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

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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 anatomic studies of diverticulosis and called the mucous membrane protrusions pseud diverticula. 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 diverticular 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

<table>
<thead>
<tr>
<th>Population</th>
<th>People with diverticulosis</th>
<th>those in need of treatment</th>
<th>those needing surgical treatment</th>
<th>author</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>180</td>
<td>7.4</td>
<td>1.4</td>
<td>0.3</td>
</tr>
<tr>
<td>Fed. Rep. of Germany</td>
<td>60</td>
<td>2.4</td>
<td>0.45</td>
<td>0.1</td>
</tr>
</tbody>
</table>
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 fibroed contracture after atrophy of muscles and

| Table 2. Systematic and relationship of pathological factors in diverticulosis. |
|----------------------------------|----------------------------------|----------------------------------|----------------------------------|
| Social factors                   | Age                              | Nutrition (food low in cellulose) |
| Biological factors               | Erect posture in walking and sitting |
| Colonic wall factors             | Muscle alterations               |
|                                 | a) primary shortening of the longitudinal taniae |
|                                 | b) secondary thickening of the circular musculature |
|                                 | Formation of so-called "segmental high pressure chambers" |
|                                 | Change from propulsive peristalsis into undulating peristalsis |
|                                 | Reduction of connective tissue and increase of fat tissue due to advancing age |
|                                 | Morphological increase of the intramural nerve plexus |
|                                 | Increased irritation of the intramural nerve plexus through: |
|                                 | a) cholinergic pharmacology |
|                                 | b) bile acids and salts |
| Intraluminary factors            | Coprostasia (extended contact with nerve plexus and musculature) |

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 Liers, 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.
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 originate in the colonic 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.
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 pharmaacoogical 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 occurred 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 not be 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 pathophysiologial and anatomical abnormalities in the in-
Fig. 2. Schematic morphology of diverticular formations.

**Complete Diverticulum**
- Free Perforation
  - (especially in segmental high pressure chambers)

**Incomplete (Intramural) Diverticula**
- Morphological correlative of diverticular disease without radiologic evidence of complete diverticula

Fig. 3. Histological demonstration of a complete diverticulum.

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

testicanl 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 diver-
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 intraluminal 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 edematous 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
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 (→) by double contrast technique.

Fig. 8. X-ray, diverticulum (●) with an infected head and stenosis of the neck.
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 Malmö-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 inflamed 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 considerably, it is a typical reaction of the surrounding area during advanced diverticular 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 a flexible 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
indicated a stenosis caused by diverticular disease (Fig. 11). The operative data confirmed this. Colonoscopy 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 diverticular 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 decades, 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 appearance in the ascending, descending and sigmoid colon was found in 6%. Further localizations or combinations were very rare.
Complications

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.

<table>
<thead>
<tr>
<th>Primary not acute</th>
<th>Acutely dangerous to life</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tumors</td>
<td>Ileus</td>
</tr>
<tr>
<td>Stenosis</td>
<td>Free perforation</td>
</tr>
<tr>
<td>Subileus</td>
<td>Fecal peritonitis</td>
</tr>
<tr>
<td>Covered perforation</td>
<td></td>
</tr>
<tr>
<td>local peritonitis</td>
<td></td>
</tr>
<tr>
<td>&quot;frozen pelvis&quot;</td>
<td></td>
</tr>
<tr>
<td>(DEÜCHER et al. 1974)</td>
<td></td>
</tr>
<tr>
<td>Abscesses</td>
<td>Colonic wall phlegmon</td>
</tr>
<tr>
<td>Fistulas</td>
<td></td>
</tr>
<tr>
<td>Occult bleeding</td>
<td>Massive bleeding</td>
</tr>
<tr>
<td>&quot;elective operation&quot;</td>
<td></td>
</tr>
<tr>
<td>or conservative treatment</td>
<td></td>
</tr>
<tr>
<td>&quot;emergency operation&quot;</td>
<td></td>
</tr>
<tr>
<td>or conservative treatment, with</td>
<td></td>
</tr>
<tr>
<td>later &quot;elective operation&quot;</td>
<td></td>
</tr>
</tbody>
</table>

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 defined 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

Criteria for classification of the clinical stages.

<table>
<thead>
<tr>
<th>Clinical Stages</th>
<th>Criteria for clinical classification</th>
<th>cases</th>
<th>Symptoms/Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>radiologic evidence</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td></td>
<td>without clinical symptoms</td>
<td></td>
<td>digestive difficulties 57 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>colic and tenesmus 54 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>occult bleeding 29 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&quot;left appendicitis&quot; 24 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>palpable tumors 13 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>stenosis 10 %</td>
</tr>
<tr>
<td>II</td>
<td>with or without radiologic evidence</td>
<td>122</td>
<td></td>
</tr>
<tr>
<td></td>
<td>with clinical symptoms</td>
<td></td>
<td>digestive difficulties 57 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>colic and tenesmus 54 %</td>
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<tr>
<td></td>
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<td></td>
<td>occult bleeding 29 %</td>
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<tr>
<td></td>
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<td></td>
<td>&quot;left appendicitis&quot; 24 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>palpable tumors 13 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>stenosis 10 %</td>
</tr>
<tr>
<td>III</td>
<td>complications which are</td>
<td>82</td>
<td></td>
</tr>
<tr>
<td></td>
<td>acutely dangerous to life</td>
<td></td>
<td>free perforation 82 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ileus 9 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>massive bleeding 9 %</td>
</tr>
</tbody>
</table>

Table 6. Degree of severity according to age.
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 three-stage-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-abrel 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 procedure is the same in the resection described by Guleke with the exception that the sigmoid loop is extraperitoneally closed.

These operative procedures are advantageous since the relatively brief operating time increases the chances of survival even in severely 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.


<table>
<thead>
<tr>
<th>Classification according to severity</th>
<th>Conservative or without therapy</th>
<th>Operative</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>69 patients</td>
<td>69</td>
<td>-</td>
</tr>
<tr>
<td>II</td>
<td></td>
<td></td>
</tr>
<tr>
<td>122 patients</td>
<td>62</td>
<td>60</td>
</tr>
<tr>
<td>Method of operation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>one stage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colostomy primary resection</td>
<td>2</td>
<td>52</td>
</tr>
<tr>
<td>two stage</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>three stage</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22 patients</td>
<td>1</td>
<td>21</td>
</tr>
<tr>
<td>Colostomy w. drainage</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
</tr>
</tbody>
</table>
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 one-stage-resection being the predominantly 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 risk factors, we have seldom performed one-stage-resection during the last 5 years. We have found the two-stage-procedure to be more favorable, preferring the incontinuity resection of the Hartmann type. The lower mortality rate as well as shorter hospitalization —55 days for the two-stage as opposed to 83 days (at that time) for the three-stage-method— speaks 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 today. 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 majority 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
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 parasympatholytics 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 colonic fistulas deserves special attention (Colcock
Fig. 13. Guide to antimesenteric incision of longitudinal myotomy according to REILLY from AKOVBIANTZ (1974).

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

<table>
<thead>
<tr>
<th>Localization</th>
<th>Frequency (n = 278)</th>
<th>Operative procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>without abscess formation</td>
</tr>
<tr>
<td>colo-vesical</td>
<td>145 - 51.2 %</td>
<td>one-stage resection</td>
</tr>
<tr>
<td>colo-cutaneous</td>
<td>90 - 32.4 %</td>
<td>one-stage resection</td>
</tr>
<tr>
<td>colo-entric a. colic b.</td>
<td>37 - 13.3 %</td>
<td>two-stage resection</td>
</tr>
<tr>
<td>colo-urethral</td>
<td>1 - 0.4 %</td>
<td>two-stage resection</td>
</tr>
<tr>
<td>colo-uterine</td>
<td>4 - 1.4 %</td>
<td>two-stage resection</td>
</tr>
<tr>
<td>colo-vaginal</td>
<td>1 - 0.4 %</td>
<td>two-stage resection</td>
</tr>
</tbody>
</table>

Collective statistics: COLOOCK A. STAHNANN 1972 (n = 1555); BEUCHER et al. 1974 (n = 152), KRAFT, KINZ A. PREXL 1976 (n = 52); own cases (n = 81)

their frequency and therapeutic treatment. The various possibilities are listed according to decreasing frequency: colo-vesical, colo-cutaneous, colo-entric and colo-colic, colo-urethral, 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 (●).

Fig. 16-17. Vascular injection with Barotrast and double contrast technique. Fig. 16. Comparative demonstration of the mesenteric vessels. a) No inflammatory changes, the vessels are still elastic and soft. b) Vessels altered by infection in diverticular disease.

Fig. 16. Comparative demonstration of the mesenteric vessels. a) No inflammatory changes, the vessels are still elastic and soft. b) Vessels altered by infection in diverticular disease.

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 al-
A comparative demonstration of an intestine segment.

a) Diverticular disease.

b) Crohn's disease.

ted 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—beginning in the colonic wall and encroaching secondarily on the mesentery. Differentiation from other inflammatory intestinal diseases 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 occurrence of diverticular complications 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 gastroenterologists should begin
at the onset of the disease in order that resection can be performed as early as necessary (Hol-
lender 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 long-
term 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. Pathophysicsy considerably influences the
prognosis. Predominant decisive factors in the diagnosis are radiological demonstration
(e.g. double-contrast study of the colon) and clinical 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 today 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 determining 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.

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