ULCERATIVE COLITIS AND CROHN'S DISEASE

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Introduction

IT IS ONLY in recent years, if indeed at all, that the need for surgical treatment in *ulcerative colitis* has gained general acceptance: and now that surgeons are more experienced they are finding that certain cases fail to conform to the general pattern both in their pathology and in their response to treatment. At the same time, there is an increasing awareness that not all the cases submitted to operation for the relief of *regional ileitis* have done as well as was expected. There is anxiety about the ileal involvement often found in ulcerative colitis and there is uncertainty about the colonic involvement found in regional ileitis. Until this confusion is resolved progress cannot be made.

If the aetiology of Crohn's disease and ulcerative colitis were known, a rational classification would be simple. Since we have no certain knowledge about causes, we can only hope to group the cases on a basis of descriptive pathology. It is surely here that we must make a fresh start and revise our definitions which have become so nebulous that the nomenclature conveys no precise meaning.

I suggest that we should for practical purposes and for convenience adopt the following definitions.

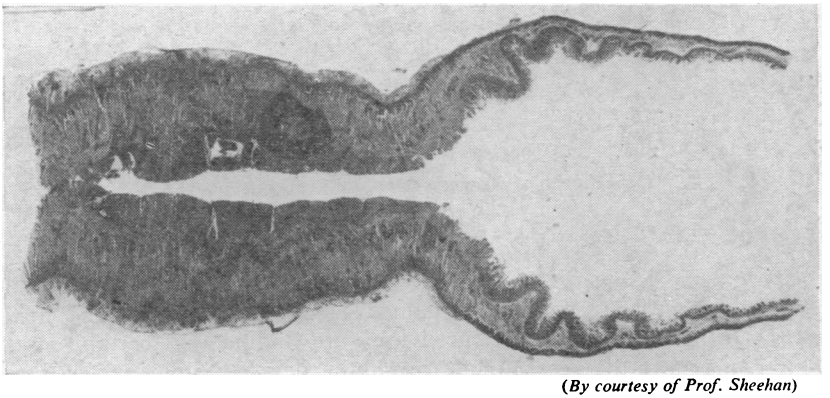
Definitions

1. Idiopathic ulcerative colitis

I suggest that the clinical entity " idiopathic ulcerative colitis " should be restricted to ulcerative states involving *always* and *in continuity* the rectum and pelvic colon and usually extending in continuity as far as the ileo-caecal valve. (Fig. 1.) This lesion occasionally involves the terminal ileum. Crohn has vividly described this extension as due to " wash-back from the caecum into the pool of the terminal ileum." Rarely, this involvement of the small intestine may be found in virtually its entire length. Such cases end fatally. These extensions of ulcerative colitis may be termed " idiopathic ulcerative coloileitis," or " colo-ileo-jejunitis." These rather clumsy terms are properly descriptive.

2. Crohn's Disease

Crohn's disease I would define as a granulomatous lesion of unknown aetiology characterised by great thickening of the bowel wall, involving



(By courtesy of Prof. Sheehan)

Fig. 1. Crohn's disease. Whole section of terminal ileum stained with H and E. Note the occasional deep fissuring of the mucosa, and the enormous thickening of the submucosa. The latter shows (area ringed) non-specific granulation tissue. The change from normal segment to diseased segment is characteristically abrupt.

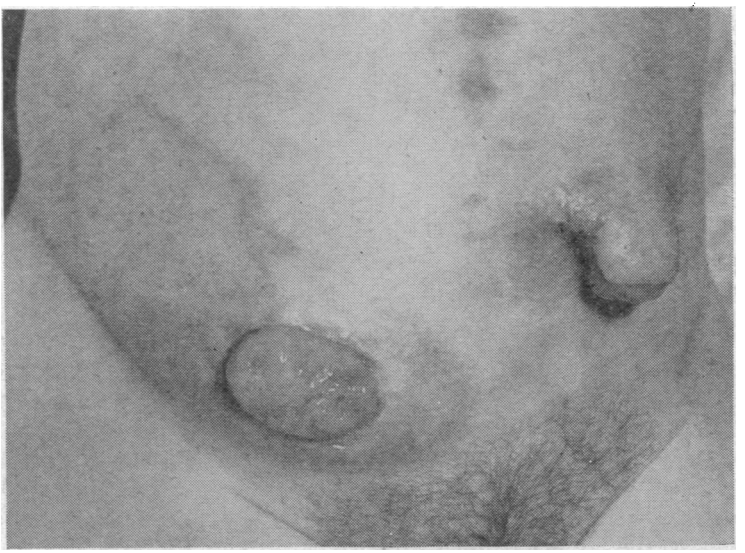


Fig. 2. Ulcerative colitis. Infra-red photograph. Double-barrelled side ileostomy with free portal-systemic anastomosis of veins due to cirrhosis of the liver. Secondary spout ileostomy (terminal) in more proximal segment of ileum. Observe robust whole-thickness skin covering upper surface of spout. A bag is worn with comfort and without leakage. Splenectomy and spleno-renal anastomosis relieved the venous congestion around the original ileostomy which had tended to bleed freely when touched. Partial gastrectomy was also done for multiple bleeding gastric ulcers. The patient, a young woman, is very well and recently was married.

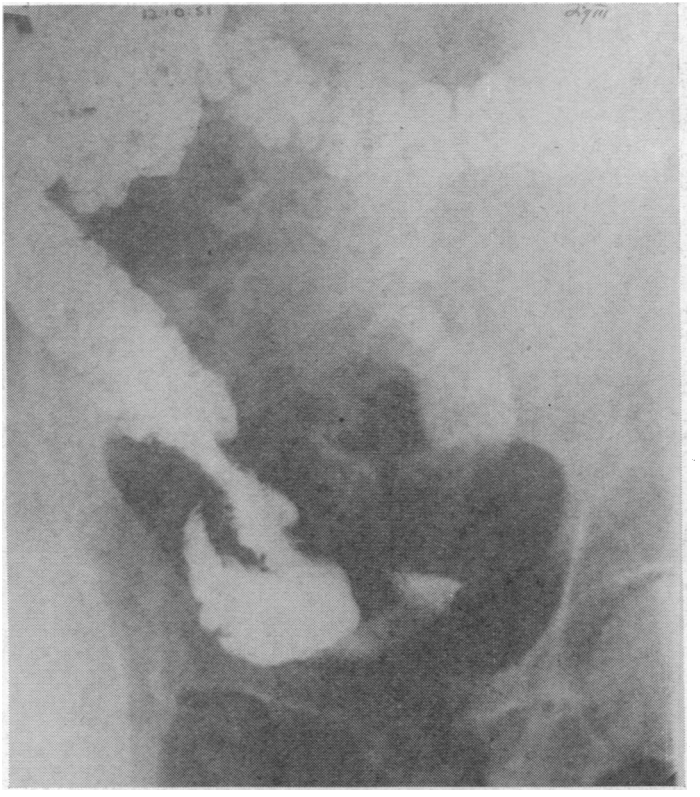


Fig. 3. Crohn's disease. Kantor's string sign in a young man who had been subject to undiagnosed intermittent diarrhoea for three years. (See Fig. 4.)

primarily some part, usually the terminal part, of the ileum. This granulomatous state may spread by " skips " to other parts of the small intestine or the large intestine. More rarely it may extend by continuity.

3. Segmental colitis

In addition to these two well-defined entities, there is a third condition which I would name " segmental colitis." Crohn employs this term but does not define it. This condition closely resembles the lesions of the colon seen in terminal ileitis (in 22 out of 306 of Crohn's own cases), but is found in patients who have no ileal or jejunal lesion. It is characterised by thickening and fibrosis of the bowel wall, patchy ulceration of the mucosa and a tendency to spread by " skips."

So far as my observations go, I am prepared to believe that although the same glandular involvement is not evident, this " segmental colitis " is a colonic form of Crohn's disease. Crohn himself, however, does

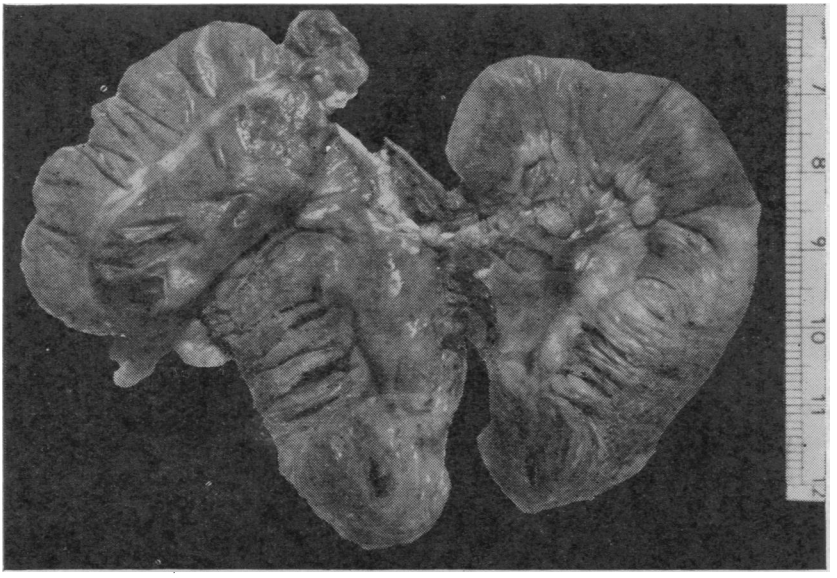


Fig. 4. Crohn's disease. Specimen of terminal ileitis removed from the patient whose X-ray is shown in Fig. 3. Fourteen inches of ileum are grossly involved, beginning abruptly and terminating at the caecum which, together with the appendix, is healthy.

not sanction this extension of the entity to which we give his name. I am therefore content to describe it as a separate entity.

Whether or not segmental colitis is a variation of Crohn's disease it, in my opinion, most certainly is *not* a variant of ulcerative colitis with which it is often identified or confused. I believe it most important to distinguish between the two. The observed differences between them are described later.

Discussion

In drawing distinctions and comparisons between these three entities we may profitably glance at the theories concerning their origins and causes. We should then compare them on grounds of pathology, clinical features and response to treatment. Throughout this discussion we shall have in mind the known causes of intestinal granulomata and specific intestinal inflammation. It is of interest that, in this country at least, the idiopathic granulomata we have named far exceed in number those of known aetiology, such as chronic bacillary dysentery, tuberculosis, bilharzia, amoebiasis and lymphogranuloma venereum.

Aetiology

First we must discuss the open question of the aetiological significance of bacillary dysentery.

Bacillary dysentery may be caused by the bacilli of Flexner, Sonne, Shiga, and others. It is associated with insanitary conditions, is highly infectious, and occurs both sporadically and in epidemics, especially among troops under difficult service conditions. The acute inflammation attacks the whole length of the colon and may, rarely, extend into the terminal ileum as well. It thus coincides with the distribution of ulcerative colitis.

In the 1914-1918 war there were many severe cases of dysentery from among whom a small number of chronic infections persisted. In these there was continuing inflammation and the causal organism was still present. This was, however, quite unusual and it is not likely to occur today because of the specific effect of sulphonamide therapy. A much greater number of sufferers continued to have changed bowel habit with variably increased frequency of stools. Such cases occur also to-day. They are attributable to change in the mucosal covering of areas of the large bowel. There is patchy fibrosis in the bowel wall and islands of modified mucous membrane may hypertrophy and become polypoid in the later stages. Eventually carcinoma may arise.

Whilst some degree of permanent damage is almost certain to follow a really severe attack of dysentery and lead to altered bowel habit for life, it is remarkable that in the more severe chronic cases, diagnosed as post-bacillary ulcerative colitis, the antecedent history is usually vague or presumptive. Thus the patients may have lived amongst dysentery but may have had no really clear-cut illness themselves.

The dividing line between severe presumed post-dysenteric colitis and true chronic ulcerative colitis cannot be drawn. Aetiologically, as has been said, a clear history is often unobtainable. It has been held by Felsen, who studied an outbreak of dysentery in New Jersey, that the original bacillary infection may be sub-clinical and that it may be spread very readily from individual to individual even some time after the original attack. Thus, he claims a high incidence of conjugal spread (between husband and wife). The greater part of Felsen's evidence is serological and turns on the question of diagnostic agglutination titres against specific strains of B. dysenteriae. He speaks of a 10 per cent. incidence of chronic ulcerative colitis or Crohn's disease among 122 patients and contacts whom he followed after the epidemic. He also claims diagnostic agglutination in 95 per cent. of these cases whereas, in 300 controls, the titres were at a similar level in less than 5 per cent.

I have found this a fascinating piece of work and so many of Felsen's straws seem to blow in the same direction that one is tempted to believe that bacillary dysentery is, indeed, the prime factor in chronic ulcerative colitis. Unfortunately, however, agglutination tests for the shigellae are far from specific and, unless the titre is very high indeed, they are not regarded as significant by experienced bacteriologists. Similarly, the finding of bacteriophage in the stool is non-specific. The dysentery

organisms themselves are, as we have seen, virtually never found in the chronic stage.

As regards pathology, the findings in chronic ulcerative colitis are compatible with a dysenteric origin. The mucosa is largely shed, regeneration is incomplete, islands of cells tend in time to become polypoid or even carcinomatous, and fibrosis is a common sequel.

The disappointing conclusion that we must reach is that nothing has been proved about the significance of bacillary dysentery in the aetiology of ulcerative colitis and no information gleaned which is helpful in treatment.

Streptococcal infection

Dr. Bargen at the Mayo Clinic has isolated a streptococcus which he regards as specific to ulcerative colitis. He has wide experience and is convinced of the truth of his hypothesis, but most other workers in this field are not in agreement with him.

Three other pieces of research deserve mention, namely (i) the production of foreign body reaction by Chess *et al.* (1950); (ii) the action of lysozyme; and (iii) the effects of vagotomy and their implications.

The foreign body reaction is a purely experimental piece of research carried out by Chess and his co-workers. In one series of animals, the ileo-caecal angle was isolated and exteriorised. Into the pouch so formed, finely divided sand or talcum powder was placed. After a number of days the animal was sacrificed and the ileo-caecal angle examined. The changes found closely resembled those of Crohn's disease, namely, thickening of the wall, narrowing of the lumen, fissuring and shedding of the mucosa and giant cell reaction in the surrounding tissues. In a further series, the irritant was given by mouth and somewhat similar changes were again found in the ileo-caecal region. The selectivity of this site is attributed to the slightly slower rate of passage of its content and the large amount of lymphoid tissue found there. The reaction found in the affected tissues is thought to be due to the property of piezo-electricity present in the crystals of the sand and talc. The application of these findings to the actual problem of aetiology is not obvious !

Lysozyme is a mucolytic enzyme found in tears and other body fluids and also in egg white from which it is prepared in pure form. It is found in great excess in the mucous membrane and in the exudate from the mucous membrane of the stomach and intestines at or near sites of ulceration. When administered intravenously, colonic mucosal congestion is caused. When injected intra-arterially into any vessel supplying the bowel, congestion and ulceration occur in the loop supplied. (Light, 1951.) There seems no doubt about the activity and importance of lysozyme. The enzyme is apparently elaborated and released at the level of the lesion in the formation of which it plays some part. There is, however, no clue to the antecedent events which *cause* the liberation of the lysozyme in excess.

It is of some interest that all the aetiological implications so far discussed apply (in so far as they apply at all !) with equal force to both Crohn's disease and ulcerative colitis.

Vagotomy. It is known that in dogs, bilateral lumbar sympathectomy causes ulcerative changes in the bowel. It is suggested that this is due to excessive spasm and uninhibited contractions. Vagotomy corrects or prevents these changes from occurring.

In man, in chronic ulcerative colitis, vagotomy relieves the symptoms in a significant proportion of cases, although very little change can be seen on sigmoidoscopy or in radiological pictures and the stool is unchanged in character. The average rate of passage of food through the small gut is slowed by only four hours from 14½ to 18½ hours. The rate of passage through the colon is unchanged. The frequency of bowel action is lowered by the combined effect of slowed rate of passage and diminished tone. The effect appears to be mainly in the small intestine, which is not surprising since most of the large bowel is not supplied by the vagus. These findings suggest that exaggerated contraction and spasm of the intestine may play some part in aetiology. Eddy (1951), who was associated with Clarence Dennis under Wangansteen at Minneapolis, found an average reduction to 1-2 from 5-15 stools daily in 42 vagotomised patients observed for a number of years.

Only early and relatively mild cases are thought to be suitable for vagotomy. One reason for refusing the later cases is that 15 per cent. of all sufferers from chronic ulcerative colitis develop carcinoma of the colon or rectum. This is regarded as a clear indication for colectomy in cases of long standing, even in patients already relieved of their symptoms by vagotomy.

The psycho-somatic concept

Finally, in this aetiological survey, we must include the concept of the psycho-somatic origin of ulcerative colitis. This has for long been accepted by many as an obvious truth. My own view is that nothing was ever further *from* the truth. These patients are among the most stout-hearted of all those with whom we have to deal. One man, who gets long periods of relief *from* rest and retention enemata, accepted a job as a steeple-jack. When forbidden this by his physician he went to the other extreme and began to work down a coal mine. A young woman patient of my own after two ileostomies, colectomy in two stages, partial gastrectomy for bleeding gastric ulcers which threatened her life, and, finally, splenectomy with spleno-renal anastomosis for portal hypertension due to cirrhosis, recently became engaged and was married two months ago. (Fig. 2.)

A third patient, again a young woman, was regarded as a psychoneurotic for two years because of her habit of biting her nails and an unfortunate, wartime, bigamous marriage with a free-French sailor.

She was ultimately found to have regional ileitis and was completely relieved by resection.

Among those who do not get better there is a high percentage with strength of character enough to take their own lives.

Pathology and clinical features of the three lesions

Crohn's disease. Turning now to the pathology of Crohn's disease, we may first discuss its typical form, terminal ileitis. Three stages are described in terms which relate pathological and clinical attributes, namely :—

(i) The early, acute, which simulates appendicitis.
(ii) The irritative, in which diarrhoea is common.
(iii) The stenotic which leads to obstruction. In this last the prognosis after operation is the most favourable.

Cases in the early acute stage are most often picked up in children. The affected ileum is thickened, red and oedematous. The illness may be quite sharp and the abdomen is usually opened for supposed acute appendicitis. The records suggest that the wisest procedure is to close the abdomen. Twenty-five per cent. of cases recover completely. Seventy-five per cent. progress and call for further surgery months or years later when they are better able to stand the operation.

In the *irritative phase*, in which diarrhoea is common, the main changes are seen in the submucous layer and there is a strong tendency to spread either by continuity or by skips. The features of this phase of the disease are intermittent diarrhoea, mild fever and general illness with loss of weight and other constitutional changes of a " toxic " nature. A mass may be palpable in the region of the terminal ileum.

The irritative phase may persist for months or years, during which the true diagnosis may go unsuspected. Once suspected, confirmation may be obtained by X-ray. The characteristic appearance is the string sign of Kantor.

The *obstructive lesion* is late in the natural history of the lesion and may be found in a matter of months or years after the onset of symptoms. As time passes, the affected loop of bowel becomes increasingly rigid and thick, to resemble, externally, a length of garden hose. The lumen becomes smaller as the submucous granulation tissue becomes more and more tough and bulky. Fibrosis is not conspicuous. The mucosa itself becomes split up into islets and has a cobble-stone appearance.

Ulceration occurs as longitudinal fissures. This is a late phenomenon which leads to quiet perforation with the formation of abscesses and fistulae, especially between the layers of the mesentery.

Sooner or later the regional glands become enlarged and rubbery. Caseation is never seen.

Skip lesions are common and may involve contiguous parts of bowel. I have, in different cases, seen involvement of " touching " parts of the ileum and ascending colon and of ileum and pelvic colon ; the colonic

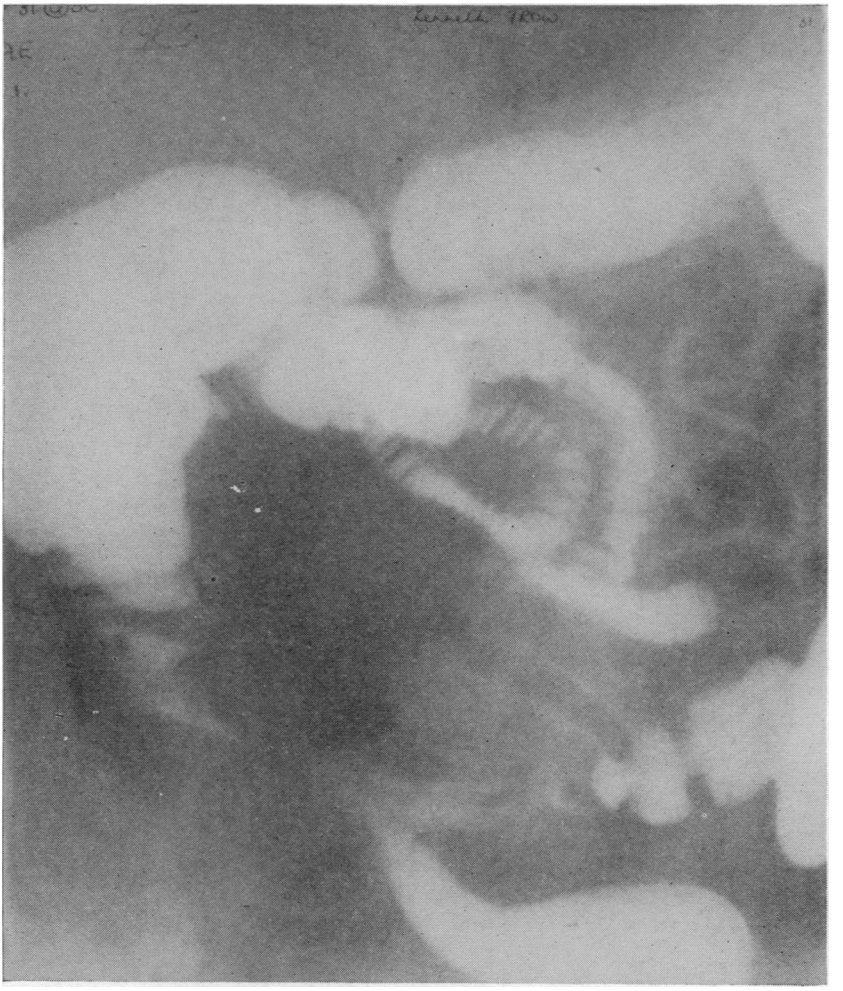


Fig. 5. Crohn's disease with skip lesion. Barium enema.

(*a*) Terminal ileitis with string sign.
(*b*) Ileo-transverse colostomy with exclusion. The blind end of ileum can be seen.
(*c*) Skip lesion to pelvic colon which is adherent to the terminal ileum.

lesions extend over three or four inches of bowel or more and produce partial obstruction by fibrosis. The peritoneum is irregularly thickened and white, giving an appearance not unlike that (quite unrelated, of course) of leucoplakia of the tongue or vulva, which has been described as " white paint that has dried and cracked." There is in the colon

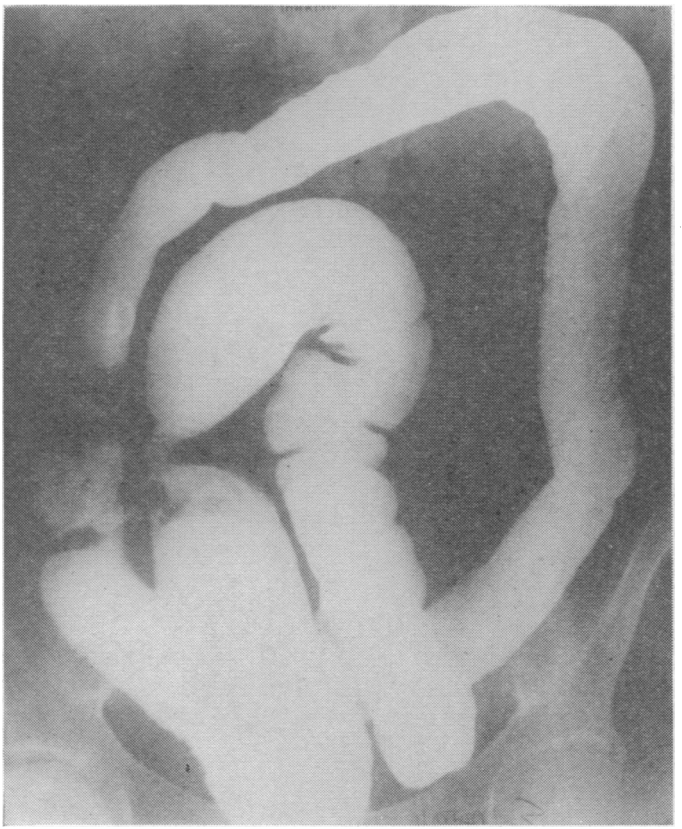


Fig. 6. Segmental colitis of ascending colon with skip lesion to pelvic colon. There was no ileal lesion.

less sub-mucosal thickening than is seen in the small intestine but more fibrosis.

The mucosa shows a change similar to that found in the ileum except that the fissures run irregularly rather than longitudinally, giving a lace-like effect. The whole affected loop is much tougher than in chronic ulcerative colitis ; skip lesions are common, and slow perforation, with fistula or sinus formation, may occur.

Ulcerative colitis. In ulcerative colitis, by contrast, the bowel is oedematous and friable and adhesions between loops are conspicuous by their absence. Sinus formation does not occur. Skip or contact lesions are never seen. The inflammation is purulent with fluctuating attacks of pain, fever and diarrhoea, with purulent and often bloody stools. Perforation is exceedingly rare and, when it occurs, leaks freely

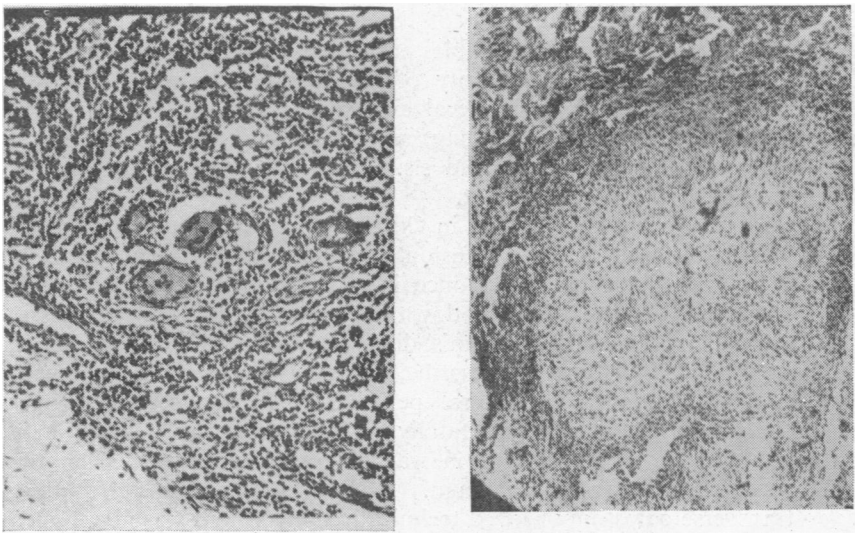


Fig. 7. Sections from non-specific intestinal granulomata showing :—
(*a*) Granulation tissue with giant cells and
(*b*) a typical " tubercle " with epithelial and giant cells. There is no caseation and the condition is not tuberculous.

without localisation into the general peritoneal cavity with the production of general septic peritonitis.

Segmental colitis is a non-specific granulomatous lesion affecting the colon without any associated ileitis. In all other respects, including the cobble-stone change in the mucosa, it closely resembles the colonic extensions seen in Crohn's disease. The bowel wall is moderately thickened, tough and fibrous with visceral peritoneal condensations. Adhesions and even fistulae are common as also are skip lesions by contact.

As in Crohn's disease, the symptoms may be irritative, with diarrhoea, simulating ulcerative colitis, or obstructive, simulating some other obstructive granuloma or carcinoma.

Microscopically, a non-specific granulomatous change is common to all these three lesions. Giant cells may be seen with or without epithelioid formations. Sometimes the giant-cell and epithelioid systems are so perfectly formed as to simulate tuberculosis very closely. Caseation is, however, not seen and all these changes are characteristic of and attributable to a foreign body reaction. This type of reaction can be seen in relation to mucosal lesions of the colon, rectum and anus, no matter what their origin or nature.

Crohn and his associates have made the most exhaustive attempts to detect tubercle bacilli by staining and by making emulsions of tissue for

inoculation to culture media and guinea-pigs. In 15 years at Mount Sinai Hospital, where hundreds of ileo-caecal granulomata have been examined, only four proved examples of ileo-caecal tuberculosis have been identified. Evidently the majority of the supposed cases in the past were either " Crohn's " with caecal extension or segmental colitis.

The microscopic picture as I have seen it does not form a sufficiently regular pattern to suggest sarcoidosis. The other and remote features of sarcoidosis are never present.

A commonly associated lesion in Crohn's disease is fistula in ano and this has lent colour to the confusion with tuberculosis. The fistula is usually strictly local but may, once in a while, come from an intra-peritoneal perforation tracking down through the ischio-rectal fossa.

A rare complication of Crohn's disease, which Crohn mentions in his monograph but does not describe, as he has not personally seen it, is acute perforation with general peritonitis. I have seen this only once and then the case ended fatally. The patient was a florid man approaching 60. The perforation was a large, transverse tear in the antimesenteric border of the ileum. After closure of the perforation, ileo-transverse colostomy proved technically difficult and was abandoned because of the poor general condition. Re-perforation occurred a week after repair. Ileo-transverse anastomosis was then carried out, but the patient did not survive.

Treatment of the three lesions

Garlock and Crohn, discussing the operative treatment of *Crohn's disease* in general, favour short-circuit with exclusion, i.e., ileo-transverse anastomosis and division of the ileum just beyond the anastomosis and short of the lesion. Lahey favours resection by the Paul-Mikulicz technique. Resection if done may be performed in either one or two stages. The case reports quoted of short-circuit with exclusion show a lower operative mortality and a recurrence rate no greater than after resection of the lesion. One cannot help feeling that these figures in some way distort the truth and my own experience suggests that short-circuit with exclusion may be followed by :—

(1) Complete and lasting relief.
(2) Continued diarrhoea due to irritation of the original mass, calling for second-stage resection.
(3) Extension of the disease either distally or, much less frequently, proximally towards the higher ileum or jejunum. This may be of a fulminating character.

I find it hard to believe that short-circuit with exclusion offers a better chance of freedom from extension than does excision. In any event, it is becoming increasingly apparent to all workers in this field that relapse or recurrence is to be expected in something approaching a quarter of the cases treated.

So far as our knowledge goes, I believe that the end results depend

more upon the degree of virulence of the disease than upon the precise operation performed. Until we know more of its true nature and aetiology we shall not be able to compile a satisfactory code of rules for treatment and prognosis.

My advice is :—

(i) to explore the abdomen widely enough to see all the lesions clearly.
(ii) to consider all the possibilities carefully before embarking on a definitive procedure.
(iii) where excision, even of two widely separated foci calling for two separate resections, is easy and likely to prove complete—to resect ;
(iv) otherwise to do a short-circuit with exclusion.
(v) to resect at a second stage, after exclusion, in the event of persistent or recurrent irritative symptoms (diarrhoea—fever—weight-loss).

The prognosis is never certain and relapse may occur even after years and even after apparently complete resection. The reasons for this are not apparent.

In planning treatment in *chronic ulcerative colitis* it is helpful to think of three grades of severity :—

(i) The mild case, amenable to medical treatment, blood transfusion, rest, diet, and retention enemata.
(ii) The fulminating case in which ileostomy can be life-saving and should be done early.
(iii) The average moderately severe case with exacerbations and remissions. The operation is *planned* during an attack but is *done* in a remission. One-stage ileostomy and colectomy is the best procedure.

At Johns Hopkins, a one-stage operation is completed by bringing the ileum to the anus. Apparently a fair degree of continence is usual, but I should like to see some of the patients myself before advocating the procedure.

My own preference is for a spout ileostomy partly covered with a skin flap and partly with a split skin graft. (*British Journal of Surgery* —in the press.) This procedure not only enables a bag to be worn in comfort but also excludes the risk of prolapse of the ileostomy. This is, otherwise, a troublesome complication.

End ileostomy with the Rutzen bag is a method of which I have no personal experience. It is undoubtedly excellent.

After making the spout ileostomy the skin of the abdominal wall and thigh is protected by nursing the patient in a semi-prone position on the right side over split mattresses. The ileal content thus falls cleanly away into a receiver until the spout is strong enough to receive the bag.

Segmental colitis. It is important to distinguish this condition from chronic ulcerative colitis, first because there is usually no need for ileostomy. The affected segments of bowel can be resected with end-to-end anastomosis, or short circuit with exclusion may be practised with or

without secondary resection. Extension and recurrence are not uncommon, but the rectum is unlikely to become involved. One is tempted to believe that it was on the basis of segmental colitis rather than chronic ulcerative colitis that the plan of operation advocated by Sir Hugh Devine was based. In it a double-barrelled ileo-colostomy is constructed with a view to subsequent ileo-sigmoid or ileo-rectal anastomosis. Certainly this procedure is not suitable in the average cases of chronic ulcerative colitis as we know it, where the rectum is invariably diseased.

Other granulomata, etc. Certain other lesions may need to be distinguished in diagnosis or may throw some light upon the aetiology of the three idiopathic conditions just described. These are: amoebiasis, lymphogranuloma inguinale, tuberculosis and endometriosis.

Amoebiasis may lead to the appearance of a rectal or recto-sigmoid or other colonic granuloma most closely simulating carcinoma. Cysts or ova should be looked for; sigmoidoscopy and biopsy should be done; the therapeutic test may prove the condition within a matter of days.

Lympho-granuloma venereum (syn. L. inguinale) is a virus disease which responds to treatment with chloramphenicol. In the early stages the proctitis resembles that of chronic ulcerative colitis and the resulting strictures also resemble this condition. Multiple peri-anal abscesses or sinuses are common; the venereal character of the infection is to be borne in mind; Frei's test is confirmatory as also is the response to therapy.

Efforts to relate chronic ulcerative colitis to this virus infection have so far failed.

Non-specific lesions resembling lymphogranuloma may arise, and I have one such case under observation now. The Frei test is negative and the response to antibiotics is nil. Such cases suggest that chronic ulcerative colitis may begin as a virus proctitis.

A biopsy from this particular case is of interest. It closely resembles all the other sections so far discussed. This merely suggests that the appearances are all non-specific and that the foreign body granuloma is a secondary effect due to gross contamination with faeces.

Tuberculosis differs from the lesions under discussion *both* macroscopically, microscopically, and biologically. The lesions have a spreading quality within the peritoneal cavity. Tubercles are visible to both the naked eye and the microscopist. Caseation and cold abscess formation are common. Oedema abounds and tissue planes disappear. Resection is impossible and short-circuit with exclusion, often difficult. It may, on this account, need to be done at a spot far removed from the site of the lesion. Organisms can be seen or cultured and emulsions of tissue are bacterially active.

The ground for aetiological confusion between tuberculosis and the three granulomata seems slender indeed.

Endometriosis has no aetiological relation to chronic colitis, but may

produce obstructive lesions leading to errors in diagnosis. Carcinoma of the colon may also be simulated. The condition must be borne in mind at operation if serious mistakes are to be avoided.

Concluding summary

A case has been made out for distinguishing as three distinct entities —Crohn's disease, segmental colitis and chronic ulcerative colitis.

Possible aetiological factors in the production of these three idiopathic states have been discussed with generally inconclusive results.

Points of resemblance to each other and to other related conditions of known aetiology have been considered. Their bearing on aetiology, pathology and symptomatology have been reviewed.

The treatment appropriate in chronic ulcerative colitis has been shown to be quite different from that applicable to the other two states. The other two have been shown to resemble each other fairly closely in their treatment and prognosis. Some practical points in treatment and management have been proposed.

Acknowledgments

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THE BUCKSTON BROWNE BENEFACTION

THE BUCKSTON BROWNE Benefaction was commemorated on Tuesday, 8th July.

Professor David Slome, Bernhard Baron Professor, delivered a lecture at 5 p.m., in which he gave a most interesting account of the origin and development of the Buckston Browne Farm at Downe, and of the research work now in progress there.

Later the same evening was held the Buckston Browne Dinner, the company of 130 being mainly composed of Fellows and Members in accordance with the donor's wish.

The toast of " The College " was proposed by Miss Patricia Hornsby-Smith, M.P., Parliamentary Secretary to the Ministry of Health, who opened with an amusing endeavour—and failure—to find common ground between the late Sir Buckston Browne and herself. After referring to the ancient traditions of the College, she praised the recent advances in surgery, particularly that of the brain, heart and lung, and plastic surgery. She thanked the College for cooperation and help given to the Ministry of Health at all times, and spoke of the increased number and better distribution of Consultants since the operation of the National Health Service. She congratulated the College on moving with the times in such matters as new examinations, the publication of the Annals, imperial connections, and all its activities as a scientific and social centre —activities which would surely be greatly increased with the rebuilding and enlargement of the College.

The President (Sir Cecil Wakeley, Bt.) thanked Miss Hornsby-Smith for her compliments and replied on behalf of the College.

" To-day," he said, " we commemorate the Buckston Browne Benefaction, and turn our thoughts to the affectionate memory of that great-hearted donor, George Buckston Browne. From 1874 to 1926 he was a Member of this College, and from 1926 till his death in 1945 an elected Fellow. It was his ideal to bring together in a family party the Fellows and Members of the College and to that end he endowed this dinner.

" Buckston Browne we can no longer have with us in the flesh, but let us at least rejoice in the company of his son-in-law, Sir Hugh Lett, and congratulate him on the jubilee of his Fellowship which he attained last month.