Dysentery: A Clinical Study.

By

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INTRODUCTION.

The following thesis is based on the notes of 246 cases of dysentery observed in the wards of the general hospital at Port Elizabeth, one of the coast towns of South Africa, during the last four years. The term dysentery probably includes several forms of disease caused by different agents just as we have different forms of disease included under the term pneumonia, and though the essential clinical features of dysentery are everywhere the same, there are slight variations according to etiology, locality or other cause. The object of this thesis is to describe the features of dysentery, more especially from a clinical standpoint, as it occurs in South Africa and to compare them with those described as occurring in other countries where dysentery prevails.

Little has been written on dysentery in South Africa and most of the writers quoted in this thesis refer to dysentery as it occurs elsewhere. Owing to the interest caused by the occurrence of this disease among the troops in the present Anglo-Boer war, there are indications that this neglect of the study of South African dysentery will soon be repaired.

Special features in certain of our cases are referred to in the course of this thesis, and
the cases quoted in more detail on pp. thirty to sixty-four, include typical cases of the different varieties of the disease and most of the cases on which autopsies were held.
DEFINITION.

Dysentery may be defined from a clinical or a pathological standpoint. In the former sense, as the name implies, it indicates a disease or group of diseases characterized by the passage of frequent stools consisting of blood and mucus, and accompanied by pain and tenesmus.

From the pathological point of view dysentery is a disease or group of diseases characterized by inflammation and ulceration or sloughing of the mucous membrane of the large and occasionally of the small intestine. No definition of dysentery can however be regarded as perfect which does not take into account the etiological factor, and it is unfortunately impossible at present to bring all cases of dysentery under one head as regards cause. The various forms of dysentery may be similarly classified clinically as acute or chronic; pathologically as catarrhal, diphtheritic or gangrenous; and as regards incidence as epidemic or sporadic.

In South Africa epidemic dysentery is unknown, but the disease along the coast line is practically endemic.

The commonest variety found is acute catarrhal dysentery, though cases of the other varieties do occur as shewn in the following pages.
ETIOLOGY.

Considerable difficulty occurred in tracing our cases to their predisposing cause or causes. This was due to the fact that the cases occurred sporadically and not in epidemic form, and in the case of native patients it is almost an impossible matter to obtain a reliable history. However, in many of them the history pointed to a definite cause or starting point of the disease.

The season of greatest prevalence appears to be in the late summer or autumn which corresponds with the experience of Osler in America. Davidson on the other hand states that "Dysentery, in India, as a whole, is a disease of the cold season."

The following table shows the average number of cases per month treated by us during the last four years:

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<tr>
<th>Month</th>
<th>Jan</th>
<th>Feb</th>
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<th>May</th>
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<tbody>
<tr>
<td>Cases</td>
<td>5.75</td>
<td>4.5</td>
<td>6.25</td>
<td>9</td>
<td>9.25</td>
<td>5.5</td>
<td>2.75</td>
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<tr>
<th>Month</th>
<th>Sep</th>
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<tbody>
<tr>
<td>Cases</td>
<td>5.75</td>
<td>2</td>
<td>3</td>
<td>7</td>
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Though these numbers are too small to draw definite conclusions from, they nevertheless

1 Osler. The principles and practice of Medicine, 1897, p. 145.
2 Davidson. Hygiene and Diseases of warm Climates, 1895, p. 555.
confirm the general experience of this part of South Africa, that dysentery is most prevalent in the summer and autumn months (December to May) and least during the winter and spring months (June to November).

A commonly held view is that the distribution of malaria and of dysentery is geographically the same and it is held that this is more than a mere coincidence. That these diseases do not invariably go hand in hand is indicated by the fact that though dysentery is an endemic and fairly prevalent disease along the coast line of Cape Colony, malaria is unknown except in cases where it has been contracted in the "fever districts" many miles inland.

Davidson\(^3\) states that malaria is unknown in the island of Rodriguez while dysentery accounts for 29.6 per cent of the total mortality.

However malaria by causing anaemia may act as a predisposing cause, as may any other exhausting or debilitating disease. We have occasionally seen acute dysentery supervene on scurvy where post-mortem the typical lesions of dysentery were discovered. Similarly we have seen dysentery occur in the course of other exhausting diseases, e.g. once in the

3 Davidson. op. cit. p. 555.
course of a case of alcoholic peripheral neuritis, once during the later stages of enteric fever, once following operation for elephantiasis of the penis and twice during prolonged suppuration after severe burns of the leg. In one of the latter, the patient was also suffering from cystitis and pyelitis due to bilharzia haematobia. In all of these cases except one the diagnosis was verified by an autopsy. Buchanan describes a form of the disease which he calls "terminal" dysentery, as occurring among prisoners in the Indian gaols, who are the subjects of some chronic disease. He quotes a number of cases where patients were suffering from some chronic illness in the course of which dysentery of a gangrenous or more rarely of an acute type supervened and hastened the fatal termination.

In the dysentery of British asylums and workhouses it is more frequently the debilitated and feeble who are attacked by dysentery.

Closely associated with a debilitated condition of the system due to exhausting disease as a predisposing cause of dysentery, is bodily exhaustion.

from protracted physical labour or mental worry. An instance is afforded in case \( \text{III} \). The patient who had recently arrived in the country was suddenly compelled to serve long hours in a newspaper office after being accustomed to regular and shorter hours in a business office.

Almost inseparably associated with bodily or mental fatigue as predisposing to an attack of dysentery is exposure to the vicissitudes of the weather. In case \( \text{IV} \) exposure appears to have had a direct bearing in this respect, and we believe that exposure and fatigue have had a potent influence in predisposing to dysentery among troops in the field (v. Cases \( \text{VI} \& \text{VII} \)).

In South Africa dysentery is most prevalent during the late summer and the autumn months—the season when there is the greatest fluctuation in the temperature, the days being hot with a sudden change to cold at night.

A most potent predisposing cause is alcoholism. This, by causing cirrhosis of the liver and consequent venous congestion of the intestinal tract, may result in a passage of blood in the stool; but apart from this it causes congestion and irritation of the intestines and especially of the large, which readily passes on to genuine dysentery. Under the heading of treatment we note that alcohol should be avoided as
a therapeutic agent in dysentery as it appears to
directly aggravate the complaint. In addition to
its inducing a condition of congestion—the first
stage of inflammation—of the intestinal tract, free
indulgence in alcohol renders the individual more care¬
less with regard to exposure or chills and at the same
time more susceptible to their influence. Of our cases
fourteen may be directly traced to this cause and of
these several have been treated during repeated
attacks traceable in each instance to this cause.

All ages, sexes and nationalities seem to be
equally liable to the disease. Among our cases there
were European, males 125, females 29; Native, males
63, females 29. When compared with the total
admissions into hospital these figures shew a slightly
higher ratio of male patients to female, while the ratio
between Europeans and Natives is about equal. The ages
of the patients ranged from seven months to seventy¬
five years.

Among the European cases, especially among
the male, we have been struck with the large proportion
of recent arrivals from Europe compared with native
born Europeans. Cases I, II & III had only been a few
weeks in the country before contracting dysentery. Our
experience both in dysentery and in enteric fever seems
to indicate that new arrivals from Europe are more susceptible to these diseases than those who have been in the country some years. This is probably due partly to the new arrival being unused to the altered climatic and other conditions and partly to his ignorance of the risks arising from impure water or contaminated food of which the older resident is well aware.

Impure water is undoubtedly an important predisposing cause to the disease, and acts by causing an inflamed condition of the intestinal mucous membrane or by directly acting as a means of conveyance of the infective agent; but it cannot be regarded as per se sufficient to cause dysentery. During eighteen months' experience of practice in an inland district in South Africa where no steps were taken to insure purity of water the town's supply and where in the country surrounding all water was obtained from artificial "dams" open to all varieties of contamination, we did not see a single case of dysentery, nor hear of one occurring; while in a coast town where the water supply is above suspicion we have seen on the average sixty cases per annum.

Where large bodies of men live together on a restricted area, as in camps, there is a tendency for dysentery to develop. A number of our cases came from railway construction camps and from ships. Frequently small epidemics break out in these camps or
on board a vessel.

Previous constipation, simple diarrhoea or any other abnormal condition of the intestine not infrequently is a precursor of dysentery.

With regard to the exciting cause of dysentery many authors have striven to shew that it is due to some parasite—coccal, bacterial or protozoon. In 1859 Lambl described the presence of amoebae in the stools of a child who had died of enteritis, but he attached no importance to their presence.

Woodward in his medical history of the American civil war described the presence of cocci arranged in chains occurring in the diseased intestine; but regarded this not as having any specific significance, but rather as secondary infection of the diseased tissues.

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5 Lambl. Aus dem Franz Joseph Kinderspitale. Bd. l., s. 363
Schafer, Prior and Petrone also describe the presence of coccii in dysenteric stools.

Besser attempted to produce dysentery artificially during a severe epidemic in Dorpat. The examination of the stools from his patients gave a negative result, and the injection of these stools into the recta of healthy dogs failed to cause dysentery. But if the rectum of the animal experimented on was previously irritated by the injection of a 9 per cent solution of ammonia, introduction of dysenteric stools caused a diphtheritic inflammation of the large intestine. Better results were obtained where cultures in liquid media were used on cats--animals which are peculiarly susceptible to dysentery. Cultures of the blood of dysenteric patients in bouillon and of stools in mud, when injected invariably caused a diphtheritic inflammation of the large intestine. In the cultures and in the diseased portions of the intestine coccii were found, and Besser considered these as the cause of the disease.

Other authors describe a bacillus as the

Schaefer. Ueber Diptheric des Darmes. Würzburg 1887
8 Prior. Centralblatt für Klinische medecin. 1885
9. Petrone. Lo sperimentale. May 1884
10 Besser. Experimentaller Beitrag Zur Kenntiss der Ruhe. Dorpat 1884.
Condorelli-Mangeri and Aradas found a bacillus in the faeces, but not in the blood or in the intestines of several patients during an epidemic. They found the same bacillus in the water of a well used by several of the patients and they infer from this that this bacillus is the cause of dysentery. Klebs has found a short bacillus which grows on gelatine plates in small non-lipid-fying colonies. Negative results were given by experiments with this bacillus on dogs and rabbits.

Ogata examined the faeces and tissues of several cases of dysentery in Japan. In all the cases cultures from the faeces on gelatine plates, gave a short bacillus which stained by Gram's method. This micro-organism reproduced the disease with similar ulcerative processes in the large intestine to those found in his autopsies, when injected into the recta of mice, guineapigs and cats.

Lately Flexner has described a short rod with rounded ends possessed of flagellae and feebly motile, which he considers the cause of dysentery. In

various media he has succeeded in isolating the bacillus from the stools of cases of dysentery from Porto Rico, Manilla and America, and he believes this microorganism is the cause of dysentery as it occurs in the Far East and America and identifies it as the same bacillus as that described by Kruse in his cases of dysentery in Germany. He found that blood serum from convalescent dysenterics from Porto Rico, Manilla, California, Philadelphia and from immunised animals, gave positive agglutinating results with his bacillus. Kruse in Germany and Shiga in Japan had previously described a similar bacillus during epidemics of dysentery in these countries, which also gave positive agglutinating results with the serum of convalescent patients and Flexner considers these bacilli to be identical with the one he describes.

Before bacilli were described as the cause of dysentery, Lôsch found a large number of amoebae in the stools of a patient suffering from dysentery. He found the amoebae in the mucus of the stools. They measured from five to eight times the diameter of the red blood corpuscles, and moved by means of blunt processes, which were never pointed or sharp. He found

15. Lôsch. Archiv für path. Anatomie 1875, Bd. 65, S.196
that the severity of the symptoms were proportionate to the number of the amoebae in the stools, and as the number of the amoebae diminished so the symptoms improved. The patient died as the result of pneumonia and no amoebae were found in the intestine. Läsch injected stools containing amoebae into the recta of four dogs. Of these only one became infected and amoebae were found plentifully in the stools. The dog was killed and congestion and superficial ulceration of the rectum was found, on which amoebae were present.

Koch found in four cases of dysentery, amoebae on the ulcers of the intestine and in sections of these. He describes them as \(1\frac{1}{2}\) to 2 times the size of white blood corpuscles.

Kartulis of Alexandria, as the result of his experience of several hundred cases in Egypt, states that he has found amoebae in every case, not only in the stools but also confirms Koch’s observation of them in dysenteric ulcers. He goes further and states that "in no case other than dysentery did I find amoebae."

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"I found amoebae in sections of the intestine of twelve cases which died of dysentery. In thirty control cases (enteric fever, phthisis, biliary typhoid, bilharzia) where the intestines were eroded, I found no amoebae. Similarly in a few cured cases of dysentery, in which the ulcers were cicatrized amoebae could no longer be found."

His description of the amoebae corresponds with that of Lösch as far as structure and movement are concerned, but they were somewhat smaller. He found similar amoebae in liver abscesses following dysentery, but not in the so-called "idiopathic liver abscesses." At first he was not successful in inoculating animals but later he succeeded in reproducing the disease with the presence of amoebae and superficial ulcers of the large intestine in the case of cats.

Kartulis also states that he has succeeded in making a pure culture of amoebae in an infusion of straw, and by the injection of this pure culture into the rectum in the case of cats, that he has produced dysentery in the same manner as by injection of the stools of patients suffering from dysentery.

Egypt, Osler and Councilman and Lafleur in America have found amoebae in the stools of dysenteric patients and in the contents of secondary liver abscesses, and generally support the conclusions of Kartulis.

Howard found amoebae in the stools of a case of chronic dysentery in England. The patient had contracted the disease in India.

He states that Manson has found amoebae also in England, in two cases of dysentery contracted in India. None were found in the pus from the secondary liver abscesses.

Councilman and Lafleur found the amoebae in the mucus of the stools and in the pus from secondary abscesses of the liver and lung. They state that they vary in appearance according as they are active or inactive. In the resting condition they are round or slightly oblong and are more refractive than the other cells which may be found in the faeces. Frequently no division into an ecto- and an endo-sarc can be seen, but simply a body enclosing vacuoles of a varying size. These vacuoles are clear and vary greatly in size, when small giving the amoeba a granular appearance.

In other cases there is a sharp division between the outer, pale, hyaline or homogeneous ectosarc and the inner more refractive endosarc which contains the vacuoles. When moving, there is great variation in the degree of their activity, the movements consisting of a progressive movement or of a protrusion and retraction of pseudopodia which consist of the same homogeneous structure as the ectosarc. These movements are greatly accelerated when the specimen is examined on the warm stage. The amoebae were often seen to contain various foreign bodies such as red blood corpuscles or blood pigment, pus cells in various stages of disorganization and bacilli and micrococci. The nucleus cannot be definitely made out when the examination is made in the fresh state, and in this view Councilman and Lafleur differ from other writers. They found amoebae in fourteen cases of dysentery in America and believe that a distinct clinical and pathological form of the disease—amoebic dysentery—exists, which differs from other varieties—catarrhal, diphtheritic &c.—in which the amoeba is absent.

We cannot accept the view that the amoebae are the cause of dysentery in all cases, or even in all cases of so-called "tropical dysentery."
Osler states that though amoebae have been found on the ulcerated mucous membrane, in the wall of the bowel, and after perforation of the bowel, in the peritoneal cavity, their etiological connection, he does not consider proved. He is inclined to think that these organisms are associated with a special form of dysentery.

Kartulis writing on Egyptian dysentery, as stated above, is most positive in his view that amoebae are the only cause and that they are found in no other disease. But other authors describing sporadic cases especially in Egypt and Italy—Celli and Fiocca—have failed to find the amoeba.

Gasser in Algiers examined 109 cases and found amoebae in varying numbers, in 41.3 per cent of acute and 38.2 per cent of chronic cases. There appeared to be no relation between the number of amoebae and the severity of the symptoms. They were found in small numbers in two fatal cases and abundantly in cases which ran a favourable course. In one fatal case with widespread ulceration of the intestine no amoebae were found either ante- or post-mortem. At the same time he found amoebae in 20 per cent of healthy individuals examined.

26. Gasser. Archiv. de med. exp. et d'anat. path. 1895, No. 2. (quoted by Scheube)
Kruse and Pasquale in their exhaustive investigations on dysentery in Egypt failed to find the amoeba in ten out of fifty cases of dysentery examined. This they ascribe to the protracted history of the cases and to previous treatment. On the other hand they found amoebae in the stools of healthy individuals in Europe, though in small numbers.

Other authors have found amoebae differing in no appreciable respect in size or appearance from the so-called "amoeba dysenteriae" in healthy individuals who have never suffered from dysentery.

Cunningham in India found amoebae in the stools of patients suffering from cholera.

In the "Proceedings of the Philadelphia Pathological Society" Dr. Joseph T. Buxton records an interesting case, in which pus from a case of liver abscesses contained amoebae while the intestine shewed no sign of dysentery at the necropsy.

We find, therefore, that though Kartulis states that he has found amoebae in all cases of dysentery and in no other condition, other writers have found them in only a certain proportion of their cases.

27. Cunningham. Sanitary report on cholera to the Govt. of India, 1870. Quoted by Councilman and Lafleur.

of dysentery, while others again have found them in other diseased conditions of the intestinal tract, and in the stools of healthy individuals.

Nor does the inoculation of animals with dysenteric stools (Kartulis, Kruse and Pasquale and others) resulting in dysentery with reproduction of living amoebae, seem to prove that the amoebae are the cause of dysentery. Kartulis, however, believes that he has made a pure culture of the amoeba, but his experiments are incomplete, and it must be remembered that amoebae themselves contain bacilli or micrococci, and these may have some influence in producing the disease.

In most of the cases of inoculation experiments dysenteric stools or pus from the liver abscesses have been used for injection into the recta of dogs, cats etc., and this infective material always contains other micro-organisms in addition to amoebae.

Further, Celli and Fiocca by means of heat, killed the amoebae in dysenteric stools, but injection of this material caused dysentery in cats.

Zancarol injected dysenteric stools and pus from liver abscesses which in both cases contained no amoebae, and also pure cultures of streptococci into the recta of cats, and produced dysentery in each case.

He also used stools containing amoebae and produced dysentery in does without reproduction of amoebae.

Petridis\textsuperscript{30} surgeon and bacteriologist to the Greek hospital in Alexandria, believes that neither dysentery nor liver abscess is caused by amoebae. He finds that dysentery can be artificially produced in kittens by injecting directly into the rectum (1) human liver pus whether sterilised or not and with or without amoebae; (2) human dysenteric stools, with or without amoebae, sterilised or not; (3) a culture of streptococci; or (4) pus from any abscess.

In regard to South African dysentery we have only been able to find five articles on the subject. These deal chiefly with the question of treatment, and are referred to later. In two the question of the amoeba was referred to. Richmond\textsuperscript{31} says "the amoeba was carefully searched for in many cases of dysentery at Deelfontein but was never once found." "It was searched for in all cases which were treated with sulphur but was never found." Washbourn\textsuperscript{32} states that at the Imperial Yeomanry Hospital, Deelfontein, he has "examined the stools of a number of cases (of dysentery) microscopically using a warm stage, but has failed to discover amoebae".

\textsuperscript{31} Richmond. The Lancet. June:15:1901, p. 1677
This corresponds with our own experience. During the last four years we have searched for the amoebae in the stools, pus from the liver and lung abscesses and in the discharge on the surface of dysenteric ulcers, and though we have not searched for them in all of our 246 cases we have done so in a large proportion of them. Our method in the case of stools is to obtain a certain amount in a warm gallipot immediately after the stool has been passed, pick out the small mucus pieces and examine them on a warm stage with a 1/6th inch objective. The result, as far as amoebae are concerned has invariably been negative. In one instance on examining a fresh stool we discovered three bodies, granular throughout and without a definite nucleus. These were fully twice the size of a white blood corpuscle but were non-motile, though examined fresh on a warm stage.

Though dysentery is probably due to an infective agent of parasitic origin, we do, not yet know the true cause; probably more than one cause exists. The presence of the amoebae in such large numbers in cases of dysentery in other countries is probably due to their finding a suitable soil for their growth in the diseased intestines. The presence of the cocci, bacilli and amoebae described is probably only secondary to the diseased processes, and though not directly the cause of dysentery, their presence in large numbers may very well aggravate the disease.
OUR NOTES ON THE PATHOLOGY OF DYSENTERY ARE BASED ON POST-MORTEM EXAMINATION OF SEVENTEEN CASES, ELEVEN OF WHICH ARE FULLY REPORTED ON PP. 30 TO 64. OF THESE CASES THREE WERE ACUTE, THREE GANGRENOUS AND THE REMAINDER CHRONIC.

THREE OF THESE AUTOPSIES INDICATE THAT THE PRIMARY LESION IN ALL CASES IS IN THE SOLITARY GLANDS OF THE LARGE INTESTINE, AND THAT IN CASES WHERE THE SUBMUCOSA, MUSCULAR AND PERITONEAL COATS ARE DESTROYED, THIS REPRESENTS AN EXTENSION OF THE DISEASE FROM THE MUCOUS COAT. ON OPENING THE ABDOMINAL CAVITY IN CHRONIC CASES THERE IS FOUND MARKED ABSENCE OF FAT IN THE ABDOMINAL WALLS, WHICH ARE THIN AND THE TISSUES DRY. A SIMILAR ABSENCE OF FAT IS NOTICED ON EXAMINING THE GREAT OMENTUM. THE PERITONEUM HAS LOST ITS MOIST GLOSSY APPEARANCE AND IS DULL AND DRY AND STICKY TO THE TOUCH. ITS VISCERAL LAYERS MAY BE CONGESTED AND REDDISH IN PATCHES OVER THE LARGE INTESTINE AND LOWER PART OF THE ILLIUM. IN CASES OF GANGRENOUS DYSENTERY THE PERITONEAL COAT IS DARK ALMOST BLACK IN APPEARANCE.

THE COILS OF THE SMALL INTESTINE MAY BE LOOSELY ADHERENT TO ONE ANOTHER, AND IN ONE CASE ADHESIVE BANDS EXISTED BETWEEN COLON AND LIVER.

IN TWO CASES PERFORATION WAS DISCOVERED.
In four of our cases there was general peritonitis—in the three cases of the gangrenous form, and the fourth was the case of an adult European admitted as suffering from general peritonitis. He died on the day after admission. Post-mortem examination showed dysenteric ulcers in the large intestine and two abscesses in the liver, the smaller of which had ruptured into the peritoneal cavity.

In two of our cases of chronic dysentery there was great distension of the large intestine with thinning of the walls. This, *intra vitam*, gave rise to distressing meteorism, and in two other cases there was great distension of the stomach, the organ reaching to the pelvic brim.

Usually, however, the intestines are found empty and smaller in calibre. At times the colon is found shrunk to less than the size of the small intestine. In about one third of the cases the wall of the large intestine was found greatly thickened. This was especially marked in the rectum where the thickness of the wall reached from one quarter to one third of an inch. The diminution in the calibre of the large intestine may be general or may be found only in parts with the intervening portions of normal size.
or slightly distended. The vermiform appendix was found in two cases to be very rudimentary, measuring half an inch in length.

On opening the intestine the typical lesions are found. These occur most frequently and in largest number in the rectum and caecum. In one case (Case 9) complicated by liver abscesses the only ulcers found were in the caecum. Next in frequency are the flexures (sigmoid, splenic and hepatic) of the large intestine. In three out of seventeen autopsies (Cases 14, 15 and 16) ulcers were found in the ileum as well as in the large intestine, extending in one case to three feet above the ileo-caecal valve. The sites of the ulcers in the large intestine appear to be, as pointed out, precisely those in which faecal masses are likely to accumulate. The nature of the diseased process appears to be essentially the same in all cases and varies only in degree. The earliest stage is congestion of the mucous membrane. This is thrown into folds and on the summit of these it is congested and swollen in appearance. This congestion is usually found in patches and not generally over the mucous membrane. Occasionally little ecchymoses are found in the mucous membrane. The
rest of the mucous membrane appears grey, whitish or slate coloured, is swollen, soft and oedematous and covered with adherent glairy mucus. We frequently found small patches of congestion scattered over the mucous membrane of the lower part of the ilium even in those cases where no ulceration occurred here.

The solitary follicles are found swollen and prominent and from them a small quantity of mucus can be squeezed. The ulcers vary greatly in size, number and extent. In one case which developed liver abscess the ulcers were superficial and did not extend through the mucous membrane. In these cases, should healing take place, no cicatrix forms.

Usually, however, the ulcers extend through the mucous and submucous coats and the bases are formed of the muscular coat. The form, size the ulcers vary greatly. Generally speaking they assume a more or less circular shape, though when two or more run together the shape may be irregular. In almost all cases small ulcers about the size of a pea are found along with others the size of a shilling or florin. Occasionally they may be 1½ to 2 inches or more in length, and at times we have seen the mucous membrane of portions of the intestine (rectum or caecum) almost
entirely denuded of mucous membrane, only small islands occurring here and there on the otherwise denuded surface.

The commonest form of ulcer found is the one about the size of a shilling to a florin, just mentioned. The edges are swollen rounded, smooth and oedematous, or congested. They do not shelve, but extend abruptly to the base of the ulcer as if the latter had been punched out of the mucous membrane. The base of the ulcer is usually formed of the muscular coat of the intestine, though it may be represented by any of the other coats according to the depth of the ulcer. It is covered with greyish mucus or bile muco-pus. Sometimes this is stained, yellowish or greenish, and contains in addition debris, small faecal lumps or undigested food particles and occasionally dark clotted blood. The mucous membrane between the ulcers may be congested in places and swollen, or whitish or grey and atrophied. We did not see any ulcer that appeared to be healing, but occasionally found smooth cicatrices in which no glandular structure was present.

Councilman and Lafleur, and Kruse and Pasquale in their works previously quoted on "amoebic dysentery", and other earlier authors describe the submucous coat as the starting point of the disease. The submucous
coat is infected by the amoeba which causes small areas of infiltration. These break down and give rise to the production of small abscesses. The mucous membrane they state is only secondarily affected, and not so extensively as the former, so that by extension of the process in the submucosa, large areas of mucous membrane become separated with here and there fistulous openings between the affected portions of the submucosa and the lumen of the bowel.

Among our cases we did not find this condition in a single instance, though special care was devoted to the point. We did not find a single ulcer with undermined edges, or a diseased area in the submucosa with two or more openings between it and the lumen of the bowel. The edges of the ulcers in our cases were always sharply defined and would not admit of a probe being passed beneath them.

In this respect our cases differed markedly from those described in India, Egypt and America.

Microscopically there is small round-celled infiltration in the solitary glands and between Lieberkühn's follicles. The surface of the mucous membrane shews catarrh and in places, active destruction of the epithelium with fibrinous exudation. This is also seen in the deeper tissues. The base of the ulcer consists of altered mucous membrane or muscular tissue,
according to its depth.

Closely associated with changes in the intestinal tract in dysentery, are the changes in the mesenteric glands. We found these invariably enlarged. Sometimes they were congested and only slightly enlarged, occasionally they were the size of a cherry or larger and caseous.

The liver shewed various changes. It was cirrhotic in one case, waxy in two cases of chronic dysentery, while at other times it was pale and atrophic, occasionally enlarged, firm, shewing chronic venous congestion. In three cases the gallbladder contained no bile, but was greatly distended with thin clear mucus. In 8 of the 17 autopsies hepatic abscesses were found. These are referred to again under the heading of complications.

The spleen shewed nothing characteristic of the disease. It was usually somewhat congested. The kidneys were sometimes pale and under the usual size. In two cases they were cirrhotic and in one there was pus in the kidney substance and in the pelvis of the ureter.

The lungs usually shewed congestion and oedema, occasionally old or recent pleurisy. In one case there
was a large abscess of the left lung. The heart
and brain shewed nothing characteristic.
CASES.

Case 1.—Acute dysentery treated by rectal irrigations.

Recovery.

W. J., European male, aged 11 years, admitted on May 4th, 1901. He stated that three days before, diarrhoea started with pain in the abdomen. He had no vomiting or headache. Stools consisted of blood and mucus from the first. Patient was a healthy looking boy with good personal and family history. He had arrived in the country only a fortnight before being taken ill. On admission, temperature was 102.2° F, face somewhat flushed, with a pained expression. He complained of frequent inclination to go to stool, with small result after much straining. The abdomen was tender over the left iliac fossa. Tongue moist and slightly furred. Rectal irrigations of two pints of warm boracic lotion twice were ordered to be given daily and a diet of chicken broth and water arrowroot. During the first day there were six stools. These were small and consisted solely of mucus, blood and shreds and were accompanied by great straining. On the fifth there were ten stools of similar consistence, but with much less tenesmus. On the 6th. and 7th., there were nine and eleven stools respectively. These contained less blood, but consisted of mucus and shreds, tenesmus absent. On the 8th. there were seven
shreddy colourless stools without blood. On the 9th. there were five stools, the later ones partly formed and partly yellow liquid with mucus. On the 10th., the temperature which had been ranging from 99 to 101 F became normal and remained so till the patient was discharged. On this day there were three yellowish partly formed stools without blood or mucus. The rectal irrigations were ordered to be given once daily. On the 12th. there were two stools, normal in appearance. The rectal irrigations were discontinued, patient feeling well and hungry. Milk and cornflower were added to the diet. From the 12th. to the 19th. when he was discharged well, patient's bowels acted only on administration of castor oil.

CASE 2.—Acute dysentery treated by rectal irrigations and magnes. sulphat. Recovery.

S. J., European, aged 12½ years, brother of previous patient, was admitted to hospital on May 7th. three days after his brother. He had always been healthy, till his arrival in South Africa, two weeks before present illness. On the day before admission he was taken ill with headache and diarrhoea. The mother states that his bowels acted every ten minutes and
he passed blood and mucus. On admission patient's face was flushed and presented a pained expression. Tongue slightly coated and tenderness was elicited in the left iliac region. T. 103 F. Patient passed 16 motions during the first 24 hours. These were small consisting of blood and mucus, and accompanied by severe straining. Urine was scanty and contained no abnormal constituents. The chest shewed nothing abnormal on physical examination. The treatment consisted of a diet of chicken broth and water arrowroot with irrigation of the rectum with 2 pints of warm boracic lotion twice daily. During the next two days the temperature ranged from 99 to 101 F. The number of motions were twenty-three and twenty-two during the 24 hours, while the character remained the same. On the 9th., i. e. the 3rd. day of treatment, Magnes. Sulphat. 1 drachm was ordered to be given two hourly, while the irrigations were continued as before. During the next four days the temperature gradually fell to normal while the number of stools was 18, 14, 10 and 8 respectively. At the same time the stools became greyish then yellowish and fecal in character, while the blood and lastly the mucus gradually disappeared from them. During the next fortnight the daily number of stools varied from two to seven in
the twenty-four hours. They still occasionally contained mucus with streaks of blood. On the fifteenth day of the disease a paronychia on the right index finger developed and four days later one on the left index finger. From this time patient made a gradual recovery and was discharged well three weeks later.

CASE 3.—Acute dysentery with great tenesmus. Treatment rectal irrigations. Recovery.

T.E., European male, aet. 33, had been in this country only a few weeks when he was taken ill with dysentery. He was admitted on April 1st. 1900 and during the night before, he states that his bowels acted 54 times. On admission he appeared wasted and ill. Temperature 100.6° F., pulse 136 thready, respirations 28. Tongue slightly coated and dry, great thirst. Abdomen soft, tender all over, constant nausea and frequent vomiting, aggravated by taking nourishment. Patient had 18 stools during the first day. These consisted of mucus with bright red blood, and were accompanied by severe tenesmus. Morph. hydrochlor. gr. 1/4 was given hypodermically. This controlled the vomiting, though it did not entirely and arrest it, relieved the tenesmus. Only small quantities of chicken broth and warm water were given by mouth.
During the next two days patient required the morphia to be repeated and by this time the nausea had disappeared. From the first day rectal irrigations of warm boracic lotion were carried out. Patient complained of pain and a feeling of faintness during the administration of the first two, so that the quantity employed was only 1 pint. After each irrigation patient stated he had freedom from the desire to go to stool which had been constant before, and this feeling lasted for some time. When the bowels acted the tenesmus was less severe. As the rectum became less irritable the quantity of boracic lotion used was gradually increased, till the usual irrigation with four pints was administered daily. The character of the motions improved, the blood, then the mucus disappeared though it was only four weeks later that the patient passed a normal formed stool. He was discharged well two weeks later, i.e. after six weeks treatment.

CASE 4.- Acute dysentery following exposure, treated by rectal irrigations. Recovery.

C. W., European, 26 years, admitted on April 13th, 1901. Three days before admission he had been to a picnic and on returning home in the evening he had been caught in a shower of rain. Next day he had
pain and straining on going to stool and passed small quantities of blood and mucus. On admission he had great pain and tenderness over the descending colon and sigmoid, and constant desire to go to stool. Temp. 99.4°F, pulse 92, respiration 20, tongue coated moist. He passed 12 small motions consisting of mucus with streaks of blood and accompanied by great straining and abdominal pain. Diet of water arrowroot and broth ordered with rectal irrigation with warm boracic lotion once daily. 14th. April. Patient had 10 stools similar in character to those described, but abdominal pain less marked. 15th. There were 6 stools consisting of mucus and a little faeces, very little blood. 16th. Three stools passed, abdominal pain and tenesmus absent. Patient hungry. From this point patient rapidly improved, having one to two relaxed motions daily and was discharged well on the 22nd. of April.

CASE 5.—Chronic dysentery treated successfully with large irrigations of solution of silver nitrate, after failure of other methods.

P. H., European male aged 26, employed in railway locomotive works, suffering from chronic dysentery. He had been treated by the medical officer to the railway works at his own home. On admission,
patient appeared somewhat emaciated, tongue moist and furred, pulse 92, respiration 20 per minute. The abdomen was somewhat retracted and tender over the course of the colon. He was passing from six to nine small liquid stools daily. These were dark in colour and contained mucus and occasionally specks of blood. There was little tenesmus. Temperature normal and only twice during the next five weeks reached 100°F. Treatment on admission consisted of a diet of milk and broth, and an enema of starch 1 oz., tr. opii m. XV daily, with a mixture of Tr. monsonia 1 drachm and Infus. Calumb. 1/2 oz. given four hourly. This treatment was persisted in for three weeks without any amelioration in the patient's condition. Rectal irrigations daily with large quantities of warm boracic lotion were added. The number of stools fell to one to three daily in the course of the next week, but the character remained unaltered. Pulv. ipecac. Co. and Bismuth subnitrate 1/2 gr. X were substituted for the monsonia for a week without effect. On the 30th. day of treatment in hospital, preliminary irrigation of the bowel with four pints of warm saturated solution of Sodii bicarb. was ordered, followed half an hour later by an injection of three pints of solution of argent. nitrat. of the strength argent. nitrat. gr. ss to Ag. destillat. 1 oz. Patient complained of slight pain
during, and immediately after, the latter injection. On the following day the number of stools pass was increased, but the consistence was firm, and no blood and mucus was present. Patient from this date improved rapidly and on the fifth day after the injection of Argent. nitrat. left hospital at his own request, apparently in perfect health.

CASE 6.—Chronic dysentery, liver abscesses. Autopsy.

F. D. J. European male aged 24, professional boxer, was admitted on June 17th. 1901. He had been engaged with the Rhodesian force about Mafeking till the relief in May 1900. He stated he had been in good health till a few weeks before admission, when he suffered from diarrhoea. This was painless and he states no blood or mucus was passed. Three days before admission he noticed a swelling in the abdomen below the ribs on the right side, and had profuse sweats. On admission he was emaciated, skin a dirty brown colour. Breathing was rapid (36), painful and alae nasi working. Pulse 120 per minute, T. 101.4° F. There was a round, smooth tender swelling the size of a coconut in the epigastrium. There was dull on percussion and continuous with liver dulness.
Bowels constipated and patient perspiring freely. Liver abscess was diagnosed and on the following day under anaesthesia an opening was made directly over the swelling in the epigastrium. This proved to be an abscess in the right lobe of the liver near the surface. Its anterior surface was adherent to the abdominal parietes. On incision a pint of thick blood-stained pus escaped. On passing the finger into the abscess cavity, numerous sloughs were felt projecting from the abscess wall. Patient's temperature fell to 97 °F on the evening of the operation, but rose the next morning to 101 °F, and varied between 102 °F and 97 °F during the rest of the illness. Large sloughs continued to be discharged daily as they became detached from the wall of the cavity. Temperature remained intermittent, sweats continued and later diarrhoea set in. At first ordinary liquid stools were passed, but on July 2nd, they contained blood and mucus. Diarrhoea continued and patient became rapidly more emaciated. On July 5th, patient had 27 liquid dark motions each containing blood. He died the same day.

An autopsy was held next day. The rectum, colon and caecum contained innumerable small ulcers, in size varying from a pins head to that of a threepenny bit. These ulcers were deep, extending down to the muscular coat, and were covered with semi-liquid greenish faeces. The edges of the ulcers were smooth rounded not undermined, and intensely congested.
The ulcers covered the greater area of the large intestine; the mucus membrane between the ulcers was dark congested and swollen. The serous coat of the ileum shewed numerous distended blood vessels. On of the lower end of the ileum the mucous membrane were several small irregularly shaped superficial ulcers, the surfaces of which were covered with mucus. The liver and stomach were adherent to the anterior abdominal parietes, and the stomach to the under surface of the liver. The liver was generally enlarged. The left lobe contained three large cavities containing pultaceous sloughing liver tissue. One of these had been opened at the operation, while the other two lay respectively posteriorly and to the left of this, and were separated only by a thin friable wall of softened liver tissue. Attached to these "abscess" walls were yellowish stringy sloughs of liver tissue. Almost the whole of the left lobe was occupied by these three abscess cavities. What remained of liver tissue was yellowish soft and friable. In the right lobe which was also enlarged, were two small areas near its upper surface about the size of cherries where the liver substance was commencing to soften. On cutting through them no pus escaped, but the cut surface had a yellow spongy appearance. On pressing with the finger the yellow area broke down into material resembling thick pus. The rest of the liver substance was pale, especially in the region of the necrosed areas just described.
The spleen and kidneys were congested and somewhat enlarged. The mesenteric glands were swollen. The thoracic organs shewed nothing abnormal.

CASE 7.—Acute dysentery. Liver abscesses. Autopsy.

F. P., European boatman, aet. 31 years, admitted on November 18th., 1898 complaining of acute pain in the right hypochondrium. He had up till three weeks ago been in good health, though exposed to the weather in his occupation. Three weeks ago "diarrhoea" with straining and passing of blood had started. At first he was not confined to bed, but on Nov. 15th. he felt a sudden sharp pain in the right side, and three days later he was admitted to Hospital.

Patient had a dark muddy complexion was perspiring freely and appeared to be in great pain. The abdomen was slightly distended and tender over the line of the colon and at the margin of the ribs on the right side, where the lower border of the liver could just be felt on inspiration. No enlargement upwards of the liver was made out. The respirations were 40, shallow and painful. Pulse 120 soft Temp. 101.6°F. There were three small offensive stools consisting almost entirely of blood and mucus, and accompanied by a considerable degree of tenesmus. A linseed meal poultice to the right side and morph. hydrochlor. gr. 1 were
ordered for the pain, and magnes. sulphot. Drachm 1
hourly. During the next few days patient passed from
20 to 26 stools similar to those described. On Nov.
22nd. rectal irrigations with boracic lotion were
added. The number of stools diminished gradually to
three per diem and the character improved; the pain
in the right side disappeared and the temperature which
had risen to 103°F, fell to sub-normal. On November
30th. patient complained of severe pain below the
right costal margin, the temperature rose to 101.6°F.
The lower border of the liver reached to three fingers'
breadth below the costal margin, and coarse friction
was readily felt over this area. The respirations were
frequent and painful. During the next week the
temperature ranged intermittently from normal or sub-
normal to 103°F, patient had profuse sweats, the
number of stools one to three daily. On December 9th.
the liver was exposed by an incision in the right mid-
axillary line over the 8th. rib, a portion of which
was removed. A large abscess was found and its con-
tents evacuated and the cavity flushed out. The
temperature dropped, but otherwise patient did not
improve after the operation. The profuse sweats con-
tinued, 35 stools daily were passed and the patient
died on December 14th.

POST MORTEM. The liver was found to be considerably
enlarged and adherent along the upper surface to the
diaphragm and to the peritoneum of the anterior
abdominal wall. The abscess cavity drained at the operation was found, while internal to it was a second abscess. Behind these two was a third abscess. All of these were in the right lobe and about the size of a large orange. The walls were ragged irregular and soft, and the liver substance forming the partitions between the abscesses was pale and friable. The left lobe contained no abscess and shewed venous congestion. The wall of the large intestine was thickened especially that of the rectum. The mucous membrane of the large intestine was ulcerated throughout its whole extent. The ulcers were about the size of a three penny bit circular with swollen smooth edges and extended down to the muscular coat. The transverse colon looked like a piece of tripe—the mucous membrane being honeycombed with small punched out circular ulcers situated regularly all over the inner coat. The spleen was congested and enlarged, mesenteric glands swollen; the other organs shewed no naked eye change.

CASE 8—Dysentery, liver abscesses. Autopsy.

C. D., European male, aged 34, was admitted to Hospital on July 23rd., 1901. He had been serving with one of the irregular Colonial Corps in the Anglo-Boer War, and was first taken ill in March 1901. He was sent to one of the stationary military hospitals suffering from acute dysentery. In July he arrived
in Port Elizabeth, and shortly after was admitted to the civilian hospital as stated. He complained of "pain in the right side with diarrhoea and occasional passing of blood". Personal history good, had never suffered from malaria. On examination the abdomen was found moderately distended and slightly tender especially in the iliac fossae. Patient complained of pain chiefly in the right hypochondriac region. The temperature was 103°F, tongue moist and covered with white fur, respirations 36, pulse 120 and small.

In the right mammary line dulness was found extending from the 3rd. interspace to a finger's breadth below the costal margin. Behind, dulness extended from the spine of the scapula to the 11th. rib, and over this area there was entire absence of breath sounds. The intercostal spaces were considerably widened especially in the mid-axillary line where there was tenderness on pressure, and slight oedema of the subcutaneous tissues. In the right axillary and mammary regions several distended sub-cutaneous veins were seen running upwards. The cardiac impulse was felt in the left nipple line. The heart sounds normal. Over the left lung crepitations were heard.

Patient had from two to three small liquid yellow stools, in appearance resembling enteric stools, daily. No blood or mucus was present. The temperature ranged from 100°F to 103°F, and the pain in the right
side was only temporarily relieved by local applications. Two days after admission, an exploring needle was inserted posteriorly in the 8th. intercostal space and some thick pus withdrawn.

On July 27th., under anaesthesia, a portion of the 8th. rib was removed in the posterior axillary line and the liver was here found adherent to the parieties. The abscess cavity was found about \( \frac{1}{2} \)" from the surface of the liver and its contents evacuated. On passing the finger into the cavity a soft area was found on its posterior wall. On this being broken down more pus escaped - the second abscess cavity having been artificially connected with the first. On the same evening the temperature was normal and the patient somewhat easier, though he complained of feeling weak. On the following day the temperature rose to 101°F, patient perspired freely and still complained of feeling weak. As patient's condition remained unsatisfactory - the temperature ranging from 100 to 102°, the sweats persisting and patient evidently becoming worse - On July 31st. he was again anaesthetised and the liver explored through an incision in the right mammary line in the 6th. interspace. Here the liver was adherent to the parieties, and immediately under the surface an abscess cavity was found and its contents evacuated. As there was marked dulness in the
epigastrium another incision was made in this region and a large abscess in the liver substance was found and opened.

The patient was suffering profoundly from shock and died the same evening.

Pus and necrotic shreds of liver substance from each of these abscesses was carefully examined immediately after the operation, and was found to consist of broken down liver cells, pus cells and red blood corpuscles, but no amoebae were seen. Post-mortem examination, held the following day, revealed the whole of the right lobe of the liver adherent along its surface to the diaphragm. The adhesions were soft and on breaking them down a small abscess between the posterior surface of the right lobe and the diaphragm was opened. On removing the liver the three abscesses drained at the operations were seen to have ragged walls formed of necrotic liver substance, and to encroach in various directions on healthy liver tissue. On the posterior border of the liver a small abscess was found. Three other areas of necrotic liver substance were found on incision - all in the right lobe. These latter could hardly be considered abscesses. The liver substance at these spots was soft and spongy of a yellow colour and the structure of the liver lobules not apparent. On gentle pressure these areas broke down into a cheesy yellowish semi-liquid material. The intervening liver substance was pale with the central vein
of the lobule clearly visible. The left lobe contained
no abscess but was pale and firm on pressure. It did
not give the reaction of waxy degeneration with tincture
of iodine. The large intestine contained many superficial
ulcers of the mucous membrane. They were mostly
oval, with fairly smooth edges, not undermined, and the
surface was covered with mucus. They were most numerous
in the rectum and descending colon. The mucous membrane
between the ulcers presented no unusual features. The
ileum at intervals shewed marked injection of the
vessels. The mesenteric glands were slightly enlarged
but not caseous. The spleen and kidneys were congested.
The right lung was collapsed and contained very little
air. The pleural cavity on this side contained 3 pints
of clear fluid. The left lung was oedematous. The
pericardial cavity contained two ounces of clear fluid.
There was a large pale clot in the right auricle,
otherwise the heart shewed nothing abnormal.

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CASE 9- Dysentery. Liver abscesses, intense jaundice.

Autopsy.

F. L., European male aged 28 years, a Tramway
Guard, was admitted on 29th. May 1901.

He had been ill for four days before admission, and
complained of headache, general malaise and constant
abdominal pain. He had taken part in the defence of
Mafeking during the siege October '99 to May 1900. He
stated he had never been ill in his life before, and denied having ever had dysentery or malaria. On admission temperature was 102 degrees F., tongue thickly coated, respiration 28, pulse 96, abdominal pain and tenderness marked all over, bowels constipated, the thoracic organs were healthy. Spleen slightly enlarged. The liver extended in the right nipple line, from the fourth rib to two inches below the costal margin, posteriorly no enlargement of the liver could be determined, but in the epigastrium the liver could be felt extending two and a half inches below the tip of the Xiphisternum, and was very tender to the touch. B-naphthal gr. 6. was given thrice daily with a diet of milk and broth, after an initial dose of calomel gr. 5. The temperature fell to 98.4°F and did not rise during the next two days. The abdominal pain continued. The bowels acted only with aperients and the result was yellow liquid stools. On the fourth day after admission the temperature rose 101.2°F and patient perspired slightly, abdominal tenderness less marked. The patient was slightly jaundiced. The jaundice increased rapidly till the skin all over became of a deep yellow or brownish yellow tint. The liver gradually increased in size downwards while the tenderness disappeared. The intercostal spaces in front and in the mid-axillary line became greatly widened. The remaining organs of the body showed nothing abnormal on physical
examination. The urine contained bile pigment, no albumin or sugar present. The patient remained constipated, appetite ravenous, pulse 120 soft, temperature remittent. Liver abscess was diagnosed and patient advised to undergo operation, this he refused. On June 22 when patient had become much worse, he consented to operation.

A portion of the 7th. rib in the right mid-axillary line was removed and a large abscess discovered in the liver. This was opened and drained, patient gradually became weaker and died on June 25th. Autopsy 12 hours after death. Body jaundiced and emaciated. Liver enlarged especially in the right lobe. The latter contained four large abscesses communicating with each other. These abscesses occupied the greater part of the right lobe, a small amount of liver substance remaining and forming the partition between the several abscesses.

The walls of the abscess cavities were ragged and contained semi-liquid chocolate coloured pus. In the small intestine Peyer's patches were visible, not swollen. Two large superficial ulcers occurred in the caecum. No amoebae were found in the abscess contents.

M. B., European male, aged 46, admitted on October 23rd., 1900 suffering from "pain in the stomach." He stated that he had been ill for a month, during which time he had been passing frequent relaxed stools, containing blood and mucus occasionally, without much straining. The temperature was 102.2°F, tongue covered with brown fur. Pressure elicited tenderness over the right iliac fossa. On the morning of the 24th., about twelve hours after admission he suddenly developed meteorism and became collapsed. The temperature fell to 100°F., pulse 128 small, respirations 32 and purely thoracic and patient died at 3 p.m. on the same day. During the 24 hours he was under observation he passed 32 small dark liquid stools which were very offensive, but contained no blood or mucus.

P. M. The body was well nourished, the abdomen greatly distended. Early peritonitis was found; the coils of small intestine congested and adhering lightly to each other. The caecum was deeply congested and dark in appearance. Its outer surface was covered with thick flakes of lymph and pus which was also found in the pelvis. On the anterior surface of the vermiform appendix.
form appendix a perforation about the size of a goose quill was found \( \frac{3}{4} \) inch from its base. There was a small concretion at the apex of the appendix. Throughout the large intestine round or oval ulcers with regular smooth edges were found. These extended across the long axis of the bowel and down to the muscular coat. They were most numerous in the rectum, sigmoid flexure and caecum. The muscular coat of the former was greatly thickened.


M. W. N., European male, aet. 60 years admitted on 18th., December, 1900. He complained of pain in the right side of the abdomen and diarrhoea which had lasted twenty-six days before admission. Patient had been a hard drinker. On examination temperature was 101.8°F, pulse 116, respirations 28 shallow. The abdomen was somewhat distended and tender especially in the right iliac fossa, where on palpation there was a sense of fulness. Patient had four dark liquid offensive stools without blood or mucus in the twenty-four hours. Tongue dry and coated. During the next four days patient's condition remained unaltered except that the tenderness and fulness in the right iliac fossa became more pro-
nounced, the symptoms and signs pointing to appendicitis. Laparotomy was performed on Dec. 22nd. A four inch incision was made over the caecal region and on opening the peritoneal cavity some thin offensive fluid escaped. The caecum and surrounding tissues were found in a gangrenous condition, the appendix healthy. The patient died the same day.

Post-mortem. The peritoneal cavity contained a quantity of dark liquid serum with thick flakes over the colon and in the pelvis. The caecum and ascending colon were dark and gangrenous as were the soft structures behind them. The appendix was healthy. Four irregular ulcers were found on the mucous membrane of the ascending and transverse colon. The liver was cirrhotic and half a dozen small abscesses were found varying in size from a pea to a walnut. The spleen was congested. Both kidneys were cirrhotic and the capsule adherent and cortex contracted. Pus was found in the substance of the kidney and in the pelvis of the ureter. The other organs shewed no abnormality.


J. S., European male, aged 55 years, admitted on Novem —
ber 22nd, 1898. Patient was a butcher and stated that he had been taken ill suddenly four days before admission, with pain in the abdomen and frequent action of the bowels. On Examination, Temperature was 100.4 °F; pulse 120 small, regular, respirations 22. The abdomen was slightly distended and tender all over especially in the right iliac region. He passed 11 dark liquid offensive stools containing dark shreds, with some old blood. Liquid diet was ordered and on account of the severe abdominal pain morphia was given hypodermically. Patient remained in this condition, passing from 11 to 17 offensive shreddy stools daily, until Novem. 26th, when the abdomen became greatly distended and tender all over. On the same day exploratory laparotomy was performed, patient being almost in extremis. Incision over the caecum shewed gangrene of the bowel which patient's condition prohibited removal of. He died the same day. The post-mortem examination shewed recent general peritonitis. This was most marked near the caecum. The anterior wall of the caecum was black and gangrenous with a linear rupture. The vermiform appendix was thickened, the serous coat inflamed but not ruptured. The mucous membrane of the large intestine shewed ulcers with characters similar to those described in previous cases. The liver and other organs were healthy.
CASE 13—Chronic dysentery. Lung abscess. Autopsy.

J. M., Native, male aged 49 years admitted on March 29th, 1901. Diarrhoea had commenced four days before, followed soon after by pain in abdomen and back and tenesmus. He had frequent call to stool and often after much straining passed nothing, or at times he passed blood and mucus. On admission he appeared in pain and very weak. Tongue thickly coated with brown dry fur, edges red and clean. It was impossible to estimate the number of stools during the first few days. He was constantly on the slipper and appeared to have no control over the sphincter. The stools were small, shreddy and contained dark coloured blood. Pulse 120, small, feeble, regular. Respiration 36, T. 97.4°F.

Diet of arrowroot and chicken broth, magnes. sulphat. drachm 1; 2 hourly and rectal irrigation with warm boric acid solution once daily ordered.

30th. Patient cold collapsed, pained anxious expression. Pulse feeble. Condition generally the same as on admission.

April 3rd. Patient has more control over sphincter. had 15 stools consisting of small bile stained faecal masses, dark blood and shreds of mucus. Tongue still dry and thickly coated.

April 7th. Tongue clean except at the centre and moist. Face fuller and patient altogether more
comfortable. He had 11 small liquid bile stained stools without blood. Milk 1 pint ordered.

April 9th. Patient had 11 yellowish lumpy stools containing undigested milk curds, no blood or mucus. Tenesmus slight.

April 10th. Eight stools, still yellowish with undigested milk, no blood or mucus. Milk ordered to be peptonized. From this point patient's condition remained practically stationary with only slight daily variations. On the 24th. the tongue became dry, smooth and glazed, otherwise condition remained unchanged. On May 8th. Pulv. Cinnamon gr. 30 ter in die was ordered. This was increased later to gr. 30 three hourly, without any noticeable change in patient's condition. On May 23rd. 1. e. eight weeks after admission he left the hospital at his own request. All pain and tenesmus were gone, but he was passing 9 or 10 liquid yellow stools daily and appeared unable to digest the simplest food. During all this time his temperature was for the most part sub-normal, with an occasional rise to 100°F. He was re-admitted on June 5th. decidedly worse than when last seen. The number of stools was fewer (from 3 to 7 in the 24 hours), but he was more emaciated, tongue dry, brown and cracked, and he complained of pain in the knees and calves of the legs. The feet were swollen and oedematous. There was no cardiac murmur, but patient was very anaemic. Urine scanty and contained no abnormal
constituent. Patient was at first ordered a strictly limited diet of arrowroot and broth and as he appeared to make no headway, this was 10 days later supplemented by a diet of meat and vegetables. Pulv. Cinnamon gr. 50 three hourly; then pulv. ipecac. Co. and Bismuth; liq. ferri perchloratis MXXX, diluted, thrice daily; liq. hydrarg. perchlor. Mxxx, also freely diluted thrice daily, were all tried in turn without any change in the character of the stools being observed.

On June 16th, patient complained of pain in the right hypochondrium and in the left side of the chest. The temperature which had been sub-normal, rose suddenly on this date to 102.2°F without a rigor, and patient had a frequent dry cough. On examination there was a hyper-resonant note over the chest, in front and behind while friction sounds could be heard in the mammary and axillary regions on both sides. The temperature continued intermittent, ranging from 103.4°F to normal, during the next three weeks. Patient began soon to expectorate, at first clear mucus, then muco-purulent matter, not very abundant. Friction sounds could still be heard over the right side of the chest in front and at the side. Breath sounds on the left side, at the apex in front, and all over the posterior surface became tubular with moist crepitations, heard most abundantly at the apex and immediately below the clavicle. The percussion note was hyper-resonant on both sides in front and behind. Later
the breath sounds became feeble or almost inaudible on the left side. Patient died on July 17th.—Four months after onset of his illness.

Post-mortem examination held 8 hours after death. Body much emaciated and on incision almost an entire absence of subcutaneous fat found. Heart small, ante-mortem clot in the right auricle.

Both lungs shew recent pleurisy. Right lung oedematous and a firm cicatrix found on the post-mortem border of the lower lobe. Left lung adherent below to diaphragm. On detaching it a large abscess cavity found in the lower lobe. The walls of this are formed of softened easily broken down lung tissue, of a yellowish putty colour and consistence, from which no air can be expressed. The upper lobe forms one large abscess cavity containing chocolate coloured pus. The walls of this are formed by pleura with almost none of the lung tissue left. The bronchial glands are enlarged, some of the size of pigeon's eggs. In the pus no amoebae or tubercle bacilli were found. It consisted chiefly of pus cells, a few red blood corpuscles and ordinary pus cocci. On opening the peritoneal cavity this membrane appeared dry. The great omentum appeared as a thin transparent membrane containing no fat. Between the folds of the mesentery is little fat, and the lymphatic glands are enlarged. Stomach greatly distended reaching to the left iliac fossa. The walls of the small intestine are thin, and the mucous membrane is congested at intervals. There
are numerous superficial ulcers of the mucous membrane of the large intestine and between these the mucous membrane is of normal colour. There are several enlarged follicles in the mucous membrane, feeling like small lymphatic glands, which appear semi-solid on section. At the splenic flexure, there is a sudden narrowing of the lumen of the colon, barely admitting the tip of the little finger. The muscular coat appears thickened at this part which is about an inch in length. The vermiform appendix is \( \frac{1}{2} \) inch long.

Spleen enlarged and friable. Kidneys are small pale, capsule strips off easily, and shews two small cysts in right kidney.
CASE 14—Chronic dysentery. Ulcers in the large and small intestine.

M.R., native woman, aged 24 years, admitted on March 12th, 1901, suffering from chronic dysentery. Patient had suffered from "looseness of the bowels" for two months previously during which period she had had no treatment.

Patient was a small woman with a pained expression making her appear older than the age stated. She was much emaciated, the abdominal walls soft flabby and retracted. The mucous membranes of lips, tongue and conjunctiva had a pale anaemic look, and she had a slight cough. From 10 to 14 dark liquid stools were passed daily, containing no blood but shreddy mucus. Temperature 96°F. on admission and only twice reached the normal line after admission. She died of exhaustion eight days later.

At the post-mortem examination the body shewed all the appearances of starvation. The skin and tissues were dry, the integument hanging in loose folds. There was no subcutaneous fat and none in the great omentum which was thin and transparent like gossamer. The glands in the great omentum and mesentery were enlarged and caseous, some reaching the size of a walnut. The vessels of the mesentery were congested. The
lumen of the intestines was diminished in size and the walls thin. The mucous membrane of the rectum was congested. Small follicular ulcers were found in the mucous membrane of the rectum, descending, transverse and ascending colon. These were about the size of a split pea and superficial. The edges were smooth and not undermined. The surface was covered with mucus and the mucous membrane between was congested giving it a purplish look. The mucous membrane of the caecum similarly injected and contained several superficial ulcers. At the base of the ileo-caecal valve were two ulcers of similar character. The serous coat of the lower end of the ileum appeared congested. On the inner surface of the ileum, three inches and also six inches from the ileo-caecal valve, several ulcers were found. These were larger and deeper than those which occurred in the large intestine. The edges were irregular smooth and not undermined, the ulcers being more or less round or oval in shape, and were situated near the mesenteric attachment. Peyer's patches were clearly seen, but not ulcerated. Three feet above the ileo-caecal valve an annular ulcer was seen running like a ring completely round the inner circumference of the ileum. This ulcer had smooth rounded and somewhat thickened and congested edges, and
extended through the mucous and sub-mucous and invaded the muscular coat. The edges were at no spot undermined, and the surface was covered with mucus and greenish semi-solid intestinal contents. The stomach was dilated. The surface of the liver was covered with lymph by which it was adherent to the under surface of the diaphragm. The gall bladder was greatly distended and contained a clear water fluid. On section the liver shewed venous congestion. The kidneys appeared large with pale cortex and firmly adherent capsule. The lungs were oedematous. No amoebae were found on the surface of the ulcers.
CASE 15. — Chronic dysentery. Ulcers in large intestine and in ileum.

M. R., native woman, aged 27 years admitted on February 15th., 1901 suffering from chronic dysentery. She had been suffering from "looseness of the bowels" for some weeks before admission for which she had had no treatment. On admission, patient was much emaciated, complained of no pain, but passed from 3 to 9 liquid brown stools in the 24 hours. These contained mucus but no blood. Magnes. Sulphat. ^ was given 2 hourly. Four days later the number of stools was one per diem; a week later patient had a relapse, passing seven or eight liquid stools as described, and she complained of pain in the left leg. This became oedematous and was very tender. Oedema round the right ankle followed. The heart and urine shewed nothing abnormal. A week later she developed periostitis of the left upper jaw and oedema of the cheek. The temperature on the first two days after admission was 101° F, after which it remained subnormal. She died three weeks after admission.

Post-mortem. The body was much emaciated, oedema of left leg, right ankle and left cheek present. The thoracic organs shewed nothing abnormal beyond oedema.
of the bases of the lungs. The stomach was dilated. The mucous membrane of the rectum was injected but not ulcerated. In the descending and transverse colon, especially in the former, numerous ulcers were found. These were circular or oval in shape and about \( \frac{1}{3} \) inch in diameter. The edges were smooth swollen and not undermined. The base, formed of the circular muscular fibres, was covered with thick adherent mucus and greenish semisolid matter. No ulcers were found in the ascending colon. The mucous membrane of the caecum was almost entirely destroyed by ulceration. The ulcers were somewhat larger than those in the rest of the large intestine, but otherwise resembled them. Narrow strips of swollen greyish mucous membrane alone remained and separated the individual ulcers. The base of the latter was formed by the muscular coat and adherent to it was mucous and green semisolid faecal matter. The serous coat of the small intestine was congested. At seven different places there were rings of ulceration extending completely round the inner wall of the ileum. The narrowest of these was \( \frac{5}{4} \) inch and the widest 2 inches in width. Some of them which appeared to be older, had smooth even bases composed of the muscular coat. The bases of others were uneven, part of the ulcer having extended down to the muscular wall, while elsewhere only the superficial portion of the mucous membrane had dis-
appeared. Some small follicular ulcers were seen. All of these ulcers were covered by similar material to those in the large intestine. Peyer's patches were readily seen but not ulcerated. The mesenteric glands were swollen. The liver was fatty; the spleen and kidneys were congested.

CASE 16. — Chronic dysentery. Ulcers in large and small intestine.

F. B., native male, aged 50 years was admitted on August 16th., 1900, suffering from chronic dysentery. On admission he was found to be much emaciated, tongue slightly coated, temperature subnormal. The abdomen was retracted and boat shaped, and tender in the left iliac fossa. He passed from four to nine liquid stools containing mucous but no blood, per diem. In spite of treatment by diet, ipecac, and purgatives patient steadily lost ground and died on Sept. 5th. — three weeks after admission. Five days before death synovitis of both wrists and right ankle joint developed. There was slight effusion into the joints, but the pain and tenderness was considerable.

Post-mortem. The wall of the large intestine was thick-
ened throughout its whole extent, especially that of the rectum. Numerous circular and irregular shaped ulcers were found extending through the mucous membrane of the large intestine. They occurred chiefly in the rectum, descending and transverse colon. The lumen of the bowel was somewhat narrowed at the hepatic flexure. The ulcers varied in size from that of a pea to that of a florin and presented elevated smooth and rounded edges. In the ileum several ulcers with similar characters, but smaller, were found extending to a foot above the ileo-caecal valve. The mesenteric glands were enlarged. No change was discovered in the other organs.
SYMPTOMS.

In describing the clinical varieties of dysentery as it occurs in South Africa we recognise:

(1) Acute Catarrhal.
(2) Chronic
(3) Gangrenous

All of these start as acute catarrhal and may pass into either of the other varieties.

(A) Acute Catarrhal. This is by far, the most frequent form met with in South Africa and is variable in its onset. After exposure to rapid changes in the temperature or fatigue, the patient may have diarrhoea or any uneasy sensation in the rectum for a day or two. The stools soon become more frequent, are accompanied by abdominal pain and straining, and the stools change in character. They become more frequent, as stated, cease to contain bile or faeces, and consist of small quantities of bright red blood and clear mucus like white of egg. The number of the motions varies from 10 to 20, in severe cases reaching as many as 70 in the twenty-four hours, and in some cases it is impossible to enumerate the numbers as the patient has a constant desire to evacuate the bowels and refuses to have the slipper removed from the bedside.

There is usually some elevation of temperature during the first three or four days, though this rarely
exceeds 101°F. In the case of children it may reach 104°F. The temperature chart is of little clinical value during the early stages of acute catarrhal dysentery though later it may be of importance as indicating the formation of pus, or the occurrence of some other complication. In the course of a few days the temperature falls to normal or below this, and does not again rise above this point. The patient frequently has a flushed and pained expression, the tongue is usually slightly covered with white fur, or it may be dry red or glazed. There is usually abdominal pain and tenderness. This may consist only of vague uneasiness with localised pain at the anus on the bowels acting, but usually definite tender spots, especially over the sigmoid, can be elicited on pressure. The abdomen is usually flat or slightly retracted during the acute stages. Usually no enlargement of the spleen can be made out on examination, except in cases which have previously suffered from malaria. The liver is usually not enlarged; but it may be congested and tender. There is loss of appetite, usually considerable thirst exists, but no vomiting. The amount of urine passed is small—sometimes only ½ to 8 ounces in the twenty-four hours for the first few days. Patients not infrequently complain of a burning pain in voiding urine. On examination, this presents nothing abnormal. Occasionally
phosphates or an excess of urates is discovered. The thoracic organs are usually not affected in the acute stage, though when the temperature is elevated the respiration and pulse rates may be somewhat accelerated. The skin as a rule is dry, though this is more marked in chronic cases. The patient usually has no rigors or headache during the course of the disease.

The diarrhoea is the principal feature of the disease, and this varies considerably in frequency. In almost all cases it commences as simple diarrhoea which in the course of one to four days changes in character. In one of our cases, a sailor, there were four days of prodromal symptoms (colic, abdominal uneasiness and occasional diarrhoea), but usually the typical stools follow within a day on the onset of the disease. These consist, as stated, of bright red blood in streaks and clear mucus which adheres to the bottom of the bed pan. A day or two later the stools become more watery and floating in this are small dark or red lumps of blood with whitish shreds of mucus, giving the appearance of fresh raw meat scrapings described by German Authors. At this stage there is a characteristic odour of the stools and round the patient's bed.

The foetid offensive odour is found only in cases of gangrenous dysentery which are rare in our experience. The characteristic odour is difficult to describe, but is of an acrid "metallic" character. Under the influence of
treatment, or without, in favourable cases the blood disappears from the stools, they become yellowish from the return of bile and contain liquid faeces and mucus. Later the mucus disappears and the stools are liquid yellow, which finally become semi-solid and then solid normal motions. The duration of these stages varies greatly. In some mild cases the stools become normal in a week or less, while more frequently the attack lasts one and a half to two weeks. At any stage the patient may have a relapse and blood or mucus re-appear in the stools. In the early stage of blood and mucus, the microscopic examination of the stools reveals red blood corpuscles, white blood corpuscles and epithelial cells, in varying proportions according to the part of the stool examined. The red masses in the stool consist almost entirely of red blood cells, perfect in shape. The whitish mucus masses consists largely of white blood corpuscles, mostly granular, of uniform size and round; and occasionally round or oval epithelial cells with granular protoplasm resembling amoebae, from which they differ in being non-motile.

Later still the microscopic appearance of the stools show nothing characteristic — debris, undigested food, leucocytes, occasionally ova of ascaris and cocci. The appearance of the stools is of the greatest importance clinically. They should be examined daily in all cases and one's prognosis and treatment based on the result.

(B) In chronic dysentery the characters of the disease vary more than in acute. Several
are quoted at length among our cases (Cases 5, 14, 13, 15, 16. ). They start with acute symptoms which occasionally in spite of treatment go on to chronic.

Chronic dysentery may supervene on acute in spite of careful treatment, but more usually results from neglect during the early stages, and this in our opinion accounts for the large proportion of chronic cases as compared with acute, occurring among native patients. All our cases of chronic dysentery were admitted as such, except in the case of two Europeans with a previous history of alcoholism, and two natives, who were admitted as cases of acute dysentery which eventually became chronic.

A previously debilitated condition of the system appears to predispose to the development of chronic dysentery. A number of our cases of chronic dysentery among natives gave a previous history of scurvy or were actually suffering from this disease on admission. Among Europeans alcoholism was frequently traced as an important factor in inducing the disease to take a chronic form.

Chronic dysentery varies often in its clinical characters in different patients and also at different times in the same patient. The number of stools as a rule is not so frequent as in the acute form. They usually number from 3 to 8 or 10 in the twenty four hours, but may be more or at times constipation may supervene for a day or more. In appearance, too, they vary. They are usually
larger than those in the acute stage, and may be yellowish or grey in appearance. They frequently contain no blood, or they may contain small bright red masses of blood and mucus. At times the blood may be intimately mixed with the stool giving it a uniform dark brown appearance. At other times the stool may appear normal in form and consistence. Floating in the liquid stool is usually found mucus in small clumps, particles of undigested food such as milk curds and shreds of mucous membrane. We have never seen the casts of the large intestine described by some authors. The stools are frequently frothy. Under the microscope, red blood corpuscles, leucocytes and debris are found in varying proportions.

Pain and tenesmus are rarely complained of though the patient may complain of local irritation through excoriation of the mucous. The appetite is variable; at times almost absent, or the patient may have a keen appetite. The tongue is usually red and denuded of epithelium; though it may be dry, covered with brown crusts and cracked, resembling the tongue in the late stage of enteric fever, or it may have a thick white or brownish coating. The voice sometimes becomes husky due probably to dryness of the larynx, and in one case the patient could not speak above a hoarse whisper. Occasionally there is bleeding from the nose or gums and in a few severe cases, we have seen petechial spots under the skin. The temperature as a rule in uncomplicated cases
remains subnormal and is of little clinical value. There may be vomiting. The patient becomes greatly emaciated and the skin dry. The abdomen is usually markedly retracted (boat-shaped), though in two of our fatal cases there was distressing meteorism which was found post mortem to have been due to distension of the colon especially in its transverse portion. The urine is scanty, but contains nothing abnormal. There is almost invariably anaemia, in which the numbers of the red blood corpuscles and the percentage of haemoglobin are reduced in equal proportions. In one case the number of red blood corpuscles shewed 2,750,000 per C.M.M.

During the later stages of the disease oedema of the feet and lungs, effusion into the joints or pain in the limbs and other complications to be mentioned later occur. Paralysis of sphincter ani—unfavourable symptom. The pulse is increased slightly in frequency, is small in volume and of low tension. If left untreated, or in spite of treatment the patient may die of gradual exhaustion, formation of bedsores or from the development of some intercurrent affection such as pneumonia or peritonitis. The duration of chronic dysentery is variable. The average in our cases was 2½ months. In one fatal case it had lasted four months. In another case complicated with taenia, which eventually resulted favourably, the duration was 8 months. Another case, which also resulted favourably, was periodically under
We have seen three cases of gangrenous dysentery which are described at length (Cases 10, 11, 12). All of these were fatal, two through perforation and peritonitis. One showed multiple liver abscesses. The course of all these cases was comparatively rapid, and all the patients showed signs of extreme collapse.

None of these cases except possibly Case 10 appear to have suffered previously from acute catarrhal dysentery. The onset and progress of the disease appears to have been rapid. The number of the stools was somewhat greater than in most cases of chronic dysentery - in one of the cases numbering 32 per diem. The character of the stools was also different. They were dark brown or backish liquid containing mucus, brown or black shreds but seldom blood, and were most offensive in odour. The patients had more abdominal discomfort and pain than in cases of chronic dysentery, and the abdomen was somewhat distended and its walls fixed. The temperature was also higher than that usually found in chronic dysentery. It was elevated above the normal in all our cases, and in one case reached over 102°F. The pulse was increased in frequency, soft and small in volume. The breathing was also increased in frequency and was chiefly thoracic. The skin in all the cases was cold and covered with clammy perspiration. The patients were restless and evidently very ill; presenting
the clinical picture of a mixture of peritonitis with septic poisoning.

The cases ran a comparatively rapid course. In cases 10 & 11 from the onset to the fatal termination, the disease lasted a month, in Case 12, it lasted 10 days.
COMPLICATIONS & SEQUELAE.

In the first place we may mention the tendency to relapse. This may take the form of an exacerbation during the course of the disease, but more usually it occurs during convalescence or long after the first attack. It may be caused by cold, error in diet or over indulgence in alcohol. Alcoholic subjects and those debilitated from any cause appear to be specially liable to a relapse. One of our cases, a European man, a hard drinker, was under treatment for 39 days for acute dysentery when he was discharged well. He was re-admitted three weeks later suffering from the effects of alcohol, and with a return of all the acute symptoms of dysentery which lasted 13 days. Another case, a male European gardener, was treated during his primary attack and two relapses lasting 43 and 19 days respectively within a year. In this case the relapses were apparently due to exposure and bad feeding. Two cases, both females, Europeans, had relapses at short intervals after the primary attack where the cause of the relapse appeared to be the presence of Taenia.

Seven of our cases passed segments of Taenia during treatment, but their presence appeared to have no deleterious effect beyond increasing the amount of mucus passed, except in the two mentioned above.
In clinical characters and duration a relapse differs in no way from the primary attack.

Of our 58 cases in female patients, three aborted. Two were native women suffering from acute dysentery, and were respectively 2 and 5 months pregnant. The third was a European woman who was suffering from chronic dysentery and aborted in the third month of her pregnancy.

Scurvy played an important rôle in a number of our cases among native patients. Many native labourers, especially those on railway construction works live solely on a kind of porridge made of whole meal, and among these natives scurvy frequently occurs. Several cases suffering from scurvy and sent down to hospital were found to be also suffering from dysentery. In these cases, 11 in all, scurvy can hardly be correctly placed under the heading of "complications of dysentery". The history invariably shewed that the scurvy preceded the dysentery. These cases shewed the spongy bleeding gums and extravasations under the skin of legs and arms and other symptoms of scurvy, along with those of dysentery. The latter differed from the usual in the voiding of more blood in the stools - in some cases nothing but pure blood being passed - and in their greater frequency. These cases usually did well on ordinary antiscorbutic treatment if seen early. When not treated at the outset there was a great tendency for the cases to become chronic.
A well marked complication in certain cases was inflammation in one or more joints. Trousseau and Davidson describe a "Rheumatic" form of dysentery in which the symptoms are swelling and effusion into certain joints. Scheube also describes joint affections as occurring in the course of dysentery. He states that they may occur during the course of an attack of dysentery, but especially during convalescence, more often in slight than in severe cases, and that there may or may not be effusion into the joints.

In seven of our cases there were well marked symptoms of joint affection. Our experience differs from Scheube's in so far as our cases all occurred during the course of severe and protracted cases of chronic dysentery and none during convalescence. All the above authors state that the knee is the joint most frequently affected and then the ankle. Our cases entirely bear this out. In each one, one or other knee was first affected then the ankle and joints of the foot. In one case both wrists became subsequently inflamed, swollen and tender. In all of our cases more than one joint was affected and frequently both knees and both ankles. The affection differed in several ways from

an attack of acute rheumatism.

With the onset of the joint affection there was never a rigor, sweats, or general pains, and frequently there was no rise of temperature. The patient complains of pain in some joint (in our cases invariably first in the knee) and later there may or may not be effusion into the joints. Case XIII developed inflammation in his knees and ankles, but although there was oedema of the feet there was no effusion into the joints. In other cases there was considerable effusion, but in no case was the joint tense and hot to the touch — the appearance in other words was not that of an intensely inflamed joint such as is found in acute rheumatism. When a second joint becomes inflamed the symptoms in the joint primarily inflamed immediately subside. Though the symptoms were not severe in all cases they were slow in subsiding. In no case did the effusion result in suppuration, nor did the occurrence of the joint affection appear to affect the dysenteric symptoms. Endocarditis or other cardiac affection was not observed in any of our cases. The cause of the joint affection is probably, as Scheube states, secondary infection by micrococci. We have already referred to perforation and gangrene of the bowel as complications of dysentery. The latter occurred in 3, the former in 2 out of our 246 cases and in each case was fatal. In this connection we may mention a thought that has frequently struck us in connection with dysentery. We have seen ulcers
extending through the mucous membrane and even through the muscular and peritoneal coats of the intestine in dysentery, and the ulcers in dysentery are numerous; but while in Enteric fever copious haemorrhage from the bowel is a not infrequent complication yet we have only once seen anything approaching this in the course of dysentery. This was in case of a European boatman aet. 28 years who attributed his attack of dysentery to eating bad fish. Three weeks after the onset he was admitted to hospital and treated with astringents. After a week of this treatment he was ordered Magnes. Sulphat. two hourly, when he passed a large black clot of blood, in appearance like that of an old haemorrhage in Enteric fever. Though both of these diseases consist in ulceration of the inflamed mucous membrane of the intestine, in the one we not infrequently have copious haemorrhage, in the other we almost never have a large amount of blood passed at one time. It is difficult to give a satisfactory explanation of this when we consider that the distribution of the blood vessels in the large is the same as in the small intestine and the ulcers may be of equal depth, and are usually more extensive in dysentery. Stenosis and obstruction of the large intestine is described as a sequela of dysentery. This would appear not to be a very frequent occurrence. Among our cases we have not seen this once, though at post-mortem examinations we have on several occasions seen the calibre of the colon narrowed at parts and dilated at
others, but never approaching any extreme degree likely to cause symptoms of obstruction. During four years' experience in a large general hospital we have not seen a single case of intestinal obstruction due to cicatrization of a dysenteric ulcer, though obstruction due to other causes has been not infrequent.

Peripheral neuritis occurred in the course of four of our cases - two in European and two in native patients. All of these patients were over 40 years of age and the dysentery had assumed the chronic form. G.B.S., European male, aged 43 years was admitted on January 5th. 1901 suffering from chronic dysentery. On January 7th. he complained of pain in the calves of the legs which were tender on pressure. The reflexes were unaltered, but movement of the lower limbs became slow and impaired and the reflexes disappeared. Sensation was impaired, and delayed, but not entirely abolished. The dysenteric symptoms improved and the patient got up on January 28th. He was, however, only able to sit in a chair at the bedside and was unable to progress even with the aid of crutches. The limbs became rapidly emaciated and after he sat up oedema of the feet and ankles supervened. As the acute symptoms passed off massage and the Faradic current were applied to the legs which under this treatment gradually improved, though the dysenteric symptoms returned
Patient's voice became very husky, then almost disappeared. He was only able to speak in a faint whisper difficult to hear, but no complete aphonia took place. All the symptoms gradually improved and patient left the hospital 3½ months after admission. He could then get about slowly by the aid of a stick.

This case illustrates the chief features presented by our cases of dysenteric neuritis, which resembled those of neuritis due to other infectious diseases, such as Enteric fever.

Abscesses of the lung, brain and spleen have been described as complications of dysentery. We have not seen instances of the two latter, but have had one instance of lung abscess on which an autopsy was held. This is fully described in Case XIII. Councilman and Lafleur in 15 cases (op. cit.) found abscess of the lung in three, and in every instance this was secondary to abscess in the upper lobe of the liver.

Out of 246 cases with 17 autopsies we found abscess of the lung in only one case and in this there was no abscess of the liver.

Our case was interesting in more respects than one. The patient was under treatment for chronic dysentery, and presented symptoms of a rheumatic nature—synovitis of knees, ankles and wrists, and later physical signs of pleurisy on both sides. At the autopsy a large abscess of the lung
was found with typical dysenteric ulcers in the large intestine. However, by far the most frequent, as well as the most important, complication of dysentery is abscess of the liver. Of our cases of dysentery, 246 in number, 38 proved fatal, and autopsies were performed on 17 of the latter, in 8 of which liver abscesses were found. To this number we must add another case of liver abscess occurring during the course of chronic dysentery which was operated on and recovered. We have seen four other cases of liver abscess. Two of these were successfully treated by operation and the other two were demonstrated at post-mortem examination. In all of these four cases there was an absence of a history of dysentery and at the autopsy on the two fatal cases no trace of a dysenteric lesion was found. They are consequently not included in the following table. We have not infrequently had cases of suppurating hydatid cysts of the liver, but these are apart from the present subject.

Our experience of liver abscess occurring in connection with dysentery may be represented by the following table.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Deaths</th>
<th>Autopsies</th>
<th>Liver abscess found</th>
<th>Do. successfully operated on</th>
</tr>
</thead>
<tbody>
<tr>
<td>246</td>
<td>38</td>
<td>17</td>
<td>8</td>
<td>1</td>
</tr>
</tbody>
</table>

Total no liver abscesses 9.
This gives an incidence of 3.66 per cent of liver abscess in all our cases and of 21\% in fatal cases of dysentery, or a little over one case of liver abscess in five fatal cases. Councilman and Lafleur, in their work on Amoebic Dysentery give statistics of cases of liver abscess collected from the writings of various authors in India and elsewhere. In India out of 1429 autopsies, there were 306 cases of liver abscess or 1 case of liver abscess in 4\% autopsies on cases of dysentery. In Algiers the proportion was 1 case of liver abscess in a little over five. In Egypt, liver abscess appears to be a very common complication of dysentery among European patients, while Griesinger, in 186 autopsies on native Egyptians did not find liver abscess in one. Kruse and Pasquale, however, found liver abscess not infrequently. All authors agree that liver abscess occurs more frequently among European than among native patients in tropical countries. In Europe and America this complication appears to be comparatively rare.

Six of our fatal cases of liver abscess occurred among European patients, and the one successfully operated on was also a European, the two remaining cases being natives.

36. Councilman & Lafleur. op. cit. pp. 490 & 504
Seven of our nine cases of secondary liver abscesses therefore occurred among European patients. All of these cases were males, whose ages varied from 24 to 60 years. In only one case was a single abscess cavity found. This was the case of a coloured man aged 60 years, admitted in a moribund condition. At the post-mortem examination the liver was found much enlarged and containing a single large abscess cavity in the right lobe containing chocolate coloured pus. The cavity was irregular in shape, and gave the appearance of having been formed of the fusion of two or more abscesses. Apart from this case all the others showed two or more abscesses, in some cases the whole of the right lobe was studded with abscesses.

In the case successfully operated on, a small abscess was opened in the right axillary line and the wall at one spot was felt to be soft and bulging. On opening through this, a second larger abscess was found, which was drained through the smaller more superficial one.

The right lobe is by far the commonest seat of the abscesses. In Case VI three large abscesses were found in the left lobe, while in the right, two were found in the process of formation. In all the other cases the right lobe was the only one affected. In the majority of cases the abscesses occurred superficially, near the upper surface, less frequently near the under surface. In most cases old or recent peritonitis was found over the position of the abscess, causing adhesions between the liver and under surface of the diaphragm, or
anterior abdominal wall. In one case the anterior surface of the stomach was adherent to the anterior abdominal wall and the under surface of the liver by broad bands. The liver as a whole appeared in most cases to be enlarged. In some cases where the right lobe was largely destroyed, the left lobe was found distinctly hypertrophied. The contents of the abscess vary. In cases 8 and 6 the process of abscess formation was seen in its early stages, as well as large old-standing abscesses with liquid contents. In the early stage, on incising the liver tissue, little pale or yellowish areas are seen, the size of which vary from that of a bean to that of a cherry or larger. From the cut surface no liquid contents escape; but meshes of soft yellowish tissue containing a semi-liquid material are seen. The whole on pressure breaks down into a thick caseous mass. This appearance does not seem to be peculiar to liver abscess formation following dysentery. We found this spongy appearance in two cases of liver abscess where there was no history of dysentery, and where post-mortem no lesion in the intestine was found.

In abscesses a little larger, on section, part of the contents escape leaving a spongy looking net work, which readily breaks down on pressure.

In the larger abscesses the contents are a thick liquid containing semi-solid masses of necrotic liver tissue. In case 6 after the operation, the discharge consisted almost
entirely of thick sloughs. The colour of the liquid contents is yellow, or more usually brown from the presence of blood. The walls are usually irregular, and on washing them under a running stream of water, dirty yellow shreds are seen attached to the walls and projecting into the cavity.

The liver substance round the abscess is usually pale and friable, and in some cases shewed the spongy condition of early abscess formation. The rest of the liver substance varied in appearance; occasionally it was congested and dark, but more usually it was pale. In one case it was cirrhotic, and in two it was waxy.

The abscess contents obtained at operation, and also post-mortem examination were frequently examined for amoebae, but always with a negative result.

In all of our cases, liver abscess had probably developed before admission to hospital.

The period at which liver abscess developed after dysentery, in our cases was from 1 to 3 months after the primary attack.

In case XI, the patient died a month after he first contracted dysentery, and post-mortem several small abscesses were found in the liver. Case 8 had first suffered from dysentery in March. In July—four months later—he was admitted with several large abscesses in the liver, which must have existed for a considerable time. Another case, a European male, aged 30 years, had been treated at home for
dysentery. Three months after he had been apparently cured, he was admitted to hospital with two abscesses in the right lobe of the liver. The severity of the attack of dysentery does not appear to have any relation to the frequency of liver abscess occurring.

Six of our nine cases had had no treatment for the dysenteric attack previous to admission to hospital.

The majority of the cases complained of pain in the region of the liver. This was sometimes of an acute nature due to perihepatitis, or of a dull aching nature, due probably to the increased size of the organ. This pain was increased on patient lying on the left side. None of the patients referred the pain to the shoulder. An early sign of formation of liver abscess is rise of temperature (v. charts)

After a period during which the temperature has been normal, or more usually subnormal, the temperature rises at night. At first there may be only an evening rise, to 100° or 103°F, rarely higher, or the temperature may be remittent, and the patient has night sweats. The pulse and respirations become increased in frequency and the patient becomes almost invariably of a sallow or dirty brown complexion. Deep jaundice occurred in case 9, but jaundice is rare in this condition.
The tongue is furred, appetite fails, and the patient becomes rapidly emaciated. On examining the liver, in the early stage no change may be found. In this case the diagnosis must be based on the previous history of dysentery, the intermittent rise of temperature and the profuse sweats. As the abscess increases in size, some enlargement of the liver can be made out. The liver dulness usually increases upwards and this increase is usually confined to the right lobe, though the left lobe may also appear increased in size though not the seat of the abscess. Occasionally the increase takes place downwards, when the liver can be readily felt below the costal margin (cases 9, 6 and 7) In case 7 coarse friction was felt over the liver on inspiration. When the abscesses are of large size or near the surface the side may be seen to bulge and the size of the intercostal spaces increased. In case VI, on admission a bulging could be seen in the epigastrium. In some cases we have seen the superficial veins of the abdominal wall and chest dilated over the enlarged liver. The heart may be displaced upwards and outwards and there may be congestion or oedema of the lungs. The only absolutely reliable diagnostic procedure is exploratory puncture and withdrawal of pus.

Councilman and Lafleur found liver abscess in six of their cases, and in three of them there was secondary abscess of the lung. In none of our cases of liver abscess
was there a secondary lung abscess; but in one case of chronic dysentery there was abscess of the left lung, but none in the liver. (Case 13)

With regard to the termination of liver abscess, we have heard of cases bursting into a bronchus, and thick brownish red pus being coughed up, or into the duodenum, or some other part of the alimentary tract. We have heard of one case - not published - in which a patient after dysentery developed liver abscess and suddenly passed a large quantity of pus per urethram. This continued daily for some weeks and the patient eventually recovered. In this case the abscess had probably burst into the ureter on the right side.

Of our cases two were admitted moribund and died a few hours after, one died of peritonitis due to rupture of an abscess on the under surface of the liver into the peritoneal cavity, in one case the abscesses were small, and discovered only at the autopsy, while the remaining five were incised and drained.

The treatment of hepatic abscess is considered later. The mortality in cases of dysentery varies considerably among the different races. Acute dysentery in South Africa when seen early and treated is not a very fatal disease as shown by the following percentages. The higher death rate among natives than among Europeans is to a large extent due to their neglecting treatment in many
cases during the early stages, and also to the great tendency among them to develop pulmonary affections when kept in bed for any cause. Several of the cases among native patients occurred in subjects debilitated by scurvy. The death rate of all of our cases was: European 7.7%, Native 28.2%.

When classified according to the variety of the disease, the mortality was: European, acute cases 2.3%, chronic 27.2%, gangrenous 100%; Native, acute cases 8%, chronic 52.3%.

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PROGNOSIS.

The prognosis of dysentery depends on the form of the disease, and on the age and constitution of the patient. Briefly, very old people and infants readily succumb.

In those debilitated from any cause such as scurvy, and in alcoholic subjects the prognosis is less favourable than in other cases. The patient stands the acute attack worse, and there is a greater tendency for the disease to assume the chronic form. In three of our cases, where pregnancy existed, all aborted and two died.

With regard to the bearing on prognosis of the form of the disease, our cases shew that acute catarrhal dysentery is not a very fatal disease, while all our cases of gangrenous dysentery ended fatally. In chronic dysentery the prognosis will depend on the personal
history, the duration of the disease, and the presence or absence of the complications already described. Hicough, vomiting, restlessness and incontinence of faeces are unfavourable signs.
The prophylactic treatment of dysentery consists, in a word, in avoiding the predisposing causes already described. We have seen that tainted food or improperly cooked food, impure water, exhaustion, cold or rather sudden changes in the temperature of the atmosphere, may all act as predisposing causes, and these should be all guarded against. With regard to the latter, we have had frequent testimony and personal experience of the value of a flannel binder or belt worn next the skin of the abdomen. Constipation or simple diarrhoea should not be neglected.

The stools of all patients suffering from dysentery should be carefully disinfected. Our rule has been to place half a pint of Jeye's fluid or chinosol (1 in 600) in the bedpan before it is used. After use, a second half pint of either of the above disinfectants is poured over the contents of the bedpan, & this is allowed to stand for 20 minutes before being emptied.

The question of the communicability of the disease from a patient to a healthy individual is still uncertain. 

38 E. Henry, F.R.C.S. quotes an interesting series of cases in this connection. An Officer of a Steamship recently returned from a voyage contracted dysentery

dysentery which subsided in a fortnight. Later his wife who had been nursing him began to pass blood & mucus, and her attack also lasted a fortnight. One of the two children remained in the house, in a separate room, seeing his parents only occasionally, and he developed the disease after the mother had recovered. The second child who had been sent away returned to the house a fortnight after the first had contracted the disease, and two days after his return, similar symptoms occurred & he died three days later. The Grandmother of the children who had been nursing them was attacked in a similar way a fortnight after the second child. Henry concludes "The infection was doubtless contracted in the first case during the voyage, and was communicated from case to case, as disinfection was necessarily imperfect & isolation impossible".

If dysentery is directly communicable from a patient to a healthy individual, we believe that with reasonable precautions, the risk of this occurring is slight. Our cases of dysentery were treated in the general medical wards, and in our experience extending over four years, only two patients admitted for other causes contracted dysentery— which lasted three days in one case, & a week in the other— while in the wards. In these cases however, there were no dysenteric patients in the same wards. Two of the nursing staff during the same period
period contracted mild forms of dysentery. One from her position, was not brought into close contact with the patients while the other who had recently started nursing had been on duty in a ward in which no cases of dysentery then were. None of these cases can we think be regarded as cases of direct infection from a dysenteric patient, and if this does occur we believe it to be rare.

Diet is of the greatest importance in the treatment of dysentery. Most writers state that this should consist almost entirely of milk, but we think that most patients in cases of acute dysentery do better without milk in the early stages. Milk readily forms curds which easily decompose in the intestinal canal, and further irritate the inflamed mucous membrane. Even where the milk has been diluted with lime water, we have frequently detected undigested milk curds in the stools of patients where this diet has been prescribed, and post mortem we have found thick curds adhering to the surface of the ulcers. The diet we have found to be best is one consisting of water arrowroot and clear chicken broth with a little common salt added. In children we frequently give only albumin water made from the white of egg, alternated with barley or toast water. Later when bile or faeces appear in the stools, Benger's food made with water or milk, or dry toast may be added, and the diet gradually increased according to the character of the stools. These should
should be daily examined and the diet modified entirely according to the result of this examination.

With regard to the drug treatment of dysentery, we intend only referring to the various drugs which have been used in this disease, and give generally our experience of their efficacy in South African dysentery. We have not arranged our cases in tables with number of cases, duration of disease & mortality for each of the several methods of treatment adopted, for these tables would be useless for several reasons. Thus the total number of our cases is too small to prove the efficacy of any particular method of treatment over another, in many cases the patients rapidly get well when placed in bed and suitably dieted, whether this treatment is supplemented by drugs or not, and further in many of our cases two or more drugs were used either in combination or at different periods of the disease, so that it was difficult to attribute the result to one drug more than to another.

Among drugs recommended for this disease the first to claim attention is ipecacuanha, introduced about the middle of the seventeenth century. This drug when first introduced had much difficulty in becoming generally accepted, but later, came to be looked upon as a specific in this disease. This was particularly the case after Docker had re-introduced the treatment by large doses in his cases of dysentery in India. The method of
of administration consisted in starving the patient for an hour or two then administering T₉. opii m xx followed twenty minutes later by pulv. ipecac. radio. gr. xxx to xl. If necessary a sinapism was also applied to the epigastrium. The patient was then recommended to remain recumbent and resist the desire to be sick as much as possible. All nourishment was avoided for two or three hours after the administration of the ipecacuanha. This method of treatment gave wonderfully successful results especially in India. Lately, however, some doubt seems to have occurred in the minds of practitioners in tropical countries as to the efficacy of this method of treatment, and papers have been published from practitioners in tropical countries comparing unfavourably the results obtained from this method as compared with those obtained from others. To obviate the distressing nausea caused by ipecacuanha, others have recommended ipecacuanha sine emetine. This, however, has from opinions collected, given rather less satisfactory results than simple ipecacuanha powder. Some of those who report favourably on the use of ipecacuan in this disease attribute the whole of its efficacy to its emetic principle.

In the scanty literature on dysentery in South Africa, Dr Washbourn writing of his experience of

of dysentery in the Yeomanry Hospital, Deelfontein, Sou. Africa states "during the acute stages (of dysentery), the symptoms usually yield readily to treatment with ipecacuanha or with magnesium sulphate".

In "a civilian war Hospital" the writer on the chapter on dysentery as observed among the troops in the present Anglo-Boer war says "the ipecacuanha treatment which is so successful in tropical dysentery seems to be much less so in the South African variety". Maberley & Richardson (op. cit.) also report on the inconvenience to the patient and inefficacy of ipecacuanha in cases of dysentery in South Africa. Our own experience entirely coincides with that of the latter writers. When we first began to treat cases of dysentery four years ago, we found a strong prejudice against the use of ipecacuanha on the part of experienced practitioners in the country. We however gave the pulv. ipecac. a fair trial in a number of our earlier cases with most disappointing results. It appeared to have no influence on the number or character of the evacuations or on the tenesmus, while it invariably caused nausea & depression. In almost all of the cases, after trying this drug for some days without effect we had to abandon it and have recourse to other methods of treatment. In certain mild cases, the patients recovered in a few days under this treatment.

treatment but most mild cases will recover rapidly when merely placed at rest in bed & suitably dieted. In acute cases of moderate severity & in all chronic cases, ipecacuanha appeared to have no appreciable influence on the disease. Before administering ipecacuanha it is the custom to give a dose of tinct. opii to prevent nausea, and this we think is a further drawback to the ipecacu¬ahan treatment.

The general experience in South Africa is that a purga¬tive line of treatment is the most satisfactory, and by giving opium one goes directly against this general ex¬perience. On theoretical grounds the practice of giving opium in dysentery is to be condemned. It certainly easies the pain & tenesmus, but by interfering with peristalsis of the bowel, & diminishing the secretions of all the glands in, and associated with the alimentary tract, it allows of accumulation of intestinal contents on the floor of the ulcers or on the inflamed mucous membrane, and diminishes the quantity of glandular secretion which acts beneficially by flushing the intes¬tine, and keeping the surface of the ulcers clean.

In practice we have found that the use of opium is only justifiable in exceptional circumstances. As a routine practice or as an essential element in the treatment it cannot be too strongly condemned. The following two cases are typical of the effect of this method of treat¬ment. P.S. European male aged 30 was admitted suffering
suffering from dysentery of three weeks' duration. A preliminary dose of Magnes. Sulphat & Magnes. carb. was given & then Tr. opii M xv 4 hourly. The number of the stools remained at from six to thirteen in the twenty-four hours. They contained blood mucus & shreds, and were very offensive. The patient complained of constant & severe "gripping" pains in the abdomen and the tongue was dry and brown in appearance. After six days of this treatment, Magnes. Sulphat. 1 drachm two hourly was prescribed. Patient passed several large dark coloured and very offensive stools with immediate amelioration of the symptoms especially of the abdominal pain. The patient then made a complete recovery. R Mc G., European male aet. 38 years admitted suffering from chronic dysentery of seven months' duration. He was ordered rest in bed, broth & arrowroot diet and opium 1 gr. thrice daily. During the next four days patient had nausea after each dose of opium, the stools remained at from seven to nine per diem, were offensive, shreddy, & accompanied by abdominal pain. At the end of this period the opium was stopped, & mages. Sulphat. 2 drachms every morning & Salol gr x thrice daily were ordered. The patient began immediately to improve. The nausea and the abdominal pain disappeared, and three days later patient was passing normal formed stools. Three weeks later all symptoms of dysentery had disappeared. Our experience of opium as a curative agent in dysentery, in any stage, is
that it is not only disappointing but positively harmful. Dysenteric ulcers are found most frequently in the situation where faeces are most prone to collect and opium by causing retention of the intestinal contents merely aggravates the complaint. In certain exceptional circumstances, however, opium is useful. In case 3, where patient was becoming exhausted from want of nourishment through sickness and loss of sleep, a hypodermic injection of morphia enabled him to retain small quantities of liquid nourishment, and to have some sleep with relief from the constant straining and desire to go to stool. In other cases where the rectum is in an irritable condition, and where this interferes with the administration of rectal irrigations, an Enema of Starch 2 oz., & tr. opii. M xxx, enables a larger amount of fluid to be injected without its being immediately expelled and consequently the ulcers are more thoroughly cleansed. In this respect, a cocaine suppository acts equally beneficially. We regard opium, therefore, as having only a baneful influence in the ordinary case of dysentery, as merely checking the evacuation of the intestinal contents is harmful. In rare cases, however, opium is useful as an auxiliary in preventing sickness, and allowing of the administration of nourishment or of other drugs or in enabling more complete lavage of the large intestine to be carried out.

In the Lancet of Feb. 6th & 13th 1897, Dr Maberley

42. Maberley. op. cit. p.368 & 433.
Dr Maberley gives a detailed account of his remarkable success in the treatment of dysentery by a tincture of the Monsonia Ovata. This plant, which is found widely spread over South Africa, is an annual, and Maberley states that it should be gathered in January & February. The whole plant is used and a tincture is prepared by macerating $3\frac{1}{2}$ ounces in a pint of rectified spirit. Of this tincture two to four drachms are given four hourly. Maberley reports 100 consecutive cases treated by this drug. Of these 90 were acute, and all of these recovered, the average duration of treatment being 2.3 days. Of the 10 chronic cases nine recovered, the average duration of treatment being 8.1 days.

We tried the effects of this tincture of Monsonia— that obtained commercially and some specially prepared from carefully collected plants— in about 40 cases. Our results were not at all as satisfactory as Maberley's. Mild cases recovered rapidly under its administration as they will with simple rest in bed & dieting. More severe cases and chronic cases, appeared to be influenced by it, and in the majority of our cases in which it was tried, it had eventually to be abandoned and some other method of treatment substituted.

The method of treatment which for long has been regarded the most successful in South Africa is the purgative, and various drugs have been used. A favourite combination
combination with pioneers is ol. ricini \( \frac{1}{2} \) oz, tinct. opii Ms.XV taken at the onset of the disease and repeated as required later. A prescription which has been used during the past twenty years in this Hospital with great success in dysentery is hydrarg. subchlor. gr.V, pulv. jalapae. co. gr. 45. One of these powders is given as early as possible in the disease, and repeated the following day if the stools still contain blood & mucus. The drug, however, held in greatest favour by those who adopt the purgative treatment is Magnes. Sulphat or Sodii Sulphat. Magnes. Sulphatis was recommended by Heberden at the end of the 18th century. Trouseau states that Bretonneau following the plan adopted by Stoll, Zimmerman & Pringle, re-introduced the treatment by purgatives in 1823, and that this plan was attended by success. Trouseau himself advocated the treatment of dysentery with the sulphates of Magnesia & Soda, or with large doses of Calomel (gr. 27) morning & evening, as recommended by Amiel in 1812, from his experience of dysentery among the British troops in Gibraltar. Trouseau further strongly condemns the use of opium except to allay pain or prevent sickness. This line of treatment was almost entirely superseded by ipecacuanha. Though many of the English textbooks mention treatment by the neutral Salts, it is always as quite subsidiary to the ipecacuanha treatment.

43. Trouseau op. cit. p. 177.
Leahy writes in favour of the Magnesium Sulphate treatment as a result of his experience of 95 cases of acute dysentery in India. He sums up its advantages over Ipecacuanha as follows:

(1). It has no depressant action on the system.
(2). It neither produces nausea nor vomiting.
(3). It quiets and soothes the patient.
(4). It acts beneficially on the mucous membrane by relieving hyperaemia & preventing the formation of ulcers, or placing those already formed in the most favourable condition to undergo a cure.

Wyatt-Smith writing on his experience of Magnesium Sulphate in the Hospitals of Buenos Ayres & Monte Video concludes, that-

(1). Ipecacuanha (in dysentery) is useless, if not worse.
(2). That opium is positively poisonous in these cases.
(3). That the treatment of dysentery is essentially purgative.
(4). That Magnesium Sulphate is practically a specific.

During the last three years we have found nine articles in the Lancet & Br. Med. Journal giving the experience of Surgeons in the Army & Navy & practitioners in Jamaica, the Niger Coast, Trinidad, Mauritius & India, all generally indicating the superiority of Magnesium Sulphate over Ipecacuanha.

over ipecacuanha in the treatment of dysentery. We first tried the Magnes. Sulphat. treatment in 1897 and the results appeared to be somewhat disappointing, or at any rate not so brilliant as those recorded in India or other tropical Countries. This result was probably due to the fact that the cases in which this drug was first tried were either subacute or chronic. We then tried the Tinct. Monsonia, and as this also proved disappointing after a lengthy trial, we again returned to the purgative treatment. The results were then so favourable that it has become our routine practice in Hospital. Of 98 cases of acute dysentery among Europeans & 30 among Natives, the death rate has been nil among Europeans, & 1 among Natives. The latter case was pregnant and aborted soon after admission. The average duration of the acute attack under this treatment was six days as compared with ten to fourteen days generally. In chronic cases this line of treatment is not so successful. A saturated aqueous solution of Magnes. Sulphat. (i.e. 1 in 2) is made, & this is given in 2 drachm doses two hourly, until the character of the stools improves and then less frequently. Aromatic Sulphuric Acid M.X may be added to each dose to prevent griping. Sodii. Sulphat. which acts as efficiently as Magnes. Sulphat. appears to be less apt to cause griping.

In the majority of cases the beneficial effects are soon evident. Sometimes before the purgative action of
of the drug occurs, there is diminution of the pain & tenesmus. Usually after six or eight doses the character of the stools is greatly improved. They become more copious, the blood and then the mucus disappears and the stools become watery, bile returns and they become yellowish or brown and later faecal. The Magnes. Sulphat. may then be gradually diminished. When the stools become bile coloured, it may be given four hourly, then thrice daily, and it is necessary to continue its administration for a day after all blood & mucus has disappeared to prevent recurrence. Should this occur the Magnes. Sulphat. should be again increased. As the character of the stools improves an intestinal antiseptic, such as Salol, should be added to the treatment. During convalescence the bowels should be kept regular by an occasional dose of Magnes. Sulphat. To get the best results with Magnes. or Sodii Sulphat. the treatment should commence early & it is then almost specific.

The action of the Sulphates is undoubtedly due to their increasing the secretion of the glands of the alimentary tract & liver. The return of bile to the stools is always a favourable clinical sign. Acting as a hepatic stimulant they cause an increased flow of bile to the intestinal canal, and this acts as a mild antiseptic & purgative. By causing a watery discharge from the intestinal glands, they diminish the hyperaemia of the inflamed
inflamed mucous membrane which is at the same time flushed & cleared of any irritating substance in contact with it.

In certain cases, especially when not treated from the commencement of the disease, the Sulphates are less effectual, and in these cases their action may be supplemented by local treatment. Many substances have been recommended in the form of enemata. Osler recommends Quinine in the strength of 1 in 5,000 and this may be specially useful in the amoebic form. Others recommend Salicylic Acid, perchloride of Mercury, dilute Silver nitrate &c. We have found great benefit from the use of Enemata in acute dysentery, but think that the whole benefit is derived from the mechanical cleansing of the diseased mucous membrane & soothing of its inflamed surface. The results obtained from the use of the above solutions seem in no way different from those obtained by using Enemata of Boracic Lotion, infusion of Chamomile flowers or plain water. We usually employ boracic lotion at the temperature of 99°F. Two to three or four pints of this are slowly injected into the rectum and the patient allowed to pass it. Occasionally the rectum is so irritable that not more than half a pint can be injected. In this case a starch & opium enema or cocaine suppository may be administered shortly before the irrigation. The unpleasant effects of this irrigation are almost nil. There is very little discomfort, but
but twice patients complained of a feeling of faintness during the irrigation. The beneficial effects are most marked. After the enema there is invariably amelioration of the pain & tenesmus, the stools become less frequent and improve in character. We have seen patients kept awake for nights from the pain and constant desire to go to stool, who after an enema of Boracic Lotion have felt comfortable and gone quietly to sleep a quarter of an hour later. In severe cases two irrigations, in milder cases one daily are usually sufficient. Several of our acute cases were treated solely by rest, diet & rectal irrigations with favourable results. Cases 1 & 3 illustrate the effects of this treatment, while in case 2 the enemata were combined with Magnes. Sulphat by mouth. The following case is interesting as shewing the rapid improvement after irrigation of the bowel when Magnes. Sulphat. had failed. A.F. aged 19, European Seaman, was admitted on Sept. 17th, 1901. He had been suffering from "looseness of the bowels" and "pain in the stomach" for seven days previously. On admission the temperature was 100·F, tongue thickly coated with white fur, and there was great tenderness over the region of the sigmoid. He passed eight stools which were faecal, bile stained and contained small lumpy blood-stained mucus. Magnes. Sulphat. 1 drachm two hourly was ordered. On the 18th. he passed eleven, on the 19th. seventeen
seventeen small stools consisting of blood & mucus only. There was severe abdominal pain and tenesmus. The pulse rate was 120, thready, & patient vomited everything he took.

On the evening of the 19th, Morph. Hydrochlor. gr. 1/6 was ordered hypodermically, and a rectal irrigation consisting of four pints of warm Boracic Lotion once daily. On the 20th., the temperature rose to 102 F. Patient felt more comfortable, no tenesmus or sickness, there was slight tenderness over the sigmoid flexure, stools contain bile, faeces & mucus. On the 21st. there were ten relaxed yellow stools, no blood or mucus, temp. 100 F. Temperature was normal on the 23rd and patient had three relaxed stools. After this he made a rapid recovery.

Many recent writers fail to make mention of the treatment by Enemata and others state that this remedy should be used with caution. Manson states that Enemata should never be used when acute symptoms are present. Osler states that he regards Enemata in cases of acute dysentery with some disfavour on account of the acute pain caused when the diseased bowel is distended with fluid. He mentions that there is also exhaustion after their use and considers them most useful in chronic dysentery.

Sandwith speaks of the failure of ipecacuanha in his cases of acute dysentery treated in Cairo, & of the

of the great value of Enemata. He had tried them in only twenty cases, using first a solution of silver nitrate and later of Copper Sulphate, and thinks that Enemata should be given as early as possible. Prentice in his cases of acute dysentery in Central Africa found Enemata relieved tenesmus.

Our own experience of this remedy in a number of cases has been entirely favourable. There is an almost entire absence of unpleasant symptoms, the tenesmus is invariably relieved and the symptoms improved. The earlier the Enemata are given the greater the relief, especially of the tenesmus. Furthermore, this treatment is the most rational. The disease is at first confined to the lower bowel, and local treatment is specially indicated, and at this stage there is no danger of perforation of the bowel from distention by the fluid. Mild non-irritating fluids such as Boracic Lotion or plain warm water, are better tolerated in the early stage than astringents, such as the silver or copper salts. Alcohol should always be avoided, as it increases the pain & tenesmus and aggravates the other symptoms. In the rare cases where a cardiac stimulant is required, Ammonia, Caffeine or Strychnine should be used in preference to Alcohol.

In the treatment of chronic dysentery, diet is of prime importance. This should consist of clear broths, diluted milk, or some artificial food such as Benger's, made with

with water & milk. If curds of milk appear in the stools the milk should be peptonized. Later dry toast, then fish may be added. The gradual increase in the diet should depend entirely on the number and character of the stools. In certain obstinate cases a purely meat diet has been recommended, but this treatment has not given favourable results in the few cases in which we have tried it. Care should be taken to keep the abdomen warm, especially during convalescence when the patient is going about. A malarial or scorbutic taint should be looked for, and treated if present. In the latter case fresh fruits such as the juice of Oranges & Grapes or Strawberries are useful. Alcohol is rarely indicated except in cases of great exhaustion, when a small quantity of Port Wine may be useful during convalescence.

With regard to treatment by drugs, this is much less satisfactory in chronic than in acute dysentery. Ipecacuanha is useless and opium harmful and should never be prescribed. There is an absence of tenesmus so that hypodermic injections of morphia are not required for the relief of this symptom. Abdominal uneasiness or colic can usually be relieved without morphia, and the administration of opium by mouth almost invariably aggravates the symptoms.

The method of treatment which has given the greatest success in our hands is a combination of Magnes Sulphate
Sulphate, and some intestinal antiseptic. Two drachms of Magnes. Sulphat. are given at the outset and this dose is repeated every morning. Some antiseptic is given by the mouth. We prefer Salol in 10 grain doses thrice daily, as Salol splits up into its components in the small intestine and is more likely to continue to exert its action in the intestinal tract than other more diffusible antiseptics.

In cases where this fails we have had good results from liquor hydrarg. perchlor. M. XXX and tinct. cannab. indic. M.30 given thrice daily, freely diluted.

In two cases where other means had failed, recovery took place after three weeks' treatment with Bismuth. Subnitrat. gr. 30 given four hourly.

Many other drugs have been advocated, such cinnamon powder in drachm doses thrice daily, Ammonium chloride, liquor ferri pernitratis; but none of these appeared to have any special influence over the disease in the few cases in which we tried them.

A most valuable adjunct to the above described methods of treatment is the local treatment by large Enemata of water, medicated or not. Many authors write in favour of this, most of them recommending some particular drug for use in the Enemata.

50 Trousseau used "styptic & caustic lavements with Sulphate of Zinc, Sulphate of Copper, & Nitrate of Silver".

50. Trousseau. op. cit. p. 181.
Wood recommends nitrate of silver (½ to 1 drachm) in two or three quarts of water.

Gallay uses silver nitrate gr. 20 to 1 litre of water, and recommends a series of daily washings of the large intestine with this solution. He gives no drugs by the mouth and allows the patient to feed himself as he pleases. He finds that if a series of sixty daily irrigations is carried out that the result is invariably a cure. One would think that if he used this as supplementary to other methods of treatment that fewer irrigations would be required to effect a cure.

Other drugs such as Alum & Copper Sulphate are recommended by various writers.

We have found large Enemata of considerable use in chronic dysentery. In case V after four months' treatment by various methods without success we prescribed an Enema of two quarts of saturated solution of Sod. Bicarb. to dissolve the mucus and clean the surface of the ulcers, then an injection of silver nitrate ½ gr. to the ounce of distilled water followed half an hour later. Patient was directed to retain this as long as possible. Five days later the symptoms had all disappeared and patient at his own request returned home.

As a rule when irrigations of silver nitrate of this strength (gr. ½ to 1 drachm) of which three to four pints

Wood. The Lancet. Aug. 22nd. 1891. p. 433

pints are slowly injected, are used, the patient complains of a little pain and desire to immediately evacuate the bowels, but the discomfort is slight. Treatment by irrigation of the bowel, as stated, has given good results in our cases, and the rationale seems to us to be sound, for by this means the surface of the ulcers is kept clear of irritating substances and the ulcer placed in a favourable condition for healing. No one method of treatment is applicable to all forms of chronic dysentery, however, and some cases resist all forms of treatment. In these cases it might be justifiable to perform colotomy, as suggested in the Lancet. This procedure would rest the ulcers by giving escape to the faeces higher up, so that they should not pass over the surface of the ulcers, and at the same time the bowel could be irrigated from the artificial opening in the colon downwards towards the anus. The treatment of the various complications and sequelae requires no special description. Hepatic abscesses require drainage at the earliest possible moment, and this is best effected by means of an open incision. The method advocated by Manson of inserting a drainage tube through a trocar has several disadvantages. It drains only one abscess and in our experience, secondary liver abscesses are more usually multiple, and the large sloughs occasionally found in liver abscesses cannot pass through a fine drainage tube.

SUMMARY

In summarising our experience of dysentery in South Africa, we note that the etiology is still uncertain. Amoebae have not yet been found in cases of South African dysentery and in clinical features it differs from those of "Amoebic Dysentery" described by other authors. The latter has more tendency from the first towards chronicity, and the characters of the ulcers also differ from those in our cases. We never found extensive detachment of the mucus membrane with fistulous openings through it.

With regard to dysentery generally, many organisms have been described as the cause, but no one cause has been shewn as the invariable originator of the disease. Probably there are several forms of dysentery due to several organisms just as there are several varieties of pneumonia. The term dysentery is still rather a clinical than a pathological one. Just as no one cause has been proved in this disease, so there is no one specific treatment.

In acute dysentery we have got the best results from frequent small doses of Magnes. or Sodium Sulphate and from rectal irrigations. In chronic cases, irrigations with large quantities of some astringent such as silver nitrate and an intestinal antiseptic by mouth appear to have the most influence over the disease.

In both forms the diet is of considerable importance. Alcohol & opium should be avoided in acute as well as in
in chronic cases, as aggravating the complaint, and ipecacuanha though not acting deleteriously, is in our experience useless alike in the acute and the chronic form of the disease.

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1895
Rectum and descending colon from a case of chronic dysentery.

Ascending colon from a case of chronic dysentery.
Part of transverse and descending colon from a case of chronic dysentery.

Calcium from a case of gangrenous dysentery.