SEGMENTAL ILEITIS*

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SEGMENTAL (OR REGIONAL) ENTERITIS is a chronic, nonspecific granulomatous disease occurring usually in young adults and most commonly found in the terminal ileum, although the jejunum or colon may be involved. "Skip" areas are frequent and are thought to be the cause of such recurrences as develop following bowel resection.

There is voluminous literature on this subject. The purpose of this paper is to review briefly some of the more significant contributions to our knowledge of the condition and to present our own experience in its treatment at the George F. Geisinger Memorial Hospital.

HISTORICAL

Although Crohn[1] is rightfully given credit for first describing regional enteritis as a clinical entity, a condition resembling regional enteritis was described as early as 1806.[2] Sporadic case reports later followed, particularly in the German[3, 4] and British literature.[5] Chronic granulomatous lesions involving the intestines were referred to in some detail by several American authors prior to Crohn's paper. Moschowitz and Wilensky[6] suggested that many cases of nonspecific granulomatous processes in the bowel are incorrectly diagnosed as tuberculosis. Mock[7] confirmed the nonspecificity of many intestinal granulomas. In 1932 Crohn, Ginzburg and Oppenheimer[1] described in detail the entity of regional ileitis as we now know it, both as to its clinical and pathologic manifestations.

Recognition that more widespread involvement of the bowel occurs led to the proposal of more inclusive terms, such as cicatrizing enteritis,[8, 9] segmental enteritis.[10] and others.[11-13]

ETIOLOGY AND PATHOGENESIS

Despite extensive researches the etiology of regional enteritis remains unknown. Various bacteria have from time to time been accused. Attempts to implicate the tubercle bacillus have been made. The similarity of the histologic appearance of chronic regional enteritis and that of hyperplastic intestinal tuberculosis is close. However, cultures from removed specimens as well as multiple sections properly stained to demonstrate acid-fast bacilli have met with failure. Indeed, the inability to demonstrate tubercle bacilli has been an important factor in the establishment of segmental enteritis as a separate entity.

That *B. dysenteriae* might be the causative agent was proposed by Felsen.[14] This investigator was able to culture *B. dysenteriae* (Sonne and Flexner types) in certain cases in which the lesion was remarkably similar to regional enteritis. Corroboration has not been reported elsewhere, it being felt by most writers that this organism cannot be considered an important factor. Other organisms have been suggested: the colon bacillus,[15] *Myobacterium Johnei,*[16] *Aerobacter aerogenes,*[17] and anaerobic streptococci.[18] Bacterial toxins[19] and viruses[20] have been erroneously implicated. Protozoa such as *E. histolytica,*[21] and metazoa such as *Giardia lamblia,*[22] trichocephalus, oxyuris vermicularis and parasites

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have all been described at one time or another as causing enteritis. Several cases have been described resulting from trauma.[23, 24]

The role of heredity has been investigated by Crohn,[25] who reported the case of familiar incidence occurring in a brother and sister. The earlier reports suggested a possible racial factor in the etiology, a predominating number of cases in the Jewish race being found. Later papers discount this.

Since the etiology of the condition is unknown, it necessarily follows that the pathogenesis is obscure. Indeed, regional enteritis is not recognized as a pathologic entity by numerous writers.[26-28] However, authorities, Warren and Sommers[29] and Owens[30] regard it as such. Hadfield[31] earlier reached the same conclusion.

Many authors feel that the histologic changes produced in regional enteritis are due to chronic lymphedema. Whether the usual lymph blockage is due to mucosal infection which spread to the intestine and causes a lymphedema of the mesentery or whether the initial lesion is a lymphatic stasis which produces secondary changes in the bowel itself is a moot point.[32]

Reichert and Mathes[24] have injected sclerosing solutions into the intestinal lymphatics of dogs producing changes resembling cicatrizing enteritis both grossly and microscopically. If *E. coli* were also injected intravenously these changes were increased in degree. These authors concluded

FIG. 1

FIG. 2

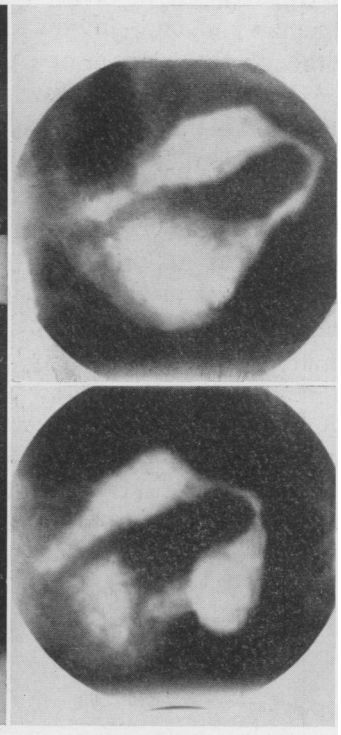


FIG. 3

FIG. 1.—The "string-like" area marked by arrows.
FIGS. 2 and 3.—Spot films illustrating the marked narrowing of the bowel lumen producing the "string sign."

that both low grade chronic infection and chronic lymphedema are responsible for the characteristic changes seen in the bowel.

Some authors have studied the role of impairment of the blood supply in the pathogenesis of regional enteritis. Ginzburg

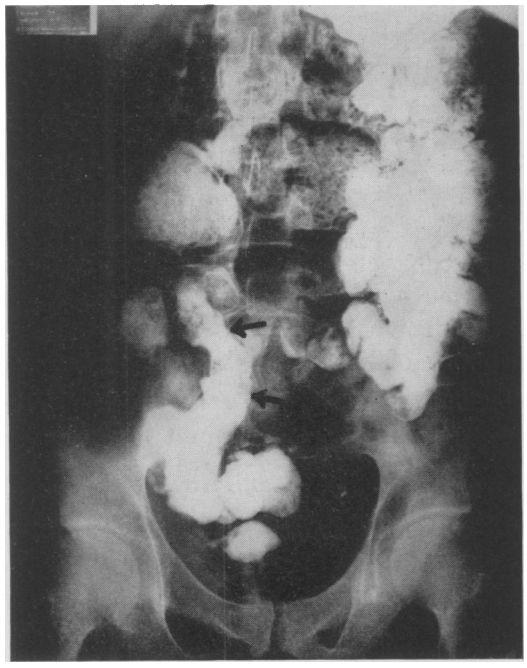


FIG. 4.—Alteration of the mucosal pattern is seen in the area indicated by the arrows. A collection of barium is seen in the urinary bladder: the fistulous tract is not evident.

and Oppenheimer[33] have described cases in which granulomatous lesions in the intestine occurred after the blood supply to the bowel has been experimentally impaired. The possibility of an anomaly of the ileocecal artery with torsion as a cause has been explored by Bockus and Lee.[34] Proliferation of the interstitial elements of the bowel, thereby reducing the blood supply and thus causing necrosis with ulceration and cicatrization, has been suggested.[35, 36] The possibility that superficial ulcers act as portal of entry for organisms of low virulence which multiply in the submucosa and muscularis has been mentioned.[37]

Pratt and Ferguson[38] feel that the original site of the disease is usually the appendix and that spread of organisms to the local lymphatics occurs from this focus. This idea is questioned by Mixter.[18]

Recently a new theory of the pathogenesis has been promulgated by Chess[39] and co-workers. They have produced changes similar to regional enteritis in the intestines of dogs by feeding finely ground sand and silica, ascribing the effects produced as due to irritation from the ingested material, resulting in a proliferative cellular response followed by lymphatic obstruction.

PATHOLOGY

The pathology of regional enteritis can best be considered under three phases—the acute, subacute, and chronic.

In the acute state the involved segment or segments of bowel are boggy, edematous, and reddish purple in color. Usually there is a sharp distal demarcation at the ileocecal valve. The mesentery of the affected bowel is thickened, edematous and contains numerous hyperplastic nodes. There is frequently an increased amount of serous fluid in the peritoneal cavity. Occasionally small ulcerations along the mesenteric border of the bowel are seen. On cut section it is the submucosal layer showing the greatest edema.

Microscopic sections in the acute phase are rarely obtainable, but have been described[1, 40, 41] as consisting of an infiltration of the subserosa by inflammatory cells—lymphocytes, leukocytes (including many eosinophiles), large monouclear cells, and plasma cells. There is marked edematous thickening of the submucosa. In this layer there is considerable dilatation of the blood vessels. The subserosa and serosa are the site of edema, interstitial hemorrhage and congestion. The mucosa may be intact or may contain shallow ulcers which usually are covered with a fibrinous exudate. The

mesenteric nodes are hypertrophied and edematous.

In the subacute phase the bowel and mesenteric lymph node changes are a combination of those found in the acute and chronic forms. Grossly the bowel shows some of the congestion and hyperemia of the acute phase as well as marked edema and thickening seen in the chronic state. Extensive ulceration extending into the submucosa may make the bowel friable.[42, 43] Microscopically there is a scattering of the acute and chronic inflammatory cells. Eosinophilic leukocytes are frequently present, but are not characteristic of this phase. Proliferation of the endothelial cells of the lymphatics becomes more pronounced and the giant cell granulomas, which characterize the chronic phase, appear. Similar changes occur in the mesentery and mesenteric lymph nodes.

The changes seen in the chronic phase are particularly marked. The involved segment of bowel is thickened, soggy, leathery, and stenotic. It has been described as comparable to a garden hose.[44-46] There is serosal thickening with extension of mesenteric fat around the involved area. The mesentery corresponding to the involved segment is thickened and contains numerous large lymph nodes. Dense fibrous adhesions between the diseased segment of bowel and adjacent structures are frequent. Usually the bowel, proximal to the involved segment, is dilated and hypertrophied. Fistula formation occurs in this stage with characteristic frequency. The mucosa is swollen and exhibits ulcerations on the mesenteric side. Whether this represents the vestiges of the ulcers which occur in the acute phase or are secondary to lymphatic obstruction and fibrosis is not definitely known.

Microscopically the chronic phase of regional enteritis is characterized by thickening of the bowel wall, the greatest degree occurring in the submucosa. There is hypertrophy of the muscular coats late in the course of the disease. The submucous and myenteric plexuses of Auerbach and Meissner are prominent. Within the submucosa there is a hyperplasia of lymphatic tissue and obstructive lymphedema. The germinal centers are replaced by reticulo-endothelial cell proliferation, and within these centers giant cells of the Langhans type appear. These have been called "giant cell systems" by Hadfield.[31]

The same type of "systems" may be found in the mesenteric nodes. Necrosis within these granulomatous lesions is rare. No caseation necrosis occurs. The giant cells may contain refractile foreign bodies and as the mucosal ulceration progresses secondary inflammation occurs, the giant cells becoming obscured by a secondary lymphadenitis characterized by diffuse inflammation and fibrosis.

CLINICAL MANIFESTATIONS

The clinical manifestations of regional enteritis vary according to the phases of the disease. In the acute stage the signs and symptoms are usually suggestive of acute appendicitis. The most constant symptom is abdominal pain, colicky in nature and frequently localized in the right lower quadrant. Pain is often accompanied by nausea, vomiting, and diarrhea. Crohn has warned that in cases simulating appendicitis and accompanied by diarrhea, the possibility of acute regional enteritis should be considered. Leukocytosis, fever, increased pulse rate, tenderness, and rigidity also occur. A palpable mass may be present in the right lower abdomen. It is not surprising that many patients with acute regional enteritis are mistakenly operated upon for acute appendicitis.

The subacute phase is sometimes referred to as the stage of "irritation."[47] Here the clinical features resemble those of an enterocolitis or ulcerative colitis. There is weight loss, diarrhea accompanied by se-

vere abdominal cramps, anemia, nausea, and vomiting. A palpable mass slowly forms in the right lower quadrant. There is missions become less frequent and of shorter duration. Symptoms of intestinal obstruction may now appear.

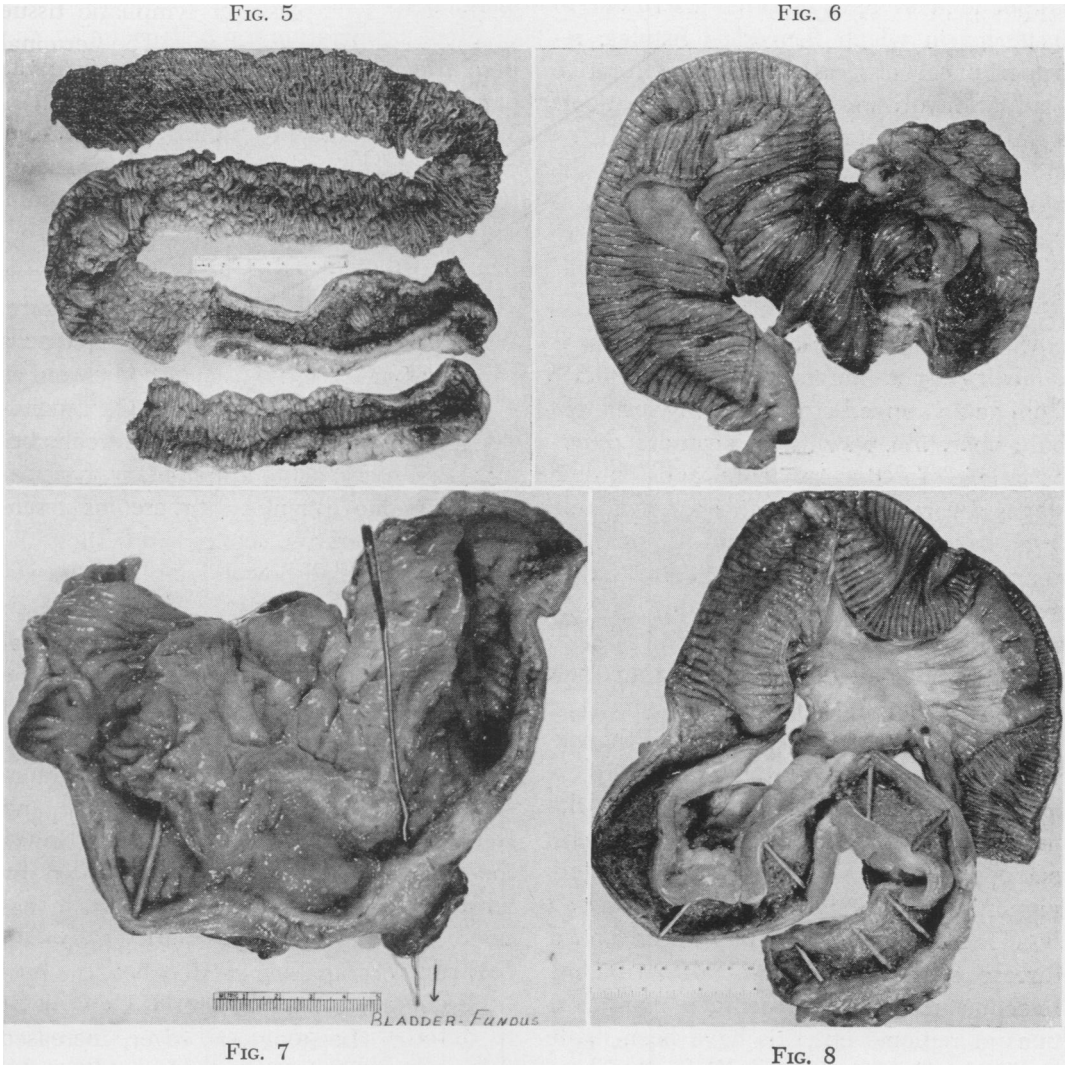


FIG. 5 FIG. 6

FIG. 7 FIG. 8

FIGS. 5 and 6.—Surgically resected specimens showing marked thickening of the bowel wall with resultant narrowing of lumen. The mucosa is intact.
FIG. 7.—Resected ileum showing fistulous tract opening into urinary bladder.
FIG. 8.—Resected specimen of lower ileum showing submucosal thickening and edematous mesentery.

moderate elevation of temperature. Night sweats may occur. Occasional melena may be present due to ulceration. Early in the disease remissions and exacerbations frequently occur. As the disease progresses re-

In the chronic phase there is, usually, a history of diarrhea and crampy abdominal soreness of several months or years duration. Severe colicky abdominal pain accompanied by vomiting and visible peristalsis is

the rule. There is usually weight loss. In addition evidences of obstruction and fistulas may now appear. Anal abscesses and fistula occur frequently.[48] Ginzburg and Oppenheimer[49] have reported a series of 13 instances in which ileovesical fistulas, retroperitoneal abscess, and nonperforative bladder involvement occurred in patients with regional enteritis. In their series the chief complaints were not related to the gastro-intestinal tract, but rather those of cystitis, pneumaturia, and fecaluria.

DIAGNOSIS

Segmental enteritis in the acute phase is difficult, if not impossible, to differentiate from acute appendicitis. In many instances only operation permits an accurate differentiation. In the subacute and chronic stages several diagnostic aids are available in addition to the history and physical examination. Roentgen examination of the intestinal tract is important. Kantor[50] in 1934 described the "string sign"—"a thin slightly irregular linear shadow suggesting a cotton string in appearance and extending more or less continuously from the region of the visualized loop of ileum through the entire extent of the filling defect and ending at the ileocecal valve." This sign is not always pathognomonic, but when present is highly suggestive. We now know that the "string sign" is a finding of relatively far-advanced disease with marked luminal narrowing. Recent studies of the roentgen manifestations of regional enteritis have been made by Brown,[51] Janus,[52] and Kiefer.[53] These authors suggest that deformity of the bowel contours, narrowing of the lumen, loss of mucosal pattern, rigidity and fixation of the involved area, displacement of the adjoining segments of bowel by pressure from pseudo-tumors, demonstration of fistulous tracts, hypermotility of the intestinal contents, and dilatation of the bowel proximal to the site of construction are all important roentgen evidences of the disease.

Proctoscopic and sigmoidoscopic examination should be carried out. The usual proctoscopic and sigmoidal manifestations of regional enteritis have been previously mentioned.

Stool examination for *E. Histolytica,* tubercle bacilli, dysentery organisms, ova, parasites, pus, and blood should be made. The Mantoux and Frei skin tests should be carried out to rule out tuberculosis and lymphopathia venereum. A chest roentgen ray examination is helpful in ruling out pulmonary tuberculosis. Cultures and smears of all draining fistulas, for tuberculosis, blastomycosis, and actinomycosis should be studied.

DIFFERENTIAL DIAGNOSIS

Regional enteritis must be differentiated from dysentery, acute appendicitis, typhoid fever, amebic dysentery, tuberculosis, sarcoidosis, ulcerative colitis, diverticulitis, neoplasm of small bowel, lymphopathia venereum, actinomycosis, and talc granuloma. Stool cultures, serum agglutinations, chest roentgen rays, proctoscopic and sigmoidoscopic examinations, and skin tests are helpful in ruling out other conditions. It is at times difficult to eliminate intestinal sarcoidosis; however, absence of generalized sarcoidosis is presumptive evidence of the absence of this condition involving the bowel. It is felt that regional enteritis and sarcoidosis are two distinct diseases (Warren).

Right-sided ulcerative colitis is difficult to differentiate preoperatively. Ulcerative colitis, however, never shows grossly the marked fibroblastic process seen in regional enteritis. Microscopically it is differentiated by its mucosal changes without involvement of the submucosa and muscular coats. Talc granuloma may produce a gross appearance indistinguishable from that of regional enteritis. Microscopically the presence of double refractile crystals surrounded by epithelioid cells should suggest this condition as a possibility.

TREATMENT

Treatment may best be discussed in terms of the phase in which the disease appears. In acute regional enteritis, if recognized preoperatively, the treatment is non-surgical, consisting of a bland, high protein, high carbohydrate, low residue diet, parenteral vitamins, appropriate anti-anemia therapy, bed rest, penicillin, and streptomycin, and removal of foci of infection.[54-57]

FIG. 9

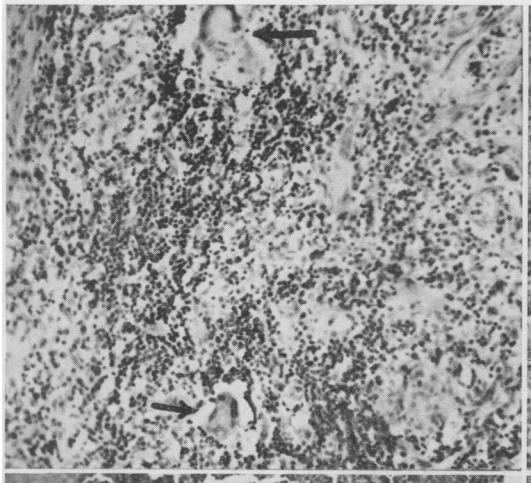
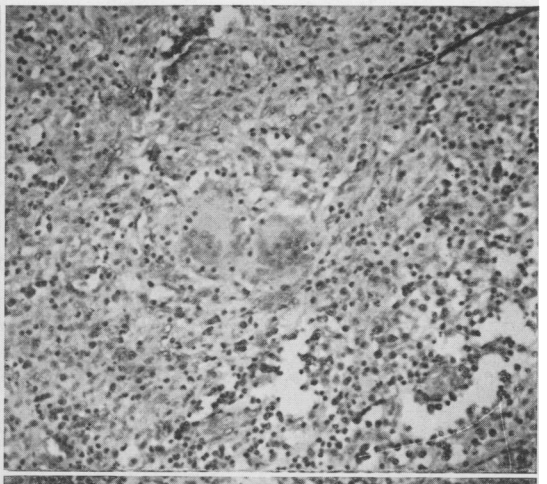


FIG. 10



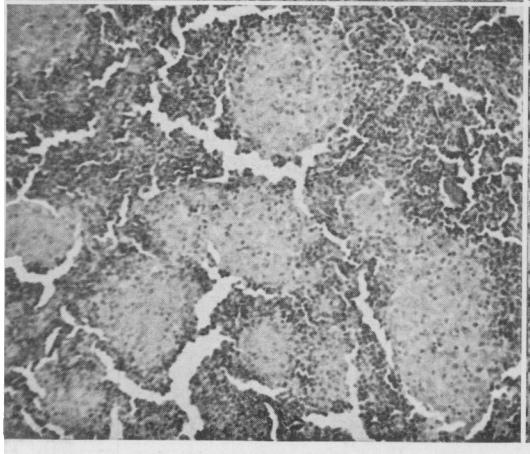


FIG. 11

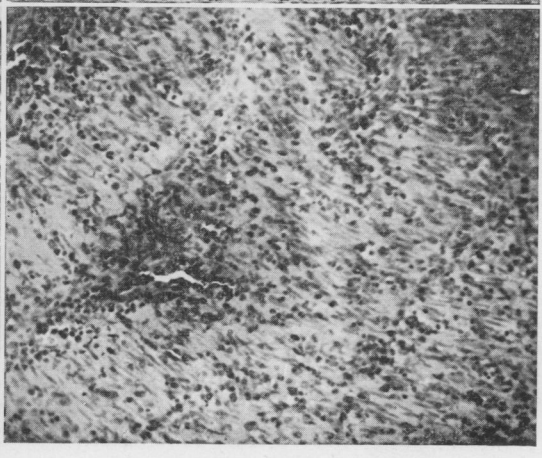


FIG. 12

FIG. 9.—Microscopic section from terminal ileum showing multinucleated giant cells (arrows). Elsewhere the submucosa is heavily infiltrated with lymphocytes, eosinophiles, and polymorphonuclear leukocytes. Considerable edematous connective tissue may be noted.

FIG. 10.—This shows marked inflammatory submucosal reaction in the terminal ileum. Two multinucleated giant cells occupy the center of the field.

FIG. 11.—A section of mesenteric lymph node. Numerous pale staining circumscribed areas are present. These appear to be undergoing fibrinoid degeneration. There is no evidence of coalescence or caseation.

FIG. 12.—Section of terminal ileum showing involvement of the muscularis. Smooth muscle fibers and their nuclei predominate. To the left of the center is a collection of lymphocytes, polymorphonuclear leukocytes and eosinophiles. Elsewhere a diffuse scattering of acute and chronic inflammatory cells is to be noted.

If the abdomen is mistakenly opened for acute appendicitis the question of whether to perform an appendectomy or not is disputed. Garlock,[58] Cave,[59] and Crohn feel that to perform an appendectomy at this time is to invite disaster in the form of postoperative fistulas. Fallis,[60] Ferguson and Pratt,[38] and Eckel and Ogilvie[61] have removed appendices in acute regional enteritis without untoward results. Pratt and Ferguson suggest that a chronically infected appendix may be the source of infection producing regional enteritis.

Treatment of the subacute phase without complications is also thought by many to be medical (Pratt, Ferguson, Cutler, Kiefer). If conservative management fails, either a by-passing operation or resection may be indicated.

Treatment of the chronic stages of regional enteritis has become a matter of great controversy. The Mt. Sinai Hospital group—Garlock[63] Colp,[62] and Garlock and Crohn[63] prefer short circuiting procedures, ileotransverse colostomy with complete division of the ileum well proximal to the involved segment. They report a low mortality rate and low incidence of recurrence. Equally experienced surgeons — Dixon[64] Marshall,[65] Pugh,[66] Pratt and Ferguson[38] and many others believe that wide resection of the bowel with as much of the involved mesentery as possible is the surest way to eradicate the disease. Nevertheless recurrence rates seem to be high, being 73 per cent, in a series followed by Hawthorne and Frobese.[41] Bockus[54] states that we may not expect permanently good results in more than 50 per cent of cases treated by resection.

Most authors agree that the cause of the high recurrence rate after either resection or short circuiting, is failure of the surgeon to resect or bypass all the "skip areas." A study by Melton[67] at the Mayo Clinic indicates that one-third to a half of all recurrences are the result of disease remaining in the bowel or mesentery at the time of the operation.

Recently reports from the Mayo Clnic[68] and Lahey Clinic[69] following the use of roentgen therapy have been published. The criteria for the use of radiation therapy at Rochester are: (1) The disease must be severe; and (2) surgical treatment must be not feasible or advisable. The results in 50 cases show 20 "favorable results." The follow-up study on these patients will probably decide whether this therapy is actually curative or is but palliative.

CASES TREATED AT GEISINGER MEMORIAL HOSPITAL

In approximately 50,000 surgical admissions to the George F. Geisinger Memorial Hospital and Clinic over a 30-year period, 17 proved cases of regional enteritis have been encountered. Nine patients were females and eight were males. (None of the patients were of the Jewish race.) The average age was 38 years—the youngest patient being nine, the oldest 69. Tables I and II summarize the chief symptom presented by the patients in this series.

In tabulating symptoms we find that 94 per cent of our patients complained of abdominal pain. It varied in intensity from dull to severe, occurring, generally, throughout the abdomen and, usually, colicky in nature. Diarrhea was not a common symptom, occurring with but 12 per cent of the patients. Weight loss was a common finding, occurring with 65 per cent of the patients; the average weight loss being 16 pounds. Weakness occurred with 35 per cent of our patients, while 30 per cent complained of abdominal distention. Forty-one per cent had constipation. One patient had a suprapubic swelling and complained of pneumaturia and fecaluria, the result of an ileo-cystic fistula. Anorexia was present in 41 per cent of the cases. Only one patient had a history of fistulae-in-ano—the patient being the youngest patient in the series, and

having been treated by a skilled proctologist for the fistula without the ileac lesion being discovered. This patient had also a draining sinus from a postoperative appendectomy wound. The average duration of symptoms of the patients in the series was 15 months, although the duration varied from three days to 13 years.

One other finding of interest in this group was that three related the onset of symptoms immediately to dental extractions. One patient stated that her bowel complaint came on after an acute pansinusitis, suggesting bacterial allergy as an etiologic factor.

A mass was palpable in nine cases or 53

TABLE I.—*Symptoms of Patients.*

Patient's Initials	Abdominal Pain	Diarrhea	Weight Loss in Pounds	Weakness	Abdominal Distention	Constipation	Suprapubic Swelling	Urinary Symptoms	Anorexia	Fistulae-in-ano	External Fistulas	Duration of Symptoms	Character of Abdominal Pain
R.K.	+	0	0	0	0	0	0	0	0	0	0	3 days	Pressure sensation in R.L.Q.
W.J.B.	0	0	?	0	0	0	0	0	+	+	+	18 mo.	0
M.C.	±	0	19	+	+	+	0	0	+	0	0	3 wks.	Moderate generalized cramplike pain
E.K.	+	0	0	+	0	+	0	0	0	0	0	6 mo.	Generalized crampy pain of variable severity
A.P.	+	0	38	0	0	+	0	0	+	0	0	3 mo.	Generalized severe crampy pain
C.F.	+	0	45	+	0	+	+	+	+	0	+	13 yrs.	Moderate generalized crampy pain
M.P.	+	+	25	0	+	+	0	0	+	0	0	3 mo.	Severe pain in R. L. Q.
L.E.M.	+	0	35	+	0	0	0	0	+	0	0	6 mo.	Dull epigastric pain
J.L.	+	0	27	0	+	+	0	0	0	0	0	3 mo.	Moderate generalized crampy pain
G.R.	+	0	0	0	0	0	0	0	0	0	0	2 mo.	Moderate generalized crampy pain
F.L.	+	0	0	0	0	0	0	0	0	0	0	4 days	Gnawing Q pain in R. L. Q.
A.B.	+	0	9	0	0	0	0	0	0	0	0	3 yrs.	Moderate crampy pain in abdomen
A.F.	+	0	26	0	0	0	0	0	0	0	0	4 days	Same
R.P.	+	0	15	0	+	0	0	0	0	0	0	3 mo.	Severe pain in R. L. Q.
B.R.	+	+	30	+	+	0	0	0	+	0	0	2 yrs.	Generalized crampy pain
O.S.	+	0	0	+	0	+	0	0	0	0	0	1 yr.	Moderate generalized pain
J.R.	+	0	8	0	0	0	0	0	0	0	0	3 days	Moderate peri-umbilical pain
Total	16	2	11	6	5	7	1	1	7	1	2		

TABLE II.—*Positive Signs Found on Examination of Patients.*

Patient's Initials	Abdominal Mass	Tenderness	Muscle Spasm	Hyperactive Peristalsis	Fever	RBC in Millions	WBC in Thousands	Hemoglobin in Per Cent of Normal
R.K.	+	+	0	0	0	4.6	15	91%
W.J.B.	+	0	0	0	+	3.7	9.8	65%
M.C.	0	+	0	+	0	4.4	4.5	84%
E.K.	+	+	0	0	0	none recorded		....
A.P.	+	+	+	0	0	6.3	25.5	110%
C.F.	+	+	0	0	+	3.1	8.2	65%
M.P.	+	+	+	0	0	5.1	9	91%
L.E.M.	0	0	0	0	0	5.5	6.1	78%
J.L.	+	+	0	+	0	4.7	10.6	98%
G.R.	0	+	0	0	+	...	14.9	....
F.L.	0	+	0	0	0	...	18	....
A.B.	+	+	0	0	0	3.9	6.1	77%
H.F.	0	+	+	0	+	4.	28.6	78%
R.P.	+	+	0	0	0	4.5	5.3	78%
B.R.	?	+	0	0	0	3.8	3.6	78%
O.S.	0	+	+	0	+	3.1	23.1	62%
J.R.	0	+	0	0	0	4.8	8.25	76%

per cent of the total (Table II). Abdominal tenderness was present in 94 per cent. Muscle spasm and muscle guarding was found in 23.5 per cent of the cases. Hyperperistalsis was rarely found. Moderate anemia was present in about 50 per cent of the cases; the average erythrocyte count for all the cases, however, was 4.4. million, average hemoglobin 81 per cent. In this series a leukocyte count averaging 10,000 was present in 50 per cent of the cases.

TABLE III.—*Roentgen Ray Findings.*

Patient's Initials	Altered Mucosal Pattern	String Sign	Partial Obstruction	Spasticity of Cecum	Fistulous Tract
W.J.B.	0	0	0	+	0
M.C.	+	0	+	0	0
A.P.	0	0	+	0	0
C.F.	0	0	+	0	+
M.P.	+	0	+	0	0
L.E.M.	+	0	0	0	0
J.L.	0	0	0	+	0
A.B.	0	0	+	0	0
R.P.	0	0	+	0	0
B.R.	+	+	0	0	0
O.S.	0	0	+	0	0

Roentgen ray findings were significant in the majority of the patients, the remainder being diagnosed at laparotomy. One patient was found to have ileitis during a routine abdominal exploration following a cholecystectomy. It is worthy of note that the most frequent roentgen finding was that of partial intestinal obstruction. The "string sign" as described by Kantor was found in only one instance. Some alteration of the mucosal pattern was noted in four patients. Spasticity of the cecum was seen in two of 11 cases and a fistulous tract was demonstrated between the ileum and urinary bladder in one (Table III).

The accompanying photographs (Figs. 1 and 2) show the salient roentgen ray and pathologic changes (both gross and microscopic) encountered in our patients.

TREATMENT

In this group of patients only three had acute regional enteritis, all being explored. In two appendectomy was carried out; with the other only an exploration was performed.

Eleven patients underwent primary extensive resection of the involved area of bowel with its mesentery and end-to-end anastomosis. Two patients had a two-stage resection consisting of a primary ileostomy followed by resection at a later date.

One patient was not explored due to a recent myocardial infarction.

TABLE IV.—*Results of Primary Bowel Resection in Ten Surviving Patients with Subacute and Chronic Regional Enteritis.*

Patient's Initials	Length of Follow-up	Weight Gained in Pounds	Diarrhea	Constipation	Able to Work	Second Operation	Excellent Result	Good Result	Poor Result
R.K.	11 yrs.	0	0	0	+	0	+	..	..
W.J.B.	6 mo.	10	0	0	+	0	+	..	..
E.K.	10 yrs.	0	0	0	+	0	+	..	..
C.F.	5 mo.	25	0	0	+	+	+	..	..
M.P.	6 yrs.	0	0	0	+	0	+	..	..
L.E.M.	12 yrs.	120	frequent +	0	+	0	..	+	..
F.L.	12 yrs.	0	0	0	+	0	+	..	..
J.R.	17 mo.	0	occasional +	0	+	0	..	+	..
R.P.	11 yrs.	40	+	0	+	0	..	+	..
B.R.	5 mo.	20	0	0	+	+	+	..	..

RESULTS OF TREATMENT

Of the two patients with acute regional enteritis treated by appendectomy, one died five days postoperatively as a result of overwhelming peritonitis. The other recovered from the operation but developed acute lymphatic leukemia and expired three and one-half months postoperatively, with no recurrence of bowel symptoms. The one patient who had an exploratory laparotomy and who was closed without appendectomy is living and well five months postoperatively without bowel symptoms. She has gained five pounds in weight.

Of the group of 11 patients with subacute and chronic regional enteritis who underwent primary bowel resections, ten are alive. There was one postoperative death due to peritonitis. Table IV furnishes a summary of the results in these ten cases.

It will be noted that the follow-ups on these patients have covered long periods. Fifty per cent of the patients have gained weight. Only three are bothered by diarrhea. All are able to work. We have regarded those who are symptom free as having excellent results. Those with some slight remaining gastro-intestinal symptoms have been listed as having good results. None had poor results. Seventy per cent of the patients who had resections have obtained excellent results. Thirty per cent have obtained good results.

Two patients required two resections. One had a recurrence one month postoperatively, the other in two months. In each case the recurrence was at the site of anastomosis. Both have been symptom free since the second resection. The one operative death makes our mortality 9.1 per cent.

There were two two-stage resections. One of these has been followed 18 years. The patient is asymptomatic and has gained 100 pounds. The other has been followed 11 years and has gained 40 pounds, and is symptom free. If we add these two cases to the ones treated by one-stage resection we find that excellent results were obtained in 75 per cent of our resections with 25 per cent good results. Our overall mortality for resection was 7.8 per cent.

CONCLUSION

1. A review of the literature on regional enteritis has been presented.

2. Regional enteritis is a rarely encountered entity at the George F. Geisinger Memorial Hospital.

3. In our experience widespread resection is the treatment of choice in subacute and chronic regional enteritis.

4. In the event of recurrence, further resection is necessary for cure.

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DISCUSSION.—DR. FRANK H. LAHEY, Boston: Dr. Foss asked Dr. Marshall to discuss this paper but Dr. Marshall was unable to be present and, since all of us have been interested in regional ileitis and have had a good many of these cases, he asked me if I would not discuss it for him.

Dr. Everett D. Kiefer in the gastro-enterologic division of the Clinic has divided these cases into three good groups, I believe: the enteric group which is the acute stage, the obstructive group which is the cicatrizing stage and the fistulous group which is the late stage. We would like to get these cases during the first two stages rather than in the late stage because it is with the late stage, so often associated with skip area involvement, that we have had our greatest difficulty. We have felt that radical removal of the lesion with a good segment of colon when it is in the terminal ileum, and a good segment of the ileum on the other side or a good segment of normal jejunum on either side when it is in the jejunum, has been the best method of treatment. We have never been able to get up any enthusiasm for the side-tracking operation because, if the same thing can be accomplished and get the lesion out, together with many of the mesenteric nodes, it seems to us it offers a lower percentage of recurrence rate, although the recurrence rate is bad with all types of surgical treatment. The mortality with this radical type of procedure has not been high. There has been a total of 182 cases operated upon and but two postoperative deaths, none in 1948, 1949 or 1950, giving an operative mortality rate of 1.1 per cent.

The thing that distresses us with regional ileitis is the fact that there is nothing really that we can do for the late stage cases, and also the fact that our recurrence rate, even with radical removal, has been 34 per cent. The reported recurrence rate of 15 per cent is, we believe, too low. Anyone who deals with this disease in any considerable number of cases has been made very aware that it is probably nearer 30 than it is 15 per cent, and whichever it is, it is certainly too high for anyone's satisfaction.