotes a most interesting case (81) in which sigmoid irrigation showed six to eight warty' outgrowths from the sigmoid flexure, and thus the diagnosis was made with certainty. In the case illustrated in *Fig. 292* irrigation was inconclusive, but feeding with bismuth showed a loaded colon with these projections. After the main faecal mass had passed on and the lumen of the sigmoid flexure was clear of shadow, the row of black spots remained for days. It would seem



almost certain, however, that the usefulness of *x*-ray examination is largely restricted by the fact that the diverticula so often have solid fæces. This must tend to prevent a bismuth-impregnated mass passing through the flexure from entering the diverticula to an extent adequate to reveal their outlines in shadow.

**CYSTOSCOPY.**—This has been of use in the bladder-fistula cases, chiefly by tending to exclude malignant disease. Giffin quotes a case from the Mayo Clinic in which two fistulæ were thus observed in the roof of the bladder, and were diagnosed as inflammatory, their edges being neither rigid nor indurated, thus enabling a correct diagnosis to be made.

The chief points in differential diagnosis from a carcinoma may be summarized as follows —

1. The absence of the "shadows of malignancy" from the general picture (Giffin).
  2. Tendency to obesity, and maintenance of good nutrition generally
  3. Long history of attacks of abdominal pain in the left lower quadrant.
  4. History of tumour formation with subsequent disappearance.
  5. Absence of blood (visible to naked eye) in stools, over a prolonged period.
  6. Presence of a vesical fistula, in which malignancy can be excluded by cystoscopy
  7. Negative sigmoidoscopy as regards malignant disease.
  8. *X*-ray demonstration of diverticula.
  9. Pyrexial attacks.
  10. Examination of blood the presence of neutrophilic leucocytosis and the absence of the specific nuclear changes characteristic of cancer.
- In carcinoma of the sigmoid, loss of flesh is early, pain and tenderness are late, and are often *preceded* by tumour.

There are certain other conditions which have to be considered in arriving at a differential diagnosis. They are (*a*) Sigmoiditis, (*b*) Hyperplastic tuberculosis, (*c*) Actinomycosis, (*d*) Syphilis, (*e*) Pelvic conditions generally.

*Sigmoiditis*, from whatever cause arising, has already been somewhat fully discussed. We confess that, apart from the three points already noted (occurrence of some cases at earlier ages, greater proportion of cases ending in spontaneous recovery, and more frequent evidences of involvement of the mucosa), we do not discover any reliable differential diagnostic criteria short of operation or autopsy findings.

*Hyperplastic tuberculosis* is extremely rare in this section of the bowel, its incidence is almost confined to the cæcum. Consequently, in any suspected cases, one must be assured (1) that diverticulitis has been carefully excluded, and (2) that the evidences of tuberculosis are such as to make that diagnosis reasonable. In this connection it is interesting to refer to the three cases described by Kidd (45, 46, 47). He does not make any allusion to diverticulitis in discussing the diagnosis, and this must under the circumstances be taken as proving that he was unaware of this possibility, in two cases there is not the slightest evidence of tuberculosis, in the third it appears to be limited to the finding of a few giant cells in the hyperplastic tissue. We

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have already shown that this is not conclusive, indeed, Nicolaysen has found them, in her hyperplastic case (26), of proved diverticular origin.

*Actinomycosis* is mostly in the cæcal region, but otherwise would in time present a different clinical and pathological picture.

*Lues* is very rare in the sigmoid flexure, and would be expected to be capable of confirmation in the usual ways. Some observers have urged that some of the obscure and so-called syphilitic strictures of the rectum have really been of diverticular origin the suggestion merits attention.

*Pelvic lesions* have already been discussed, and there is little to add to the dictum that all left-sided (and some right-sided) examples of supposed pelvic disease must, after the age of 30, be thought of in connection with morbid processes in the sigmoid, and in particular in relation to diverticulitis.

### VI.—SOME UNUSUAL COMPLICATIONS.

*Pulmonary Embolism.*—This complication has been noted in four cases of our series (29, 30). The occurrence of left-sided *phlebitis* in pelvic and sigmoid suppuration has also been noted, and is probably directly associated with this grave complication. *Pylephlebitis*, leading to multiple liver abscesses, was observed in Whyte's case (62). *Fat necrosis* in the neighbourhood of an acute diverticulitis has been observed once (34). *Severe rigors*, simulating those of malaria, were observed in Case 7 (Ewald). *Suppuration in a hernial sac* has been already alluded to.

### VII.—TREATMENT.

This is comprised in a single word—surgery—unless operative interference is specially contra-indicated. We shall not venture to indicate what particular surgical procedure is indicated in special cases, but content ourselves with more general remarks.

In view of the potentiality for mischief of diverticula, all diverticulum-bearing gut should be removed. At operation, search should be made, whenever possible, for further diverticula. Recurrence after operation has occurred, presumably from neglect of this point. There seems to be a special liability to post-operative peritonitis, possibly due to the chronic infection of the inflamed tissues in some cases.

Care should be taken in handling the gut, as at least one instance has occurred of rupture of a diverticulum by surgical traumatism. The good results already obtained in certain bladder fistulæ should stimulate surgeons to deal with such cases. In at least one very successful case the possibility of successful intervention had been negatived by several surgeons of eminence.

Lastly, no case of supposed carcinoma of the lower bowel must in future be regarded as inoperable, either before or at laparotomy, unless diverticulitis has been remembered, fully considered, and systematically investigated.

### VIII.—GENERAL SURVEY.

It is now possible, after the perusal of the foregoing text, to appreciate the pathological and clinical inter-relationships and connections which are summarized in the schema we have drawn up. It is, of course, evident to all

who study the subject at all closely, that diverticulitis has only recently emerged from obscurity into recognition, and, as is usually the result, the searchlights of many observers together tend to give it an undue or false prominence. The apparent complexity of the schema may at first convey this impression in regard to a condition for which no great frequency is claimed. But do we know its frequency? Even so, there are two facts which impart importance to the condition quite apart from the question of frequency (1) The very complexity of pathological relationships, which makes recognition of the diverticulum process an essential to differential diagnosis in so many different directions, (2) The undoubted gravity of the condition when present.

A conception of the successive action of various factors upon the formation of diverticula has thus been presented. Commencing with a simple mechanical defect in the bowel, there are seen two main lines along which pathological changes can proceed, and each line may branch off into three or four directions until we arrive at definite morbid changes, whose clinical manifestations are decided and have been discussed at some length. This mode of presentation has brought us steadily to the right-hand side of the schema, and provides us with a tangible explanation of the manner in which each clinical type has arisen.

It is useful now to gather together the conceptions of the pathological and clinical aspects of the subject, and sharpen them into a stereoscopic image by a brief consideration of a hypothetical case of the disease, with the object of demonstrating the principles upon which a clinical examination of the case is to be made. Our patient will be middle-aged, and probably well-nourished, and seeks our help because of more or less severe abdominal pain, which is most likely localized over one or other of the areas indicated in *Fig. 289*. According as the case is acute or chronic, the investigator's mind is drawn backwards along either the upper or the lower series of conditions delineated in red and black respectively upon the schema. In either case, a decision must be made upon the probable anatomical site of the disturbance—whether generalized, or restricted to a definite anatomical area such as the appendix, the cæcum, the transverse colon, the sigmoid, etc. It would be desirable to reach still more refinement in this direction, deciding, for instance, whether the lesion were in the mesentery or in an epiploic appendage, but it is more important—because more practicable—to search for indications of such a type as the pelvic syndrome and the facts we have brought to light in relation thereto.

The next step in the mental process of study of a case takes us to a consideration of the *nature* of the lesion. The middle portion of the schema reveals the fact that there are two main types to discuss (1) The inflammatory, and (2) The neoplastic. The former group includes a series of cases of inflammation which are successively advancing in severity, branching off into the direction of abscess formation on the one hand, and of gangrenous change, with perforation, on the other. In each case a definite series of clinical phenomena is produced. But it also includes a second line of phenomena, which become progressively less and less ominous pathologically, until we arrive at a quiescent condition of fibrosis—whose effects are apt to be none the less

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serious for the patient, by reason of the mechanical disturbances which they produce. The neoplastic group again reveals a dichotomous chain of events, the one relating to the existence of genuine malignant disease, while the other—mimicking the cancer—links up with the quiescent type of inflammatory process just referred to.

The investigator will now find himself able to proceed with confidence along these main avenues of thought, using the special data discussed under each heading in preceding pages, in combination with the general principles of medical diagnosis. Confronted with a case of this disease, he will now find himself set with some precision along the roads which terminate in an exact assessment of the given case—a precision which is the more concrete the closer the grasp of the exact nature of the pathological roads whose termini are indicated at the right-hand end of the schema.

We once more maintain that the clear delimitation of sigmoiditis from diverticulitis is essential to the understanding of left lower quadrant syndromes. Sigmoiditis is, so to speak, the older disease, and one or two observers (e.g., Ransohoff) have mourned its threatened eclipse by diverticulitis at a time when it—even perforative and suppurative sigmoiditis—was so firmly established. We ourselves, while constrained to admit the existence of these cases, are by no means as familiar with them clinically as certain series of cases would lead us to expect, possibly because sigmoiditis may own a special (e.g., a dysenteric) and local bacterial origin, which can scarcely be expected to determine the formation of diverticula. A study of the literature of sigmoiditis leads us, as it has led others, to suspect that had the contributors been fully aware of diverticulitis, much would have been written otherwise. As a result of eighteen years' observation and study of diverticula, we feel that it far outweighs in clinical importance any other pathological condition in the left iliac fossa, with the sole exception of carcinoma, and even here there is something in need of revision.

### X.—A SELECTED LIST OF ILLUSTRATIVE CASES.

*Case 1 (ALBRECHT, 1907).—Female, 36. Previously under medical treatment for a tumour in the abdomen. Present attack began with sudden severe cramps, rigors, nausea, pyrexia, leucocytosis (15,000). Obstinate constipation. Abdomen tense and tender. Vaginally, a tumour on the left side of the uterus, the size of a fist and a half. Right tube, thickness of finger. Laparotomy showed a mass adherent to the right side of the pelvis. The sigmoid was a rigid tube and showed no carcinomatous change in the mucosa. The walls were very greatly thickened (up to 3 cm.), and there was a recent fibrinopurulent deposit on the appendices epiploicæ. There were a few diverticula present.*

Apparently there had been several attacks of pelvic peritonitis depending on a gonorrhœal pyosalpinx, perisigmoiditis had resulted, and this, coupled with the formation of diverticula from constipation, gradually produced more and more fibrosis of the walls of the gut. Acute infection of the pyosalpinx led to renewed peritonitis.

*Case 2 (ABBÉ, 1914).—Male, 60. Sudden partial obstruction, with acute tenderness over a hard resisting mass in the left iliac fossa. Pyrexia. Leucocytosis (22,000). X rays showed the presence of diverticula. There had been loss of weight. Numerous diverticula were found at operation, and one formed an abscess in the mesentery outside the intestinal wall. (Fig. 289, <sup>9</sup> black.)*

*Case 3* (ALBERTIN, 1912).—Female, 39. A chronic sufferer from abdominal pain and digestive trouble. Present attack presents very acute lumbar pain, worse at night, colic, occasional vomiting. Vaginal examination showed a large *mass in the left fornix* hardly distinguishable from the uterus. The mass is tender. Operation revealed a very fat sigmoid, with fat epiploic appendages and much congestion. A very large diverticulum was found, just about to perforate. Recovery (*Fig. 289, 6 black.*)

*Case 4* (ANSCHÜTZ, 1909).—Male, 23. Had an attack like appendicitis in March, another in May, and a third (very severe one) in June. Previous health good. Operation showed a tumour in the sigmoid, which proved on *microscopic* examination to be diverticular. One had perforated.

*Case 5* (BARBAT, 1910).—Male, 56. Very obese. The symptoms were those of acute strangulation, complete constipation had existed for three weeks. *Salines* were given by proctoclysis, and *led to the rupture of a diverticulum*. Enemas previously had extracted some small round masses of hard fæces which may have come from within diverticula. Operation revealed a dirty grey sloughing mass of fat at the junction of the anterior layer of the mesosigmoid and the root of the mesentery. A perforation had occurred at this place. Death six days after operation.

*Case 6* (CAMERON and RIPPMMANN, 1910).—Female, 73. Stout. She had an irreducible right femoral hernia, severe abdominal pain, constipation, subnormal temperature. Pus was found over the hernial sac, and the pelvis was half full of pus owing to an abscess behind the sigmoid. A *bile-stained fish-bone* projected into the gut from a diverticulum half an inch long, whose apex was perforated, thus setting up general peritonitis. There were three other diverticula higher up.

*Case 7* (EWALD, 1912).—Male, 60. Taken ill with a sudden rigor fourteen days before, *rigors recurred, the illness simulating malaria*. There was 2 per cent glycosuria, and 7 gm. of albumin per litre (rapidly falling to nil). The patient was too ill to justify operation, and he died in three weeks. Numerous diverticula were found, as well as a large abscess round the colon, extending up to the spleen and diaphragm, and involving the pancreas. He was a chronic sufferer from gout. (*Fig. 289, 7 black.*)

*Case 8* (EBNER, 1909).—Male, 65. Nutrition bad. The illness began suddenly while *lifting sacks of grain*. There was slight pyrexia, increasing constipation, and frequent vomiting. The abdomen was markedly tender. Laparotomy showed a twisted omental mass. The intestinal walls were notably pained. Pea-sized diverticula along the whole length of the sigmoid. There was a *sinus running in the walls of the bowel*, opening at one spot into an abscess, in another into a perforated diverticulum. Death occurred rapidly (*Fig. 289, 10 black.*)

*Case 9* (ERDMANN, 1914).—Male, 48. Began like acute appendicitis, after eating a hearty Christmas dinner. Pyrexia. Leucocytosis (19,500). Pain and extreme tenderness in the left iliac fossa. Operation on the fourth day showed an exudate along the greater curvature of the sigmoid, with a small abscess. Two epiploons were engorged, one nearly gangrenous, and perforated. Recovery (*Fig. 289, 7 red, 9 black.*)

*Case 10* (ERDMANN, 1914).—Male, 34. Had sudden severe pain in left flank. A tender mass felt as in *Fig. 298, 7 red*. Pyrexia. Operation showed acute diverticulitis. One diverticulum was tied off. Recovery

*Case 11* (RICHARDSON, quoted by GRAVES, 1911).—Male, 70. Pain in the epigastrium for two days, and then in the right iliac fossa, with tenderness. Leucocytosis (17,000). Rectal examination showed a resistant tender *tumour at the right pelvic brim*. Operation showed pus and fæces in the pelvis. A partly gangrenous epiploic appendage, the size of a plum, was found adherent to the rectum and the coils of intestine. Its cavity opened into the bowel. Death twenty-two days later. (*Fig. 289, 5 black.*)

*Case 12* (RICHARDSON, quoted by GRAVES, 1911).—Male, 52. Symptoms of fistula between bladder and intestine. There had been *attacks of sudden fever for five*

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or six years. Eighteen months ago there was very severe pain in the bladder. X rays showed no evidence of calculus at that time. Operation revealed a thick infiltrated rectum. One diverticulum was resected. Another was not inflamed. Death six months later. There was a fistula into the bladder, a small abscess cavity being present in the adhesions between the two viscera, there were two holes into the rectum, and one into the bladder.

*Case 13 (GRAVES, 1911).—Female, well nourished. Had a faecal fistula into the bladder, which allowed gas but not faeces to pass. Tender mass behind the uterus, and tenderness on deep pressure over the left iliac fossa. Operation showed adhesions to posterior wall of uterus. There was a necrotic area on the sigmoid, with two small diverticula near by. Recovery.*

*Case 14 (HARTWELL, 1910).—Boy, age 6, suffering from an infective condition which proved fatal. At autopsy the colon was found to have sacculated haustra. One diverticulum was found, 2 cm. in diameter (see Fig. 279), opening into the intestine by a circular hole. There were no special symptoms.*

*Case 15 (HARTWELL, 1910).—Female, 43. Nutrition good. Had been ill with pain in the right lumbar quadrant for a week. Constipation. Attacks of abdominal pain for ten years, two or three times a year. A hard adherent tender mass in the right quadrant. Uterus and vagina fixed posteriorly. Operation revealed a mass round the caecum and appendix, associated with a diverticulum which had perforated. This is a case of diverticulum in the ascending colon.*

*Case 16 (LE JEMTEL, 1909).—Female, about 45. There is no record of clinical symptoms (anatomical cadaver). There was perforation of the large intestine at the end of the sigmoid flexure, and adhesion of the Fallopian tube to the site of perforation, so that faeces could pass through into the uterus and vagina. No peritonitis. The left ampulla (Fallopian tube) was much dilated. The mucosa appeared to be continuous from one lumen into the other. The uterine orifice was no larger than would admit an ordinary penholder. Uterus normal in size.*

*Case 17 (KOHN, 1914).—An old man, supposed to have appendicitis. Operation showed hundreds of diverticula, varying from a pin's head in size to that of a cherry stone. One—situated on the transverse colon—had perforated. (Fig 289, 10 red, 10 black.)*

*Case 18 (KOHN, 1914).—Male, 67. Symptoms of chronic obstruction. Pain and tenderness in the left side of the abdomen. Attacks of obstruction for four years. There was a mass in the left lower quadrant which proved to be adherent to the bladder and the pelvic brim. The wall of the sigmoid was very thick, and numerous diverticula were found, varying in size. The stenosed portion was thickened. The case resolved for a time with high enemas. He lived for six years after the operation, thus proving it was not a case of cancer. Death occurred subsequently from oedema of the larynx. (Fig. 289, 9 red, 9 black.)*

*Case 19 (LILIENTHAL, 1910).—Male, 45. There was a febrile illness for weeks. Then the abdomen became suddenly distended, especially in the left iliac fossa. A hard mass adherent to the abdominal wall was found. There was a small abscess between the omentum and sigmoid. There were two inflamed diverticula, one communicating with the abscess. Other diverticula occurred which were not inflamed.*

*Case 20 (MURRAY, 1913).—Male, 67. Taken suddenly ill while at stool. He had to be assisted to bed. The abdomen became slightly distended and very tender all over. Laparotomy showed general peritonitis due to the rupture of a gangrenous diverticulum of the large intestine. There were numerous diverticula into the pelvic colon—some very small, others large enough to project from the walls. The faeces are noted to have been like rabbit-droppings.*

*Case 21 (LITTLEWOOD, 1910).—Male, 50. Had passed faeces and flatus per urethram for four years, during which time he had been unable to work. Stone in bladder. Mass found in sigmoid, attached to apex of bladder. A portion was removed, and showed chronic peridiverticulitis. Recovery.*

*Case 22 (LITTLEWOOD, 1909).—Female, 65. Had passed feces and flatus per urethram for twenty years. There was also a fistula to the abdominal wall, through which feces and urine passed. Chronic thickening of the sigmoid was found, and a funnel-shaped diverticular opening into the bladder and on to the surface of the body in the left hypogastric region. Operation. Recovery*

*Case 23 (MUMMERY, 1910).—Female, 37. Taken ill with signs of acute general peritonitis after a dose of castor oil. There was no previous illness. Operation showed a perforation of the colon in the left iliac fossa. It was bound down by adhesions. A diverticulum had perforated. There were many others throughout the colon. Death in a few hours.*

*Case 24 (MURPHY, 1916).—Male, 53. Ten months ago while in a motor, the patient received a heavy jolt. Shortly after, cramps came on, radiating from the left lower quadrant to the rectum. The cramps were aggravated by food. Medical treatment was begun a month later, and relieved the patient for nine months. Then recurrence, after a heavy meal. Constipation. Stools like sheep's. Mass in left iliac fossa. Leucocytosis (11,400). Operation verified the diagnosis of an inflamed diverticulum. There were adhesions around, and pus amongst them. The sigmoid was indurated and contracted. The mesentery was infiltrated, thickened, and short. *B. coli* was isolated from it.*

*Case 25 (MEARA, 1914).—Age and sex not stated. Cited to show that diverticula will retain bismuth for forty-eight hours after a meal. A further twenty-four hours still showed marked sacculations, in another twenty-four hours the shadows had disappeared. This is instanced to indicate that a long series of plates is needed for x-ray diagnosis.*

*Case 26 (NICOLAYSEN, 1914).—Female, 36. Pains in the lower abdomen for two days, these subsided, but recurred. Again pain, and again subsidence. Then a very severe attack, but no pyrexia, and no constipation. A mass was felt per vaginam. Laparotomy showed a tumour in the mesentery, due to dense fibrosis of the sigmoid, nearly the size of an egg. There was no stenosis of the sigmoid. The mass was adherent to the left ovary and tube (recently). Recovery*

*Specimen removed. The mucosa is normal. The tumour consisted of fibrosed mesenteric fat close up to the wall of the gut. The microscope revealed diverticula. In one section giant cells were found.*

*Case 27 (NEUPERT, 1908).—Female, 23. Patient suddenly experienced severe abdominal pain, with vomiting, and later, diarrhoea. Loss of weight. There had been an attack of very obstinate constipation once before. A hard nodulated mass was felt below the umbilicus the size of an apple, it was movable. Laparotomy showed diverticula full of feces, lying in the large omentum, and connected with the transverse colon. Recovery. Later a relapse, with a tumour in the same place. A second operation showed several small diverticula in the descending colon and three in the caecum. Resection. Recovery (Fig. 289, <sup>3</sup> black.)*

*Case 28 (MORLEY, 1913).—Male, 50. Severe pain in the lower abdomen. Micturition was painful. Absolute constipation. Vomiting. Symptoms of general peritonitis originating in the left iliac fossa. Operation revealed an unusually short mesocolon, with perforation of a gangrenous diverticulum on the antimesenteric border of the pelvic colon. There were other small ones near by. Adhesions to bladder and pelvic colon. Death later.*

*Case 29 (POWERS, 1912).—Male, 49, very obese. Had acute appendicitis seventeen years ago. Now has a 'bilious attack,' with exquisite tenderness between umbilicus and anterior superior spine (see Fig. 289, <sup>4</sup> red). Leucocytosis (26,000). Laparotomy shows a gangrenous (diverticular) mass the size of an apple, half way along the sigmoid. No stenosis. The mass was excised. Death from pulmonary embolism on the seventh day. No peritonitis at autopsy*

*Case 30 (DE QUERVAIN, 1914).—Male, 69. There had been attacks of colic. Present illness that of an 'acute abdomen.' Tenderness, slight resistance, and mobile*

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dullness on the left side. Laparotomy showed a very thick colon, and a very red appendix epiploica, which had not perforated. There was no peritonitis. There were very minute diverticula in the upper rectum. Death took place from *embolism of the pulmonary artery*.

*Case 31 (DE QUERVAIN, 1914).—Male, 61. Indigestion for twelve months. Increasing constipation. The case was examined by x rays, and it was found possible to demonstrate the presence of diverticula thereby (see Fig. 292). There was no mechanical obstruction of the colon. Operation confirmed the finding.*

*Case 32 (REICHEL, 1913).—Male, 34. Seized with violent pain in the left groin while jumping. After this diarrhoea came on, with pyrexia. There was a hard, tender mass above the left Poupart's ligament. Operation revealed numerous diverticula the size of a hazel nut, concretions within them. The perforation of three of these led to a great thickening of the walls, which were riddled with fistulae and abscesses. Death later from peritonitis. (Fig. 289, <sup>2</sup> red, <sup>2</sup> black.)*

*Case 33 (RIXFORD, 1908).—Male, 60. Recurrent attacks of abdominal inflammation. Death occurred from peritonitis following a dose of castor oil. Examination showed that a rectal diverticulum had perforated into the bladder. There was a pelvic abscess opening into the peritoneum and bladder, and communicating with the rectum via the diverticulum.*

*Case 34 (ROBERTS, 1914).—Female, 46. Wasting. Pain in lower abdomen one year. Absolute constipation for one week. No tumour felt. The case proved to be one of multiple diverticula along the whole large intestine. One of these had perforated, producing an abscess cavity which was filled with faecal matter. There were also two in the lower end of the ileum. There was an adenocarcinoma seven inches from the anus.*

*Case 35 (RISEL, 1913).—Male, 49. Violent pain in the appendix region, beginning six days before death. Appendicectomy was performed within fourteen hours. The operation revealed turbid fluid in the peritoneum, but no pus. The diffuse peritonitis was due to gangrene of a diverticulum, with necrosis of the fat of the epiploic body and the mesentery near by. There were numerous typical diverticula in the sigmoid. The feature of interest is the displacement of the sigmoid by becoming adherent horizontally along an old bubo incision, thus reaching over to the right side, and hence causing the right-sided symptoms. (Fig. 289, <sup>10</sup> black.)*

*Case 36 (SHOEMAKER, 1914).—Female, 63. History of pelvic inflammatory attacks. There was a diverticulum in the rectum, and bean-sized indurations along the rectum (presumably other diverticula). There were very firm adhesions to the broad ligament, the ovaries, and the tubes.*

*Case 37 (SHOEMAKER, 1914).—Female, 43, short, muscular, and obese. Came with pelvic symptoms, which proved to be due to a blockage of the pelvis by a large round firm mass. 'Wiredrawn stools' for five years. Operation showed a pus sac provided with very thick walls, not lined by mucous membrane. There was a second abscess near by. There were adhesions between the uterus and rectum.*

*Case 38 (STANTON, 1913).—Male, 30. Previous history was 'unimportant.' Three attacks of left-sided appendicitis. The last one showed a mass as indicated in Fig. 289, <sup>2</sup> <sup>4</sup> black, <sup>4</sup> red. It was movable, and tender, but subsided in a few days. It returned again, and an operation was decided on. A mass the size of half a lemon was found on the middle part of the sigmoid. There were fresh adhesions. Marked peridiverticulitis was found. Oedema of the bowel wall was present. The diverticulum contained a faecal mass the size of a marble. There had never been constipation.*

*Case 39 (SUDECK, 1914).—Male, 52. Painful flatulence for one year. Six months ago a tender mass appeared in the left fossa. There was sugar in the urine, and hence an operation was not deemed advisable. X rays showed numerous diverticula in the sigmoid and ascending colon. Not confirmed by operation. Recovery*



*Case 40 (SUDECK, 1914).—Male, 60.* Signs of stenosis of the sigmoid present. There was mucus in the stools, and a *trace of blood*. Bismuth showed the existence of absolute stenosis in the lowest part of the sigmoid. Operation revealed a circumscribed tumour of the flexure, with diverticula near. There were adhesions to the bladder.

This is a case of '*cancer, with recovery.*'

*Case 41 (SIMMONDS, 1911).—Male, 57* Attack of *acute intestinal obstruction* a year ago. A *second attack* began a fortnight ago. Operation revealed a marked stenosis of the sigmoid (thickening of the muscular, serous, and perisigmoid tissue). There was no ulceration of the mucosa. There were numerous little pits, but none of them had perforated.

*Case 42 (BALL, 1908).—Adult male.* Had intermittent fever for some days, followed by the appearance of an inflammatory swelling at the root of the penis. There was pain during defæcation. The case proved to be one of fistula formation into the bladder, due to a diverticulum, but the latter had become obliterated by the inflammation as in acute appendicitis, and hence there were no bladder symptoms. The *diverticulum was in the rectum, and it led to a perirectal abscess*. It had undergone violent gangrenous inflammation.

*Case 43 (CHUTE, 1911).—Male, 53.* *Sudden attacks of fever for five or six years.* There was also bladder discomfort. The urine had been turbid for some years. Latterly, pneumaturia. Operation showed the sigmoid adherent to the posterior wall of the bladder, well down towards the bottom of the cul-de-sac. The sigmoid was thick and indurated, and there was a pocket of pus between it and the bladder. Two perforations occurred from the sigmoid into the abscess cavity and one from the cavity into the bladder. There was satisfactory recovery. But three months later a similar series of symptoms arose, because another diverticulum behaved in the same way. This was satisfactorily treated, and in twelve days the same thing happened again with another diverticulum. After this death took place.

*Case 44 (CULLEN, 1904).—Female, 60.* Had been liable to attacks of pyrexia, with slight difficulty in defæcation. Vaginal examination showed the uterus slightly enlarged, and a globular mass posterior to it, and apparently continuous with it. The *mass appeared like a myoma*. Operation revealed the fact that this was a case of *rectal diverticula* which had ruptured into the surrounding perirectal fat, producing a definite inflammatory mass. There was a small abscess between the tumour and the pelvic floor. *Fig. 291* shows two diverticula opening into the inflamed cellular tissue. The small abscess between the tumour and the pelvic floor was due to the extension of the inflammatory process to the peritoneum of Douglas's pouch. The diverticula were lined by atrophic mucosa.

*Case 45 (KIDD, 1907).—Male, 49.* Had been ill for about nine weeks with pain in the epigastrium and hypogastrium, steadily increasing in severity, but with intervals of complete freedom. Three days before admission there was difficulty of micturition. Pyrexia. Indefinite swelling in rectovesical pouch. Slight cedema of rectal mucosa. Urine normal. Incision opened an abscess surrounding the sigmoid flexure, which was said to be affected by a carcinomatous ulcer with perforation. Death in four hours.

*Autopsy.*—Omentum adherent to anterior abdominal wall, shutting off upper part of abdominal cavity. Mesentery œdematous. Cæcum and small intestine dilated. Transverse and descending colon contracted. Descending colon passed into a hard mass which filled the pelvis and fixed the bladder. Foul pus in the pelvis. No gross perforation of the bowel. Sigmoid narrowed for six inches, from much thickening of its walls, the increase being almost entirely in the subperitoneal coat, which was three-eighths of an inch thick, pale, hyaline, and hard, *almost cartilaginous*. The muscle was thickened, but not the submucous tissue. Mucosa not ulcerated. Lymph nodes behind the gut contained ragged cavities full of offensive pus, and appeared gangrenous. They were the source of the peritonitis. Microscopic examination showed the most change in the subperitoneal coat, which presented a close

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matting with young fibrillæ, produced by numerous fibroblasts. Small round cells were moderate in number. The picture was one of slowly organizing granulation tissue confined almost entirely to the subperitoneal coat of the gut.

*Case 46 (KIDD, 1907).—Male, 57, brought in dead. Well nourished. Cæcum acutely dilated, its wall œdematous and infiltrated with hæmorrhages. The rest of the colon as far as the sigmoid was distended. The lower end of the sigmoid was firmly bound down amidst a mass of new-formed tissue, which appeared to consist of a mixture of fat and firm hyaline tissue. The lumen was considerably narrowed at this point. It was considered to be a case of carcinoma of the sigmoid, but subsequent examination showed no sign of ulceration. The chief change was in the subperitoneal coat, which was nearly three inches thick in places. The thickening was produced by new fibrillar tissue and fibroblasts forming bundles between which were groups of spindle cells, and chronic inflammatory cells. The mucosa was intact, and there was no evidence of carcinoma.*

*Case 47 (KIDD, 1907).—Female, 7. Had measles three years ago, followed by vaginal discharge due to ulcer. A fæcal fistula appeared into the vagina. Operations were unsuccessful. A further attempt to close the fistula by first performing colostomy, revealed conversion of the sigmoid into a firm indefinite mass about six inches long. The subperitoneal fat and appendices epiploicæ were greatly thickened, and the gut appeared buried in a mass of fat. The whole mass was extremely vascular. The growth was subsequently cut away as being a cancer. At the site of stricture there was a loss of mucosa, elsewhere it appeared healthy. The subperitoneal coat was greatly thickened, and appeared hyaline, and *almost cartilaginous*. Microscopically there was a marked hyperplasia of the connective-tissue elements. Tubercle bacilli could not be found. The child improved markedly*

*Case 48 (REICHEL, 1913).—Male, 64. Had an attack of sudden pain in the left iliac fossa three years before. Constipation. A fistula developed subsequently. There was a hard, tender tumour, which proved to be due to an abscess between the sigmoid and bladder. The patient was well a year later.*

*Case 49 (WALDVOGEL, 1902).—Male, 35. Long history of difficult urination. Signs of fistula about a year before death.*

*Necropsy.*—Marked stenosis of the sigmoid flexure from inflammatory thickening. Above this is a row of deep diverticula. The thickened and stenosed area is adherent to the bladder, six openings—two lined by mucous membrane—lead from the gut into the tissue intervening between it and the bladder, and two of these enter the bladder directly. The gut also communicates with an abscess cavity containing fæculent material and a comparatively fresh apple-seed.

*Case 50 (HERCSZEL, 1889).—Male, 48. Irregular bowel action. Five months' symptoms of entero-vesical fistula with passage of air and fæces per urethram. Stricture of the bowel 12 cm. from the anus, tumour felt in the left iliac fossa. Carcinoma of the sigmoid flexure diagnosed, at operation the bowel was found to be adherent to the bladder. Subsequent colotomy and death.*

*Necropsy.*—Multiple diverticula containing fæcal matter in the whole of the rectum, one had given rise to fistula, stenosis of the bowel, small abscess cavity outside of it. The fistula was 26 cm. above the anus.

*Cases 51, 52 (RIXFORD, 1904).—Epiploic inflammation.* Inflammation of appendices epiploicæ due to infection conveyed from neighbouring false diverticula. The inflamed appendices were palpable as tumours (one of the size of a walnut and one of the size of a hen's egg) and were removed.

*Case 53 (SYDNEY JONES, 1858).—Male, 64. Began to pass fæces and straw-like bodies in the urine for ten months before death. At the necropsy the sigmoid flexure was somewhat contracted and presented a large number of diverticula of unequal length. One had ulcerated at its extremity and had given rise to an abscess outside the gut opening into the bladder. In this there was a fæcal calculus of the size of a horse-bean.*

*Case 54* (SCHREIBER, 1902).—Male, 37 Stout, alcoholic. Six months' abdominal pain, loose stools.

*Necropsy*.—Multiple diverticula (20) of the sigmoid flexure, in two vertical rows, one had perforated. Bowel somewhat thickened and mucosa of the sacs necrotic, contained faecal concretions. Some fibrosis of the mesosigmoid.

*Case 55* (MERTENS).—Male, 45. Eight days' pain in the lower abdomen, with constipation. As a result of *straining at stool* evidence of perforative peritonitis. Origin not located at the operation, but at necropsy found to be due to a perforation of false diverticulum of the lower end of the sigmoid flexure.

*Case 56* (ROTTER, 1899).—Male, 58. Tumour, abscess, stenosis. *Diagnosis carcinoma of the cæcum*. Rumbly and pain in the cæcal region, with alternate diarrhoea and constipation for some time. Obstruction for nine days. In the ileo-cæcal region a large tumour of the size of a child's head, fixed, solid, irregular.

*Operation*.—The sigmoid flexure found to be adherent to the right side of the sacrum, ileosigmoid anastomosis. Tumour was formed by sigmoid flexure (20 cm.) and its mesentery, in which is an abscess. Great thickening of gut with stenosis. Numerous diverticula, six enter abscess cavity.

*Case 57* (ROTTER, 1897).—Male, 53. Stenosis, sigmo-ileic fistula. Symptoms of obstruction for six months. No previous constipation. Operation as for perityphlitis, artificial anus at end of ileum.

*Necropsy*.—In the upper rectum a stricture 4 cm. long and 3 cm. thick, allowing a lead pencil through. Mucosa healthy, but above the stenosis it was rugose, with several fine openings leading to fistulous tracts, these were narrow and communicated with the lumen of the gut and with a small abscess cavity, this was adherent to the vermiform appendix and was in fistulous communication with loop of small gut. *Condition regarded as secondary to a former appendical abscess.*

*Case 58* (STIERLIN).—Aged female. Multiple diverticula above a stenosing carcinoma of the sigmoid flexure. This case is interesting from its association with carcinoma. (*Fig. 289, 11 red.*)

*Case 59* (W J MAYO, 1905).—Female, 65. Stenosis, tumour (diagnosed as ovarian). Chronic constipation with attacks of pain in the lower abdomen for some time. Mass lay down in the left side of the pelvis. At operation found to be the sigmoid flexure, eight inches of which were resected, thought to be carcinomatous. High-grade stenosis of sigmoid flexure due to a fat-containing inflammatory mass in subserosa round numerous thin-walled diverticula. (*Fig. 287.*)

*Case 60* (FIEDLER, 1868).—Female, 22. Nutrition good, chronic constipation. Abdominal pain, distention, and tenderness, maximal in the sigmoid region, diarrhoea. Large abscess in left iliac region extending to the right of the pelvis, numerous perforations due to diverticula, leading from the colon and especially from the sigmoid flexure. Some only ulcerated and contained faecal masses. Adhesion to neighbouring coils. (*Figs. 275 and 276.*)

*Case 61* (STIERLIN 1902).—Male, 71, stout. Incarcerated inguinal hernia. At the operation, *suppuration in the sac* from perforation of a diverticulum of the incarcerated piece of sigmoid flexure, the bowel was much thickened, with an infiltration of the subserous fat sheets. Regarded as carcinomatous and resected. Sudden death in fifteen days from cardiac degeneration.

*Case 62* (MACKIE WHYTE, 1906).—Male, 65. Alcoholic, stout, four weeks' illness with rigors and pyrexia. A limited necropsy revealed *multiple abscesses of the liver*, no pylephlebitis. In the wall of the sigmoid flexure was a cavity of the size of a thrush's egg, with smooth lining, communicating with the lumen of the bowel, the cavity was a diverticulum, and the inflammation was due to a faecal concretion. There were numerous diverticula in the neighbourhood, mostly near the mesenteric attachment, but there was a row on the opposite side. Two or three of the size of a pea or bean contained faeces.

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*Case 63* (HOCHENEGG, 1902).—The patient, age 70, had suffered from constipation. A carcinoma of the sigmoid flexure was resected, the flexure showed for a great distance numerous cherry-sized diverticula, filled with somewhat hard, plastic faecal matter. The carcinoma was regarded as resulting from the chronic irritation and ulceration of the diverticula due to the retained faeces.

*Case 64* (G. W. WATSON, 1907, quoted by TELLING, 1908).—Female, 60. Body stout. No previous history. Left pyonephrosis. In the pelvis a faecal concretion of the size of a small bean, lying free. Another concretion of similar size and shape attached to the sigmoid flexure by a filamentous band about three-quarters of an inch long (*Fig. 284*).

*Case 65* (KOCH, 1903).—Male, 64. Chronic bronchitis. For one year constipation and abdominal pain, with much mucus and *blood in stools*. A tender movable tumour in the left iliac fossa. Diagnosis stenosis from cancer.

*Operation*.—Colotomy. Death from peritonitis. Twenty diverticula of the sigmoid flexure, two entering an abscess cavity adherent to the bladder. Stenosis of the gut, *chronic sigmoid mesenteritis*. No venous stagnation.

*Case 66* (BLAND-SUTTON, 1903).—Female, 60. Chronic intestinal obstruction, mobile tender lump in the left iliac fossa, loose stools. Diagnosis carcinoma.

*Operation*.—A hard oval body in the sigmoid flexure. The adjacent walls of gut were much infiltrated and regarded as carcinomatous, resection. The oval body was found to be a thickened epiploic *appendage containing a foreign body (straw)* and communicating with the lumen of the gut by a narrow channel (*Fig. 271*).

*Case 67* (BLAND-SUTTON, 1903).—Male, 40. Acute pain, swelling, and tenderness in the left iliac region, suggesting *appendicitis with transposed viscera*. At the operation there was an inflamed diverticulum on the descending colon. This entered an epiploic appendage and contained faeces and a *foreign body*. Resection of the appendage, recovery.

*Case 68* (GEORGI, 1901).—Male, 64. For some years alternate constipation and diarrhoea. In the left lower abdomen an ill-defined immobile tumour, diagnosed as carcinoma. At the operation the tumour was deemed to be *inoperable because of adhesions*. Death from peritonitis following straining at stool.

*Necropsy*.—In the lower half of the sigmoid flexure a stricture 3 cm. long was discovered. Above and below the stenosis were several diverticula admitting the tips of the little fingers, one of these entered an abscess cavity (5 by 2 cm.) between the muscularis and the much-thickened serosa. This communicated with several smaller diverticula, so that the *strongly thickened serosa* in its whole extent was *undermined by fistulous tracts and abscesses*. One of these had perforated, giving rise to acute general peritonitis.

*Case 69* (GUY'S HOSPITAL MUSEUM, No. 945).—Male, 65. Vesico-sigmoid fistula. Admitted for passage of wind (twelve years) and faeces (six weeks) from the urethra. Colotomy, death in three days. The patient had been in the tropics but had not had dysentery.

*Necropsy*.—Signs of former ulceration were observed six inches from the anus, but none higher up. Description in catalogue "Much thickened sigmoid which is contracted to some extent, unduly rugose but practically normal mucosa. There is a very chronic perisigmoiditis and a large mass of inflammatory tissue between it and the bladder, a small fistulous communication exists between them." This is an instructive and typical specimen the thickened, stenosed, and rugose bowel, with two inches of inflammatory thickening between the bladder and the bowel. Into this can be seen extending in two or three places diverticular passages, cut more or less obliquely across, and lined with smooth mucous membrane. Beneath the rugæ, and not obvious until searched for with a probe, are the small orifices of many diverticula half an inch or more in depth. The fistula is a tortuous passage communicating with a small nut-sized abscess cavity.

*Case 70* (1907).—Male, 54. Well nourished. No constipation. *Chronic obstruction of the small intestine* by adhesion to the sigmoid flexure. Illness began December,

1905, with sudden abdominal pain, rigor, and vomiting several times. Diarrhoea for three days. Afterwards colicky pains at intervals. Steady loss of weight. At Easter, 1907, he had another attack of vomiting with rigor and pain in the lower abdomen. On July 19 another attack of abdominal pain was experienced, but no vomiting. On July 26 there was marked distention in the right iliac region, when an attack of colic was experienced this tumour became as large as a child's head and there were peristaltic movements to be observed over it. No solid tumour was to be made out. A diagnosis of chronic intestinal obstruction, possibly of the small gut, was made, carcinoma was not thought probable owing to the length of history (twenty months) and the absence of the general appearance of cancerous cachexia. He was operated on the same day.

*Operation.*—The small intestine was obstructed by adhesion to a mass in the left side of the pelvis, and this mass was found to be the lower part of the sigmoid flexure. It was thought to be malignant, and *owing to the extensive adhesion removal was deemed impossible*. Lateral anastomosis was performed. Death took place on Aug 2 from general peritonitis, due to stercoral ulceration.

*Necropsy.*—The lower two-thirds of the sigmoid flexure presented numerous small diverticula, arranged mostly in two lateral rows. Some contained faecal material. There was chronic inflammatory perisigmoiditis, the lowest three or four inches of the gut having a wall half an inch thick. Some narrowing of the lumen existed, but no real obstruction. There was marked adhesion to the wall of the pelvis. On separating this a small abscess cavity was found into which at least one of the diverticula led directly. There was strong adhesion to the bladder, and between it and the bowel a 'double' abscess cavity existed, with a similar communication with the bowel. There was much general matting of the parts around, whereby a loop of small intestine had become adherent and partial chronic obstruction had been caused by stenosis due to the inflammatory exudate. The small gut above the obstruction showed much thickening of its walls from muscular hypertrophy. There was some dilatation, below, the walls and lumen were normal. The mucous membrane of the diseased sigmoid flexure was normal, except for marked rugosity with a tendency to polyposis of the folds of the mucous membrane. Many of the diverticula were concealed beneath these folds. The orifices were small in the thickened area (*Fig. 286*).

*Case 71 (GRASER, 1898).*—Symptoms and diagnosis of carcinoma of the sigmoid flexure. Stenosis of the sigmoid flexure 5 cm. in extent, with much thickened walls. Adherent to the bladder. Thirty-five diverticula present in the stenosed area, as well as others above and below the stenosis.

*Case 72 (MOYNIHAN, 1906).*—Male, 52. Chronic constipation. Symptoms of chronic duodenal ulcer. *Recurrent intestinal obstruction, complete on third attack*, led to supplementary diagnosis of carcinoma of the large bowel. A mass felt in the sigmoid flexure adherent to coils of intestine and the abdominal wall, five inches excised. Great thickening (one and a half inches) of a piece of gut due to cicatricial connective tissue causing stenosis. The mucous membrane was intact but showed numerous diverticula (*Fig. 285*).

*Case 73 (W. J. MAYO, 1906).*—Female, 52. Chronic constipation and obstruction. Periodic attacks of pain in the lower bowel for five or six months. A left-sided pelvic tumour, operation for chronic obstruction. Death 18 days after from general peritonitis. Resected sigmoid flexure 17 cm. long and 6 cm. thick. Thickening of the sigmoid flexure due to chronic inflammation from the presence of numerous diverticula. Many contained enteroliths, *none showed inflammation within the sac*.

*Case 74 (LOOMIS, 1870).*—Male, 61. Well nourished, constipated. In the sigmoid flexure and the descending colon were multiple diverticula filled with faecal matter. They were much thinned out but had *not* perforated, nevertheless, there had resulted general peritonitis.

*Case 75 (SIR ERASMUS WILSON, 1884, No. 2455D, ROYAL COLLEGE OF SURGEONS' MUSEUM).*—Portion of the colon with dilated haustra, upon these are numerous

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diverticula varying in size from one-third to three-eighths of an inch in diameter. They contain faecal concretions.

*Case 76 (SCHREIBER, 1902).—Male, 67 Stout. Chronic bronchitis. Carcinoma of pylorus and liver. About 60 diverticula from the splenic flexure to the sigmoid flexure, where they are much more numerous and at the end of which they stop abruptly, reach the size of a cherry in places. Most are near the attachment of the mesosigmoid, some into the appendices epiploicæ, some elsewhere.*

*Case 77 (EDEL, 1894).—Female, 73. Chronic constipation, fair nutrition, arteriosclerosis. Seven diverticula in the jejunum, containing faeces. Many in the whole extent of the large intestine, most numerous in the sigmoid flexure. Mostly at the mesenteric attachment, but some on the free surface. All enter the appendices epiploicæ and contain faeces.*

*Case 78 (WALLMANN, 1858).—Male, 69. Two diverticula in the sigmoid flexure, three in the descending colon, three in the transverse colon, one in the ascending colon, and seven on the convex surface of the bowel.*

*Case 79 (FISCHER, 1900).—Male, 40. Chronic passive congestion in cardiovascular disease. Twenty-five diverticula in the rectum.*

*Case 80 (HANSEMANN).—Male, 85. Always thin, no constipation. Death from pneumonia. Four hundred diverticula in the jejunum and upper ileum (where empty), in the transverse colon to the sigmoid flexure (most numerous in the latter), containing faecal lumps. Situated chiefly on the mesenteric border, but some at the sides entering the appendices epiploicæ.*

*Case 81 (G. W. WATSON, 1907, quoted by TELLING, 1908).—Male, 65. Death from ruptured aortic valve. The whole sigmoid flexure is fatty and presents marked dilatation and sacculation of its normal haustra, in addition there are numerous diverticula which in most cases enter the appendices epiploicæ. The pouching stops abruptly at the commencement of the rectum. The sacculation and diverticula are filled with hard faecal masses, the mucosa is normal. Some of the diverticula present small orifices at the bottom of the dilated haustra, in some cases the haustra themselves diminish gradually and end as diverticula entering an appendix epiploica. There are certainly two distinct conditions (1) Sacculation of the haustra; and (2) Diverticulum-formation. Externally many of the appendages present inflammatory changes, some are adherent one to the other (in one case forming a 'ring'), in one there is a hard, cherry-sized faecal concretion, inflammation around which has led to obliteration of the orifice of the diverticulum and to the formation of a pedicle—that is, the first stage in the process of detachment and the formation of a free body in the peritoneum.*

*Case 82 (1906).—Female, 73. Death from strangulated femoral hernia. Obese. Much fat on the large gut, the last three feet of the descending colon and sigmoid flexure present numerous diverticula, arranged for the most part in two rows, entering the fatty appendices epiploicæ. But some are at the sides of the appendages or into the mesosigmoid, thus there may be several diverticula at the same level. They cease abruptly at the commencement of the rectum. The orifices are much smaller than the pouches and are somewhat 'lipped' by mucous membrane, this latter is unduly rugose but otherwise normal. The diverticula are most numerous and largest in the lower twelve inches of the sigmoid flexure, in places they appear 'bunched' together (when the appendical fat is removed) like clusters of currants, which is the prevailing size, though both smaller and larger are found. They contain faeces. Where most thickly placed the gut wall is thickened and there is some narrowing of its lumen (Figs. 269, 270).*

*Case 83 (MOYNIHAN, 1901).—Male, 55. Acute obstruction of the small intestine by adhesion to the sigmoid flexure. Admitted with five days' history of abdominal pain, vomiting, and constipation. Moderate abdominal distention, with peristalsis due to distended coils of small intestine. At the operation the small intestine, some*

distance above the ileocaecal valve, was found to be obstructed by an adhesion in the pelvis, the nature of this was not determined, but the adhesions were separated. Double enterotomy was performed, and the intestinal contents were evacuated. Death in five days from general peritonitis.

*Necropsy.*—General suppurative peritonitis, the coils of intestines above the obstruction were gangrenous. There was much thickening of the bowel, commencing four inches from the anus and extending upwards for about five inches, the thickening lessened from below upwards. There was *considerable periproctitis* and higher up peri- and meso-sigmoiditis, with adhesion to the pelvic walls. In the sigmoid flexure were numerous diverticula arranged in two rows and mostly entering the appendices epiploicae, they varied from pea- to nut-size, they contained faeces, and in the lower part of the sigmoid flexure were mostly ulcerated inside. Several had perforated and led into small abscess cavities which were in the midst of the inflammatory thickening above noted and had evidently given rise to it. The inflammatory changes in the diverticula had led to strong adhesion to a loop of small intestine, and this had thereby become kinked, leading to the acute obstruction.

*Case 84 (GUY'S HOSPITAL MUSEUM, No. 1071).*—Male, 51. Strangulation by band. Admitted for anal fistula. Acute intestinal obstruction occurred after the administration of an enema. At the necropsy two appendices epiploicae of the sigmoid flexure were found to be adherent to the mesentery of the ileum just above the caecum. *This adhesion formed a band* below which was strangled a loop of ileum eight feet in length. On examining the specimen its diverticular origin is apparent. A diverticular sac leads into one of the adherent appendices. Below the aperture of this are two other diverticula, one of which has a depth of half an inch into another epiploic appendage. There are also some thickening and narrowing of the gut.

*Case 85 (JAMIESON, quoted by TELLING, 1908).*—Male, 76. Well nourished. Death from chronic bronchitis. Adhesion of omentum to rectovesical pouch. Lowest ten inches of the sigmoid flexure presents two rows of diverticula entering the appendices epiploicae for the most part and containing a little faecal material. Two inches from the lower end of the sigmoid flexure is a *polypus of the size of a cherry*, and there is a marked *hypertrophy of the longitudinal muscle bands*, increasing from above downwards to the point of attachment of the polypus, where the hypertrophy abruptly ceases. There are a few diverticula below the polypus, they cease at the commencement of the rectum.

*Case 86 (MOURE et DESBOUI, 1909).*—Male, 70. Was seized suddenly with very violent abdominal pain (*Fig. 289, 3 red*), pyrexia, rapid pulse, and respiration. Proved to be an acute perforating sigmoiditis. Histological examination proved its origin in a diverticulum.

*Case 87 (MUMMERY, 1910).*—Female, 38. Developed sudden severe pain ten days before while at work. At the end of a week the pain was extreme, and pyrexia and rapid pulse were present. A mass was felt as in *Fig. 289, 1 black*. There was marked leucocytosis. An abscess was found extending backwards to the posterior abdominal wall, and along the inner side of the descending colon. Recovery.

*Case 88 (STANTON, 1913).*—Male, 48. Was taken ill with nausea and vomiting and slight pyrexia. There was a tender area as shown in *Fig. 289*, and a large tender movable mass, the size of an egg, as shown at 2 black and 2 red. The condition gradually disappeared without operation.

*Case 89 (SOUTTAR, 1914).*—Female, 59. Was seized with sudden abdominal pain. There was extreme deep tenderness over the whole right side (*Fig. 289, 12 red*), with a mass to be felt in the right iliac fossa. There was a perforation of a diverticulum of the ascending colon.

*Case 90 (DUNN and WOOLLEY, 1911).*—Male, 45. Recurrent attacks simulating appendicitis, but on left side. Leucocytosis. Indicanuria. Tenderness as in *Fig. 289, 1 and 4 red*. Proved to be an abscess under the sigmoid, due to chronic perforative diverticulitis. Fatal.

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**Case 91 (EISENBERG, 1914).**—Male, 45. Attacks like renal colic. Mass and tenderness as in *Fig. 289*, <sup>5</sup> red. Operation revealed a very adherent tumour produced by thickening of the sigmoid, with four diverticula embedded in the wall.

**Case 92 (HARTWELL, 1910).**—Male, 42. Had signs of acute obstruction. There was a tender mass as in *Fig. 289*, <sup>6</sup> red. Operation showed a walled-off abscess outside the sigmoid, arising from a perforation of a diverticulum. The walls of the sigmoid, and the mesentery, were much thickened, but there was no stenosis of the lumen of the gut.

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