DIVERTICULAR DISEASE OF THE COLON. PATHOGENETICAL, PATHOPHYSIOLOGICAL, RADIOLOGICAL AND SURGICAL ASPECTS.

Dept. of General Surgery (Head: Prof. Dr. B. Löhr) andDept. of Radiology (Head: Prof. Dr. H. Gremmel).University of Kiel, D-23 Kiel, Fed. Rep. Germany.

von Löhr, B., Thiede, A. and Poser H.

Introduction

100 years ago diverticulosis of the large intestine was known (Cruvellier, 1849; Habershon, 1857) but was hardly considered a source of disease. Graser (1899) was the first to make extensive anatomical studies of diverticulosis and called the mucous membrane protrusions pseudoiverticula. In recent decades an increase of this large intestinal abnormality has been noted, frequently accompanied by complications; the condition is known as diverticular disease (Vega, 1976). Besides radiologic analysis, clinical symptoms play a predominant role in the diagnosis of this disease (Goulard and Hampton, 1954;Wolf et al. 1956).

If the assumption is correct that changing nutritional habits are a major factor in the pathogenesis of diverticalar formation (Parks, 1975), then the advent of modernized method of food processing in countries such as Japan, where the disease has been relatively unknown until now, will undoubtedly be accompanied by a rise in diverticular disease, similar to that observed in Europe and the U.S.A. in recent years.

Epidemiology

Classen (1973) reported that by 1980 the U.S.A. is expected to have 7.4 million people with diverticulosis, a fifth of whom (about 1.4 million) will show symptoms of the disease and about 300,000 (a large number!) will require surgical intervention (Table 1). According to Strohmeyer (1976) the figures are expected to be 2.5 million, 450,000 and 100,000 respectively

	Population	People with diverticulosis	those in need of treatment	those needing surgical treatment	author
USA	180	7.4	1.4	0.3	CLASSEN 1973
Fed.Rep. of Germany	60	2.4	0,45	0.1	STROHMEYER 1976

Table 1. Epidemiology U.S.A.-Federal Republic of Germany (Figures in millions).

1977年9月

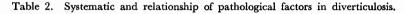
97(555)

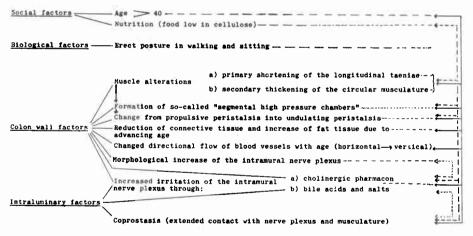
for the Federal Republic of Germany. These figures alone testify to the importance of exact analysis of all prognostic, diagnostic, pathophysiological and therapeutical criteria of this disease. Today it is not sufficient that the surgeon intervene only the cases with complications, rather he should participate in the early stages with consideration of pathogenetical and pathophysiological aspects for treatment. He is, therefore, not only responsible for therapy in the later stages, but should also be consulted at the onset of the disease. The following discussion concerning the pathogenesis of diverticular disease will substantiate this viewpoint.

Pathogenesis

The pathogenesis of diverticulosis is determined by several closely related processes which can be classified as social, biological, colonic wall and intraluminary factors (Table 2).

The social factors include advancing age and nutritional habits. With increasing age, changes occur in the wall of the colon which enable protrusions to break through the muscular wall with simultaneously increased muscular tonus. Stelzner and Liersf (1976) called this a "myostatic contraction" as opposed to fibrosed contracture after atrophy of muscles and



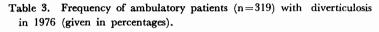


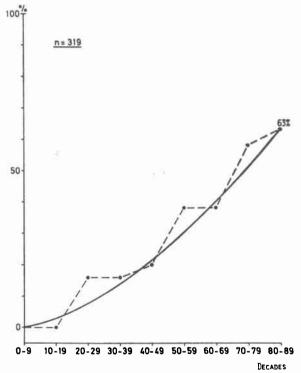
pharmicochemically induced contracture brought about by drug poisoning or changes in ionic concentration. Because of high inner pressure, mucous membrane is forced outward through the breaches in the muscular wall forming so-called pseudodiverticula.

Burkitt et al. (1972) claims that changed nutritional habits, i.e. low intake of roughage, play a significant role as a predisposing factor. Foods low in cellulose —lacking fiber pass through the bowels at a much slower rate than foods with high bulk content (Parks, 1974). This slow passage leads to spastic colonic musculature and thus to increased inner pressure (Stelzner and Lierse, 1976). On the other hand bulky a stool with much undigested material requires shorter passage time and as a result inner pressure is greatly reduced. 98(556)

Whether an erect, sitting, or walking posture is actually a factor as Becker (1976) postulates, must remain questionable since it is possible to induce diverticulosis in animals through appropriate diet (Hodgson, 1972).

Certainly the greatest number of mutually related factors orginate in the colinic wall itself (Table 2). According to Morson (1963) and Morson and Dawson (1974) changes occur in the muscle structure. Primarily there is shortening of the longitudinal taenia and secondarily a thickening of the circular muscle layer (Williams, 1967). By filling up the left side of the colon with silicone foam, Hughes (1969a, b) was able to obtain a three-dimensional





view of the altered colonic anatomy and thereby secured evidence supporting this opinion. Comparative histological studies have confirmed the abnormalities in the muscles and nerve plexus (Becker, 1976). The anatomical alterations are accompanied functionally by the formation of "segmental high pressure chambers" as described by Reifferscheid (1967). These are formed by extreme haustration and lead to high segmental pressure which Parks and Connel (1969) were able to demonstrate by open-end catheterization. Slack (1962) and Reiffersheid (1967) drew particular attention to changes in the vascular course which occur with advancing age and are simultaneously accompanied by a reduction of connective tissue.

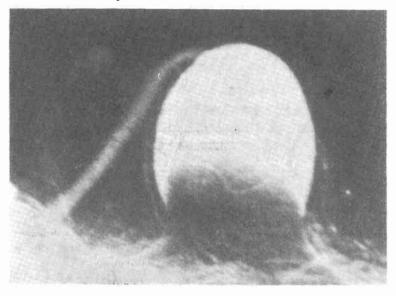


Fig. 1. Radiographic demonstration of the relationship between a diverticulum and vessel. Microscopic evaluation.

We have developed a special radiologic examination technique whereby it is possible to show the formation of diverticula on the vasuclar breaches in the muscular wall (see Fig. 1).

Whether the morphological increase in the intramural plexes observed by Becker and Brunner (1974) is a primary or secondary phenomenon remains undecided. Functionally, it has been established, however, that increased irritation in the circular colonic muscles in the diverticular intestine is due to physiological and pharmalcoogical substances (Painter and Truelove, 1964).

An increase in frequency and a rise in the intensity of pressure waves after injections of morphine and prostigmine were observed through a combination of pressure measurements and cineradiography. The greatest pressure occured again in the "segmental high pressure chamber".

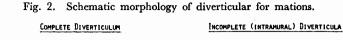
A similar stimulating effect may be attributed to bile acids and salts, especially through relatively high concentration and extended contact, both of which are caused by coprostasis.

Of course, the numerous factors leading to diverticular formation should be not considered by themselves. The formation of diverticula is brought about through the coincidence of several predisposing factors. This must also be considered with regards to the respective choice of treatment —whether conservative or operative—.

Pathophysiology and Anatomy

The relationship between pathophysiological and anatomical abnormalities in the in-





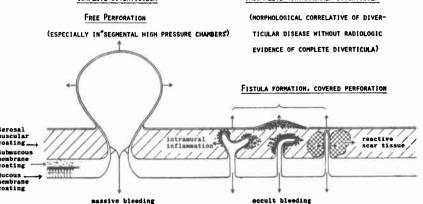


Fig. 3. Histological demonstration of a complete diverticulum.

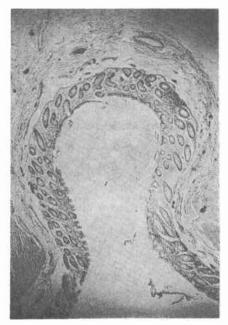


Fig. 4. Histological demonstration of an incomplete diverticulum with signs of intramural inflammation.



testianl wall explains the clinical progress of diverticular disease. As can be seen from the studies of Schreiber et al. (1966), Reifferscheid (1967) Schumpelick and Koch (1974), there are various forms of diverticula which exhibit distinctly different prognostic significance (see Fig. 2).

Complete diverticula are mucous membrane prolapses with a serosal coating and are, strictly defined, not true diverticula but so-called Graser pseudodiverticula (1899); true diverticula protrude through all layers or the intestinal wall (see Fig. 3). Complete diverticula are mostly responsible for free perforations which are often due to pressure necrosis caused by fecal stones of their early stages accompanied by simultaneous increase in intraluminary pressure. Secondly, ulcerations lead to the erosion of larger vessels in the neck area resulting in massive bleeding which cannot stop of its own accord because of the relatively wide lumen (see Fig. 2).

Incomplete or intramural diverticula, a second type of diverticular formation, are often T-shaped invaginations of the large intestinal mucous membrane which communicates with the intestines by way of a very narrow lumen. Unlike the complete, transmural diverticula, they are sheathed with a strong layer of muscle. According to Schumpelick and Koch (1974). rejected epithelial cells, fecal remains and leukocytes are found in the lumen where they are highly predisposing to infection. Already in the early stages of development, lymphocyte patches can be identified on the base, along with perifocal swelling of the surrounding muscles and mucous membrane abnormalities. In later stages, in which most of the complete diverticula are still fully free of infection, microabscesses may be found in the surrounding area (Fig. 4). The results are oedematic swelling of the intestinal walls and elimination fissures with microscopic blood clots in the venous drainage vessels. The surrounding tissue becomes scarred and then fibrosclerotic leading to contraction of the walls and finally to stenosis. Covered perforations or fistula formations are frequent and there is often occult hemorrhaging in the lumen. Incomplete diverticula are predisposers and as such are the anatomical-pathological correlates of perisigmoiditis. If diverticular disease is brought about as the result of incomplete diverticula, then it must be understood that complete recovery is hardly possible. rather the disease will continue to progress.

Radiologic Diagnosis

A reliable diagnosis of the disease and its complications is largely dependent on radiology. The preferred method presents the large intestine by means of the Malmö-technique (Welin and Welin, 1976) which is based on the work of the Kiel surgeon A.W. Fischer (1925). In our opinion, other techniques are inadequate and may lead to false interpretations. Examples are given in Figs. 5–10.

Especially important for the surgeon is the preoperative differentiation between diverticulosis and diverticular disease with their complications.

Diverticulosis is usually found in segments of the sigmoid and descending regions of the colon. This is a typical condition in the Federal Republic of Germany, comprising nearly 60% of the cases. Pronounced spasticity and lack of elasticity in the affected segment with relatively good flexibility in the neighboring large intestinal region is typical for diverticular

102(560)

Fig. 5. Double contrast study of the colon with the Malmö-technique. Segmental diverticulosis in the sigmoid area.



Fig. 6. GRASER (**(**) diverticula in the caecum and ascending colon inflammatory reaction on the tip of the caecum.



Fig. 7. Demonstration of incomplete diverticula (\rightarrow) by double contrast technique.

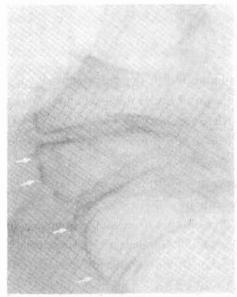


Fig. 8. X-ray, diverticulum (\Rightarrow) with an infected head and stenosis of the neck.

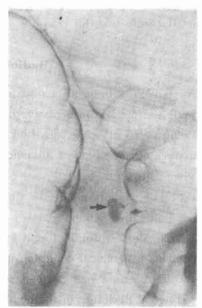
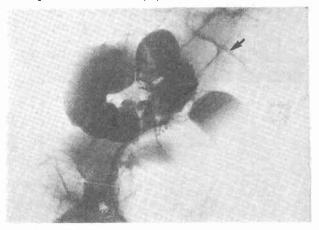


Fig. 9. X-ray; covered perforation (\mathbf{P}) .

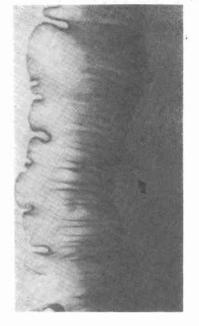
Fig. 10. X-ray; typical pericolic reaction between posterior rectal wall (→) and Os sacrum (←).



disease of the left colon (Fig. 5). The disease seldom involves the caecum (Fig. 6). Radiologic demonstration of incomplete diverticula is only possible through careful employment of the Malmöo-technique (Fig. 7).

Inflammatory determination of the diverticular head and stenosis of the neck can be shown by radiology and thus provides evidence of diverticular alterations caused by inflammation (Fig. 8). Covered perforations are among the serious complications (Fig. 9). If the tissue surrounding the large intestine becomes inflammed during this disease, retrorectal widening of the area results as soon as the spreading reaction reaches the sigma-rectum-area (Fig. 10). Normally, the distance between the sacrum and the posterior rectal wall should not exceed 1 cm; if this distance widens considerable, it is a typical reaction of the surrounding area during advanced diverticual disease of this intestinal segment. This can be visualized by X-ray.

It is often difficult to differentiate between infected stenosis at the base of diverticula and carcinomatoid stenosis. A procedure that we have developed (Thiede et al. 1977) can be of great help in making clear, reliable diagnoses. Our procedure involves pin-pointed coloscopic/radiologic demonstration of the large intestine. A tri-iodized contrast medium, Conray- 80 for example, is injected through afl exible coloscope. This allows demonstration of the stenosed large intestinal segment without its being overshadowed. An example can demonstrate this. The segment which has been extremely altered by diverticular disease simulates a complete tumorous stenosis. The special, combination examination technique



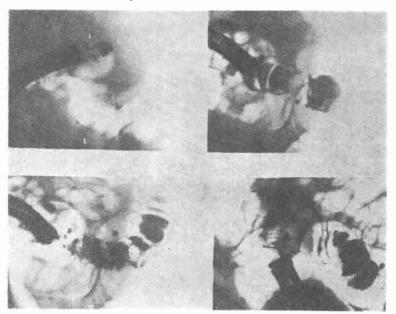


Fig. 11. Combined coloscopic-radiologic demonstration of a sigmoid stenosis in diverticular disease in 4 phases.

indicated a stenosis caused by diverticular disease (Fig. 11). The operative data confirmed this. Coloscopy alone plays but a minor role in the diagnosis of diverticula. Only the localization and morphology of a beginning diverticular neck can be determined endoscopically.

Radiologic Evaluation

An evaluation of double contrast studies of the colon carried out on a total of 319 patients in 1976 revealed some noteworthy data on diverticual disease. Table 3 shows the relative frequency of this disease according to decades of life. Although we found no instances in the first and second decases, there was a steady rise from the third to the ninth decades, increasing to 63% of all patients examined. The number of cases was relative low; nevertheless, it seems to us that these figures are still representative for the population of our country as a whole. The evaluation also yielded some exact data concerning the localization of diverticula. In a total of 116 patients with diverticulosis or diverticular disease, diverticula formations were distributed throughout the colon as follows (see Fig. 12): 57.8% were found in the sigmoid and descending colon (a), 16.4% in the transverse and the left side of the colon together (b), 8.6% in the entire large intestine (c) and 6.9% in the ascending colon (d). A combined appearence in the ascending, descending and sigmoid colon was found in 6%. Further localizations or combinations were very rare.

e)

8,6% n = 116 16,4 %

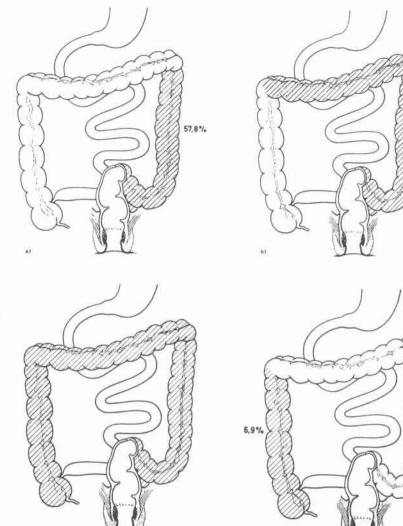


Fig. 12. Diagrams showing the radiologically determined frequency of the localisation of diverticulosis.

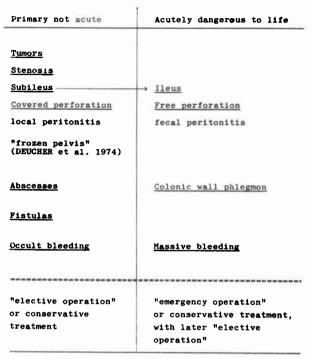
Complications

4)

Complications are diagnosed by clinical, laboratory technical and particularly by radiological criteria. In diverticular disease, complications occur which we differentiate primarily as not acute or acutely dangerous to life (Table 4). Tumors, stenosis, subileus, covered perforations, abscesses, fistulas, and occult hemorrhaging are not considered mortally dangerous and maybe treated operatively or even conservatively. On the contrary, ileus, free perfora



Table 4. Complications of diverticular disease.



tion with fecal peritonits, colonic wall phlegmon and massive bleeding can usually be contr olled only by emergency surgery. However, in the cases of massive bleeding, one should always firstattempt a procedur in which the intestine is washed out with an ice-cold saline solut ion to which 1–2 ampules Suprarenin– have been added per 1000 ml. In 90% of the cases the hemorrhaging will stop and recurrence can be prevented by elective operative intervention afterselective diagnosis has been carried out. According to Taylor and Epstein (1969), bleedin grecurs in 20–40% of the cases after purely conservative treatment.

In cases with complications, radiography is the most important examination technique, whether through total abdominal view by perforations with free air in peritoneal cavity, or as a double-contrast enema with a tri-iodized contrast substance, e.g. Urovision-, to disclose the presence of tumors, stenosis, covered perforations or fistulas. On the contrary, angiography had been a disappointment in discerning between carcinomas and stenosis of the sigmoid region caused by diverticula. Differentiation through vascular demonstration is not possible preoperatively. At best, angiography can be a diagnostic aid in localizing heavy diverticular hemorrhaging and enables preoperative localization of the source of bleeding in 30-50% of the cases, only, however, in patients with massive bleeding (Heuck, 1976).

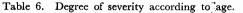
Surgical Aspects

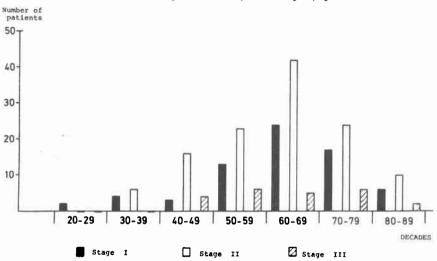
We have classified the hospitalized cases at the Kiel Clinic from the last 14 years into 3 stages of development from the standpoint of clinical observation. This classification is, in our opinion, the best method of showing the therapeutical aspects; it is clearly difined and understandable (Table 5).

The statistics for the years 1963–1976 are presented in the table as well as the symptoms in percentages. Table 6 gives a diagram of the stage of severity corresponding to the age of

Clinical Stages	Criteria for clinical classification	cases	Symptoms/Findings		
I	radiologic evidence <u>without</u> clinical symptoms	69			
II	with or without radiologic evidence with	122	digestive difficulties colic and tenesmus occult bleeding "left appendicitis"	57 54 29 24	
	clinical symptoms		palpable tumors stenosis	1 3 10	
111	complications which are	82	free perforation	82	
	acutely dangerous to life		ileus massive bleeding	9 9	

Table 5. Hospitalized patients with diverticulosis and diverticular disease 1963–1976. Criteria for classification of the clinical stages.





108(566)

the hospitalized patients. Stage III was not observed before age 40; after 40 there was a rise in frequency increasing with advancing age.

The operative procedure depends on whether the patient has a complicated or an uncomplicated form of the disease. While the uncomplicated diverticular disease is usually treated by one-stage-resection, this procedure is considered too risky for the treatment of the complicated form for which various other methods are more suitable (Summary by Parks, 1976).

There are several methods of multi-stage-resection which can be applied selectively according to the symptoms and the clinical and anatomical condition of the patient. The threestage-procedure of Schloffer (1. step=colostomy+drainage of the peritoneal cavity; 2. step= resection; 3. step= closure of the colostomy; Baumgartel at al. 1972) is accompanied by a high mortality rate (Smiley, 1966; Heberer et al. 1974). The source of infection continues to contaminate the surrounding area in spite of fecal elimination (Deucher et al. 1974).

The two-stage-incontinuity-resection (Miller and Wichern, 1971) with excision of the diseased segment can be applied in several modifications. In the procedure according to Mikulicz, the proximal and distal sigmoid loops are sewn to the left underside of the abdomen in double-abrrel form. In the second step, the two large intestinal loops are rejoined. In Hartmann's resection the proximal sigmoid loop becomes a terminal anus and the distal sigmoid loop is intraperitoneally closed.

The procendre is the same in the resection described by Guleke with the exception that the sigmoid loop is extraperitoneally closed.

These operative proceedres are advantageous since the relatively brief operating time increases the chances of survival even in severly ill, older patients. Parimary resection with protective colostomy (Madden, 1965) should only be attempted on younger patients in relatively good general condition, as it requires longer operating time which involves additional risks for the patient. Here again the colostomy must be closed in second step.

Classification according to severity	Conservativ or without therapy	operative				
I 69 patients	69	-				
2000 C				Method	of operation	
		60	one stage		multi-stage	
II 122 patients	62		Colostomy 2	primary résection 52	two stage	three stage
111			Colostomy a. drainage			
22 patients	1	21	7	4	6	4

Table 7. Therapeutical procedure in 213 patients (1963-1976) hospitalized.

1977年9月

The therapeutic procedures carried out on 213 patients in Kiel during the years from 1963 to 1976 are given according to clinical stage in Table 7.

All persons in Stage I either received no special treatment or were treated conservatively, that is by diet control. Nearly half of the patients in Stage II underwent surgery, with onestage-resection being the predominatly elected course. The 22 patients in Stage III, those with mortally dangerous complications, were treated by various methods; the procedure was dictated by local findings and above all by the general condition of the individual patients. In almost all cases operative measures were undertaken (also in a case with heavy hemorrhaging while another such case was treated conservatively). The patients receiving only a colostomy and drainage died of therapeutically resistant fecal peritonitis. A multi-stage operative procedure had been indicated. Due to the high rick factors, we have seldom performed one-stage-resection during the last 5 years. We have found the two-stage-procedure to be more facorable, preferring the incontinuity resection of the Hartmann type. The lower mortality rate as well as shorter hospitalization —55 days for the two-stage-resection.

Our primary hospital mortality rate was, at that time, about 6.5%; in 3.2%, i.e. two patients, suture insufficiency was the cause of death. The primary mortality rate of patients in Stage III is rather dismal. In 7 of 11 cases, therapeutic resistant fecal peritonitis was responsible for death. The ability to control this factor is exceptional even tody. There is a chance of survival only if the time between perforation and operation is short. The situation is hopeless if 3-4 hours have elapsed since perforation. Neither conservative nor operative therapy are sufficient to check a neglected fecal peritonitis.

Long-term Observations of Conservative Versus Operative Therapy

The high mobility of the working population in the Federal Republic of Germany makes it difficult to observe development over longer periods of time; many patients could not be traced 5 years or more after hospitalization. Therefore, the figures given in table 8 may only be understood as tendencies, that is, they are not truly representative.

Of the 14 patients who were classified in Stage I and who did not undergo surgery, 9 still showed Stage I symptoms 5 years later, while 5, that is 1/3, had progressed to Stage II. The tendency of group II patients is impressive: Of 20 conservatively treated cases, 18 had remained stationary, i.e. by exact examination of their case histories they continued to show symptoms of Stage II. Two patients, however, had died in the meantime of free perforation, followed by consecutive peritonitis and had, therefore, progressed to Stage III. The large majoritiy of patients in Stage II who had been operatively treated showed few or no symptoms of the disease 5 years later. This was also true of the few patients in Stage III whom we were

Clinical stage at hospital admission	Treatment	Clinical stage at follow-up ex 5 years later	amination Cause of death
I n = 14	none or conserva- tive	I 9 II 5	
II	conserva- tive n = 20	II 18 III 2 (death)	2 fecal peri- tonitis
n = 34	operative n = 14	I completely 12 free of symptoms II 2	
III n = 4	operative n = 4	I completely 3 free of symptoms	
		II 1	

Table 8. Long-term observations. Conservative therapy versus operative treatment.

able to trace.

The long-term results of our conservatively treated patients were quite good, that is, no deterioration of the disease in 77%. Improvement was achieved in 90% of the patients receiving operative treatment. In comparison, statistics collected by Reifferscheid (1976) showed good long-term results after resection in 98.5% (762 cases), while only 66.5% (992 cases) of the conservatively treated group were described as good.

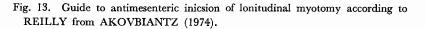
Myotomy

Myotomy of the sigmoid intestine in the early stages has recently been propagated as a new technique. This technique was developed by Reilly (1965, 1966, 1975) who referred to it as longitudinal myotomy. The separation of the muscles is supposed to lead to reduction of pressure in the sigmoid colon. In the original procedure, however, only the thickened circular fibers between the taenia were separated (Akobviantz, 1974; Fig. 13). The method is controversial because the mortality rate lies at 5% (Parks, 1974); and the intraluminal pressure reduction remains effective for only about 3 years. After that time there is a recurrence of the original condition (Smith and Ruckley, 1971; Prasad and Daniel, 1971) unless paraysmpatholytics and a high cellulose diet are prescribed.

The transverse myotomy method, which has gained current popularity (Hodgson, 1973; Kyrle, 1976), and the somewhat similarly evaluated method of spiral myotomy (Parks, 1974) can be documented only by a few short-term observations as no long-term data are available at this time.

Colonic Fistulas in Diverticular Disease

The therapeutic approach to diverticular colinc fistulas deserves special attention (Colcock



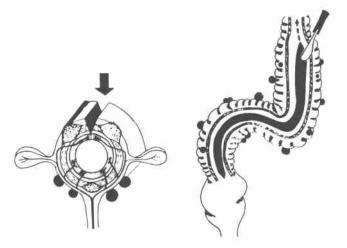
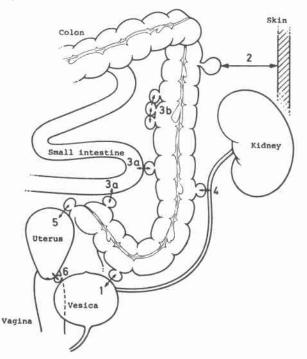


Fig. 14. Schematic fistula formation in diverticular disease.



and Stahmann, 1972; Kraft-Kinz and Prexl, 1976). General recommendations can only be arrived at through collective statistics. Neighboring organs in which diverticular fistulas have been known to develop are shown in Fig. 14 and Table 9. Fistulas appear in about 15% of the cases to be operated. Fig. 14 and table 9 demonstrate the different fistula formations,

112(570)	
----------	--

Localization	Frequency (n = 278)	Operative procedure		
		without abscess formation	with abscess formation	
colo-vesical	145 - 51.2 %	one-stage-resection two-stage-resection		
colo-cutaneous	90 - 32.4 %	<u>one-stage-resection</u> two-stage-resection		
colo entric a. colic b.	37 - 13.3 %	two-stage-resection	alvays two-or three- stage-resection	
colo-uretherale	1 = 0,4%			
colo-uterine	4 = 1,4%	two-stage resection		
5 colo-vaginal	1 - 0,4 %			

Table 9. Appearance and treatment of colonic fistulas in diverticular disease. 278 patients out of a total of 1840 had preoperative fistulas.

 $Collective \mbox{ statistics: COLCOCK a. STAHMANN 1972(n = 1555); \mbox{ DEUCHER et al. 1974 (n = 152), } \\ KRAFT-KINZ a. PREXL 1976 (n = 52); \mbox{ own cases } (n = 81)$

their frequency and therapeutic treatment. The various possibilities are listed according to decreasing frequency icolo-vesical, colo-cutaneous, colo-entric and colo-colic, colo-uretheral, colo-uterine and colo-vaginal fistulas. 98% are found in the first three categories; the last 3 are very rare as the collective statistics for 278 cases indicates (Table 9).

The mode of surgery is still essentially determined by the existing abscess formations. When abscesses are still in evidence, a multi-stage-procedure is called for; a one-stage-resection may be considered only if fistulas are present between the large intestine and the bladder or the skin, and if the abscesses are no longer in evidence.

Intraoperative Combined Evaluation of the Resected Segment

For almost a year now we have been analyzing each resected large intestinal segment intraoperatively by means of a combined technique. A mucous membrane demonstration is achieved with Barotrast and simultaneously, a vascular injection is carried out. Up to now we have been able to collect some remarkable facts about diverticular disease. The excellent X-rays obtained through this technique are evaluated microscopically; some of the capillaries may even be evaluated by this method.

Some of the data we have acquired can be documented by a few examples. To our knowledge, such examinations have not been reported before in the literature. The question of whether incomplete diverticula are still present at the edge of the resection can be clarified intraoperatively by microscopic evaluation of the X-rays; if necessary, these pathogenically crucial incomplete diverticula can then be resected immediately in order to achieve radical surgery (see Fig. 15). Incomplete diverticula do not exceed the intestinal wall niveau. Signs

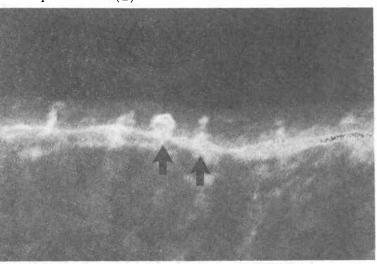
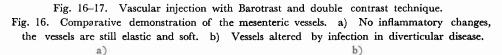
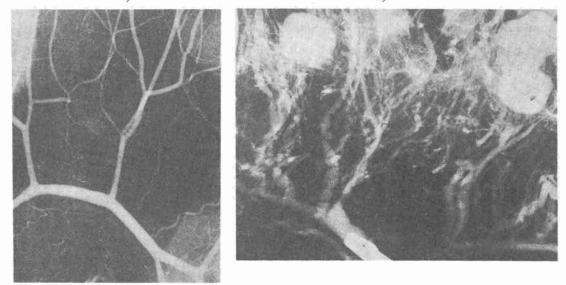


Fig. 15. Demonstration of small incomplete diverticula through X-ray and microscopic evaluation (1).





of infection are indicated on the diverticular neck, causing abnormalities in the area. Further information about the inflammatory involvement in the mesentery can be obtained by radiology. Figs. 16a) and b) are exemplary: The diverticular disease illustrated in Fig. 16a) is still confined to the wall of the colon. The mesenteric vessels show no inflammatory changes and are still elastic and soft. Fig. 16b) shows the vessels in a mesentery which have been ala)

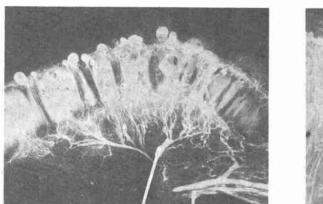


Fig. 17. Comparative demonstration of an intestine segment. Diverticular disease. b) Crohn' 8disase



tered by infection in a case of segmental diverticular disease of the colon. Irregularities and stenosation of the vessel walls can be recognized. Such examinations are a great aid in understanding the tendency of diverticular disease to spread —begininng in the colonic wall and encroaching secondarily on the mesentery. Differentiation from other inflammatory intestinal disaeases is also possible with this technique. A comparative demonstration documents this impressively. (Fig. 17a and b). 17a) shows a specimen from diverticular disease and 17b) an intestine infected with Crohn's disease. Arteriovenous aneurysms, in particular, are typically located in the intestinal wall itself during Crohn's disease. The mesentery vessels, however, show a different kind of abnormality when affected by diverticular disease.

Trends in Contemporary Treatment of Diverticular Disease

The abundance of pathological-anatomical, radiological and functional data (Graser, 1898; Schreiber, 1965; Parks, 1969; Heberer et al. 1970; Becker a. Brunner, 1974; Heuck, 1974; Ottenjann, 1974; Becker, 1976; Stelzner, 1976 and others) has considerably influenced the attitude towards diverticular disease. Our own studies (Thiede et al. 1975) as well as those of Kümmerle and Pross (1974) have shown that the primary mortality of Stage III patients can be as high as 50%. Surgical techniques can not substantially reduce this high death rate, since the patients succumb to therapeutically resistant complications of the disease. In view of these facts, two demands must be made of the conservative and operative therapists:

1. When possible, the occurence of diverticular comlications must be prevented through diet and medical treatment. This can be accomplished by the constant prescribing of wheat bran and foods high in cellulose as laxatives e.g. Agiolax- (Brodripp and Humphreys, 1976; Schellerer, 1976; Strohmeyer, 1976).

2. Close cooperation between the internal and surgical gsatroenterologists should begin

at the onset of the disease in order that resection can be performed as early as necessary (Hollender et al. 1974). It has not yet been established whether the myotomy procedure (Reilly, 1971) and its modifications in the early stages are of therapeutical significance, since longterm observations are not available (Deucher, 1976).

Summary

The recent sharp increase in the number of people with diverticular disease, particularly in the Western industrial nations, necessitates the analysis of all epidemiological, pathogenetical, anatomical, and pathophysiological parameters of the disease. A number of mutually realted pathogenetic factors, classified as social, biological, colonic wall and intraluminary, are responsible for the appearance of this disease. Pathophysiology considerably influences the prognosis. Predominant decisive factors in the diagnosis are radiological demonstration (e.g. double-contrast study of the colon) and cilnical symptoms. Subtle X-ray diagnosis also assists in the acquisition of information about the relative frequency and localization of the of the diverticula. Distribution into three clinical stages is based on therapeutical observation. Operating techniques are standardized tody and as is shown in this study, can be applied differentially in Stages II and III. For patients in Stage II, primary resection is the "elected" procedure, while Stage III usually calls for multi-stage resections, the incontinuity resection of the Hartmann type being the preferred method. Because of poor prognosis in cases with complications, resection should be carried out in the early stages of the disease; inflammatory involvement caused by incomplete diverticula also speaks for early resection. Insufficient data are available to evaluate the various myotomy techniques. Colonic fistulas, which appear in about 15% of the cases of diverticular disease, require a multistage operational procedure in the majority of the cases. Combined intraoperative evaluation of the resected segment is advantageous in determing the achieved operative radicalness, especially regarding the pathogenetically important incomplete diverticula. This procedure is also an aid in bringing to light new aspects concerning the genesis and spread of diverticular disease.

Literature

Akoveiantz, A.: Die Myotomie in der Behandlung der Kolondivertikulose und divertikulitis in: Kolondivertikulitis, Reifferscheid, M. Thieme, Stuttgart (1974.)

Baumgartel, F., Kremer, K. and Schreiber, H.W.: Spezielle Chirurgie für die Praxia. Thieme-Stuttgart (1972).
Becker, V.: Pathologisch anatomische Aspekte zur Entstehung von Divertikeln und ihren Komplikationen.
Langenbecks Arch. Chir. 342 (1976) 401.

Becker, V. and Brunner, H.P.: Divertikulose, Divertikulitis. Pathogenese und Patholgoische Anatomie in: Kolondivertikulitis. Hrsg. Reifferscheid, M. Stuttgart, Thieme (1974).

Brodripp, A.J.M. and Humphreys, D.M.: Diverticular disease: Three studies. Brit. Med. J. 1 (1976) 424.

Burkitt, D.P., Walker, A.R. and Painter, N.S.: Effect of dietary fiber on stools and the transit times and ts roe in the cunsation of disease. Lancet 1972 II, 1408.

Classen, M.: Divertikel des Darmes. In: L. Demling (ed): Klinische Gastroenterologie, Bd. I Stuttgart:

Thieme 1973.

Colcock, B.P. and Stahmann, F.D.: Fistual complicating diverticular disease of the simgoid colon. Ann. Surg. 175 (1972) 838.

Curvellier, J.: Traité d Anatomie descriptive. Paris méd. 1 (1849) 593.

- Deucher, F.: Rundtischgespräch: Colondivertikulitis. Langenbecks Arch. Chir. 342 (1976) 453.
- Deucher, F., Blessing, H. and Fartab M.: Die chirurgische Behadnlung der Kolondivertikulitis. Bericht über 152 Fälle, in: Kolondivertikulitis: M. Reifferscheid Thieme-Stuttgart 1974.
- Ficher, A.W.: Über die Röntgenuntersuchung des Dickdarmes mit Hilfe einer Kombination von Lufteinblasung und Kontrasteinlauf. Langenbecks Arch. Chir. 134 (1925) 209.
- Graser, E.: Entzündliche Stenosen des Dickdarmes bedingt durch Perforation multipler falscher Divertikel Zbl. Chir. 25 (1898) 140.
- Graser, E.: Über multiple falsche Darmdivertikel in der Flexura sigmoidea. Münch. med. Wscher. 46 (1899b) 741.
- Goulard, A. Jr. and Hampton, A.O.: Correlation of the clinical pathological and roentgenological findings in diverticulitis. Am. J. Roentgenol. 72 (1954) 213.

Habershon, S.O.: Observations on the alimentary canal. London 1857.

Heberer, G., Brehm, H.Y and Hirshfeld, J.: Die Divertikelerkrankungen des Dickdarmes. Chirurg 41 (1970) 252.

Heberer, G., Hoffmann, K. Bary, S.V. and Nakano, H.: Zur operativen Therapie der Dickdarmdivertikulitis. Münch. med. Wschr. 116 (1974) 1075.

- Heuck, F.: Die Divertikulitis in Röntgenbild, Langenbecks Arch. Grri 342 (1976) 421.
- Hodgson, J.: An interim report in production of colon diverticula in the rabbit. GUT 13 (1972) 802.
- Hodgson, J.: Transverse taeniomyotomy for diverticular disease. Dis. Col. & Rect. 16 (1973) 283.
- Hollender, L.F., Meyer, C. B, F. and Marrie, A.: Plädoyer für die Frühresektion der Sigme-Diverticulitis. in: Kolondivertikulitis. Hrsg. M. Reifferscheid Thieme-Stuttgart (1974).
- Hughes, L.E.: Post-mortem survey of diverticular disease of the colon Part. I. Diverticulosis and diverticulitis. GUT 10 (1969a) 336.
- Hughes, L.E.: Post-mortem survey of diverticular disease of the colon Part II. The musculer abnoramlity in the sigmoid colon. GUT 10 (1969b) 344.
- Kraft-Kinz, J. and Prexl, A.J.: Die komplizierte Divertikulitis und ihre Behandlung. Langenbecks Arch. Chir. 342 (1976) 431.
- Kümmerle, F. and Pross, E.: Ein- oder mehrzeitiges Vorgehen bei der komplizierten Divertikulitis. in: Koloudivertikulitis. Hrsg. M. Reiffescheid Thieme-Stuttgart (1974).
- Kyrle, P.: Die Myotomie als Behandlung der Divertikulitis. Langenbecks Arch. Chir. 342 (1976) 445.
- Madden, J.L.: Primary resection and anastomosis in the treatment of perforated lesions of the colon. Amer. Surg. 31 (1965) 781.
- Miller, D.W. Jr. and Wichern, W.A. Jr.: Perforated sigmoid diverticulitis. Appraisal of primary versus delayed resection. Amer. J. Surg. 121 (1971) 536.
- Morson, B.C.: The muscle abnormality in diverticular disease of the sigmoid colon. Brit. J. Radiol. 36 (1963) 385.
- Morson, C.B. and Dawson, I.M.P.: Gastrointestinal pathology. Muscular disorders. Chapter 35. Blackwell Scientific Publication, Oxford, London, Edinburh, Melbourne (1974).
- Ottenjann, R.: Divertikulose und Divertikulitis des Dickdarmes. Münch. med. Wschr. 116 (1974a) 1069.
- Ottenjann, R.: Divertikulose und Divertikulitis aus interner Sicht. in: Kolondivertikulitis. Hrsg. M. Reiffercsheid, Thieme-Stuttgart (1974b).
- Painter, N.S. and Truelove, S.C.: Intraluminal pressure pattern in diverticulosis of the colon. GUT 5 (1964) 201.
- Parks, A.G.: Divertikulitis, Pathophysiologie und chirurgische Indikation. In: Kolondivertikulitis: Hrsg. Reifferscheid, Stuttgart-Thieme 1974.
- Parks, A.G.: Ätiologie und Pathogenese der Divertikulose. Schweiz. med. Wschr. 105 (1975) 825.
- Parks, T.G.: Natural history of diverticular disease of the colon. A review of 521 cases. Brit. med. J. 4 (1969) 639.
- Parks, T.G.: Diverticular disease of the colon. Chapter 84. Part II. Surgery of diverticular disease of the colon. in: Gastroenterology. A.L. BOCKUS. Saunders C., Philadelphia-London-Toronto (1976).

1977年9月

Parks, T.G. and Connel, A.M.: Motility studies in diverticular disease of the colon. Part I. Basal activity and response to food assessed by opened tube and miniature balloon techniques. GUT 10 (1969a) 534.

Parks, T.G. and Connel, A.M.: Motility studies in diverticular disease. Part II. Effect of colonic and rectal distension. GUT 10 (1969b) 538.

Prasad, J.K. and Daniel, O.: Recurrence of high intracolic pressure following sigmoid myotomy. Brit. J. Surg. 58 (1971) 304.

Reifferscheid, M.: Pathogenese der Sigmadivertikulitis und die Indikation zur Resektionsbehandlung. Langenbecks Arch. Klin. Chir. 318 (1967) 134.

Reiffersheid, M.: Die Frühresektion der Divertikulitis. Langenbecks Arch. Chir. 342 (1976) 439.

Reilly, M.: Sigmoid myotomy for acute diverticulitis. Dis. Colon & Rect. 8 (1965) 42.

Reilly, M.: Sigmoid myotomy. Brit. J. Surg. 53 (1966) 859.

Reilly, M.: Sigmoid myotomy for diverticular disease of the colon. In: Irvine, W.T. (ed): Modern Trends in Surgery. ED. 3. London Butterworth & Co. Ltd. (1971).

Reilly, M.: Sigmoid myotomy. Clin. Gastroent. 4 (1975) 1.

Schellerer, W.: Nachuntersuchungsergebnisse konservativ behandelter Divertikulitis-Patienten. Langenbecks Arch. Chir. 342 (1976) 449.

Schreiber, H.W.: Neue Gesichtspunkte zur Divertikulitis des Dickdarmes. Dtsch. med. Wschr. 90 (1965) 1998.

Schreiber, H.W., Koch, W. and Oestern, H.F.: Über den Krankheitswert der inkompletten Dickdarmdivertikel. Zbl. Chir. 91, (1966) 1740.

Schumpelick, V. and Koch, G.: Die Bedeutung des inkompletten Dickdarmdivertikels für die Divertikulitis. Langenbecks Arch. Chir. 336 (1974) 1.

Slack, W.W.: The anatomy, Pathology and some clinical features of diverticulosis of the colon. Brit. J. Surg. 50 (1962) 185.

Smiley, D.F.: Perforated sigmoid diverticulitis with spreading peritonitis. Am. J. Surg. 111 (1966) 431.Smith, A.N. and Ruckley, C.V.: Disease of the colon. Scott. Med. J. 16 (1971) 130.

Stelzner, F.: Strukturveränderungen der Colonwand als Ursache der Divertikulose und Divertikulitis. Langenbecks Arch. Chir. 342 (1976) 411.

Stelzner, F. and Lierse, W.: Über die Entwicklung der Divertikulose und Divertikulitis. Langenbecks Arch. Chir. 341 (1976) 271.

Strohmeyer, G.: Internistische Aspekte der Divertikulose und Divertikulitis. Langenbecks Arch. Chir. 342 (1976) 412

Taylor, F.W. and Epstein, L.I.: Treatment of massive diverticular hemorrhage. Arch. Sarg. 98 (1969) 505.

Thiede, A., Brieler, H.S. and Hantschmann, N.: Divertikulose, Divertikulitis und Perisigmoiditis. Zbl. Chir. 100 (1975) 1082.

Thiede, A., Schewelises, S. and Poser, H.: Gezieltere Dickdarmdiagnostik durch kombinierte coloskopisch radiologische Dickdarmsegmentdarstellung. in Vorbereitung 1977.

Vega de la, J.M.: Diverticular disease of the colon. Chapter 84. Part I Medical aspects. in: Gastroenterology II. H.L. Bockus. Sanunders C., Philadelphia-London-Toronto (1976).

Welin, S. and Welin G.: The double contrast examination of the colon and experiences of the Welin-modification. Thieme-Stuttagrt (1976).

Williams, J.: Mass movements (mass peristalsis) and diverticular disease of the colon. Brit. J. Radiol. 40 (1967) 2.

Wolf, B.S., Khilnani, M. and Marshak, R.H.: Diverticulosis and diverticulitis. Rogentgen findings and their interpretation. Am. J. Roentgenol. 77 (1957) 726.