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Diverticulosis and diverticulitis of the large bowel

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DIVERTICULOSIS And DIVERTICULITIS

Of The Large Bowel

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TABLE OF CONTENTS

INTRODUCTION

> "The romance of medicine lies in inductive philosophy, in which tomorrow is the great day. Yesterday furnishes the deductive philosophy, which acts as a compass to keep our directions true." Mayo(85)

Diverticulosis and diverticulitis are more than meets the physician's eye. They are problems which will test anyone's diagnostic acumen and ability. The following pages contain a brief resume of some of the more important literature written about diverticulosis and diverticulitis. Similar phases of the literature have been grouped into sections. Certain phases, especially etiology and diagnosis, have been emphasized more than the others.

Despite the fact that diverticulosis and diverticulitis, as gathered from the literature, are found only in a small percentage of the population, it is my opinion that it is more common than the majority of physicians think. The mere fact that there is no definite method of diagnosis and treatment which could be gathered from the contradictory views mentioned in the literature, denotes that the last word has not as yet been conceived concerning various phases of diverticulosis and diverticulitis.

DEFINITION

Berman and Bauer (13), without exception, probably had the simplest definition of diverticulum. They believed that literally it meant a by-path. Yet to be exact, one should no doubt consider the definition proposed by Edwards (36). He stated that the word diverticulum came from the Latin "diverto"-I turn aside- and the "culum" is a diminutive ending equivalent to the English "icle", as in follicle and cuticle. Thus a diverticulum means literally "a small turning aside", that is, a small pouch. When diverticula are present in the large bowel, the condition is termed "Diverticulosis". If these diverticula become inflamed, the condition is termed "Diverticulitis".

Gant (49) presented an interesting definition when he said that a diverticulum is a non-neoplastic outpouching of intestine having a lumen that does or did connect with the bowel. However, one can find fault with that, as a diverticulum is not just found in the intestine. Buie (18) considered a diverticulum as a blind tube or sac but he said they may branch from cavities or canals. Brown (15) had the same idea when he claimed that a diverticulum

may extrude from the wall of any hollow viscus.

Abell (2) was of the opinion that diverticula were noted at all points in the intestinal tract from the pharyngo-esophageal junction to and including the rectum.

It also is of interest to note the following two definitions. Fraser (47) quotes the "Century Dictionary" and "Oxford Dictionary" as giving a diverticulum a designation of " a way-side shelter or lodging", with from the context the underlying meaning that they are "houses of ill repute where trouble is apt to brew". From the complications of diverticulosis as affecting the colon he could vouch for such an evil reputation, also for their annoyance both to their owners and to the doctors called in to try to establish law and order.

One appreciates why Haggard (56) spoke of sigmoid diverticula as a "blowout of the sigmoid", because almost everyone has watched with anxious expectation a diverticulum of his bicycle or automobile tire.

The fact that in their symptomatology, di-

verticula offer a counterpart of other diseases common to the intestinal tract at the points at which they occur lends an added interest to their study. They occur as sacculations projecting from varying portions of the circumference of the intestinal tube-mesenteric, antimesenteric and lateral - and range in number from one to several hundred.

HISTORY

Ochsner and Bargen (91) believed that the and incidence of diverticula of the large intestine had offered a fertile field for study and speculation for several centuries. However, the first complete description to be found in the literature of diverticula, in which no doubt existed as to the nature of the diverticula in question, was by Cooper in 1844. Cruveilheir (27) described diverticula of the colon in 1849. Others who reported on diverticula were Virchow in 1853, Habershon in 1857, and Jones in 1858. The condition appears to have excited little comment at that time, and very few other references are to be found until the first decade of the twentieth century, although Osler reported a case in 1881 and Fraser presented a paper in 1889.

Cooper (25) described numerous pouches of the upper part of the jejunum in a man of 65.

Virchow (108) described certain pathologic changes involving the descending colon and the sigmoid characterized by isolated circumscribed adhesive peritonitis. He even described possible complications yet, he did not note the presence of

diverticula as the original cause, he considered constipation as the etiologic factor and he did not even attempt to describe the clinical picture.

Habershon (54) published the first account of diverticulitis in English.

Jones (65) reported a striking case of acute diverticulitis which resulted in a fistulous connection between the bladder and the sigmoid.

Osler (92) found at postmortem in a man of 65, who had suffered from rumbling noises after food, 55 diverticula in the upper part of the jejunum, ranging in size from a cherry to a large apple.

Graser (51) emphasized the frequency and significance of the disease. He emphasized the importance of, and 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. The first comprehensive treatise, embodying both etiology and classification was brought out by Beer (11) in 1904. This has been followed by papers written by Drummond, Telling, Hartwell, Brewer, Rogers, Mailer, and others. (85)

CLASSIFICATION

Abell (2) decided upon a simple classification and concluded that diverticula had been described as congenital or true, and acquired or false, the former containing all the coats of the intestinal tube, the latter representing protrusions or herniations of the mucous and submucous coats through apertures in the muscularis. The term diverticulosis connotes the presence of such sacculations while diverticulitis implies the varying changes which occur as the result of irritation and inflammation.

Several writers questioned the accuracy of any such terminology and attempted to bring forth other classification which they thought would clarify the subject.

Levitt and Goldstein (75) divided diverticula as follows;

1. True congenital. (Wall composed of all coats of the intestine)
2. Acquired.
   (A) Primary. (Hernial protrusions of the mucous and submucous coats through a tap in the muscular coat)

(B) Secondary.

(a) associated with disease in the intestinal wall.

(b) traction, due to adherent fibrous tissue.

Buie (18) stated that congenital or acquired diverticula may either have the muscular coat or not have it, therefore, he suggested the following classification:

1. Prenatal diverticula: Under which he implied that there were two types: (A) the first type form as true anomalies, and all walls of the colon are included in the walls of the sacs. (B) the second type are hernias of the mucous membrane between the developing muscular structures of the colonic wall and therefore, their walls consist of mucous membrane and serosa.

2. Postnatal diverticula : Here he also believed that there were two types. The walls in both types, however, consisted of the same layers, namely, mucous membrane and serosa. Therefore his separation into two types did not depend on the presence of or absence of the muscular layer but, depended on why and when they formed. (A) The first type form be-

cause the wall of the colon is weak between imperfectly encircling muscular structures. These are seen in adolescence. (B) The second type form because the physiologic processes within the colon are sufficiently abnormal to force the mucous membrane through or between what would be presumed to be adequately encirling muscular structures. These are seen in adult life.

Diverticulitis does not have a definite classification either. Jones (68) classified diverticulitis into three groups: (A) Diverticulitis with enterospasm. (B) Diverticulitis with infiltration. (C) Diverticulitis with perforation. Yet, he believed that these were merely progressive stages of the same pathologic process and that two or more stages may be present at the same time.

Case (21)(22) likewise divides diverticulitis into three quite similar groups: (A) Enterospasm type, which includes that type of case in which the diverticula even though numerous are scattered and where the symptoms are apparently only those of enterospasm, kept up by continuous renewal of intestinal irritation from the retained contents of the diverticular sacs. (B) Hyperplastic type,

where due to being situated fairly close together and due to the peridiverticular inflammatory reaction with its consequent production of connective tissue, tends to produce a tumor with resulting organic obstruction. (C) Pseudoappendicitis type, where one or more of the diverticula undergoes an acute inflammatory process analogous to that occurring with acute appendicitis.

Probably the best suited classification for a general study of diverticula is the one proposed by Newton (90) in which he combined the clinical picture with the pathologic changes. He divided diverticula into five groups: (A) Cases showing no clinical symptoms. (B) Cases showing symptoms but no abscess, fistula or peritonitis. (C) Cases showing peridiverticulitis with abscess, fistula, and peritonitis included. (D) Cases showing peridiverticulitis plus obstruction. (E) Cases showing coincident carcinoma.

ETIOLOGY

Regardless of the cause - upon which there is no unanimity of opinion - Abell (2), Rankin and Brown (98), and several others seem to have the correct idea when they admit that some may be congenital in origin, others acquired, or, both may be present at the same time. They do not specifically state that diverticula are due to one cause as several authors have done.

While diverticula of the colon may be single, they are as a rule multiple, varying in number within rather wide limits. Practically all of those observed in the colon are of the false or acquired type, the sac consisting of mucosa and submucosa covered with peritoneum.

However, the fact that they are of the acquired type and occur late in life has not prevented the assumption by some that the fundamental cause is an inherent weakness of the intestinal musculature which has existed since birth. In other words, that they are essentially congenital in origin. Since they are of the pulsion type, it is logical to assume that muscular weakness, whether congenital or acquired, is the fundamental factor and increased

intracolonic pressure the exciting cause in their production. It has been argued that they occur as the result of the weakening of the intestinal musculature by the piercing of the muscles by the vessels of the bowel wall. Their appearance at points other than those corresponding to the entry and exit of vessels would clearly indicate that other factors play a part.

Erdman (42) and Heinz (59) simply concluded that all diverticula are congenital. Bunch (19) stated that a congenital diverticulum occurs in one of every two hundred persons and is present at birth, but didn't consider any other etiological agent or mention whether diverticula were present other than above.

Other opinions could be taken literally to mean that all diverticula are congenital, yet the author did not state it as such. For example, Guthrie and Brown (52) stated that diverticula were mostly congenital. Lynch (80) stated that in his mind, the evidence in favor of the acquired theory is not convincing. Jones (66) came to the conclusion that after looking over the literature it seemed more than likely that there is some congenital weakness

of the bowel wall, either an irregular distribution or thinning of the circular muscular fibers, or unusually large openings about the perforating vessels leaving an area of weakness.

Evans (44) reported a case of ileocecal cysts. He concluded that they were developmental enterogenous cysts because any cyst having the structure of gut must have been derived from the primitive intestinal tract. He believed that the cysts in his cases originated either in the vitello-intestinal tract, or in the diverticula which are found in the developing entoderm of the embryo.

Lewis and Thyng (77) mentioned that the bowel was developed from a diverticulum and from it, as diverticula, arise the liver, pancreas and the cecum with its degenerate end, the appendix. Sporadic secondary diverticula may occur in regions where diverticula are a normal developmental necessity or they may be merely structural imperfections of early fetal life, as in the case of the outgrowths of intestinal epithelium perforating the muscular coat, which occur about the third month and which may later on, be responsible for diverticula.

Poncher and Milles (96) provided a theory that diverticula or accessory epithelial nodules present in embryo's probably ordinarily disappear. They believed that these may originate in one of two ways: (1) They may arise as sequestrations in the process of development of the anterior abdominal wall, narrowing and finally pinching off the vitelline sac. (2) They can be accounted for as supernumerary outgrowths, the prototypes of which give rise to the lungs, pancreas, and liver.

Haggard (56) spoke of sigmoid diverticula as a "blowout of the sigmoid" and believed that the cause of the herniation of the mucosa was the replacement of the muscle by fat tissue. Erskine (43) stated that this theory was utterly fantastic because the walls of the colon were very tough and it seemed inconceivable that great enough pressure could be generated to produce a blowout since the colon is essentially a tube, more or less open at both ends, and provided by nature with a vent at one end to take care of just such emergencies. He also stated that if a weak spot in the wall were expanded into a pocket, its wall would continue to become thinner and weaker. The gas pressure

would not be diminished and therefore the pocket would likely continue to expand until it reached the bursting point, in the same way as the inner tube blows out. Perforation, however, does not occur in this way, therefore, he prophesied that eventually diverticulosis of the colon would be considered a congenital anomaly.

Bearse (9) implied that diverticula are probably an inherent weakness is the structure of the colon, especially in young persons.

Spriggs and Marxer (102)(103) presented many points that were supportive to the congenital origin such as : (1) There is often no evidence of inflammation, dilatation or hypertrophy of the adjacent bowel. (2) The inner concave aspect of the horseshoe shaped duodenum, from which most pouches arise, is supported by the pancreas and should be less liable to extrusions from weakness of the wall than the convex aspect. (3) In some diverticula the mucous membrane at the apex is thrown into folds, which seems to make it unlikely that these are herniae from internal pressure. (4) The pouches are frequently single. (5) The

outgrowth of the pancreas and liver as outgrowths from the embryonic gut suggests that other outgrowths are not improbable.

They also presented the alternative view that diverticula are caused by extrusion of the parts of the bowel wall which are weak naturally or are made so by ulceration or other pathological processes. However, they believed that the etiology of the pre-existing diverticula was as yet unknown and offered evidence that the multiple colic diverticula were preceded by a local condition, the prediverticular state, in which there was the appearance of irritation if not of inflammation involving sometimes the whole circumference of the gut.

To them, the prediverticular state implied the formation of the small herniae which then push their way through the weak places in the bowel wall. This is the important active stage of early diverticulosis. After progressing past the prediverticular state, the haustra and segments tend to lose their symmetry probably due to the muscle fibers being thinned in places with corresponding bunching of the part which retains its elasticity.

It appears, probably, that damage to muscle fibers occurs at this stage and, as a consequence, the mucous membrane herniates at weak places to form the little pockets.

The definitely localized appearance of areas of bowel in the prediverticular stage indicates a local irritation in the early stage of the disease, and a bacterial cause is the first one that suggests itself. Such a cause would be more likely to be found particularly if the area were in reach of a sigmoidoscope and the secretion lying over the affected part could be studied.

Another view supporting an inflammatory origin of diverticula associated with bacterial activity is that diverticula occur most frequently in the sigmoid flexure. This is the part of the bowel where fluid fecal matter is in contact longest with the mucous membrane of the gut and would be particularly favorable for toxic damage and bacterial invasion of the bowel wall.

They presented 58 cases of diverticulosis and showed the common association of diverticulosis with septic teeth, especially apical abscesses, and with arthritic changes in the spine. They

believed this might be explained on the view that they are both due to infective or both due to degenerative processes. They reported the presence of spondylitis in 21 of 58 cases, apical abscesses in 18 of 58 cases, apical peridontitis in 12 of 58 cases and the teeth nearly gone in 24 of 58 cases. They concluded that the occurrence of the prediverticular state and afterwards of formed diverticula in patches, in various parts of the bowel, suggests a local disease rather than any mechanical cause. The isolated diverticula occasionally seen, especially in the ascending colon and hepatic flexure, may be accidental pulsion pouches.

Lockhart-Mummery (78) admits that there are congenital diverticula and states that no one should doubt their origin. However, he does not believe that diverticulitis occurs in congenital diverticula but that it occurs only in acquired diverticula. Facts against their congenital origin are that multiple diverticula practically only occur after middle age, and that since many cases have been watched by X-rays over a considerable length of time it is obvious that the condition is

often, if not always, progressive. He had a marvelous opportunity to observe diverticula formation in his surgical work and it was his opinion that the development of multiple diverticula is the primary stage and is unaccompanied by any inflammatory change as presented by Spriggs and Marxer (102)(103). The earliest stage observed by him in the development of diverticula was the presence of a large number of small millet seed projections arranged in rows on the outside of the colon. These were along the edges of the longitudinal muscle bands, and occured at the points where the lymphatics and blood vessels perforated the muscular coat. Only in one case was there any inflammation noted about the diverticula and this was probably the earliest stage of an extensive diverticulitis. In other cases there was no inflammation present at all.

The process which starts the formation of diverticula is only conjecturable, but it seems probable that some degeneration of the bowel wall, probably from age and unnatural conditions of the bowel function, is the initial factor. Then the resulting pulsion hernia through the bowel wall

and later retained fecal content is likely to set up inflammation which spreads to the neighboring tissues. This secondary inflammation may never occur, or it may take place at almost any stage in the development of the pouches.

David(28) stated that there were several anatomical arrangements in the colon that may predispose to diverticulosis. The three tinea of the colon include practically all of the longitudinal muscle of the colon. Between these tinea isolated bits of the longitudinal muscle may be found, but in the main the muscularis in these areas is composed of circular muscle alone. The pleated or gathered appearance of the colon is due to the formation of three rows of sacculations between the longitudinal muscle bundles. These sacculations are separated by falsiform ridges composed of all layers of the intestine, as much mucosal as muscular. The circular muscle is reinforced in them and augmented in volume. These sacculations and ridges of the colon retard the passage of feces.

Willey (115) believed that the build and temperament of the patient as well as any temporary mental worry or powerful emotional disturbance had

a very close connection with pathological conditions of the large intestine. Impassioned temperament was considered as being the most powerful predisposing factor. He stated that the colitis of worry evidently resulted from diminished trophic innervation and tissue resistance, while at the same time the natural digestive ferments are faulty in quantity and quality. The food and mucous lining become morbidly septic, the B. coli becomes more and more virulently active. Probably many follicles are infected, which may account for the diverticuli being multiple.

Dixon (31) said that the method of formation is not known exactly but he thought it was a herniation of the mucosa to a subserous position through the muscular layers of the intestinal wall. This herniation probably occurred at the sites of least resistance probably at or near the openings through which the blood vessels penetrate the wall of the bowel.

Edwards (37) presented an extensive review which corraborates Dixon's opinion. He discussed two factors which he considered as the causal factors of the herniations: (1) The presence of a weakened

area - a locus minoris resistentiae - in the bowel wall. (2) A pulsion force acting from within the bowel which initiates the process of herniation.

Under locus minoris resistentiae he implied that the consistency with which these diverticula are associated with the blood vessels refutes the possibility of coincidence. The arteries supplying the intestine form a series of arches about one inch or more from the margin of the intestine and from these, segmental arteries pass to the intestinal wall. The arteries pierce the intestinal wall in pairs and on either side of the mesenteric line and a variable distance from it, but not more than three sixteenths of an inch away. (Refer to figure I on following page.)

In most cases the arteries of each pair are exactly opposite each other, but in other cases they alternate, therefore, there is not a completely regular arrangement. When the arteries reach the wall of the intestine, on either side of the mesenteric line, they pierce the muscular coat to reach the submucosa. Some fine twigs are given off before the main vessels pierce the muscular coat and pass toward the antimesenteric border

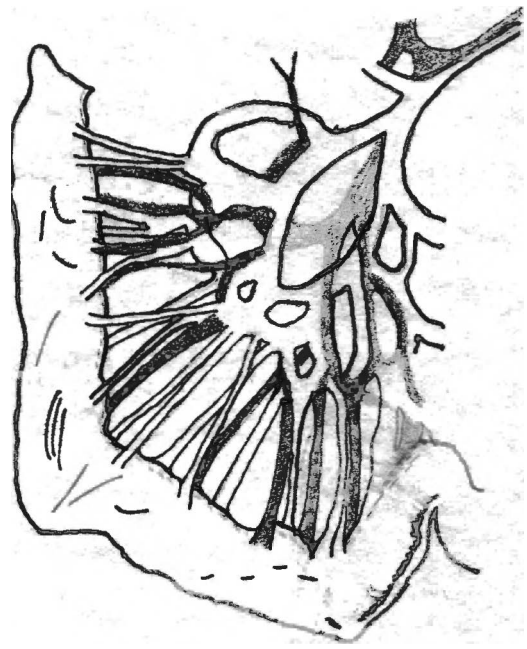


Fig. 1. "Dissection of the vascular supply of the jejunum. The midline is indicated by the interrupted line. The terminal ve sels enter the bowel on either side of the midline." Edwards (37).

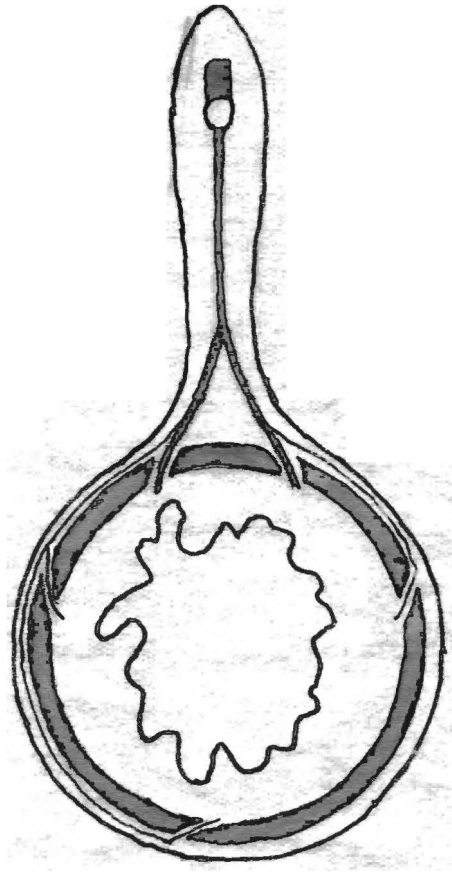


Fig. 2. "Diagrammatic representation of the mode of entry of the blood vessels through the muscular wall of the bowel." Edwards (37).

between the muscular and serous coats. The blood vessel passes through both muscular coats producing a well defined gap. The entrance is about 60 degrees to the muscular coat so that the gap is not a very oblique one. (Refer to figure II on preceding page.)

Practically the whole blood supply to one-half of a segment of intestine is conveyed by this single vessel, which terminates in branches in the submucous coat. These terminal branches are somewhat congested as the result of injection under pressure. There are thus only two large gaps occasioned by entry of the blood vessels in each segment of intestine, and they are situated on either side of the mesentery, and a short distance from it. The angle of penetration of the vessels, moreover, is such that the opening in the longitudinal coat is directly in line with that in the circular coat. Where any branches pierce the muscular wall farther away from the mesentery, not only are the gaps much smaller as the vessels are smaller, but the angle of penetration is far more oblique, and thus does not tend to create the same degree of weakness in the intestinal wall. Thus his

thought that diverticula are hernias of the mucous membrane through the muscular wall, at the point where a gap is caused in the latter by the entrance of blood vessels, is indisputable.

Under pulsion force he stated that passive distention of the wall from pressure of its contents was not a factor but the pressure was due to the action of the muscular coat. Under normal conditions of contraction there is no prolonged increase in pressure. Therefore, in order to get an increase in pressure one must postulate some irregularity in muscular contraction. If there is an irregular contraction of the muscular layers, so that one set of fibers is in spasm and the adjacent fibers are relaxed, there is alteration from the normal, both in local pressure inside the lumen and the resistence of the wall to that pressure. He stated that such local contractions and relaxations of the muscularis have been witnessed in the colon.

During the maximum contraction of the muscle, the vascular gaps are temporarily obliterated as they pass through two sets of muscle fibers contracting at right angles to one another. During the wave

of relaxation the vascular channels gape to their maximum extent and constitute a very weak area in the bowel wall. Under normal conditions there is no tendency toward herniation of the mucous membrane through these areas as the contents of the bowel are hurried along and there is none but a temporary rise of pressure inside the lumen of the bowel.

There may be considerable local increase in pressure if there is persistent irregular contraction of the bowel. Such a condition would be fixed spasm occurring in two segments of intestine, with the intervening segment relaxed. The pressure in the relaxed portion is increased as the bowel content can not be passed on and the contents are forced against the mucous membrane. This tends toward herniation of the mucous membrane through any weakness of the muscle fibers. The vascular channels, gaping to their fullest in the relaxed muscle wall, offer the mucous membrane loci minoris resistentiae.

Butler (20) thought the pull of the vasa recti of the superior and inferior mesenteric arteries as a result of sclerosis was an important factor in

the formation of diverticula.

Berman and Bauer (13) remarked that there was no proof of their idea but they believed that since the veins were larger than the arteries and were easily distended by passive congestion, they may be a factor at the onset of diverticula formation.

Graser (51) also emphasized passive hyperemia which dilated the veins and thus increased the space of their penetration through the muscularis of the bowel.

Brown (15) decided that it was obvious that increased inward pressure and weakness of a portion of the intestinal wall were physical reasons for presence of diverticula but the "why" was not understood.

Poate (95) was another who thought gaseous distention played some part, yet, he remarked that he'd never seen or heard of diverticulosis associated with Hirschsprung's disease, with its immense dilatation and obstinate constipation, however, he stated that this may be due to hypertrophy of the bowel wall in this condition.

Telling (105) and Perry and Shaw (94) thought internal pressure was the major factor because

most diverticula occurred in the sigmoid flexure and it was there that the longest retention of fecal material occurred, and consequently, the pressure from within was liable to be the greatest.

David (28) stated that age and constipation are outstanding factors. He mentioned that adiposity had often been considered as a factor, yet, many cases of diverticulosis have been observed in thin patients.

Young and Young (116) believed that age, weight, irritations and putrefactions caused fatty degenerations of the musculature, with loss of elasticity and proliferative trabeculations, irregularities and fissures of the mucous membrane. It was these degenerative changes that caused diverticula.

Beer (11) worked on the intestines obtained at autopsies and showed that when pressure was exerted within the intestine, diverticula occurred at the entrance of the blood vessels. However, he concluded that the dead intestine presented altogether different conditions than the live intestine. In the dead intestine, the blood vessels

are empty and the distention caused by the injection of a stream of water is entirely different than the normal distention of the bowel within the body. Therefore, he thought the cause must be sought in a muscular deficiency.

Buie (18) surmised that diverticula came when age, with its attendant wear and tear and atrophy of tissues, had thinned the muscle fibers and separated them. Therefore, diverticula may occur not only as a result of unusual strain and so forth, but also because of the effect of normal processes on tissues which have developed abnormal characteristics.

Spriggs and Marxer (103) did not believe that constipation was a primary cause of diverticulosis because straining at the stool causes a general increase of intra-abdominal pressure which would fall equally upon the peritoneal surface of the bowels and would protect rather than cause the small extrusions.

Willard and Bockus (114) were also of the opinion that constipation was not a factor in causing diverticula because in their practice the occurrence of constipation did not steadily increase

with age, as did the age distribution of diverticulosis. They also mentioned that a number of patients, who had had a life long tendency to defecate after meals or excitement, had developed diverticula.

Poate (95) stated that the appendices epiploicae contained the artery and that their fatty content lifted the peritoneum, thus adding to the lowered resistance of an already weakened area and aiding diverticula formation. He also considered fatty infiltration as playing a part in the formation.

Fat deposits were also considered a factor by Finney (45) who believed there was what may be termed a pathological weakness. That is, destruction of at least a portion of the normal wall by an ulcer, or an infiltration or replacement by fat deposits with resulting weakening of the normal muscle fibers.

Hurst and Rowlands (62) stated that muscular atrophy and the presence of excessive fat in the walls of the colon aided in diverticula production. They accounted for this because diverticula were usually seen in obese people. They also believed that diverticula were still more easily produced if an excessive development of fat had for any reason

rapidly disappeared.

Davies-Colby (29) assumed that the intestinal fat was in some way primarily responsible for diverticula formation. His assumption was based on three possible explanations: (1) The subperitoneal fat weakens the intestinal wall by inducing a simple atrophy of the muscle from pressure. (2) The fat invades and separates the muscle fibers and so produces weak spots. (3) The fat becomes infected probably by the intestinal contents and the muscle in contact with it is weakened by an inflammatory process.

Chapman (23) was opposed to Kleb's (71) theory of diverticula developing at the points where vessels perforated the intestinal wall because he thought Klebs made this statement to explain the mesenteric position of most diverticula. Chapman thought that the vessels, instead of penetrating the muscle at once, divided into two branches which coursed over the muscularis just beneath the serosa, sending branches through the muscles at intervals until the antimesenteric point is reached.

Chapman also believed the traction theory was unsatisfactory becauses if it were of great import-

ance one would expect the transverse colon to be the seat of election for diverticula, with the stomach in perhaps second place, since ptosis is common in both of these; yet, both these organs are relatively immune. The most frequent sites for diverticula are the sigmoid and the duodenum, both of which are rather firmly held in position.

Chapman could see no great reason that sclerosed and shortened vessels would produce traction to cause diverticula because there was no reason why sclerosis should be limited to the superior and inferior mesenteric arteries.

Chapman made note that Beer (11) had mentioned that the submucosa was the last coat to yield before rupture. Chapman then quoted Lord Moynihan (89):

> "The layer which it is of the chiefest importance to secure, in order that the suture may hold well, is the submucous coat. This is of great strength and toughness."

He also quoted Waterson (110):

> "The submucous coat is a loose but strong layer of areolar tissue....on which chiefly depends the strength of the intestinal wall."

Chapman therefore believed weakening of the submucosal layer to be the cause of diverticula.

He compared this with the arteriosclerotic aneurysm. In both, a hollow tube is subjected to varying degrees of pressure. The lining layers (mucosa and endothelium followed by submucosa and subendothelial connective tissue) are surrounded by layers of muscle and elastic tissue. An aneurysm either begins in the media or in the intima from which it extends to the media, with resultant weakening of the elastic structure, while the systoles produce a hammer-like pounding within. He tried to explain diverticula on the same basis; changes of connective tissue, loss of elastic tissue, something analogous to hyaline changes in the submucosa, and finally hammer-like changes in pressure due to rapid contractions alternating with periods of complete emptiness. If true, he believed that there might be a parasyphilitic origin of some intestinal diverticula. Also, diverticula are therefore so frequent in old people, whose elastic and connective tissue very characteristically undergo atrophy.

Chapman thought it might be plausible that there be a possibility of hereditary or congenital maldevelopment, yet these adjective had served

so long to excuse lassitude that consideration of them should be altogether omitted until other sources are exhausted.

Mackoy (81) suggested the possibility of a hereditary tendency to the development of diverticula of the colon when he reported on a family of seven (maternal grandmother, two maternal aunts, one cousin, a niece, and a sister of his patient). The similarity of the symptoms presented by all was so apparent that the intestinal trouble became a family tradition. The oldest members had died without a definite diagnosis but the three sisters of the third generation had X-ray examinations, and all three showed multiple diverticula of the colon.

Wierda (113) recorded an experiment in which he had fed rats on a high fat diet beginning at the time of weaning. He had eight rats which were members of the 3rd generation. He killed five at 25 weeks of age and they showed no signs of diverticula. The remaining three were allowed to live for 90, 107, and 111 weeks before being killed. In each of the three was a diverticulum within about 2 cm of the cecum.

McCoy (86) conducted an experiment on rats by feeding them balanced diets or a diet that was restricted in amounts for a varying length of time, as long as one thousand days for some rats. None of the rats developed diverticula even though some had reached an age beyond that equivalent to a human being being 109 years of age.

Lubbock, Thomson, and Garry (79) performed a long-term experiment with rats on a human dietary. Two groups of rats were reared under similar conditions except for diet. One group received a ration closely approximating that eaten by a working class community in Scotland; the other group received the same diet with an additional supply of milk and green food. In the rats fed on a low grade human diet an unusual kind of epithelial proliferation was observed in the small and large guts. This resulted in multiple diverticula which penetrated the entire thickness of the bowel wall.

Diverticulitis, although a likelihood, does not always follow diverticulosis. Everyone is born with a vermiform appendix, but everyone does not

get appendicitis.

Edwards (36) thought diverticulitis was always due to retention of fecal contents and it was most common in the sigmoid colon because; (1) diverticulosis is more common here than elsewhere, and, (2) the sigmoid contents are solid.

Bearse (9) believed that food may be a factor in causing diverticulitis. Potatoes and milk may cause irritation by forming a relatively large mass and red meats may increase the bacterial activity within the intestine.

Morrison (88) presented a case where the patient had known diverticula. When the patient would eat bran he would develop a diverticulitis and once the bran was discontinued he was free of symptoms. The patient had remained free of symptoms since being placed on a high fat diet and mucoid in consistency.

Telling (106) believed that trauma had a definite diagnostic value when diverticulitis was suspected. He mentioned that the acute manifestations of diverticulitis have occurred while straining at the stool, following a dose of castor oil, during the administration of an enema, following

a heavy meal, jolting in a motor car, while lifting a weight, jumping, and while at work.

INCIDENCE

Anything simulating a fair appraisal of the frequency with which diverticula develop in the colon probably will never be made.

Kocour (72) thought diverticulosis was the most common pathological lesion seen in the large intestine and was responsible for the majority of cases of disease of the colon brought to the attention of the surgeon and the roentgenologist. He reviewed 7,000 consecutive autopsies and found 127 cases, or 1.81 per cent of diverticulosis. 120 of these cases were 41 years of age or over. From this he concluded that the incidence of diverticula increased gradually in number after 40 years of age, and after 60 years of age the increase became very pronounced. Two-thirds of all his cases over 40 years of age were found in persons over 60 years of age. He found only two-thirds as many colored persons with diverticula as white persons. There was no greater incidence of peptic ulcer, carcinoma of the colon, or essential hypertension in the patients with diverticulosis than in the entire group studied. However, he did find that the incidence of gall-bladder lesions in

patients over 40 years of age was doubled in those with diverticula. Therefore, although he could not explain it, he believed there existed a relation between abnormal colon function and cholecystopathy.

Willard and Bockus (114) reported on 72 cases seen first in their office. They found no predilection for any particular habitus. Of their cases, 44 per cent had constipation and 15 per cent had a persistent tendency toward diarrhea. They made a comparison between functional colopathies and diverticulosis and found that 53 per cent of their cases showed some type of functional colonic abnormality; whereas, diverticula occurred in the colon in only 8.27 per cent. Therefore, they believed that this refuted any idea which might be advanced that diverticula are more prone to develop in colons which are the seat of a neuromuscular irritability.

Coleman and Capps (24) observed thirty patients with diverticulosis during a four year period and found all but three of them in the fifth, sixth, and seventh decades of life. All of the patients were of the white race and 60 per cent were males.

Finney (46) and Brown and Marclay (17) found that most cases fell in the higher age groups, from fifty years up, with the decade from sixty to seventy furnishing the greatest numbers.

Jones (67) reported diverticula more prevalent in males, the ratio being about three to two. He stated that diverticula were predominate between fifty and seventy years of age and seldom troublesome before the age of forty.

Wierda (113) stated the diverticula found in the rat resembled diverticula of man in several respects. They are seldom reported in man before the fortieth year and in his rat experiments they were not found until the ninetieth week, which is equivalent to the fifty-second year in the life span of the human being.

Bailey (5) believed diverticula to be present in 5 per cent of all hospital patients. He reported the ratio of males over females as two to one. Walkling (109) thought the sex ratio of diverticulosis was equal.

Bargen (7) stated that diverticulosis was rare before thirty-five years of age and that it was more likely to be found in an obese patient. While

Spriggs and Gordon-Watson (102) found no evidence that diverticulosis patients were fatter than others of the same age in their series of two hundred and six patients.

Newton (90) found that diverticula were as evenly distributed among the better class as the poor.

Bearse (9)(10) reported 5.9 per cent of his patients developed diverticulosis of the colon, while the percentage under the age of thirty was only 0.31. He stated that it was possible, however, that diverticulosis and diverticulitis occurred more frequently in young adults than indicated. This was because forty-five years of age had been given as the average age of patients who have X-ray examinations made of the gastro-intestinal tract and also because special efforts have to be made to demonstrate diverticula. He concluded that the incidence of diverticulosis in persons under forty could be determined only by X-ray studies of large numbers of persons within this age group even though they have no abdominal symptoms.

Buie (18) stated that no hint of the presence of diverticula is ever given until they become the site of another pathologic process, and although

it was impossible to determine the exact frequency with which this happened, it was likely that the secondary pathologic development is of extremely rare occurrence.

Babcock (4) found diverticula to be more prevalent in men (66 per cent) of lax fiber, who are or had been obese. He mentioned that physicians were frequently affected. He estimated that 10 to 12 per cent of patients with diverticulosis would at some time develop diverticulitis.

Slesinger (101) reported that diverticulosis was present in 10 per cent of the patients over forty years of age but considered the risk of developing diverticulitis a small one because actual diverticulitis was comparatively rare.

According to Rankin and Brown (98) diverticulitis occurs almost entirely in persons of middle age who are inclined to be corpulent and who lead sedentary lives. While Eggers (38) considered diverticulitis as being more frequent in the age group between 50 and 60 years.

Lloyd (76) found the incidence of diverticulosis of the colon to be 6.6 per cent in 3,000 roentgen-ray examinations. He estimated that one-fourth of the cases of diverticulosis developed diverticulitis,

and about one-fourth of the diverticulitis cases would require surgical treatment.

Laufman (74) determined from clinical data that 10 to 20 per cent of patients with diverticulosis develop diverticulitis.

Shipley and Gerwig (100) thought it was interesting to note that although a large percentage of patients with diverticulosis had multiple diverticula, there was little clinical evidence of diverticulitis attacking different sections of the colon in the same patient.

Bearse (10) thought that the likelihood of diverticulitis should increase in proportion to the number of diverticula, yet, in two patients operated on for perforation and abscess he could only find a single diverticulum.

The following was taken from Erdmann (41):

> "The presence of a diverticulum in the intestine merits no more attention than does the existence of the appendix or gall-bladder, except that there may be many diverticula, whereas there is only one appendix and one gall-bladder. Therefore, ordinarily the chance of acute diverticulitis is greater, but clinical evidence shows that diverticulitis is far rarer than disease of the gall-bladder or appendix."

ANATOMIC SITUATION

It was Telling's(106) opinion that diverticula were seldom observed on the outside of the gut, except by the trained eye, because they mainly entered the appendices epiploicae and the gut in which they occurred was very frequently fat-ladened necessitating careful dissection to discover them at all. He mentioned that adhesions to other structures and peridiverticular hyperplasia acted further to conceal and obliterate their presence.

Drueck (34) reported that diverticula may be single or multiple and may be confined to a single intestinal segment or scattered over several.

David (28) considered the large bowel to be the seat of diverticula formation over any other structure in the body. Hurst and Rowlands (60) believed that diverticula were rarely found above the middle of the descending colon because they considered that the distribution of diverticula corresponded with the position in which accumulations of hard feces most commonly occurred.

Mayo (84)(85) presented the following:

> "In mammals the testis is the primitive procreative organ, and be-

> cause of its long heredity it is relatively free from disease; the ovary, secondary to the testis, is a more recent acquisition which has not yet achieved the same resistance. So, too, the sigmoid, a convenient storage organ but of more recent development, has not yet achieved the stability of the primitive small intestine."

He then stated that all of the diverticula originated in the descending colon and sigmoid, between the splenic flexure and the rectum, structures which were derived from the primitive hind gut.

Lynch (80) reasoned that although diverticulitis may occur at any point where a diverticulum had formed, it occurred most often in the sigmoid. This was because the sigmoid was a loose movable organ and may be up in the abdomen one moment and in the pelvis the next and, therefore, it was subject to twists, volvulus and variations in position. Since the sigmoid usually contained hard fecal matter and was subject to variation in position, it was more liable to injury than any of the other parts of the colon.

Buie (18) also believed that diverticulitis was seldom found elsewhere than in the sigmoid because of the prevalence of diverticula in the

sigmoid, the lower part of the sigmoid and the upper part of the rectum often are not covered by the serosal coat, and, because the patients usually suffer from constipation with the associated full rectum.

Edwards (36) places the position of diverticula of the colon away from the mesenteric border, in two rows immediately to the mesenteric side of the anterior and postero-lateral muscular taenia, at which point the blood vessels enter the submucosa layer.

Bunch (19) reported that diverticula are single and spring from the antimesenteric portion of the intestine. Erdmann (42) likewise stated that diverticula were more frequent on the convex and lateral aspects of the colon and rarely in the mesenteric folds.

Rankin and Brown (98) revealed that there was a wide divergence of opinion as to whether diverticula occurred more frequently on the side of the colon opposite the mesenteric border, or whether they were more often associated with openings between blood vessels which come in from the mesenteric side. Pepper (93) believed in the same

thing, but he considered the point of origin to be of importance because if diverticula occurred on the mesenteric side and then underwent perforation, the infection would spread extra-peritoneally and result in a localized abscess outside of the peritoneum. While, if diverticula occurred on the antimesenteric side and then underwent perforation, a generalized peritonitis would be likely to result. Therefore, he concluded that undoubtedly diverticula occurred in both these areas, perhaps with different etiology and different potentialities in pathology and symptomatology.

PATHOLOGY

The rapidly growing literature on diverticulosis and diverticulitis indicates the increased attention being paid the subject in recent years.

Berman and Bauer (13) stated that diverticula may be covered with mucosa and submucosa only, or with fibrous tissue and a few longitudinal muscle fibers, subserous fat and serosa, but hardly ever by circular muscle. They may be microscopic in size and evade detection by X-ray or gross inspection and still give rise to symptoms.

Edwards (35) pointed out that the difference between a congenital diverticula and an acquired diverticula, from the clinical standpoint, was that the former possessed a complete and active muscular coat and was thus able to empty itself readily of intestinal contents and there was no tendency to stagnation; while, the latter was merely a herniation of the mucous membrane through the muscularis and thus had no muscular coat at all.

Bunch (19) stated that a congenital diverticula was quite similar in size and shape to the human thumb. The lumen varied little in size from base to tip, being at the base about the size of

the lumen of the intestine itself. The acquired diverticula differed from this in that it was balloon-shaped with a greatly enlarged lumen beyond the somewhat constricted base due to muscle tissue. In the latter, a vicious circle was established, the decreasing resistance to distention caused increasing intestinal stasis, which in turn results in greater distention of the diverticulum.

Wierda (113) mentioned that diverticula which developed on the antimesenteric surface of the colon were much larger than the ones which developed toward the mesentery. This was probably due to the lack of adjacent restraining structures ventrally as compared to the presence of the mesocolon on the dorsal part.

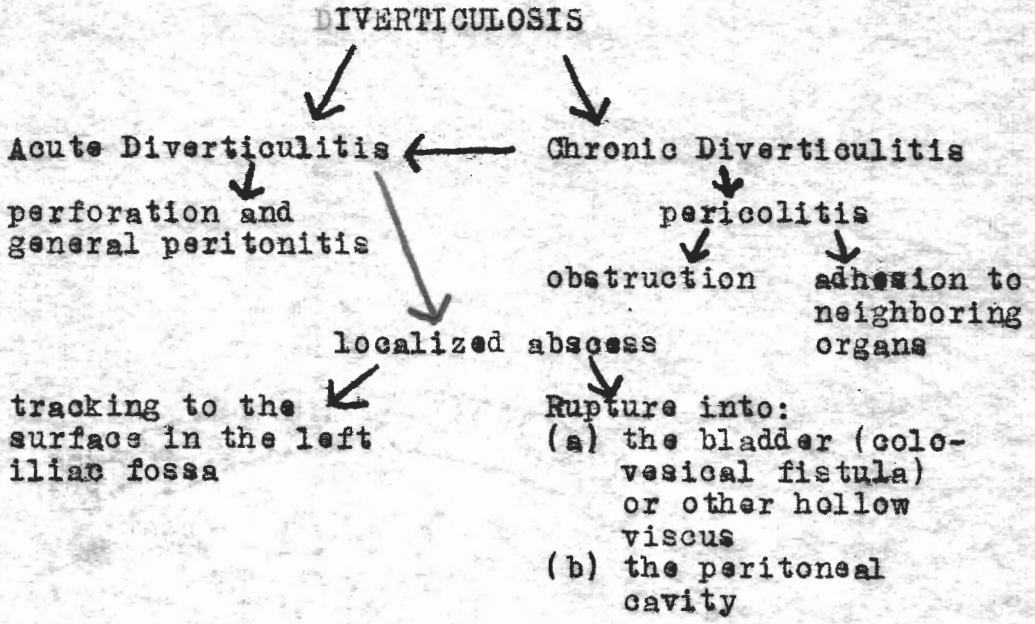
Druesk (34) regarded acquired diverticula as being flask-shaped, and once feces had found their way into it, there was a tendency for it to get gradually larger. He stated the diverticula may remain healthy or undergo secondary changes (diverticulitis) that may extend to surrounding tissues (peridiverticulitis). He also mentioned that on the internal aspect of the diverticula were round or oval openings, and in others short transverse

clefts. Gelatin plugs could be expressed from these and the presence of thin walled pouches demonstrated. Elevations on the outer surface of the diverticula corresponded to these openings on the inner surface.

Corkhill(26) reported an interesting experience in which a man was operated on twice for other than diverticulitis. The two operations were fourteen years apart. Diverticula were noted at the first operation and fourteen years later, the only change noted was an enlargement of the diverticula. No symptoms referable to the diverticula had even been noted.

According to Enfield (40) there was usually no blood in the stools because ordinarily no ulceration of the lining of the diverticula ever occurred.

Edwards (36) presented the following outline for consideration:



Telling (106) reviewed the secondary processes in diverticula and presented the following outline:

Groups of Secondary Processes

A. Mechanical

1. Formation of fecal concretions in the diverticula.
2. Torsion of the diverticulum.
3. Lodgement of foreign bodies within the diverticular sacs.
4. Perforation.

B. Inflammatory

1. Diverticulitis: (a) gangrenous, (b) acute, (c) subacute, (d) chronic, and (e) 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: (a) general peritonitis, (b) local abscess, (c) fistula, especially into the bladder, (d) suppuration in a hernial sac.
5. The formation of adhesions, especially to:

(a) the small intestine, (b) the bladder, (c) the female genitalia.
6. Chronic peritonitis, local.
7. Chronic mesenteritis of the sigmoid loop.
8. Metastatic suppuration.
9. The secondary development of carcinoma.

He stated that the fecal contents form a bacterial nidus, a reservoir of toxins and a source of mechanical irritation. Thus secondary changes may or may not supervene, if not, the condition remains a harmless pathological curiosity and gives rise to no symptoms. He considered the main secondary processes which did occur were : (1) ulceration, (2) perforation, (3) adhesions, (4) peridiverticular fibrous hyperplasia.

According to Telling, the culminating interest is reached in the production of the fourth type of lesion, the peridiverticular hyperplasia. It is due to a chronic leakage of toxins or bacteria, or both, through the sac walls, producing a hyperplastic fibrous tissue formation somewhat similar to that found in hyperplastic tuberculosis of the cecum, or more closely resembling a scirrhous cancer. A tumor is often produced because of the hyperplasia which may amount to an inch or more in thickness. Since it is fibrous, stenosis of the bowel

may result from contraction. This thickening and stenosis may be so local that it tends to make an annular stricture and resemble carcinoma, thus, in this way, many of the cases of so-called "cured" cancer in this region may be explained.

Abell (2) reported that regardless of cause, once the diverticulum is formed it becomes a bottle-shaped process. He estimated that 10 to 20 per cent of people with diverticula ultimately develop diverticulitis and present subjective symptoms. The symptoms are caused by inflammatory changes in the sac and surrounding structures, notably in the mesentery, dependent upon inadequate drainage of the sac.

Young and Young (116) stated that once inflammation sets in, the narrow neck of the diverticulum becomes edematous, preventing easy emptying. The localized inflammation then spreads to the adjacent segment of bowel causing irritability, spasticity, and tenderness.

Brown and Logan (16) reasoned that the cause of diverticulitis was not definitely known but believed there were two types of inflammation. The first begins as a superficial infection of the

mucosa, with resulting irritability of the bowel, diarrhea and pus and blood in the stools. The second, and more common, begins deeper in the walls of the diverticula, which results in thickening of the walls of both the diverticula and colon, intestinal spasm, more or less tumefaction with resulting constipation and even obstruction.

According to Hailes (55) the swelling of the bowel or cellulitis is caused by the inflamed diverticulum. The cellulitis may be localized to one portion of the sigmoid colon, or involve the whole sigmoid, or even in extreme cases the whole of the descending colon. Cellulitis of the bowel pursues the same course as cellulitis elsewhere in the body, and may lead to: (a) resolution, (b) local abscess formation, with or without rupture, or (c) obstruction. Resolution is the most common.

Buie (18) stated that although feces in the diverticula is the usual cause of diverticulitis, it may also be produced by inflammation of the sigmoidal mucous membrane spreading into a diverticulum. Once the mucosal lining of the diverticulum becomes involved it begins to disintegrate, and ultimately ulcerous transformation may develop.

Then the inflammation may or may not extend in any direction. If the neck of the diverticulum becomes obstructed it will likely go on to rupture.

Mayo (84) differentiated between diverticulitis and peridiverticulitis. He considered the former as a lesion primarily of the mucosa, caused by bacteria to which it is not immune. It causes no material reduction of the lumen of the bowel and tends toward acute perforation into the peritoneal cavity. Peridiverticulitis, according to him, was only an inflammatory reaction of the peritoneum set up by the leaking through the diverticular walls of toxins or bacteria, with which the mucosa is constantly familiar. It causes marked reduction in the size of the lumen with chronic obstruction. Perforation rarely occurs because the reparative process is so predominate.

Coleman and Capps (24) had thirty patients who suffered irregular periodic attacks of diverticulitis. Eleven of these had rheumatoid arthritis as a major complication. They examined the eleven patients for foci of infection; one had chronic prostatitis, another had chronic cholecystitis, but they found

nothing in the nine remaining except their diverticulitis. They ran stool cultures to check for streptococcus hemolyticus colonies by which they could have a rough index of the activity and grade of the focus or foci of diverticulitis. They were also an index of the result of treatment. The colony counts varied from two to one hundred and fifty but from ten to fifteen colonies were the initial average incidence. They then treated the patients with a low residue diet and oil enemas. During such treatment, they found many negative stools and a few that merely showed sharp reduction in the number of colonies. This cultural result paralleled the improvement noted in the general condition of the patient and in the subsidence or disappearance of the symptoms due to the rheumatoid arthritis.

Ballin (6) thought diverticulitis predisposed the patient to cancer because there was apt to be a slow, attenuated, chronic inflammation and persistent irritation with continuous epithelial and connective tissue hyperplasia which was similar to the pathogenesis of other intestinal cancer. He stated that if a cancer did evolve from a pre-existing diverticulitis, it did not grow toward or encroach upon the lumen and therefore gave no filling defect in

the roentgenogram. Thus, the earliest symptoms of this type of cancer are slow perfor tion and abscess formation outside of the bowel.

Drueck (34) and Mellon, Soble, Davidson, and Fowler (87) also believed there was a predilection of diverticulosis for the development of carcinoma. If the theory that cancer begins from "tissue tension alteration" has any bearing, diverticulosis is an excellent example.

> "In diverticulosis, the l byrinthine windings of the mucosa, together with the obli ty and consequent separation of the muscle bundles, bear testimony to the interstitial strains that must be the resultant of these processes. And in a mucosal hernia as it exists in a viscus, more or less constantly contractile, interstitial strain must again be unavoidably associated with the histopathological changes in evidence, viz., passive congestion, hemorrhage, and dilatation of the incarcerated gland structure.
>
> Nutritional disturbances are the logical sequel of this mechanical stress, which in their turn invite infection, a factor of neoplastic importance." (34)

SYMPTOMATOLOGY

Hurst and Rowlands (60) stated that diverticula of the iliac and pelvic colon give rise to no symptoms unless pathological changes occur in them. Yet, Lynch (80) believed that all people with diverticulosis have symptoms, but the symptoms are overlooked and are considered as being due to dietary indiscretions and the like.

Devegney and Bailey (30) and Edwards (37) were also of the opinion that the presence of diverticula resulted in symptomatology. The two outstanding symptoms are: (1) vague abdominal discomfort and sometimes pain, at an interval after meals and, (2) flatulence, corresponding in time incidence with the pain, sometimes very pronounced and associated with loud borborygmi. They stated that relief from the above symptoms was obtained after operation and removal of the diverticula. The symptoms were probably due to retention of feces rather than infection.

Spriggs and Marxer (103) thought symptoms of diverticulosis were probably from stretching of the walls by distention with food, or irritation by either acid or putrefying material. Other

factors may be pressure on surrounding structures and the active movements of the bowel may also drag upon a taut accessory pocket in a region sensitive to tension or pressure. They reported on the symptoms of eighteen patients suffering from diverticulosis:

12 had flatulence, fullness, and distention
6 had pain or aching
6 had nausea
3 had nausea with vomiting
2 had nausea with vomiting and hematemesis

Willard and Bockus (114) reported that the number of diverticula found had no effect on the incidence of symptoms collectively. However, the occurrence of symptoms thought to be due to diverticulitis was almost twice as great in the cases with many pouches as those with few.

According to Guthrie and Brown (52) symptoms of associated gastric, pancreatic, or biliary tract lesions may entirely cover up the symptoms of diverticulitis, therefore, the symptoms are not clear cut and hence not of great aid in diagnosis.

Finney (46) stated that the discomfort from diverticulosis came in attacks, without periodicity or any particular cycle connected with ingestion of food, and often with long intervals of

entire comfort between the attacks.

Dixon (31) and Mailer (82) regarded the symptoms of diverticulitis of the distal descending portion of the colon and sigmoid as typical of left-sided appendicitis. They are local tenderness, muscular rigidity, often a palpable mass in lower left abdominal quadrant, increased body temperature, and a leukocytosis.

Pottenger (97) reported a case of diverticulitis which he had suffered from. He stated that he suffered from cutaneous vasoconstriction, slight chilliness, elevation of temperature, aching, tiredness, nervousness, prostration, insomnia, lack of appetite, inhibition of digestion, nausea, and vomiting. This he concluded was a toxic syndrome consisting of most of the usual symptoms which accompany severe toxemia. He did notice, however, that his pulse was slow and he contributed the idea that this was probably due to reflex stimulation through the vagus which was able to overcome the central stimulation of the sympathetics; or possibly some vagotropic substance, which acted upon the heart, was released from the inflamed diverticulum. He also noted that this toxic

syndrome, in its peripheral expression, produced effects similar to the body's reaction to fear, anger, pain, and rage, which is expressive of widespread sympathetic stimulation and parasympathetic inhibition.

Bearse (10) reported that age was not a factor in the symptomatology of diverticulitis as the symptoms were similar in young adults or older patients.

Willey (115) stated looseness of the bowels was almost as common as constipation.

Willard and Bockus (114) reported that 11 per cent of the cases with diverticulitis had blood in their stools. Spriggs (102) reported 5 per cent and Jones (66) had 3 to 8 per cent. Rankin (98) considered blood to be in the stools of 17 per cent of the patients with diverticulitis.

DIAGNOSIS

Gaither (48) stated that because of the lack of any particular syndrome and the paucity of outstanding and distinctive symptoms there was a great handicap in making an early diagnosis of disease of this portion of the digestive tract.

Case (22) thought it depended on one's conception of what constitutes symptoms as to how large a group of cases will fall under the heading of diverticulosis. The mildest group under the diverticulitis classification is made up of the enterospasm type of cases, and it is difficult to draw a sharp line of differentiation between the cases of purely symptomless diverticulosis and those of the enterospasm type of diverticulitis. He stated that the early symptoms of diverticulitis characterized by spasm of the colon are common to the early stage of many abdominal diseases.

Edwards (37) believed that the symptoms of colonic diverticulosis are not sufficiently characteristic to warrant a diagnosis of diverticulosis on clinical grounds alone, and roentgenologic examination is the final criterion. All other possible causes should be eliminated before holding the presence of divertic-

ula as responsible for abdominal symptoms.

Erdmann (42) reported that the onset of diverticulitis was characterized by pain in the abdomen, which was more definitely located, early, in the left lower quadrant than is the localization in appendicitis.

It was Babcock's (4) impression that a chronic, intermittent type of abdominal discomfort, characterized by the accumulation of gas in the colon, with a sense of distention, epigastric fullness, excessive expulsion of flatus and constipation, is frequently indicative of catarrhal diverticulitis. The attacks may last for a few days to two or more weeks and are often precipitated by certain foods, dietetic excess, or by mental strain. A positive diagnosis of catarrha diverticulitis may be difficult to make. It is often surmised, but has heretofore rarely been proved.

He implied that the symptoms of perforative diverticulitis develop spontaneously and rather gradually or suddenly as during defecation, coughing or other muscular strain. The symptoms may or may not be acute with severe local and general reaction. The local reaction is usually referred to the left lower quadrant, but may, from the position of the

involved portion of sigmoid, be located in the right pelvis, right lower quadrant, or other part of the abdomen. Once a perforation is suspected a barium enema should not be used as the barium may escape into the bladder, ureter, abdominal cavity or other viscus, and lead to complications.

Bargen (7) considered it difficult to say which is cause and which is effect when one considers the number of human beings who come into the office of any internist with the complaint of constipation and the fact that only 40 per cent of patients with diverticulosis complain of constipation.

According to Kozinn and Jennings (73) blood in the stools is a more frequent symptom in children than in adults when one is considering a diagnosis of diverticulitis.

Willard and Bockus (114) claimed that only by actually visualizing the diverticula could a positive sigmoidoscopic diagnosis be made. The failure to see them sigmoidoscopically is due primarily to their location above the reach of an ordinary 10 inch sigmoidoscope.

Poate (95) knew of two cases in which the sigmoidoscope had been pushed through the bowel wall with fatal results. Therefore, he considered it un-

wise ever to conduct a sigmoidoscopic examination in acute or subacute conditions, as the bowel wall and the walls of the diverticula are inflamed and rigid. In any case in which a sigmoidoscopic examination is carried out, do not use a general anesthetic, desist if pain is caused, use minimal inflation of the colon.

Stewart and Illick (104) did not report the value of the sigmoidoscope in the diagnosis of diverticulitosis but stated that if it was done, do it before giving patient a barium meal.

Gaither (48) frankly stated that a proctoscopic examination was of no value in the diagnosis of diverticulosis. Rankin and Brown (98) said it was of relatively little value. Heinz (59) thought it was of value and it at least gave a chance to take a biopsy. Brown and Marclay (17) believed it to be of value. Judd and Pollack (69) claimed that it was of value only as a method of ruling out other conditions. For the same reason Erdmann (41) claimed it to be of value because he had never seen mucosa of the colon involved in a case of diverticulitis.

Shipley and Gerwig (100) held a conservative view. They reported a proctoscopic examination is helpful in some cases, diagnostic in a few, and neg-

ative very often.

Lockhart-Mummery (78) regarded a definite annular stricture without any break in the lining membrane, or, a normal appearance of the mucous membrane without any blood in the higher part of the bowel and associated with marked fixation of the colon as positive proctoscopic findings of diverticulitis.

Jackman and Pumphrey (63) reported that proctoscopic findings of immobility of the bowel, narrowed lumen, mucosal edema, angulation and extrarenal mass, though of some value, are too frequently associated with some previous pelvic inflammatory process to be more than presumptive evidence of diverticula.

Jackman and Buie (62) presented five proctoscopic signs leading to diverticulosis: (1) limited mobility of a segment of bowel which is normally freely movable, (2) angulation of the bowel, (3) reduced lumen and adherent mucosal folds, (4) sigmoidal sacculation, (5) seeing the diverticula. They reported three cases of colonic diverticula in which the diverticula were seen by a proctoscope but were not revealed by the primary roentgenographic studies. From one to six years later, roentgenologic studies of the colon revealed the presence of diverticula.

They thought this early visualization may have represented a prediverticular stage.

Buie (18) reported on the same five proctoscopic findings but he stated that it was a collection of several of the findings and not just one that was indicative of diverticulitis.

According to Enfield (40) the diagnosis depends on the X-ray examination for finality, except in the acute attack, as the history is at best only very suggestive. Characteristic and conclusive physical signs are wanting. Definite laboratory evidence is not obtainable.

Edwards (37) stated that colonic diverticula are more difficult to detect by roentgenography than are duodenal diverticula, except in the comparatively rare cases in which there is retention of barium in them, and they will, therefore, be more often missed than not.

Ballin (6) reported diverticulitis of the sigmoid as frequent but often unrecognized because the diverticula are so small, and after inflammatory changes have taken place, are not distinguishable from the thickened intestinal wall. Roentgenography will show only a certain percentage of diverticula. They are best visualized three or four days after an

opague meal, when barium remains in the sacculi after the bowel is emptied.

Case (21) also believed diverticula were seen best on the second or third day after the opague meal, when by careful screen study, especially during manipulation of the iliac and pelvic colon, one may detect their presence.

Hurst and Rowlands (60) stated that a barium enema may give an exaggerated idea of the degree of obstruction, as the narrowing caused by the contraction of newly formed fibrous tissue and the presence of inflammatory material may be greatly increased by spasm; the latter is temporary and is likely to be increased by the mechanical stimulus caused by the distention following the injection of the enema. Whether there is organic or functional narrowing, it is clear that diverticulitis and not merely diverticulosis is present.

Enfield (40) thought it was desirable in cases of suspected diverticulosis to make a preliminary film of the entire abdomen over the Bucky diaphragm in the antero-posterior projection. This would serve to establish the presence or absence of abnormal densities which might later in the examination happen to be so placed in relation to the colon as to

simulate filled diverticula. Among possible causes of such densities should be mentioned single dense gallstones, calcified glands, phleboliths, and atypical kidney or ureteral stones.

Buie (18) stated that diverticula are seen as rounded, pouch-like shadows along the contour of the bowel on roentgenograms when a barium enema is used instead of a barium meal. On using Weber's modification of the double-contrast technic of Fischer, other characteristic signs are observed which increase the efficiency of the diagnosis. The inflammation irritates the involved segment, and the resulting hypermotility varies with the intensity of the process. There may appear only sharp, serrated haustra, or the involved segment may present a narrowed lumen, or one may even see extreme occlusion. These deformities may manifest themselves as a false filling defect, owing to spastic narrowing which may become so severe as to approach complete occlusion, or an actual filling deformity produced by infringement on the lumen of the bowel by formative inflammatory development around its circumference.

Case (22) presented three X-ray findings

which he considered indicative of diverticulosis: (1) visualizing the diverticula themselves, (2) prediverticular stage, (3) displacement of the small intestinal coils upward and toward the right.

Under the second finding he discussed coming upon a peculiar serrated appearance along the descending colon and sigmoid. This is associated with more or less narrowing of the lumen and may extend over several inches of gut. These serrations are small, close together, and with a rather sharp point, presenting at times a saw-tooth appearance. Repeated examinations reveal this appearance to be constant. Furthermore, palpation under the fluoroscopic screen usually shows that the intestine is more or less like a rigid tube. He inferred that the serrated appearance is due to inflammatory thickening and induration of and about the intestinal wall, secondary to the presence of diverticula; the diverticula themselves not being visible.

Under the third finding he discussed the presence of fibroids, ovarian cysts and other pelvic tumors, pathological and physiological, as lifting the small intestine out of the true pelvis high-

er up into the abdomen. For years he had observed a similar roentgen appearance in certain cases of diverticulitis of the sigmoid. With the above possibilities ruled out, the amount of displacement is such that one can suspect a fat-laden sigmoid covered with swollen and edematous or inflamed epiploic appendages, the total forming a mass of considerable proportion. The idea that this finding may be due to the peridiverticular manifestations, is strengthened in some cases by carefully noting the gas content of the area of sigmoid under suspicion. This area doesn't exceed one-half inch in diameter, and yet the vacant space in the left iliac region due to the displacement of the small intestine is at least three inches in diameter. Therefore, it is probable that the difference between the three inches of vacant space and the half inch of colonic lumen represents the mass made up of the fat-laden, enlarged epiploic appendages attached to the already thickened walls of the colon.

Willard and Bockus (114) mentioned a peculiar appearance of angulation and fixation of one border of the bowel silhouette without any appreciable

distortion of the other margin which they saw in cases of diverticulosis. They suspected this X-ray finding may result from contraction of the bowel wall due to a small cicatrix at the site of a previously inflamed diverticulum. It is probable that this may in some instances result from exceedingly deep irregular haustral contractions. But, regardless of the mechanism responsible for its occurrence, they considered its frequent association with diverticulitis was worthy of recording.

DIFFERENTIAL DIAGNOSIS

The most important point in reaching a diagnosis is to bear the condition in mind when patients with suggestive symptoms present themselves. Lower abdominal pain with cramps, gas, fever, and perhaps alternating constipation and diarrhea, should cause one to think of a sigmoid lesion. Appendicitis, pelvic disease, tuberculosis, syphilis, and simple ulcerations have to be considered. The two important lesions which come under consideration are diverticulitis and carcinoma.

Lynch (80) stated that of the other conditions which simulate diverticulitis, a justifiable and quite natural error in diagnosis is thinking it to be appendicitis. Because of the mobility of the lower sigmoid, the symptoms may be entirely right sided.

Jones (68) and Hare(57) presented the finding, as an aid in the differential diagnosis, that the pain in diverticulitis is generally below the umbilicus, whereas, epigastric pain is present early in appendicitis with localization later.

Berne and Pattison (14) pointed out the analogy between complications of diverticulosis and those of duodenal ulcer. The basic lesion of either may be clinically silent, or there may be chronic symptoms due to local muscle spasm. An acute process with threatened perforation may appear in either. This process may slowly perforate and produce a local abscess, the surgical drainage of which may be followed by a fistula. Also, in both conditions, acute free perforation with general peritonitis is an important complication. Both may produce a chronic or an acute intestinal hemorrhage.

Devegney and Bailey (30) presented a case of a woman who had a typical ulcer syndrome with attacks of abdominal pain accompanied by much flatulence for fourteen years. A laporotomy was done, but no ulcer was found and the gall-bladder was normal. X-rays were taken and a diverticulum was excised and she was freed of all symptoms.

Hare (57) and Huston (61) and Wetherell (112) reported that diverticulitis of the mobile sigmoid

colon may assimilate the symptoms of pelvic inflammatory disease. History and X-ray are the best means of differentiating between the two.

Mailer (83) discussed two cases of diverticulitis and tuberculosis. Both are capable of producing almost identical pathological pictures. When coexistent, there is great difficulty in assessing the part which each plays in the production of the granulomatous tumor. Hyperplastic tuberculosis of the colon is a rare condition. There are two types, the submucous and the subserous. In the latter, the subserous coat is the seat of fibrous connective tissue hyperplasia and lymphocytic infiltration. It is this type which most closely mimics chronic diverticulitis and peridiverticulitis. The submucous coat may be almost unaffected, and the mucous membrane usually shows no ulceration. The symptoms of hyperplastic tuberculosis of the sigmoid are identical with those of diverticulitis and a differential diagnosis of these conditions is not possible until after operation when miliary tubercles and giant cells are seen under the scope.

Eggers (35) in discussing the differential diagnosis between diverticulitis and carcinoma believed that a prolonged history of disability, with progressive symptoms of constipation, cramps, blood in the stool, a palpable tumor and loss of weight, favored the diagnosis of carcinoma. If the symptoms were of short duration, if pain was an outstanding symptom, and if there was fever and leukocytosis, he believed a diagnosis of diverticulitis was more likely. Both affect the same age groups. He stated that neither condition is an emergency and one may follow conservatism until a definite diagnosis is possible.

Stewart and Illick (104) and Eliot (39) stated that the distinguishing factors between the two were: (1) carcinoma is progressive without remission, while, diverticulitis has recurring attacks, (2) in carcinoma, the X-ray shows a sharp, sudden demarcation between normal and pathological involvement. The demarcation is gradual in diverticulitis.

Lynch (80) did not believe loss of weight in carcinoma should serve as a distinguishing feature in comparison with diverticulosis because he had found very few cases of carcinoma except in the

advanced stages, in which there was loss of weight.

Dixon, Deuterman and Weber (32) reported one of the predominant symptoms of malignancy was the passage of a large amount of blood and mucous with the stool.

Bargen and Dixon (8) observed that noticeable bleeding by rectum was quite a rare symptom of diverticulitis in a large series of cases which they reviewed.

Brown and Marclay (17) and Erskine (43) reported that bleeding from the bowel was more frequent in carcinoma of the colon than in cases of diverticulitis.

Heinz (59) merely stated anemia is associated with carcinoma and not with diverticulitis.

Erdmann (41) thought one could eliminate carcinoma with a proctoscope inserted to its full length. This is possible in those patients in whom carcinoma starts in the mucosa, recognizing the fact that only a minimal number of cancers of the sigmoid or colon ever arise outside the mucosa.

Jones (67) stated that if obstruction results from diverticulitis, it is due to extrinsic inflammatory reaction and contraction of the underlying

coats of the bowel. This is in contra-distinction to that produced by ulcerating carcinomas.

According to Schatzki (99) the roentgenologic differential diagnosis between carcinoma and diverticulitis is easy in most cases, difficult in some, and impossible in a few.

Golden (50) reported that in diverticulitis, the gut in the involved area is often so irritable that it does not remain well filled with barium and because of spasm may appear to have a narrowed lumen. A shadow with irregular saw-tooth margins is produced by marked swelling of the mucosa and exaggeration of its folds. This is often interpreted as carcinoma. However, on using the fluoroscope in the examination of the gut as it fills, one notes changes in the mucosal contours of the wall and evidences of peristaltic movement which would be impossible in the presence of carcinomatous infiltration.

A malignant growth of the colon usually manifests itself by a narrow, sometimes slightly irregular constriction in which the above described saw-tooth appearance is absent and no mucosal contours can be detected. On either side of the constric-

tion the gut may be irritable. Both diverticulitis and carcinoma may cause complete obstruction to the flow of the barium enema.

Buie (18) thought one who was inexperienced may be confused because of the similarity of these irregularities. Yet, if it is borne in mind that in inflammatory disease the involved segment of bowel is likely to be long and that the contours are concentric, whereas, in cancer the outlines are sharply irregular and the involved segment is much shorter, much of the difficulty will be avoided.

Upson and MacGregor (107) believed it was self evident that in untreated cases of diverticulitis, the inflammatory process would become more severe. He considered this a predisposing cause of malignancy.

Peate (95) and Dixon (31) expressed similar views when they reported the occasional occurrence of both carcinoma and diverticulitis in the same segment of the intestine. So far as they knew, diverticula are not the precursors of cancer.

Rankin and Brown (98) stated there was little evidence to support the view that carcinoma is the result of diverticulitis but he saw no reason why

carcinoma should not develop in the mucous membrane of the diverticulum.

Abell (1) reported the incidence of carcinoma developing on diverticulitis at from 1.7 to 8 per cent. Slesinger (101) stated that the incidence of carcinoma developing on diverticulitis is no higher than in any other person and the fear of developing carcinoma need not in any way influence the treatment of diverticulitis.

Case (22) reasoned that unless diverticulitis has been remembered, fully considered and systemically investigated, no case of supposed carcinoma of the lower bowel must in the future be regarded as inoperable either before or at laparotomy.

TREATMENT

Abell (1) considered the wisest course to pursue in the treatment of an acute diverticulitis as a debatable question. Some prefer to pursue a conservative medical management until complications arise which necessitate surgical intervention, while others advocate immediate operation, with excision of the diverticulum and closure of the opening. The removal of one diverticula that presents acute inflammation affords no immunity to the remaining ones against similar changes.

Pepper (93) stated that only recently has the treatment of diverticulitis received from the internists the attention it deserves. He believes that it has unfortunately been considered a surgical subject for too many years.

Kozinn and Jennings (73) and Lynch (80) reported that the value of surgical treatment is disputed, yet, there is no effective medical therapy for diverticulosis than there is any medical therapy that will abort appendicitis.

Conservative medical management by means of dietary precautions and prevention of stagnation of hard feces in the diverticula was implied in the

papers of Slesinger (101), Walkling (109), Bailey (5), Johnson (64), Laufman (74), and Hurst and Rowlands (60).

Lloyd (76), Douthwaite (33), and Brown (15) thought rest was a prime requisite, as in any other inflammatory process. Therefore, they stopped all food by mouth for an appropriate length of time, followed at the proper time by a non-residue diet. Intravenous glucose and saline was given to maintain normal chemical balance. They also thought that large doses of olive oil by mouth and warm olive oil retention enemas were far superior to mineral oil.

Brown and Logan (16) questioned the use of mineral oil in treatment as in many cases it merely leaks through and is annoying to the patient. Also, another point is whether the constant use of mineral oil may not occasionally be an irritant.

Jones (67) and Kimpton (70) frequently noted that patients with an irritable bowel due to diverticulitis said they felt better following the roentgen examination, consequently, they gave a tablespoonful of barium sulfate in water twice a week to their patients in order to help keep di-

verticula filled with barium and to replace the infectious material.

Abell (1) found that medical treatment based on that for duodenal ulcer plus the use of belladonna is most likely to give good results.

Finney (46) questioned the advisability of surgery in most cases, because the patient is usually of such age as to make operation wise only on definite or imperative grounds.

Hayden (58) thought surgery was indicated only in chronic diverticulitis. Guthrie and Hughes (53) and Erdmann (41) believed in surgery even in the acute condition, however, Erdmann did not always predict a clean bill of health from occurrence or recurrence of the disease.

According to Jones (68) there is no abdominal ailment that taxes one's surgical ingenuity or calls for more individualization than diverticulitis and its complications.

Judd and Pollack (69) and Wetherell (112) reported the mortality from radical operations for diverticulitis has been very high. Therefore, they recommended preliminary colostomy with resection later, after the infection and inflammation has de-

creased in severity.

Poate (95) thought it best to let the acute stage subside as far as possible and then use surgery when it can be carried out deliberately and under the best possible conditions.

Weible (111) thought drainage through the anterior rectal wall was far the better method of drainage of acute perforated sigmoidal diverticulitis.

Brown (15) brought up the importance of the economic factor in cases in which operation is required. Not infrequently the better part of a year is spent in the hospital, especially in the cases in which multiple operative stages are required. Although complications will develop in some cases in spite of medical treatment, the long period required for the operative treatment should spur one to do everything possible to control diverticulitis by medical means.

COMPLICATIONS

Brown (15) presented an interesting question: "why do complications which require surgical treatment develop in some cases of diverticulitis but not in others?" According to him, this is as unanswerable as it is in cases of duodenal ulcer. One cannot predict that a certain percentage of patients with diverticulitis will eventually require surgical treatment because the ratio of patients who require surgical or medical treatment is purely dependent on the cases studied.

Berman and Bauer (13) reported that simple inflammation may be severe enough to interfere with the blood supply, causing gangrene with perforation.

Bearse (10) stated that chronic thickening of the mesentery can result from extension of the inflammatory process, and at times, after the swelling of the mesentery has subsided, the bowel may become kinked and cause obstruction.

Brown, Dixon, and Waugh (12) added that obstruction may also result from the formation of enteroliths within diverticula; from pressure of an inflammatory mass associated with divertic-

ulitis; or from stricture or adhesions produced by recent or ancient diverticulitis.

Arnheim (3) discussed complications under the following headings: (1) peritonitis resulting from passage of organisms through inflamed diverticula without perforation, (2) perforation of inflamed diverticula, (3) fistula formation between the colon and any other adjacent viscus, (4) peridiverticulitis with thickening of the wall of the colon, (5) metastitic suppuration, (6) carcinoma arising from diverticula of the colon.

PROGNOSIS

Rankin and Brown (98) thought the prognosis was good because it usually runs a chronic course with several exacerbations and yields satisfactorily to dietary and medical treatment.

Willard and Bockus (114) considered the prognosis of diverticulosis and uncomplicated diverticulitis as good. While Bargen (7) stated the prognosis of diverticulitis may be grave but the prognosis of diverticulosis need never be viewed with alarm.

Brown and Marclay (17) held a guarded prognosis because they thought diverticula which may appear harmless at the moment may have been, or will be at the seat of inflammation.

Bearse (10) remarked that the longer the life expectancy of the patient, the greater the possibility of subsequent inflammation and its possible recurrence.

Grant (49) stated that the diverticula may remain inactive for years, or at any time become infected and inflamed. He believed the mortality to be nil with the removal of uninflamed, inactive

diverticula. The mortality is rather high in cases in which operation is performed during an acute crisis complicated by marked obstruction, peritonitis, or abscess.

Eggers (38) considered diverticulitis a serious problem and one must be prepared at any time to see a recurrence or a complication requiring surgical intervention. While Erdmann (42) found in his series of cases that multiple attacks of diverticulitis will eventually result in gangrene of the involved segment and thus call for a very guarded prognosis as to the future.

CONCLUSION

Although the exact etiology of diverticulosis is unknown, apparently many factors are known to contribute to its occurrence. These may be classified as follows: (1) embryological, (2) anatomical, (3) physiological, (4) pathological, and (5) physical.

The cause is probably a combination of factors due principally to an increase of intraluminal pressure and a decrease in the resistance of the intestinal wall to this pressure. The former may be influenced by the smallness of the bowel lumen at the junction of the rectum and the sigmoid. Constipation, spasm of circular muscle and obesity may also be influencing factors on increased intraluminal pressure.

The decreased resistance of the bowel wall may be influenced by congenital defects, wear and tear of muscle as age is increased, and the large canals for the entrance and exit of vessels.

Most of the evidence reveals that diverticula are usually acquired. They are usually found in the pelvic and iliac colons, but may occur throughout the large bowel. They are usually found on the

mesenteric side of the anterior and posterolateral taenia, where vessels penetrate the wall of the colon.

As far as diverticulosis is concerned, it seems quite evident that is a condition which, if found, is not greatly to be feared, particularly after the sixty year period, as only a small number of surgical complications occur in the later years. The majority of them are the chronic infiltrating condition which leads to stenosis and may be mistaken for carcinoma.

The younger the individual, the greater the risk of an acute surgical emergency and also the greater the mortality. Once symptoms have developed, the patient should be warned of the necessity for a lifetime dietary control, and if patients will adhere to this advice it seems that very few will ever develop diverticulitis of sufficient severity to demand surgical measures.

BIBLIOGRAPHY

1. Abell, I.: Diagnosis and treatment of diverticulitis and diverticulosis, Surg., Gynec., & Obst. 60:370-378, (Feb.) '35.

2. Abell, I.: Diverticulosis of colon, Am. J. Surg. 46:158-161, (Oct.) '39.

3. Arnheim, E.E.: Diverticulitis of colon, with special reference to surgical complications, Ann. Surg. 112:352-369, (Sept.) '40.

4. Babcock, W.W.: Diverticulitis, Rev. Gastroenterol. 8:77-92, (Mar.) '41.

5. Bailey, F.W.: Acute diverticulitis, Surg. Clin. North America 18:1349-1367, '38.

6. Ballin, M.: Diverticulitis of colon, Am. J. Surg. 2:130-141, (Feb.) '27.

7. Bargen, J.A.: Diverticulosis and diverticulitis, Rocky Mountain M.J. 36:537-542, (Aug.) '39.

8. Bargen, J.A. & Dixon, C.F.: Quoted by Dixon, Deuterman, & Weber (32).

9. Bearse, C.: Acute perforative diverticulitis of colon in young persons, J.A.M.A. 113:1720-1722,(Nov.) '39.

10. Bearse, C.: Diverticulosis and diverticulitis of colon, with particular reference to patients under 40, Rev. Gastroenterol. 7:318-321, (Aug.) '40.

11. Beer, E.: Some pathological and clinical aspects of acquired (false) diverticula of the intes-tines, Am. J. Med. Sc. 128:135-145, '04.

12. Benson, R.E., Dixon, C.F., & Waugh, J.M.: Non-meckelian diverticula of jejunum and ileum, Ann. Surg. 118:377-393, (Sept.) '43.

13. Berman, J.K., & Bauer, T.B.: Diverticulitis of colon, J. Indiana M.A. 35:197-209, (April) '42.

14. Berne, C.J., & Pattison, A.C.: Diverticulitis of colon, California & West. Med. 52:225-229, (May) '40.

15. Brown, P.W.: Treatment and prognosis of diverticulitis of colon, Am. J. Surg. 46:162-170, (Oct.) '39.

16. Brown, P.W., & Logan, A.H.: Medical treatment of diverticulitis of sigmoid, Proc. Staff Meet., Mayo Clin. 11:37-40, (Jan.) '36.

17. Brown P.W., & Marcley, D.M.: Prognosis of diverticulitis and diverticulosis of colon, J.A.M.A. 109:1328-1333, (Oct.) '39.

18. Buie, L.S.: Diverticula of colon, New England J. Med. 221:593-598, (Oct.) '39.

19. Bunch, G.H.: Congenital factor in acquired diverticulosis of jejunum and ileum, South. M.J. 32: 919-921, (Sept.) '39.

20. Butler, R.W.: Quoted by Dixon, Deuterman, & Weber (32).

21. Case, J.T.: Diagnosis and treatment of colonic diverticula, Am. J. Surg. 4:573-596, (June) '28.

22. Case, J.T.: Roentgen study of colonic diverticula, Am. J. Roentgenol. 21:207-220, (Mar.) '29.

23. Chapman, J.: Diverticulosis of small intestine; report of case with critical review of recent literature, Ann. Int. Med. 7:1376-1390, (May) '34.

24. Coleman, G.H., & Capps, J.A.: Diverticulitis of the colon, a study of its potential role as a focus of infection in systemic disease, Arch. Path. 26:207-209, (July) '38.

25. Cooper, A.: Quoted by Edwards (37).

26. Corkill, N.L.: Extensive diverticulosis without symptoms, Brit. M.J. 1:184, (Jan.) '27.

27. Cruveilheir,: Quoted by Mayo (85).

28. David, V.C.: Diverticulosis and diverticulitis with particular reference to development of diverticula of colon, Surg., Gynec., & Obst. 56:375-381, (Feb.) '33.

29. Davies-Colby, R.: Etiology of diverticula of intestine, Brit. M.J. 1:282, (Feb.) '26.

30. Devegney, F.E.C., & Bailey, J.: Diverticulum of jejunum, Brit. M.J. 2:98, (July) '42.

31. Dixon, C.F.: Diverticulitis, Am. J. Surg. 46: 600-603, (Dec.) '39.

32. Dixon, C.F., Deuterman, J.L., & Weber, H.M.: Diverticula, Surg., Gynec., & Obst. 66:314-321, (Feb.) '38.

33. Douthwaite, A.H.: Gastro-intestinal diseases, Practioner 143:461, (Nov.) '39.

34. Drueck, C.J.: Pathology of diverticulosis, J. Iowa M. Soc. 19:372-375, (Aug.) '29.

35. Edwards, H.C.: Clinical significance of diverticula of small intestine, Clin. J. 63:231-237, (June) '34.

36. Edwards, H.C.: Diverticulosis and diverticulitis, Practitioner 149:145-150, (Sept.) '42.

37. Edwards, H.C.: Diverticulosis of small intestine, Ann. Surg. 103:230-254, (Feb.) '36.

38. Eggers, C.: Acute diverticulitis and sigmoiditis, Ann. Surg. 113:15, (Jan.) '41.

39. Eliot, E.: Clinical features of sigmoid diverticulosis, South. M.J. 26:58-63, (Jan.) '33.

40. Enfield, C.D.: Diverticulitis of colon, with special attention to diagnosis, Radiology 7:371-378, (Nov.) '26.

41. Erdmann, J.F.: Diverticulitis, New England J. Med. 223:846-851, (Nov.) '40.

42. Erdmann, J.F.: Diverticulitis and diverticulosis, J.AM.A. 99:1125-1128, (Oct.) '32.

43. Erskine, A.W.: Diverticulosis of colon, J. Iowa M. Soc. 26:255-257, (May) '36.

44. Evans, A.: Developmental enterogenous diverticula, Brit. J. Surg. 17:34-83, (July) '29.

45. Finney, J.M.T.: Diverticulitis and its surgical treatment, Inter-state Post-Grad. M.A., North America 57-65, '29.

46. Finney, J.M.T.: Duodenal diverticula; their significance and treatment, South. Sur. 11:513-(Aug.) '42.

47. Fraser, I.: Quoted by Poate (95).

48. Gaither, E.H.: Diagnosis and treatment of lesions of small bowel, M. Clin. North America 23;499-511, (Mar.) '39.

49. Gant, S.G.: Diverticula, diverticulitis and peridiverticulitis of small intestine, cecum, colon, sigmoid flexure and rectum, J. A.M.A. 77:1415, (Oct.) '21.

50. Golden, R.: Diverticulosis, diverticulitis and carcinoma of colon; roentgenologic discussion, New England J. Med. 211:614-623, (Oct.) '34.

51. Graser, E.: Quoted by David (28).

52. Guthrie, D., & Brown, M.J.: Diverticula of proximal intestine, duodenum and jejunum, Am. J. Surg. 40:128-139, (April) '38.

53. Guthrie, D., & Hughes, F.A.: Diverticulosis of the small intestine, Surgery 1:595-609, (April) '37.

54. Habershon,: Quoted by Mayo (85).

55. Hailes, W.A.: Cellulitis of colon; manifestation of diverticulitis, Australian & New Zealand J. Surg. 8:270-279, (Jan.) '39.

56. Haggard,: Quoted by Ballin (6).

57. Hare, E.R.: Appendicitis, diverticulitis and ectopic pregnancy with special reference to differential diagnosis, Minnesota Med. 4:714, (Dec.) '21.

58. Hayden, E.P.: Surgical problems in diverticulitis, New England J. Med. 222:340, (Feb.) '40.

59. Heinz, T.E.: Differential diagnosis between diverticulitis and carcinoma of rectosigmoid, M. Clin. North America 17:1665-1672, (May) '34.

60. Hurst, A.F., & Rowlands, R.P.: Diverticula of colon, Guy's Hosp. Rep. 75:462-484, (Oct.) '25.

61. Huston, H.R.: Diverticulitis of colon in women, Arch. Surg. 26:1111-1117, (June) '33.

62. Jackman, R.J., & Buie, L.A.: Diverticula of colon; proctoscopy as aid in diagnosis and differential diagnosis, J.A.M.A. 121:1144-1146, (April) '43.

63. Jackman, R.J., & Pumphrey, R.E.: Significance of proctoscopic findings in diagnosis of diverticula of colon, Proc. Staff Meet., Mayo Clin. 14: 596-599, (Sept.) '39.

64. Johnson, .R.: Is diverticulitis of colon a surgical disease? North Carolina M.J. 2:657-661, (Dec.) '41.

65. Jones, S.: Quoted by Mailer (82).

66. Jones. D.F.: Diverticulitis of colon; relation to carcinoma, New England J. Med. 203:459-461, (Sept.) '30.

67. Jones. T.E.: Diverticulitis and diverticulosis of colon, New York State J. Med. 39:1846-1851, (Oct.) '39.

68. Jones, T.E.: Surgical treatment of diverticulitis, Ohio State M.J. 34:1225, (Nov.) '38.

69. Judd, E.S., & Pollock, L.: Diverticulitis of colon, Ann. Surg. 80:425-438, (Sept.) '24.

70. Kimpton,: Quoted by Young & Young (116).

71. Klebs,: Quoted by Chapman (23).

72. Kocour, E.J.: Diverticulosis of colon; its incidence in 17,000 consecutive autopsies with reference to its complications, Am. J. Surg. 37:433-436, '37.

73. Kozinn, P.J., & Jennings, F.G.: Jejunal diverticulitis; occurring in 2 year old girl, Am. J. Dis. Child. 62:620-623, (Sept.) '41.

74. Laufman, H.: Surgical management of diverticulitis of colon; five year collective review, Internat. Abstr. Surg. 73:222-233, '41.

75. Levitt, A., & Goldstein, K.: Diverticulosis; analysis of 86 cases, Rev. Gastroenterol. 8:439-446, (Dec.) '41.

76. Lloyd, T.P.: Diverticulosis and diverticulitis, New Orleans M. & S.J. 90:196-199, (Oct.) '37.

77. Lewis, & Thyng,: Quoted by Lynch (80).

78. Lockhart-Mummery, J.P.: Etiology of diverticulitis, Lancet 1:231-232, (Feb.) '30.

79. Lubbock, D.M., Thomson, J., & Garry, R.C.: Epithelial overgrowths and diverticula in gut of rats fed on human diet, Brit. M.J. 1:1252-1253, (June) '37.

80. Lynch, J.M.: Diverticula and diverticulitis, J.A.M.A. 98:973-977, (Mar.) '32.

81. Mackoy, F.W.: Family diverticulosis of colon, Radiology 7:498-499, (Dec.) '26.

82. Mailer, R.: Diverticula of colon; pathological and clinical study, Lancet 2:51-57, (July) '28.

83. Mailer, R.: Diverticulitis of sigmoid associated with tuberculosis; report of 2 cases, Am. J. Surg. 2:142-146, (Feb.) '27.

84. Mayo, W.J.: Acquired diverticulitis of large intestine, Surg., Gynec., & Obst. 5:8-15, '07.

85. Mayo, W.J.: Diverticula of sigmoid, Ann. Surg. 92:739-743, (Oct.) '30.

86. McCoy,: Personal Communication to Wierda (113).

87. Mellon, R.R., Soble, N.W., Davidson, S.C., & Fowler, W.F.: Incidence of carcinoma in gastro-intestinal diverticulosis; detailed report of case of gastric diverticulosis, Surg., Gynec., & Obst. 33:177, (Aug.) '21.

88. Morrison, J.R.: Self medication (use of bran) with bad results; diverticulitis, Kentucky M.J. 28:156-157, (Mar.) '30.

89. Moynihan,: Quoted by Chapman (23).

90. Newton, F.C.: Acquired diverticula of colon; end-results in 44 cases, Arch. Surg. 18:1339-1358, (April) '29.

91. Ochsner, H.C., & Bargen, J.A.: Diverticulosis of large intestine; evaluation of historical and personal observations, Ann. Int. Med. 9:282-296, '35.

92. Osler, W.: Quoted by Edwards (37).

93. Pepper, O.H.P.: Diverticulitis of colon; diagnosis and medical treatment. Pennsylvania M.J. 42: 1043-1046, (June) '39.

94. Perry, & Shaw,: Quoted by Spriggs & Marxer (103).

95. Poate, H.R.G.: Diverticulosis and diverticulitis, Australian & New Zealand J. Surg. 11:110-122, (Oct.) '41.

96. Poncher H.G., & Milles, G.: Cysts and diverticula of intestinal origin, Am J. Dis. Child. 45: 1064-1078, (May) '33.

97. Pottenger, F.M.: Personal experience with diverticulitis of sigmoid; study in visceral neurol-ogy, Ann. Int. Med. 4:1295-1307, (April) '31.

98. Rankin, F.W., & Brown, P.W.: Diverticulitis of colon, Surg., Gynec., & Obst. 50:836-847, (May) '30.

99. Schatzki, R.: Roentgenologic differential diagnosis between cancer and diverticulitis of colon, Radiology 34:651-662, (June) '40.

100. Shipley, A.M., & Gerwig, W.H.: Acute diverticulitis, Surg., Gynec., & Obst. 69:474-483, (Oct.) '39.

101. Slesinger, E.G.: Prognosis of diverticulitis, Lancet 1:1458-1459, (June) '35.

102. Springs, E., & Gordon-Watson, C.: Discussion on diverticulitis, Proc. Roy. Soc. Med. 20:57-89, (Mar.) '27.

103. Spriggs, E., & Marxer, O.A.: Intestinal diverticula, Quart. J. Med. 19:1-34, (Oct.) '25.

104. Stewart, W.H, & Illick, H.E.: High and low power roentgenographic differentiation between diverticulitis and cancer of sigmoid, Am. J. Digest. Dis. & Nutrition, 1:738-742, (Dec.) '34.

105. Telling, W.H.: Acquired diverticula of the sigmoid flexure, Lancet, 1:843-850, '08.

106. Telling, W.H.: Diverticula discussion, Lancet 1:85, (Jan.) '20.

107. Upson, W.O., & MacGregor, A.E.: Roentgenological aspect of various types of colonic diverticula, Radiology 16:30-38, (Jan.) '31.

108. Virchow,: Quoted by Guthrie & Hughes (53).

109. Walkling, A.A.: Acute diverticulitis of colon, Pennsylvania M.J. 44:1318-1320, (July) '41.

110. Waterson,: Quoted by Chapman (23).

111. Weible, R.E.: Acute diverticulitis of sigmoid, Am. J. Surg. 28:142-144, (April) '35.

112. Wetherell, F.S.: Diagnosis and management of diverticulosis and diverticulitis of pelvic colon in women, Am. J. Obst. & Gynec. 35: 417-425, (Mar.) '38.

113. Wierda, J.L.: Diverticula of colon in rats fed high fat diet, Arch. Path. 36:621-626, (Dec.) '43.

114. Willard, J.H., & Bockus, H.L.: Clinical and therapeutic status of cases of colonic diverticulosis seen in office practice, Am. J. Digest. Dis. & Nutrition 3:580-585, (Oct.) 36.

115. Willey, C.H.: Etiology of diverticulitis, Brit. M.J. 1:631, (May) '20.

116. Young, E.L., & Young, E.L. (III): Diverticulitis of the colon, New England J. Med. 230:64, '44.