ULCERATIVE COLITIS

(SO-CALLED ASYLUM DYSENTERY.)

Being a Thesis for the Degree of M.D. of Edinburgh University.

by

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or

ASYLUM DYSENTERY

From time immemorial contagious diseases have aroused special interest, not only among those engaged in the study of medicine, but in all classes. Ulcerative Colitis must be looked upon as a contagious disease; this alone would be a sufficient inducement for its comprehensive study, but in addition it embodies certain special characteristics, which lend a charm to its special investigation.

It is a disease which, although unrecognised for many years, must have existed ever since it has been the custom to congregate in the same building large numbers of the insane. It is almost always an Institution disease and particularly an asylum one.

The literature of this subject, though scattered in the extreme, has been of a particularly copious nature during the last fourteen years, which is a manifestation of the importance it has assumed in the minds of those engaged in Asylum work.
As another proof of its importance it will be shewn in my statistical tables, that in the Asylums under the control of the London County Council alone, ulcerative colitis was responsible for 89 deaths during the period of twelve months.

The subject was brought vividly to my notice during a period when I was assistant medical officer at a large London asylum (Claybury) where it raged with the utmost fury, and assumed all the characters of an epidemic.

Definition:

Although ulceration of the colon occurs in many diseases; as a result of typhoid fever, tuberculosis, tropical dysentery, &c., ulcerative colitis is the name applied to a condition (generally met with in Asylums) characterised by intermittent pain, nearly always abdominal; fluid, offensive and occasionally bloody stools; with great exhaustion, and a marked absence of tenesmus; accompanied by febrile disturbances and consisting pathologically of ulcers of the large intestine, particularly of the colon, the ulcers being generally numerous, irregularly shaped, and penetrating the submucosa. Gemmel(24) defines the above disease as follows:— "Without attempting anything like a complete and exhaustive definition (ulcerative/
(ulcerative colitis) may be designated as a specific primary colitis, characterised by ulceration and sloughing of the mucous membrane, by more or less intractable diarrhoea, the stools containing and often entirely consisting of blood stained mucus and in the later stage of the disease becoming very offensive and possibly containing sloughs and shreds of membrane; by a varying degree of fever; and by abdominal pain and uneasiness, with in many cases tenesmus and strangury.

Gemmel divides ulcerative colitis into two groups:

1. Idiopathic ulcerative colitis, a specific disease with its varieties fulfilling the requirements of dysentery, by which term it may be called.

2. Secondary ulcerative colitis, which is further divided into

(a) Specific, in the same sense that it is due to a specific cause, and occurs in the course of a non-dysenteric specific disease. Examples of such complications of the colon are seen in colo-typhoid, tubercular and syphilitic disease, diphtheria and measles, &c.

(b) Non-specific, as occurs in impaction of faeces, presence of foreign bodies, and in toxic and irritant poisoning from diseased food, corrosive sublimate, uraemia, &c.

Another definition that may be given is the following:

An/
An inflammation, with ulceration of the mucous membrane and submucous connective tissue of the colon, producing lesions undistinguishable from those of dysentery; it is primarily a non-specific local affection, commencing in the nerve tissue and subsequently attacking the mucous membrane with its glandular structure. As in dysentery the inflammation induces gangrenous destruction and ulceration of the mucous membrane and underlying cellular tissue.

Mott, in an address recently delivered before the Epidemiological Society of London on December 13th, 1901, defines ulcerative colitis as follows:

A disease characterised by febrile onset, and by inflammatory lesions of the intestine, affecting persons of different ages and bodily health and tending to occur in outbreaks. As a rule it is more liable to affect the old, the infirm, and the bedridden (this, however, was not the case at Clayburg) but no particular form of insanity is more liable than another.

Mott also divides the disease into seven classes:

1. The acute case with preliminary fever lasting till death supervenes in two or three days or within a week.

2. The acute case with preliminary fever and a temperature which falls rapidly as the collapse proceeds.

3. The case with mild fever, 101-103° and diarrhoea.
5. Diarrhoea for a day or two, accompanied by diarrhoea with blood and slime in the stools for a few days, terminating however with recovery.

4. The mild case without fever, but with diarrhoea, accompanied with blood and slime, lasting over two days. In some of these cases there may have been initial fever which was overlooked.

5. Cases of varying degree of severity in which after an interval of a few days, symptoms recur, sometimes with fatal results and sometimes with recovery.

6. Cases which do not clear up after the first week or two, but which become chronic, the patients continuing at more or less intermittent intervals, to pass bloody, slimy, or diarrhoeal evacuations for months.

7. Cases of intermittent or prolonged diarrhoea, in which neither blood or slime has been noticed in the stools, and yet post mortem dysenteric lesions of a similar nature have been found.

Historical Note:

Ulcerative colitis, Asylum dysentery, abdominal coryza, or by whatever name the disease is known, is a disease of comparatively recent times, or at any rate only a recognised disease.

The earliest record I can find of what evidently was this disease is in 1670-71 and onwards, when it was called by Sydenham\(^{(38)}\) "dry grypes": he remarks that "the disease sets in with chills and shivers, then the heat of fever, then griping of the belly, and lastly stools. Occasionally there is no fever. The stools are more slimy than steroraceous, also streaks of blood, but not always
- sometimes, indeed, none from first to last. Notwithstanding — provided that the stools are frequent, slimy and attended with griping, the disease is a true bloody flux or dysentery."

The above remarks by Sydenham evidently shew that he recognised various sorts of diarrhoeal diseases, and in this case he described what we would now probably call ulcerative colitis.

It was in 1887 that particular attention was drawn to Ulcerative Colitis in an article by Hale (32) White, in which he gave symptoms, post mortem appearances, &c., in a series of cases: since that date numerous cases have been given in various medical journals; all of these cases occurred in sane patients and were therefore — being rare — looked upon with some interest: asylum medical men, and especially those belonging to the asylum to which I was attached, know the disease well and if all the cases of ulcerative colitis which came under their notice were read before societies, the societies' reports would contain nothing but the above disease.

Whether ulcerative colitis is really dysentery is not known: personally, I do not believe that the diseases are the same, and therefore I shall keep the subject of dysentery out of this thesis and deal exclusively with ulcerative colitis, or the disease known/
known by that name.

That the disease is limited almost entirely to the insane is, to the best of my belief, unquestionable; occasionally, of course, cases crop up in various hospitals, but this proves nothing. In days of old when dysentery was very rampant in the country, no doubt, numerous cases of ulcerative colitis occurred in Asylums, alongside regular dysentery; but owing no doubt to the bad diagnosis of disease in these days, it went unrecognised and all these illnesses were put down under the heading of dysentery.

In support of my above statement, I may quote Wilks & Mox(in(36) p.427-28. The term colitis is sometimes used as though synonymous with dysentery; our usual language has been too indefinite, nay too incorrect, in speaking of all affections of the large intestine as dysenteric, for the true dysenteric process, although in many features like simple ulcerative colitis, is a disease having certain definite characters. There is quite as much reason to regard febrile epidemic dysentery as a disease distinct from simple ulcerative colitis, as there is to regard febrile epidemic diphtheria as a disease distinct from idiopathic croup. Dysentery produces a variety of colitis distinguished from others by a special/
special course and by clinical phenomena, though fever when the dysentery has passed off, the state of colon which is left is much the same as that of simple ulceration.

It is sometimes said that lunatics are especially liable to dysentery or ulcerative colitis. (Mott & Durham 23) It does not seem that there is any very definite support for such a statement. The character of their surroundings and their frequently debilitated state of health, both of which are merely predisposing factors, do not shew that lunacy has any direct share in the matter. Moreover, the sane attendants and even gardeners, medical officers, and other officials do occasionally suffer. Some have gone further and insisted that certain forms of mental disease are particularly apt to be followed, or rather concluded by, an attack of dysentery. General paralysis of the insane is supposed to be a disease in which this is the case. According to mental diagnoses which have been furnished to us, in connection with reports of cases*, there are singularly few general paralytics; indeed it appears from these that there is no limitation whatever, according to mental condition: epileptics (some in excellent physical condition) acute and chronic melancholics/

* N.B. I find that in 50 cases dying from ulcerative colitis, nine suffered from G.P.I., i.e. 18%
ics and maniacs, as well as dments of various de-
scriptions all figure in the list.

If the above statement be true, re lunatics
not being especially liable to suffer from ulcerat-
ive colitis, how is it that we do not get epidemics
of ulcerative colitis in workhouses, where as a rule
the inhabitants are old and feeble, the drainage sys-
tem frequently bad, and there is generally over-
crowding, where the nursing is deficient and often
highly unskilled and the food not at all up to the
mark. I have attended workhouses for some years
and have never seen a case of ulcerative colitis in
one of these houses, whereas in the asylum at which
I had the honour to be medical officer for a short
time, cases of the above disease regularly cropped
up and often the disease assumed the form of an
epidemic. In this particular asylum the buildings,
drainage, etc., were new and supposed to be perfect,
the feeding good and the nursing much above the
average merit.

Ulcerative colitis attacks patients of all
adult ages and length of residence in an asylum is
apparently of no account in the disease: the facts
will be pointed out in my analytical and statistical
tables at the end of this thesis.

With the above remarks, I will close this
historical/
historical and partly introductory statement, and will examine into the disease as ulcerative colitis and not dysentery.

Symptoms:

It is nearly always the state of the bowels which seemingly directs the patient's attention to his illness and the first symptom which he remembers is abdominal pain. "Chills and a rigor accompanied by headache and frequent vomiting, sometimes of a bilious character, associated with high fever, indicate the onset." (Mott 42) In the case of the insane, when the senses are to a great extent dulled to pain, no complaint of pain may be made, but in a few cases, where the patient may be more or less intelligent, he will often complain of pain, chiefly around the umbilicus. Constipation and a rise of temperature, often up to a hundred, are invariably present, often accompanied by vomiting according to my experience, and when this happens I am always in a state of anxiety if any resulting diarrhoea occurs; pain may also be felt in the back and loins, but in the majority of cases in the insane, pain is felt, as I stated above, in the abdomen only. If the temperature continues for twenty-four hours, diarrhoea/
11.

diarrhoea supervenes, of a more or less severe character—the bowels, it may be, being open 20 times in the 24 hours (v. chart 4, 6); tenesmus, which is a common symptom of dysentery is generally absent, though it may be complained of to a certain extent. The evacuations are foul-smelling, fluid, dark, and consist generally of water and slime, with often a great admixture of apparently fresh blood. In cases which are acute and pass only blood and slime, one notes the remarkable scarcity of bacteria present (Mott, (42); Osler (8) also notes this. Now and again amongst the dejecta are a few scybalous masses, enclosed as it were in a capsule of slimy mucus and some mucus membrane, or the scybalus mass may be covered with blood only. The stools never, in my experience, resemble dysenteric or choleraic stools, and in fact it is due to a considerable degree to the character of the stools that a correct diagnosis can be made: attendants state that they can diagnose a case by the smell of the stools (Mott (42)).

I believe that when small scybalous masses, with blood adherent to them are passed in the motion, these masses will be found to have stuck to the walls of the intestine over the parts in which are the ulcers, hence the stained appearance of these masses. Mucus is not often present to any extent, at times, however, /
however, we may have sloughs (apparently coming from the surface of the ulcers) thrown out with the motions, but more frequently these sloughs will be found post mortem, to be adherent to the edges of the ulcers. There may be reflex vomiting, though this appears to occur more in the sane than the insane; when it does occur, I believe it to be due to the irritation caused by the secretion of the intestine acting on the exposed nerve fibres at the base of the ulcers.

The patient generally lies on his back, with legs drawn up; the face is pale and generally damp and cold, and he has the appearance of one suffering from collapse. The tongue at the beginning of the illness is generally covered with white fur, but as the disease advances this fur is replaced by a brown discoloration, the tongue itself becoming brown and dry, and it may be cracked. As the disease advances and diarrhoea persists, the patient becomes weaker and more anaemic day by day. The pulse is generally over 100 and soft; the temperature as a rule varies greatly and no fixed rule for diagnosis by temperature can be formulated; generally, however, it will be found to rise during the day and fall at night. Occasionally (v. Chart 8) the temperature will run up with a rush, immediately before death.
and at other times it falls lower and lower till it falls to it may be 96.5 or 96, the patient dying in a state of collapse. The highest temperature I have seen immediately before death has been 104.4 and the lowest 96.2, but it appears to me that there is no regular temperature attached, as it were, to this disease; the cause of death is usually exhaustion, accelerated it may be a good deal by haemorrhage. At times the diarrhoea ceases the day before death, but in these cases in which this occurs the patient is generally in an extremely debilitated state.

Extreme thirst with a sensation of dryness of the throat is at times a most distressing symptom. Some of the patients, of more than usual intelligence complain of a bitter taste in the mouth and not infrequently of a metallic taste at the end of the tongue.

The urine as a rule presents nothing remarkable; however, when the temperature is high we find it of a deep colour and loaded with urates; in these cases the smell of the urine is very noticeable and reminds one of a stable; as a rule albumen is absent, when present we find that we have to deal with some kidney mischief as well as the intestinal illness; we sometimes find traces of biliverdin and bilirubin in the urine.

Delirium/
Delirium is very rarely present; however we now and again get coma and utter collapse at the end of the illness; this generally occurs in very acute cases.

As far as pain is concerned, Hale White and Coleman both mention cases in which pain was referred to the back; personally I have never seen this.

The duration of this illness varies and does not, strangely enough, seem to vary with the age of the patient; I have seen cases lasting three weeks die, and cases lasting three days end fatally; the average duration of an acute case is ten days from the onset of the disease.

Cases however become chronic when they take unto themselves the characters of chronic or recurrent diarrhoea, more than those of ulcerative colitis.

I shall now give the opinions on reflex vomiting, etc. of various writers, all of whom have more or less studied the subject of ulcerative colitis. It is to be noted that all these cases occurred in the same, whereas my cases occurred in a lunatic asylum, and I have necessarily to give symptoms which I have personally observed.

As regards the reflex theory of vomiting, Hale White remarks that "the vomiting is reflex, as/
as is also the diarrhoea, for if they were not they
would bear some relation to the amount of ulceration,
which they do not, and furthermore the reflex theory
also explains the alternating attacks of constipa-
tion.

Tenesmus occasionally appears to exist, though
I personally have not noted a case; it is a very
prominent symptom in dysentery, and is I believe one
of the chief means of differentiating this disease
from dysentery. Of course my experience has been
limited to the insane, who are naturally not so sen-
sitive to pain as those not suffering from mental
disease.

In the case of a middle aged woman constant
tenesmus existed with acute and persistent diarrhoea
(Sharkey7)

Robinson(22) and Hale White(32) quote very sim-
ilar cases.

Prickett(21) had a case in which the disease was
characterised throughout by total absence of tenes-
mus and pain anywhere except in the parotid gland.
There was also absence of sweating and rigors and
although at first there was a good deal of vomiting,
necessitating very frequent feeding, yet the patient
(a girl) afterwards and up to the date of her death,
took nourishment frequently and well; this attack of/
of ulcerative colitis came after an attack of influenza and is, I believe, the only one on record.

Cases of ulcerative colitis all more or less interesting are given by Howard Tooth, Lazarus Barlow, Caley, Coleman, Omerod, Targett, again by Hale White, and Ogle.

Note: As I have said above it is due to the character of the stools, to a very great extent, that we can diagnose ulcerative colitis. I have had personally a considerable experience of tropical dysentery and to a certain extent of the so-called English dysentery and find that we can distinguish dysentery from ulcerative colitis by the presence of the following in dysentery and necessarily by their absence in ulcerative colitis:

1. Convulsions and rigors at the onset (Mott disagrees with this.)
2. The incessant desire to go to stool.
3. Violent straining.
4. Patient passes little or nothing.
5. The evacuations consist of mucus and blood, with perhaps one or two scybala; pure pus appears towards the end of the illness.
6. The complete prostration and fainting at stool.
7. Occurring in epidemics: this, I think however, somewhat doubtful, as I have seen ulcerative colitis occurring in what might have been called an epidemic.
Mild cases of ulcerative colitis run a very similar course to those of acute cases, only that after a week or ten days the diarrhoea stops and the temperature becomes normal; many cases, however, become chronic and last a considerable time.

Personally, I am very doubtful as to whether true ulcerative colitis ever gets well.

We frequently find that with the colitis we have other diseases, which add to the complication of the original disease.

The commonest of all is implication of the lung due to pneumonia, croupous and catarrhal; we also often find a certain amount of congestion and oedema of the lungs, granular kidney, fistula ani, bed sores.

In my statistical table at the end of the thesis, the post mortem report in 50 cases which have died of ulcerative colitis, will be analysed, and the percentage of granular kidney, phthisis, &c. will be shewn.

I have never seen a case of metastatic abscess of the liver occurring in ulcerative colitis or as a result of ulcerative colitis.

I shall now clinical notes of nine cases of ulcerative colitis, all of which died except one; the notes refer chiefly to the condition of the bowels: temperature charts are attached.
A.M. Female, aet. 64.

On 8th inst. patient suffered from constipation with rise of temperature. No vomiting, no pain, pulse 120, tongue white and furred, enema of soap and water given; some small scyballoous masses evacuated; same evening diarrhoea set in, very watery.

9th: Diarrhoea almost continuous; rise of temperature; boric acid enema given, followed by one of opium and starch; brandy, milk, eggs, internally.

11th: No change; tr. opii given internally.

12th: No change; being fed up.

13th: Patient vomited; bowels very free, indeed motions consisting of water and slime and some blood.

14th, 15th, 16th, 17th, 18th, 19th: No material change on these days; diarrhoea extremely profuse.

20th: Patient died this day, temperature at death being 100.2

V. Chart 1., P.M. record No. 46.

J. J. Female, aet 75.

19th: Patient suffered from diarrhoea; tr. opii acid S. dil. given internally; no improvement.

20th: Temperature 101.2; passing blood per rectum/
rectum with water and slime; pulse 108; tongue furred; no lung mischief: brandy, milk, eggs given internally every two hours; some albumen in urine;

21st: Starch and opium enema given; no improvement; patient getting weaker; diarrhoea profuse.

22nd to 28th: No improvement.

29th: Patient died this day of exhaustion.

V. Chart 2, Post mortem record No.20.

J. F. Male. aet 80.

On 11th patient suffered from diarrhoea; temperature 100.8 at 6 a.m.; bowels open three times during 24 hours: enema of boric acid given; patient pale; complains of no pain; skin clammy; exhausted.

12th: Pulse 120, weak and thin; temperature varies; patient fed up on eggs and brandy; internally acid S, dil. and Tr. opii; diarrhoea now profuse; chiefly consisting of water, slime, and slight amount of blood; no vomiting; no tenesmus.

13th: Diarrhoea more profuse; temperature dropped suddenly at 10 o'clock to 97.2; hypodermic of strychnine given and warmth applied to body.

14th: Patient died this day; temperature at death 100.4; some albumen in urine.

V. Chart 3; Post mortem record No.4.
A. M. Male, aet 52.

4th inst.: patient complained of constipation at 6 a.m.; was given black draught; at 2 p.m. temperature 100; enema of soap and water given; some scybalous masses came away; at 10 p.m. temperature had risen to 100.6; diarrhoea came on profusely; motions consisting of water and slime with slight blood; no vomiting; no pain; no tenesmus; pulse normal in rate, but weak; brandy; beef tea, milk, eggs given internally; bismuth and salol given every four hours;

5th: Bowels open 13 times; pulse 108 weak, tongue brown and dry.

6th: Temperature dropped subnormal and there was a certain amount of collapse; strychnine and brandy hypodermically; warmth applied; bowels open 12 times.

7th: No change; temperature varies; bowels open 20 times; pulse 140; no albumen.

8th: Died this day; temperature at death 104.4. V. Chart 4, Post mortem record No.3.

J. B. Male, aet 52.

The patient, a fairly intelligent man, complained of pain in stomach and constipation at 9 a.m. on 10th inst.; was given a dose of salts; at 6 p.m. temperature 99.6; slight vomiting and pain intermittent/
mittent: no tenesmus: at this time enema of soap and water was given and gut cleansed out; patient was put to bed and a diet of cold boiled milk was given; bowels (somewhat diarrhoeaic in character) opened four times.

11th: Typical diarrhoea of ulcerative colitis, set in, bowels being opened 9 times in 24 hours; motions consist of blood (slight) water and slime; tongue red and furred: no pain; no tenesmus; pulse 100.

12th: Temperature rising, at 6 o'clock reached 101.4; bowels opened 14 times during the 24 hours; teaspoonful doses of saturated solution of Mag. Sulph. were administered internally every hour during waking hours; beef tea, brandy, eggs internally.

13th: Bowels open 15 times in 24 hours: temperature varies; same medicinal treatment; patient rather weak.

14th: Temperature falling; same medicinal treatment; appetite improving; bowels open 4 times in 24 hours.

15th: Temperature normal; bowels open once in 24 hours; same medicinal treatment.

16th: Bowels opened once, normal motion. From this date onward patient rapidly picked up and became convalescent. The Mag. Sulph. treatment is to be particularly noted in this case. V. chart 5.
C. S. Male, aet 65.

On 2nd patient suffered from typical ulcerative colitis; passing blood, slime and water; no pain; no tenesmus; pulse 104; tongue furred; bowels open 5 times; was given enema; bismuth and salol internally; brandy, Beef tea, and milk.

3rd: No improvement; temperature very little above normal; bowels open 12 times;

4th: Temperature rose to 99.6; bowels open 18 times; pulse 120.

5th: Bowels open 25 times; pulse 123; temperature subnormal; slight albumen.

From this day till 7th inst., on which day he died, pulse became more rapid and temperature subnormal.

V. Chart 6; post mortem record 2.

J. C. aet 77.

On 24th patient suffered from constipation, was given an enema.

25th: diarrhoea with blood appeared; temperature ran up to 101; pulse 120; bowels open 5 times; was given bismuth and salol internally; brandy and milk by mouth.

26th: Bowels, which are now of ulcerative colitis character, open 8 times; pulse 128; perchloride of mercury injection; no benefit; Salol/
Salol, gr. x., t.d.s.; no blood with motions.

27th: Temperature persists; bowels open 7 times; pulse 130; tongue becoming brown; patient feeble; given M. cretae amm. and tr. opii; no vomiting; no tenesmus; no blood with motions.

28th: Diarrhoea more frequent; bowels open 12 times with slight blood; being fed up; pulse 130.

29th and 30th: No improvement; patient died with temperature 101.4.

V. Chart 7, post mortem record 36.

M. A., female; age 86.

On 24th inst. she had diarrhoea with blood; was given one perchloride of mercury injection with no benefit; on this the bowels were open 5 times; pulse 80; was given milk, arrowroot and brandy; no tenesmus; no vomiting.

25th, 26th, 27th: The pulse varied considerably, but on these dates there was no blood: internally bismuth and tr. opii were given; motions consist of water and slime.

28th: Blood again appeared in motions, bowels open 14 times in 24 hours; pulse 94; was taking brandy, milk and eggs internally; slight albumen in urine.

29th: Pulse and temperature ran up and patient died.

V. Chart 8, post mortem record 35.
The patient had been suffering for the previous three weeks from diarrhoea with blood; there was no temperature; on 5th her temperature ran up to 99.8; nothing appeared to check the diarrhoea; the bowels on this date were open 11 times and became watery and slimy with a little blood: no pain; no vomiting; no tenesmus; Although he was fed up and various drugs were tried, he went from bad to worse and finally died on the 16th inst. from exhaustion, temperature being subnormal. V. chart 9; post mortem record No.6.

Morbid Anatomy:

In ulcerative colitis any part of the large intestine may be affected, but generally no particular distribution of the ulceration can be noticed. (36) Wilks and Moxon note that they have seen cases, attended by a discharge of mucus and blood, where after death, the whole internal surface of the colon presented a highly vascular, soft red surface, covered with tenacious mucus and adherent lymph and here and there shewing a few points of ulceration. Hale White (Allbut's System of Medicine29) notes a case in which in addition to the ulceration of the colon, there were ulcers in the vermiform appendix.

In/
In an extreme case the muscular coat is exposed and the ulceration is so extensive that only islets of mucus membrane are left here and there. Often these are considerably swollen and consequently they look taller than they otherwise would, and frequently they are more or less sessile because the ulceration undermines them. The result of this is that a careless observer concludes that the islets of mucous membrane are polypoid growths and that the exposed muscular coat is the level of the colon. The vessels of the mucous membrane are dilated and sometimes the membrane is black as though from long standing congestion. The muscular coat is hypertrophied. The large intestine is as a rule the only part of the gut affected, but this is not always so. Goodhart notes a case in which the large intestine from the ileocaecal valve downwards was extensively ulcerated, the ulceration running down the bowel in a linear manner, not destroying the mucous membrane in toto, but by small ulcers separated by tuberculae of mucous membrane.

"The intestinal ulcers are surrounded by haemorrhagic infiltration." (Stengel).

Ulceration may begin in the caecum and work downwards or it may begin in the rectum and work upwards, though the latter condition is rare.
The process of ulceration, according to Coleman begins by the mucous membrane becoming of a bright red colour, swollen and soft, then probably some suppuration, ulceration and sloughing occurs at various of the solitary follicles. At this stage the intestine would be dotted with small ulcers somewhat regularly placed. The ulceration spreads both laterally and deeply, the edges of the ulcers becoming undermined and their bases irregular and exposing the muscular and peritoneal coat. The latter may slough and become perforated. The thickness of the wall of the gut may be increased several times, this being caused by inflammation, oedema and infiltration.

"Generally at the autopsy no sloughs are to be seen, but in a few cases one or two ulcers have shewn a ragged surface, indicating that a slough has recently been detached, and if the patient has died early in the disease, they may be seen adherent to the ulcers." (Allbut's System of Medicine 29.)

In my post mortem cases I find that in one case sloughs were found adherent to the ulcers (v. Case 8)

It is extremely rare to see any attempt at repair. Case 11 in my post mortem records, under the heading "large intestine and appendix" shews that, from the caecum to the rectum, the gut is thickened, all over the mucosa are remains of old ulcers, healed and/
and healing; round these the gut is pigmented. Most of the ulcers occur near the caecum, and near this is also one firm large cicatrix; there was some cicatricial contraction near the ulcers.

In many cases the small intestine is congested but it is rare to find it actually ulcerated; in the 50 cases I have recorded I find that in eight only were there any ulcers in the small intestine, viz. cases 1, 13, 24, 27, 30, 31, 32, 46. In one case, however, case 39, it was found that the Peyer's patches were large and congested, but that no actual ulcers existed.

Sharkey (7) quotes a case in which the post mortem appearances shewed a large rent in the caecum and numerous small holes; the ulcers in this case were large and irregular and clean cut; the earlier ones were long and ran transversely across the bowel.

Cases are related by Robinson (22)
Hale White (31) and Bannatyne (16) who discusses the relationship between ulcerative colitis and granular kidney, and arrives at no results.

Cases are given by Lazarus Barlow (2) and Omerod (20) quotes an interesting case in which the gut was perforated for 5 inches in length, the hole involving quite/
quite half the circumference of the gut.

Targett\(^{(41)}\), Ogle\(^{(4)}\) Prickett\(^{(21)}\) Forth\(^{(1)}\) and Allchin\(^{(15)}\) all quote cases and give some post mortem results, all these cases being more or less similar; in many of these cases the ulceration was extensive and the destruction of the mucous membrane sometimes extraordinary.

I shall now give minute details of the macroscopic character of the intestines in 50 cases examined by me, all of which died of ulcerative colitis.

1. **W. G. N.**: Male, aet 37.

   **Small intestine**: congested; towards the caecum there was ulceration.

   **Large intestine and appendix**: extensive ulcerative colitis, leaving a few patches of the mucosa unaffected; the destruction involves nearly the whole of the large intestine.


   **Small intestine**: normal.

   **Large intestine and appendix**: the large intestine was adherent at the lower third of the transverse colon to a coil of the small gut at the point it was all but perforated, tearing easily on manipulation. The lower half of the large gut was in an advanced state of ulcerative colitis, beginning above the disseminated follicular glands and ending below in a much thickened condition of the gut, the mucosa of which was honeycombed throughout its whole length, with deep circular punched out ulcers with much congested bases, some of which run together; where the mucous membrane is left it is much congested; the last three inches of the rectum are not affected.
3. A. M., Male, aet 52

Small intestine: Normal.

Large intestine and appendix: There were ulcers beginning in the caecum (where the gut was not much thickened) and going down to the rectum; the gut was green and decomposing and the naked eye changed much obscured; so far as could be seen there was follicular ulceration, with thickening of the gut towards the lower end.

4. T. F., male, aet 80.

Small intestine: Normal.

Large intestine and appendix: The mucous membrane was congested and the follicular eroded in places; there were a few ulcers with necrotic patches; there was no thickening of the intestinal wall.

5. A. E. G., Male, aet 38.

Small intestine: Normal.

Large intestine and appendix: The caecum was covered over with small punched out ulcers, especially around the appendix and ileo-caecal valve.

6. S. W. Male, aet 55.

Small intestine: Normal.

Large intestine and appendix: There was advanced chronic ulcerative colitis; the mucosa was thickened and honeycombed from rectum to caecum; the epithelium had practically disappeared.

7. T. Z., Male, aet 67.

Small intestine: Normal.

Large intestine and appendix: There was extensive ulceration: numerous small punched out ulcers, increasing in number from above downwards, so that the lower gut (sigmoid and rectum) was honeycombed; the gut was thickened where the ulcers chiefly lay; there was no congestion.
8. J. B., Male, aet 65.

Small intestine: Normal.

Large intestine and appendix: The upper part of the sigmoid was free from disease; at the sigmoid were a large number of ulcers lying in the long axis of the gut, to which thickened yellowish slough was adherent. There was not much thickening of the mucosa, which was unaffected between the ulcers; there was no congestion; the edges of the ulcers were not thickened and were sharply cut off from the healthy mucosa around.

9. S. T. R. Male, aet 34.

Small intestine: Normal.

Large intestine and appendix: Very severe ulcerative colitis existed from the caecum to the rectum; the gut was honeycombed with ulcers and much thickened.


Small intestine: Normal.

Large intestine and appendix: Extensive ulceration from caecum to rectum; the ulcers were scattered above, but ran together below; some were covered with adherent sloughs.

11. J. S., Male, aet 50.

Small intestine: Normal.

Large intestine and appendix: From the caecum down to the rectum the gut was thickened, all over the mucosa were remains of old ulcers, healed and healing; round these the gut was pigmented; most of the ulcers occurred near the caecum; there was one large and firm cicatrix near the caecum and some cicatricial contraction near the ulcers.
12. W. K., male, aet 33.

Small intestine: normal.

Large intestine and appendix: Very congested; there were small shallow irregular ulcers in the sigmoid and rectum, with no particular distribution and scattered all over the mucosa; they were quite superficial and there was no thickening of the gut.

13. R. P., male, aet 45.

Small intestine: Many ulcers, great congestion.

Large intestine and appendix: There were a number of large ulcers in the gut, particularly in the caecum and upper part of the rectum; some thickening of gut.

14. W. W., male, aet 73.

Small intestine: Normal.

Large intestine and appendix: There was acute inflammation, haemorrhages and ulcers, the latter being more marked towards the lower end of the gut.

15. H. E. H., male, aet 36.

Small intestine: Normal.

Large intestine and appendix: There was ulcerative colitis with numerous ulcers and false membrane over the whole of the large intestine; the rectum was not affected and there was no thickening of the gut.


Small intestine: normal.

Large intestine and appendix: There were many small punched out ulcers found on the whole extent of the colon, from the rectum to the ileocaecal valve; they were shallow and lay transversely to the length of the colon, were non-perforating and scattered evenly over the whole surface; no noticeable thickening.
17. H. A. B., Female, aet 60.

Small intestine: normal.

Large intestine and appendix: At the lower part of the descending colon there was a rather extensive patch of ulceration and one or two small patches existed further up in the colon; the descending colon was very much dilated (probably by faeces.)


Small intestine: Normal.

Large intestine and appendix: From the ileo-caecal valve to the sigmoid flexure the colon was in a state of membranous ulceration; the ulcers were raised, rough and not well defined; there were small, deeply punched out stercoral ulcers with raised edges, in the sigmoid flexure.


Small intestine: Normal.

Large intestine and appendix: There were many small deep punched out ulcers in the transverse and descending colon; there were no ulcers in the ascending colon; gut somewhat thickened.

20. T. T., Female, aet 75.

Small intestine: Normal.

Large intestine and appendix: There was ulceration extending along the whole length of the large intestine; this more marked about the caecum than anywhere else.

21. P. F., Female, aet 68

Small intestine: Normal.

Large intestine and appendix: There were ulcers extending the whole length of the large intestine; they were more marked about the transverse colon, where there were patches through which the ulceration perforated the gut through its whole thickness.
22. E. B., Female, aet 60

Small intestine: Normal.

Large intestine and appendix: The rectum was extensively ulcerated and there were patches of congestion in the colon; the ulcers in rectum extended round the gut.

23. M. A. M, Female, aet 73.

Small intestine: Slight congestion at lower part.

Large intestine and appendix: The large intestine was thickened throughout; there were numerous patches of inflammation and on all these patches were ulcers scattered along the whole length.

24. E. S. H, Female, aet 37.

Small intestine: The mucous membrane was congested throughout; the duodenum showed a worm-eaten appearance and there was marked ulceration, not like typhoid, above the caecum.

Large intestine and appendix: The mucous membrane all through was congested; there were scattered round ulcers of fair size in the large gut; there were fair sized areas in the sigmoid flexure denuded of epithelium.

25. C. P., Female, aet 67.

Small intestine: Congested in region of caecum.

Large intestine and appendix: There were numerous scattered superficial ulcers in the caecum; there were somewhat older ones throughout the large intestine; the intestine was a good deal congested; near the rectum there were tiny ulcers scattered closely together all over the mucous membrane.
26. M. D., Female, aet 32.

Small intestine: Congested in parts.

Large intestine and appendix: There was a honeycombed appearance from ulceration especially marked in caecum, in the whole colon, and in the lower end of ileum; there were scattered around ulcers higher up. The ulcers varied in size from a millet seed to a threepenny bit. The average size was that of a split pea; the floors of the ulcers in the caecum were quite transparent.

27. M. A. C., Female, aet 42.

Small intestine: Was somewhat congested; there was early ulceration close to caecum.

Large intestine and appendix: Large gut was congested; the colon was reddish throughout; there was a somewhat honeycombed appearance in the caecum and rectum, and there were ulcers with shallow cavities about the size of millet seed.

28. A. D., Female, aet 44.

Small intestine: Normal.

Large intestine and appendix: The caecum was congested and there were signs of early ulceration; the large intestine became more ulcerated as we went downwards and it had a honeycombed appearance; there was a good deal of congestion and the ridges between the ulcerated surface in the upper part of rectum had a black, gangrenous appearance.

29. M. H., Female, aet 58.

Small intestine: Some congestion in places near caecum.

Large intestine and appendix: There were ulcers scattered throughout the large intestine, some the size of a threepenny piece of different shapes; there was some slight induration at the edges and some general congestion of the gut; in the caecum were granular and small more recent ulcers.
30. A. C., Female, aet 45.

Small Intestine: Congested for the last two feet, especially so in places where there was slight desquamation of epithelium and a few small ulcers.

Large intestine and appendix: There was extreme congestion throughout to within one foot of anus; there were many small ulcers scattered throughout the gut; there was much desquamation and sloughing of the epithelium.

31. S. S., aet 63. (Female)

Small intestine: adherent in two places to bladder; there was congestion with slight pitting and there were early ulcers just above the caecum.

Large intestine and appendix: The gut was intensely congested, the congestion being most marked just above the sigmoid flexure and rectum; there were bright red small elevations the size of large pin heads to hemp seed scattered about; the follicles were inflated; here and there were small ulcers; there was no thickening, but the gut was honeycombed.

32. L. M., Female, aet 30.

Small intestine: slightly congested in its lower half; a circular black gangrenous patch occupied the last two inches of the gut.

Large intestine and appendix: There were numerous small ulcers scattered over the surface of the large gut; the gut was somewhat thickened; there was only slight congestion of the surrounding tissue.

33. E. F., Female, aet 48.

Small intestine: normal.

Large intestine and appendix: In the lower part of the large intestine were three or four very large ulcers which extended
through the mucous membrane; higher up there were a few patches of congestion, but no distinct ulceration; the gut was slightly thickened in places.

34. C. A., Female, aet 54.

**Small intestine:** slightly congested in lower part.

**Large intestine and appendix:** The large gut was riddled in its whole length with small round ulcers; some of the ulcers formed deep pits with overhanging edges; some thickening of walls of gut in places.

35. M. A., Female, aet 86.

**Small Intestine:** Normal.

**Large intestine and appendix:** The vessels were much injected and the solitary follicles swollen; very many small ulcers were found in the rectum: the gut was thickened in places and in others translucent.

36. I. C., Female, aet 77.

**Small intestine:** normal.

**Large intestine and appendix:** The caecum was worm eaten and there was extensive ulceration near this part; the edges of the ulcers were much thickened; there were several small ulcers scattered throughout the large intestine; the rectum was worm eaten and had a false membrane over its ulcerated surfaces.

37. I. G., Female, aet 75.

**Small intestine:** Normal.
Large intestine and appendix: There was much congestion of transverse colon, descending to colon and rectum, with several small circular ulcers in their surfaces; the gut was thickened in places.

38. M. A. B., Female, aet 67.

Small intestine: Over the last six inches the mucous membrane is much congested and there are some points of extravasated blood, but no ulcers are visible.

Large intestine and appendix: There are extensive patches of ulceration along the caecum and all along the large intestine including the rectum: the patches of ulceration are arranged irregularly, the lymph follicles being swollen, but not ulcerated. The ulcers are irregular in shape and size, varying in size from a sixpenny bit to a half crown; the floor is for the most part formed of muscular coat, but in some of the patches it is formed of thickened peritoneum: around many of the ulcers are minute spots of extravasated blood; the edges of most of the ulcers are raised above the surrounding mucous membrane. To the naked eye there does not appear to be any attempt at healing. The wall of the caecum seems generally thickened where there is no ulceration.

39. S. B., Female, aet 55.

Small intestine: Congested towards caecum: the mucous membrane was almost destroyed in places, but there were no actual ulcers: Peyer's patches were large and congested.

Large intestine and appendix: The large gut was all congested; in the ascending colon there were several small ulcers; about three inches above the caecum they were irregularly distributed; all the large intestine was inflamed: the gut as a whole was thickened.
40. L. C. D., Female, aet 46.

**Small intestine:** Normal.

**Large intestine and appendix:** The coat of the large intestine was thickened and the mucus membrane much inflamed; there were many small ulcers; some punctured out, and small and regular in shape; others were irregular in shape; these changes extend over the whole large intestine and caecum.

41. T. R., Male, aet 74.

**Small intestine:** Normal.

**Large intestine and appendix:** There was advanced colitis, mostly at the rectal end and extending up to caecum: there was extreme honeycombing of the lower end of the gut and in the gut were scattered punched out ulcers with areas of healthy mucous between. There was slight thickening.

42. S. H., Male, aet 50.

**Small intestine:** Normal.

**Large intestine and appendix:** There was ulceration in the rectum apparently in the lymphoid patches with punctured out discrete ulcers in the large gut, corresponding to the lymph follicles: higher up in the caecum, these follicles are enlarged without ulceration; there was no appreciable thickening of the gut.

43. G. A., Male, aet 42.

**Small intestine:** Throughout small, there were occasional congested areas and there were numerous little white spots about the size of a pin head.
Large intestine and appendix: The large intestine was congested throughout, but no definite ulcers could be made out; coat of gut not thickened; some of the follicles very much swollen.

44. A. M., Female, aet 64.

Small intestine: Was ulcerated to within two feet of caecum.

Large intestine and appendix: The whole of large gut was covered with ulcers of various sizes, some regular, some irregular in shape; the gut was also covered to a certain extent with sloughy membrane, thickened in places.

45. E. C., Female, aet 63.

Small intestine: Normal.

Large intestine and appendix: From the caecum to the end of the rectum, the intestine was much thickened and its mucus membrane was almost entirely destroyed by ulceration, only bands of normal mucus membrane dividing one ulcer from another.

46. M. A. K., Female, aet 72.

Small intestine: Normal.

Large intestine and appendix: In the large gut were numerous punched out irregular ulcers with overhanging edges, varying in size from a pin head to a sixpenny piece; they were scattered throughout the whole length of the gut and extended down to the rectum: the gut was thickened in places.
47. H. S., Female, aet 30.

**Small intestine:** Normal.

**Large intestine and appendix:** Almost the whole length of the large gut was congested; about a foot above the rectum, there was a patch of ulceration about three inches long; the mucus membrane came away in sloughs; there was much thickening in places.

48. S. W., Female, aet 63.

**Small intestine:** Normal.

**Large intestine and appendix:** Slight superficial ulceration of rectum and lower part of colon; no perforation.

49. F. M., Female, aet 67.

**Small intestine:** above iléocaecal valve small intestine was congested.

**Large intestine and appendix:** Extensively ulcerated; mucus membrane greenish grey and very worm eaten; peritoneal surface, here and over large part of small intestine intensely congested and almost black.

50. A. W., Female, aet 65.

**Small intestine:** Normal.

**Large intestine and appendix:** From iléocaecal valve to rectum the large intestine was much inflamed and thickened. The mucus membrane was largely destroyed and the other parts presented a honeycombed appearance, but there were no separate ulcers.
Microscopical Examination of the Gut:

In a case given by Targett (41) the microscopical examination revealed the following changes in the wall of the colon: there were abundant inflammatory changes in the walls of the sub-mucous tissue and between the bases of the Lieberkuhn's follicles. At the edge of the ulcers the mucous membrane showed much catarrh of the lining epithelium of the tubules; their mouths were blocked and their acini distended with secretion; the lymphoid nodules were much swollen.

Coleman (10) in Guy's Hospital Gazette, gives the following microscopical changes in the case of ulcerative: the mucous and submucous layers were infiltrated with leucocytes, especially the submucous layer. It seemed that the infiltration took place along the lines of least resistance, such lines appearing to be the submucous layer and the solitary follicles, especially the lymph channels around the follicles; there was catarrh of Lieberkuhn's follicles, their orifices becoming choked and the glands distended with secretion.

In two cases of general ulcerative colitis read by Howard Teoth (1) a portion of the intestine was embedded in celloidin and cut transversely to the long axis of the bowel, stained in Ehrlich's haematoxylin: when there was no actual ulceration, the follicles of Lieberkuhn presented anatomical appearances/
appearances which might be produced by post mortem changes: their epithelium is very indistinct and the lumen is filled with fibrinous matter which takes the stain very deeply. In places the follicles are only half their length as if the tops had been rubbed off. Generally speaking, the bases of the follicles are in better condition than their mouths, the nuclei of the epithelium can be seen distinctly. The submucous layer is however three or four times its ordinary thickness, very vascular and generally infiltrated with leucocytes which invade the intervals between the follicles on the one hand, and the muscular coat on the other. Here and there may be seen an apparently natural gland. Where the ulceration is most extreme the internal muscular coat is bare and ragged, with a thin layer of fibrinous material, containing leucocytes in its meshes. The peritoneal coat is also thickened. In one place the small celled infiltration seemed to have led to complete disappearance of both muscular coats.

In the second case also given by the above writer, sections were made longitudinally at the most ulcerated parts of the bowel, so as to include two of the little polypoid tags which were all that remained of the mucous membrane. They appeared pedunculated/
pedunculated in shape like a mushroom: the stalk which consisted of inflammatory leucocytes, was in continuity with those lying on the denuded muscular coat all round it. The main mass of the tag is submucosa, much thickened, but it is coated with a perfect layer of Lieberkühn's follicles. Sections made through the less ulcerated portion of the bowel, the lower part of the descending colon, show another stage in the condition. Great swelling of the submucosa with multiplication of blood vessels and in many places haemorrhages, at the same time complete disappearance of the mucosa. This will explain the spongy swollen condition of the mucous membrane which is such a characteristic condition of the disease.

Microscopical examination supports the notion that the inflammation attacks the submucosa primarily, the mucosa being thrown off as a slough.

Mott (42), in a recent address to the Epidemiological Society of London, remarks that large mononuclear macrophage cells are found in sections of the colon: microscopical examination of the intestine reveals acute inflammation of the submucous coat, especially with engorged vessels and profuse infiltration which extends into the mucous membrane:
brane; already necrotic changes can be seen to have affected the epithelium, the cells of which are swollen, opaque, bursting or disintegrated. The scars of healed ulcers microscopically show nothing much beyond the wasting of the mucous follicles and increase of interstitial cells. The solitary follicles are frequently seen in these cases surrounded by pigment or a zone of redness.

Ogle in speaking of a case of ulceration of the colon associated with paraplegia, remarks that the vessels in the submucosa, especially those just beneath the mucosa, were engorged with blood. There was round cell infiltration in the muscularis mucosae, and the mucosa was full of extravasated blood and was in part necrotic and in other regions ulcerated away. Of the bacteriology of the disease little or nothing is known.

Kruse isolated a bacillus which he regarded as the cause of the disease; it closely resembled bacillus typhosus; Durham with Mott found no agglutinating reaction with the blood of dysenteric patients at the dilution of 1:100 with B.Typhosus or B.Enteritidis; he did not, however, use the B.Dysenteriae (N.B. In this case Mott believes ulcerative colitis and dysentery to be one and the same disease.)

Allchin found large numbers of bacteriae stainable/
stainable by Graves method and by alkaline methylene blue: many of these exhibit bulbous polar expansions, and some a central bulging as well.

Kruse in *Weitere Untersuchungen*, quoted by Flexner (34) examined material from twelve stools and two necropsies and comes to the following conclusion: "I was not in any case able to find bacilli possessing all the properties of dysenteric bacilli: on the other hand, I obtained from one stool and from both necropsies a bacillus whose morphological and cultural properties could not be distinguished from those of B. Dysenteriae; the only point of difference was found in the reaction of the specific dysentery - blood serum. The blood of the Asylum cases (11 out of 15) agglutinated the three strains of bacilli in dilution of 1-100 or more. But this blood had no effect upon the true dysenteric bacillus. Blood from an animal (sheep) immunised to the B. Dysenteriae had only slight effect on Bacilli of pseudo dysentery (1 to 50 and strong effect upon Bacilli of true dysentery 1 to 250) Contrariwise animals treated with the bacillus of true dysentery agglutinated these stains strongly without reacting with B. Dysenteriae. The conclusion drawn is that "in asylums a form of dysentery occurs that has nothing to do with true dysentery, but which probably/
probably owes its origin to similar microorganisms, of which there are several distinct types.

I must however regard the above conclusions as somewhat premature.

Flexner again has studied two cases of dysentery or ulcerative colitis which developed in the wards of the insane at Philadelphia Hospital and the blood of these patients gave positive results. Goodliffe of Lancaster Asylum, quoted by Gemmel, has isolated a bacillus which he believes to be the cause of ulcerative colitis; it is a short rod-shaped organism with rounded ends, usually from 1 to $2 \mu$ long and about $0.5 \mu$ broad. Often, however, it is almost as broad as long and is frequently seen in pairs, so that it looks like a diplococcus. It stains easily with Loeffler's aniline blue; it is not decolorised and only with difficulty by Gram's iodine solution and this forms one of its chief differences from B. Coli and B. typhosus. The B. Coli was frequently present in the stools and various bacilli were met with from time to time, closely resembling it, but differing in some mode of growth, etc., etc.

If ulcerative colitis is due to a specific organism, we have to determine whether it exists in the normal tissues and contents of the bowel or whether/
whether it is introduced from without. The B. Coli we know to exist normally in the healthy intestine and to it has been ascribed inflamed piles and osteomyelitis. The latter affection has been produced experimentally in rabbits, in which it also induced diarrhoea and collapse; this experiment I was unable personally to verify. If the above organism can cause these various diseases, it is quite possible that, when it attacks a part, the nervous energy of which is lowered, owing to disease of the central nervous system, it can take to itself a new and so to speak a malignant action and cause ulceration of the bowel; it is quite possible that in the insane and more particularly in the debilitating form of insanity, that the gut suffers from a certain amount of paresis and consequently there is a tendency for faeces to accumulate and dry up and so cause mechanical pressure; this mechanical pressure in time, no doubt, wears away the surface of the gut and so admits these various microorganisms, which eventually cause the ulceration.

Clay Shaw, in an address on Asylum Dysentery or Colitis says, "I believe that there is a degeneration of the mucous coats of the intestine due to nerve degeneration, and that as ulcer of the stomach occurs without apparent cause, so we may have ulceration/
ation of the intestine without its being septic in origin. It is not disputed that pathogenic organisms, micrococci and bacilli are generally present in these cases, but no specific germ has been isolated and it is certain that when once degeneration of tissue has set in, bacterial products are readily formed; and this is probably the order of events in the cases to which I am now referring, first degeneration, owing to lowered vital tone, and then the presence of bacteria due to this katabolic state. For this assertion we ought to be able to give some cogent reason. Unfortunately the investigation of the nerve distribution of the intestines is very difficult and tedious; I cannot adduce any actual observations to prove that there is actual nerve degeneration, but judging from the analogy of the tissues it is extremely probable that this is the case.

Mott(42) entirely disagrees with the above theory: the following facts according to Mott are entirely opposed to the theory that ulcerative colitis is due to nerve degeneration.

1. There is no essential difference between the lesions of the chronic ulcerative colitis one sees in the sane (which I should regard as chronic dysentery) and the sporadic cases of ulcerative colitis met with in asylums.

2./
2. If the condition were due to nerve lesion, there should be a proportionate relationship of sporadic cases among the insane of all asylums, which is not the case. According to my experiences, these sporadic cases are much more numerous in asylums where there has been previously an epidemic .......... Examination of the splanchnic nerves of the most severe cases of chronic dysentery I have ever seen, reveal no degeneration. It may be presumed that ulceration in this case was not associated with primary degeneration of nerves. I have also examined the sympathetic nerves in the mesentery, without discovering any recognisable change, although the patient had suffered from chronic dysentery.

3. One would expect intestinal lesions to occur in the tabetic form of General Paralysis if this theory were correct; I have recently examined a case of this kind .......... and yet the internal lining of the whole of the intestine was normal.

4. My experience coincides with that of Griesinger: that neither acute nor chronic dysentery appears to have any effect upon the mental symptoms of the insane."

Mott/
Mott in the above statement uses the term dysentery to what I call ulcerative colitis.

Out of the 50 post mortem records which I have collected of patients dying of ulcerative colitis, it will be noticed that 32 are females, the remainder, males; i.e., 64 per cent. are female. Again, looking at the deaths from ulcerative colitis during the last twelve months in 69 Asylums of Great Britain and Ireland, we find that out of a total of 137 deaths, 100 were females and 37 males, or 72 per cent. were females.

From the above statement it will be seen that a very much greater proportion of those dying from ulcerative colitis belong to the female than to the male sex. In normal sane human beings, women are known to be naturally more constipated than men, and this probably applies proportionately to them when they become insane, hence acting on the above theory (of a certain amount of stasis of the intestine - in the insane - and accumulation of hardened faeces, and consequent denudation of the mucous membrane) we naturally find that more women die of this disease than men. The above statement may seem rather far fetched, but still on examination it will be found to be true and to back up by theory as to the primary cause of ulcerative colitis.

As/
As I have stated above, I do not believe that ulcerative colitis is the same disease as dysentery: (Mott believes them to be one and the same disease) no doubt the resulting ulcers are similar and to a certain though mild extent, the symptoms, but this to my mind proves nothing beyond the fact that there is some agent at work which erodes the bowels and sets up the familiar stinking discharge from the intestinal canal: no definite bacillus has as yet been found as the cause of this disease, but undoubtedly B. Dysenteriae does not cause it. When a patient has died from ulcerative colitis with the disease and destruction of bowel not very advanced, we invariably find that the ulcers, that is to say, the commencement of ulceration, is found on the surface of the rugae of the large intestine, this being the part which would naturally be scraped away (as it were) by hardened masses of faeces. Constipation is often found to precede the diarrhoea, this being accompanied by a rise of temperature; this I always look upon as a danger signal and promptly empty the bowel with an enema. The temperature often falls and the danger is averted. If however, the state of constipation with fever is allowed to go on, diarrhoea generally appears and in the majority of cases the patient dies. By clearing out the scybalous masses from the intestine we hinder any rubbing action/
on the intestine and so mechanically prevent what would turn out to be an attack of ulcerative colitis.

**The Contagiousness or otherwise of Ulcerative Colitis:**

I believe it will be found that if one case of ulcerative colitis occurs in a ward, other cases are sure to follow; this to my mind is something beyond coincidence and shows the infectiousness of the disease. In spite of this, this belief does not clash with my theory of the disease, for one case is quite capable of developing microorganisms, which may be carried by air or by other means and settling on, so to speak, prepared surfaces, i.e. the worn gut of the patient, set up the above disease irrespective of drainage, etc.

Clay Shaw remarks that "if colitis is invariably infective disease, perhaps of the nature of swine fever, brought on by the defective hygienic conditions, and of necessity to be treated by isolation, then no words of opprobrium are too strong for its occurrence in an asylum, and it is possible that a legal action for damages might lie against the authorities for neglect of proper precautions, excluding of course, all cases of disease sent into the asylums. If however, ulceration of the intestine is found to occur in insane persons of a very degraded/
ed condition, when the Institution is practically free from diarrhoea and dysentery, if it is unfrequent and found in patients placed in different parts of the building, between whom there has been no communication, then it would seem that it is not of infective origin, and is only a part of the condition where a man is insane to the "tips of his fingers."

Mott\(^{42}\) denies, of course, entirely the assumption that ulcerative colitis, or as he calls it, dysentery, is a special disease of the insane; he grants the infectiousness of the disease and looks upon air as the means of conveyance, the drainage having nothing to do with it, but at Claybury Asylum some of the drains were found to be very defective. He however admits the fact that a patient can communicate the disease to another.

Mott and Durham\(^{23}\) in a combined report look upon this disease as one of the acute infective diseases due to a microorganism.

Epidemiological condition:

Consideration of various factors which might help the spread of infective disease within asylums:

1. Conveyance through air (dust, fomites, &c.)
2. Conveyance through ingestion of polluted water.
3. Conveyance by milk.
4. Conveyance by food.

A further classification according to another basis whereby the above is included in other ways:

Defective sanitary arrangements and precautions:
1. Unsound or otherwise imperfect drainage.
2. Want of cleanly surroundings.
3. Imperfectly regulated personal cleanliness.
4. Undue contact in persons as occurs in over-crowding.
5. Imperfect attempt to separate the sick from the healthy.
6. Imperfect routine of destruction and disinfection of articles which are known to be or which may have been contaminated.
7. Occupations which involve the hand being in contact with infected matter.

Acting on the belief of the microorganism theory of this disease, Mott and Durham believe that, though there is no definite evidence for the belief that the disease is carried by air, still they are inclined to believe in this mode of conveyance.

Milk, water, food, all in their turn were examined and found wanting, as far as the conveyance of the disease was concerned.

So far no definite result has been arrived at as to the cause or mode of conveyance of ulcerative colitis.

My own theory I have stated above: of course it may/
may be wrong, but having seen a great number of cases of this disease and of dysentery, and also having been medical officer to various workhouses (where no cases of this disease occurred) I still keep to my above theory as to the causation of this disease, and also to my belief that it is a disease chiefly of the insane.

Treatement:

Recommendations and Prophylaxis: All sanitary arrangements should be carefully examined into, drains tested and wards kept absolutely clean. All patients suffering from ulcerative colitis should be immediately isolated, special wards which should be frequently cleaned and fumigated being kept for this disease alone and for no other.

Any case of diarrhoea should be looked upon as a serious case and so treated. Patients who have recovered from ulcerative colitis should not be sent into any ward where ulcerative colitis has not previously existed.

Particular facilities should be given for attention and supervision of patients, as to their cleanliness. "Patients should be made to wash their hands before meals." (Mott & Durham 23).

All patients who might assist in laying out meals should be seen to cleanse their hands satisfactorily/
torily before being allowed to handle edibles, &c.

All patients who may be called in for ward duties (especially cleansing and changing other patients), should be seen to cleanse the hands in an efficient manner.

Clean and neat lavatories should be supplied and used.

All articles of clothing used by any patients who have suffered from any form of diarrhoea should be most carefully disinfected.

All beds used by patients suffering from any form of diarrhoea should be unpicked and carefully disinfected. No overcrowding should be allowed in Wards.

No patients (except those in padded rooms) should be allowed to sleep on the floor.

Padded rooms should be most carefully cleansed and disinfected, at least once in the 24 hours.

As far as possible, patients should be prevented from eating any form of foreign matter.

Treatment: The greatest essentials of treatment are rest in bed.

Food should consist of milk and soda water or peptonised milk; beef tea should not be given as a rule, in some cases however it is allowable.

Brandy/
Brandy should be freely administered when necessary and hot water bottles applied to the body and limbs are at times absolutely necessary.

Vomiting may be treated with M. Bismuthi and Morphia or with an effervescent hydrocyanic acid mixture.

Jellies, blancmanges, or some food (e.g. Benger's) is at times useful. Occasionally Dover's powder in 10 gr. doses, given every four hours or oftener will be found useful.

Ipecacuanha, Tr. Opii, Acid Sulph. dil., have been all tried in turn and as a rule found wanting.

At the beginning of the disease it is always advisable to give an enema of soap and water, and if the diarrhoea needs slight checking, an enema of starch and opium.

A saturated solution of Mag. Sulph. in teaspoonful doses every waking hour will be found most efficacious. This should not be given for more than two days running. (I have however given it oftener than this.) In the interval an enema of starch and opium may be given and the patient's strength kept up with milk, brandy, etc., etc.

An enema of silver nitrate gr. v. to 1 oz. I have found quite useless.

Enemas of boric acid will be found at times to be/
be very useful, especially when bismuth and salol are given internally.

Poultices applied to the abdomen will be found useful in relieving pain. Thirst should be relieved by a little lemon water. I believe also in letting the patient drink as much water as he likes, provided the water has been previously boiled and is not given too cold.

Mercury, internally seems to be of little use. Quinine given in 5 gr. doses every hour seems occasionally to do good. Venesection has been tried, but found wanting.

Ipecacuanha, in my experience, is quite useless: in true dysentery I have found it often acting almost as a specific.

To conclude, of the above drugs nothing seems really of any use except magnesium sulphate, which has given good results. To the best of my belief the best treatment is rest in bed, absolute cleanliness, maintenance of strength, and mag. sulph. internally, with every third day a boric acid enema.

Prickett (21) advises opiates at first with Ipecacuanha.

Prognosis:

Of the prognosis of this disease nothing very definite can be said. If the patient's strength is well/
well maintained and the temperature falls in a few days a good prognosis, as a rule, can be given.

At the end of this thesis will be found the bibliography used in its compilation. The names with numbers attached, refer to the numbers in the bibliography.

Statistical tables of various sorts will also be found at the end of this thesis.
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STATISTICAL TABLE.

A.
Analysis of 50 cases of Post Mortem Examination on those dying from Ulcerative Colitis. The following cases are not arranged in any particular order; but both male and female cases cover the same number of years.

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<tr>
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Males, 18.
Females 32.

Small intestine, congested, 16 cases, i.e. 32%.
Small intestine, ulcerated, 8 cases, i.e. 16%.
Tubercle of lung, both, 4 cases, i.e. 8%.
Tubercle of lung, one, 7 cases, i.e. 14%.
Lungs pneumonia or congestion, 6 cases, i.e. 12%.
Kidneys, granular contracted, both, 13 cases, i.e. 26%.
Kidneys, granular contracted, one, 6 cases, i.e. 12%.
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<th>Annul, athrombosis</th>
<th>Basal vessel</th>
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The following 34 Institutions are chosen as representative Asylums in Great Britain and Ireland. The asylums chosen are those which number as nearly as possible 1000 inmates.

**Analysis**

Total cases treated during the year in 34 Asylums .................................. 51,576.

Total number of deaths in the above Asylums from all causes (during the year) ........ 3,823

Total number of deaths from ulcerative colitis in these asylums in 12 months ........ 137

Percentage of deaths from ulcerative colitis compared with deaths from all causes ................................................................. 1 in 28.

Percentage of deaths from ulcerative colitis as regards all patients passing through the above asylums (in 12 months) 1 in 376.

Total number of females dying in the above Asylums from Ulcerative colitis ............ 100.

Total number of males dying from Ulcerative colitis in 12 months .......................... 37.
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<td>1901</td>
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<td>8</td>
<td>34</td>
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<td>2505</td>
<td>3063</td>
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<td>8</td>
<td>12</td>
<td>20</td>
<td>165</td>
<td>1425</td>
<td>1850</td>
<td>1901</td>
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<tr>
<td>Stann House, Ipswich</td>
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<td>10</td>
<td>43</td>
<td>700</td>
<td>862</td>
<td></td>
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<tr>
<td>Asylum</td>
<td>M</td>
<td>F</td>
<td>Total</td>
<td>Deaths from disease of body</td>
<td>Labour in Asylum</td>
<td>Labour in other</td>
<td>Year</td>
<td></td>
</tr>
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<td>-------------------</td>
<td>---</td>
<td>---</td>
<td>-------</td>
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<td>-----------------</td>
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<td>0</td>
<td>675</td>
<td>342</td>
<td></td>
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<td>0</td>
<td>0</td>
<td>970</td>
<td>1384</td>
<td></td>
<td>1900</td>
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<td>0</td>
<td>0</td>
<td>200</td>
<td>894</td>
<td></td>
<td>1900-1</td>
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<td>0</td>
<td>0</td>
<td>912</td>
<td>1206</td>
<td></td>
<td>1900</td>
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<tr>
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<td>708</td>
<td></td>
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<td>0</td>
<td>0</td>
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<td>881</td>
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<td>1900</td>
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<td>148</td>
<td>2141</td>
<td>2545</td>
<td>1900</td>
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<td>1</td>
<td>1</td>
<td>143</td>
<td>1562</td>
<td>1824</td>
<td>1900</td>
<td></td>
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<td>Limerick</td>
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<td>1</td>
<td>1</td>
<td>622</td>
<td>730</td>
<td></td>
<td>1900</td>
<td></td>
</tr>
</tbody>
</table>

Grand total: 137 3323 39400 51576
STATISTICAL TABLE

D.

(Dealing exclusively with Claybury Asylum)
Depraved New Year decline in death rate of patients (male & female) suffering from contagious illness.

Sept 14- Aug 31/99

Claybury Asylum

The diagram indicates the total number of deaths.
Red part indicates death in recurrent cases.
Brown part denotes new cases.

The increased death rate is due to isolation, disinfection.
A table showing relation of deaths with dysentery lesions, and the total death rate at Claybury Asylum.

<table>
<thead>
<tr>
<th></th>
<th>Sept 1st 1898 to Aug 31st 1899</th>
<th>Sept 1st 1899 to Aug 31st 1900</th>
<th>Sept 1st 1900 to Aug 31st 1901</th>
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<tbody>
<tr>
<td>Death</td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
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<tr>
<td>Total</td>
<td>116</td>
<td>14.4</td>
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<tr>
<td>Deaths</td>
<td>32.96</td>
<td>5.0</td>
<td>36.7</td>
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<td>4.7</td>
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<tr>
<td>Deaths</td>
<td>21.6</td>
<td>4.6</td>
<td>10.2</td>
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</tbody>
</table>

The above statistics probably represent the maximum, as the intestines were opened and carefully examined in every case. The dysentery lesion was not only to chronic or active process, which in a large number of cases was the actual cause of death, but also to chronic, partially healed or entirely healed processes, affecting the large intestine (rect).
Recor/s of Temperature, Pulse, Respiration, Stool/s and Urine from Day of ft/V\~Sf

In the case of fa fa

Ayed

Occupation 18

fiesu/t re.

Cent

Cent

Cent

Cent

In the case of a 29.1°

Records of Temperature, Pulse, Respiration, Stool and Urine From Aged 64

Day of Result

Result 18
Records of Temperature, Pulse, Respiration, Stools, and Urine from Day of In the case of

Aged 75. Occupation: [Redacted]. Day of
Records of Temperature, Pulse, Respiration, Stools, and Urine from Day of In the case of J S (To, Occ. M)
Records of Temperature, Pulse, Respiration, Stools and Urine from Day of In the case of J. Ayed

Occupation

Day of Result

18

Cent 42° 41° 39° 38° 37° 36° 35°

Time of Day

PM 11:00
Records of Temperature, Pulse, Respiration, Stools and Urine from Day of Occupation 18

In the case of aged 77
PMU

TeH:

Records of Temperature, Pulse, Respiration, Stools and Urine from Day of In the case of R-flM)

Aged 33. Occupation Kjpjtt

VT~

M°—

40s-

3flP-

267x577

>.8&

15x566

S5»-J

487x537

Time Dy

of Disease.

107°

106°

105°

103°

102°

101°

100°

99°

Norma/

98°

97°

96°

95°

94°

93°

92°

91°

90°

89°

88°

87°

86°

85°

84°

83°

82°

81°

80°

For


PMU

TeH:

Records of Temperature, Pulse, Respiration, Stools and Urine from Day of In the case of R-flM)

Aged 33. Occupation Kjpjtt

VT~

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91°

90°

89°

88°

87°

86°

85°

84°

83°

82°

81°

80°

For