REMARKS ON

COLITIS

WITH SPECIAL REFERENCE TO

ITS INCIDENCE AND TREATMENT IN SOUTH AFRICA.

THESIS

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by

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Introduction:

The condition known as Colitis or Mucous Colitis is of frequent occurrence in general practice.

Although much has been written on the subject, there are many points connected with the disease which need elucidation.

During a period of 17 years general practice in South Africa I have had under my care a considerable number of cases of Colitis of varying types and in the present theses I have attempted to give my views on the subject, based on my experience in South Africa.

Let it be remembered that conditions, in some instances very primitive, differ materially from conditions in the old country. Owing to the open air life we find a race more sturdy constitutionally than in England.

Facilities for successful treatment, owing to the distances, are also very variable in South Africa.

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Classification.

The text books describe Mucous Colitis and Ulcerative Colitis as distinct entities, but in my experience the condition Mucous Colitis may quite readily become Ulcerative in type though there are a number of cases which commence as Ulcerative Colitis without any previous non-ulcerative stage.

I will adhere to this subdivision in the present thesis and discuss first the type known as Mucous Colitis and then the condition known as Ulcerative Colitis.

It must be remembered that there are many other conditions in which Colitis occurs - Thus Exogenous Ulceration due to an abscess rupturing into the bowel, e.g. an appendix abscess, malignant ulceration, stercoral ulcers, diverticulitis, typhoid and paratyphoid ulcers, uraemic ulcers - Tubercular ulceration and dysenteric and amoebic ulcers.

My observations will deal chiefly with the types usually described under the term mucous colitis and idiopathic ulcerative colitis, but a few cases will be appended in which the Entamoeba histolytica was the etiological factor.

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Colitis

The term "mucous colitis" is in my opinion an insufficient one to define the condition in all its diverse manifestations. It is quite true that at certain stages of the disease, this term expresses the condition present, viz. an inflammatory state of the colon with the passage of an abnormal quantity of mucus, but the condition has so many other manifestations that the name mucous colitis only presents a very inadequate picture of the entire disease. The same applies to the longer name Mucomembranous Entero-Colitis which seeks to express the existing pathological and clinical state more fully - but this also, though more accurate, is only descriptive of one phase of the whole syndrome. With these reservations the term "mucous colitis" will for convenience be retained in the present study of the subject, as it has been used by practically all who have contributed to the literature of the disease.

Before proceeding to give my views of the nature and pathogenesis of this affection I will give a brief account of a typical case:-

The patient generally a woman of the leisured class complains of abdominal pains with the passage of mucous in the stools: The latter symptom is

probably the one which has led her to seek medical advice. The history shows a period of "trouble with the bowels", of several years duration, for which various kinds of laxatives, purgatives, etc. have been used with more or less success. The abdominal pains may be confined to the caecal region and a diagnosis of appendicitis is often made and in many cases the patient is found to have already had her appendix removed, during a previous attack of so-called appendicitis as in the first of my series of cases. On examination the patient is generally found to be thin and poorly nourished as a result of a restricted diet on which she has been living. There are numerous neurasthenic symptoms, depression, anxiety, worries over domestic affairs. There is malaise and loss of appetite. The abdominal wall is flaccid - there may be some enteroptosis of the abdominal viscera - the outline of the whole colon may be palpable as a thickened cord. On examination of the stools the appearances are very striking. Sometimes the dejecta contain large quantities of mucus which may be watery in appearance or like the white of raw egg. In other cases the mucus is white like boiled white of egg or there may be membranous shreds or large pieces of membrane which

may be confounded with taenia. In some cases the shreds are haemorrhagic and there are clots or streaks of blood. In other cases the haemorrhage is more serious and the patient is very gravely ill, the clinical appearances are those of an Ulcerative Colitis.

After a period varying from one to five or six weeks, under suitable diet and treatment, the pain subsides, the mucus and shreds disappear from the stools and the patient is more or less restored to her previous health; There may be a history of several previous attacks of this kind varying in severity from mild attacks which have passed off in a few days to very grave attacks which have lasted two months or even longer. During the periods between the attacks the patient may be free from pain or the passage of mucus.

In addition to the actual symptoms of mucous colitis patients often give a history of certain other affections from which they have suffered at some period or other. Thus such patients are frequently found to have suffered at times from Haemorrhoids, Asthma, Dyspeptic troubles, Floating Kidney, etc.

The above is very briefly a typical history of

a case of mucous colitis, but there are wide variations in the duration and severity of the attacks and in the periods of intermission. The patient may go on for many years suffering from repeated attacks resulting in chronic invalidism - In other cases less common the attacks gradually increase in severity, eventually taking on an ulcerative type and only surgical intervention can prevent a fatal termination.

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Etiology and Pathogenesis.

The one important feature present in all cases of mucous colitis is a history of long standing constipation. It may be perfectly true that many cases of constipation do not lead to mucous colitis or any other disease, Some patients may suffer from constipation for years and show no ill effects; it is equally true that a careful inquiry into the history of cases of mucous colitis discloses a long pre-existing period of constipation. I do not intend within the limits of this thesis to deal with the vast subject of constipation, which embraces all varieties from simple transient cases of retarded evacuation to the comprehensive syndrome which has been described by Lane and others under the term Chronic Intestinal Stasis, but it is important to note here that constipation may exist and yet daily or even more frequent actions of the bowels may occur.

To borrow a classification from another department of medicine, cases of constipation may be divided into "manifest" and "latent". In the former the diagnosis is obvious, but in the latter the constipation may be hidden either by co-existing diarrhoea due to decomposition of long retained

faeces or by frequent incomplete evacuations.

It is well known that many patients may go on for years with evacuations occurring only every two or three days or even longer intervals without any obvious ill effects. In other cases if a daily evacuation does not occur the patient suffers from malaise, loss of appetite, nausea and other symptoms.

In order to understand this phenomenon it is important to consider a few points relating to the functions of the various parts of the large intestine in man:-

The food as it passes from the ileum first fills up the ascending colon which becomes distended. The distension causes a stimulation to contraction. It has been observed under the X-Rays that this contraction passes along from the hepatic flexure backwards towards the caecum, that is by a wave of anti-peristalsis. These contractions have the effect of forcing the food into the caecum. Regurgitation into the ileum is prevented by the oblique ilio caecal valve. As more material enters the large intestine the caecum gets distended and contractions occur in the normal direction, thus there are continual waves of anti-peristalsis and peristalsis, affecting the Caecum and ascending colon which

cause a thorough churning up of the contents which are gradually forced along the transverse colon. Every wave of peristalsis of the caecum begins in the tip of the appendix, passed along the whole organ into the caecum, and converts the appendix into a hard, stiff, cord-like structure.

I was fortunate enough on one occasion to observe this peristalsis while I was removing a huge faeco-lythic appendix, the size of a normal thumb.

During this kneading and mixing process the greater part of the water in the intestinal contents, as well as nutrient material, is absorbed and as the mass approaches the splenic flexure it becomes drier. Distension of the transverse colon and splenic flexure produce waves of peristalsis which drive the dessicated mass into the descending colon. Observations by X Rays show that the descending colon is never distended, the function of this segment of the colon is merely propulsive, and the intestinal contents are finally driven into the sigmoid flexure which may be considered as a storehouse of the faeces.

Finally increasing distension of the sigmoid excites reflexly through the pelvic visceral nerves a complete evacuation of this portion of the gut

and the rectum.

It will be obvious from this brief sketch that the functions of the colon differ widely in different parts and the active part of the large intestine is on the right side - viz. the caecum and ascending colon - Any disturbance of function there, will result in a different series of phenomena from those due to a disturbance of function on the left side. In either case stagnation may occur, but whereas on the left side it may be due to mechanical trouble or failure to respond adequately to a normal stimulus, on the right side it is due to a disturbance of the motor activity of the caecum and ascending colon. Stagnation in this part of the colon where the fluid contents are rich in microbes is obviously of more serious import than stagnation of a dessicated mass in the sigmoid, the microbes of which are probably all dead.

Hence retention of faeces in the caecum and ascending colon gives rise to greater absorption of microbial toxins and the resulting symptoms of toxaemia are more marked than in cases of left sided stagnation.

Now in mucous colitis an important feature is the toxaemia shown in mild cases by lassitude, mal-

aise, headache, etc. and in severer cases by vomiting, pyrexia and sweats - Consequently it is fair to assume that the right side of the large intestine is originally the seat of the trouble in such cases. And this is shown clinically also by the frequency with which the symptoms of mucous colitis are ushered in by an attack of so-called appendicitis.

What is the essential nature of this disturbance of the right side of the colon? Here we enter into realms of mere speculation. Is it the nature of the food?, or the mode of life?. The loss of tone of the abdominal musculature with consequent entero-ptosis? Is it a mechanical factor due to certain type of caecum? Is the primary trouble in the sympathetic nervous system as the Plombieres School believe?

Our knowledge at the present time does not permit us to answer these questions satisfactorily though much may be said in favour of each of these theories. They must be left for further research.

To summarise briefly my views as to pathogenesis:- Mucous Colitis is the result of long continued constipation acting on the right side of the large intestine leading to irritation of the mucous lining and the outpouring by the intestinal glands of large quantities of mucus which may become in-

spissated and be discharged as casts of the bowel wall. Continued irritation leads to superficial erosions and later there may be interstitial inflammation and ulceration of the mucosa, with organic changes in the muscular wall of the intestine leading to the grave symptom associated with Ulcerative Colitis.

All states are met with clinically - The neurotic symptoms may be due to the abnormal stimulation of the nerve terminations of the sympathetic in Auerbach's and Meissner's plexuses.

The next stage of the subject is a consideration of the mode of development of the later features of mocous colitis.

As previously mentioned the characteristic feature of mucous colitis is the passage at intervals of quantities of mucus in the stools. What is the cause of this? The lining membrane of the large intestine contains large numbers of simple tubular glands. The greater number of the cells, lining these glands are typical "goblet" cells and contain plugs of mucin. The secretion of mucus not only aids the passage of the faeces along the gut but probably inhibits the growth of bacteria. It can readily be understood that these goblet cells may by abnormally increased activity, secrete very

large quantities of mucin. It is well known that although bacteria are numerous in the faeces it is difficult to cultivate any large numbers, most of them being dead.

In herbivora the large intestine has an important absorbent function. The nutritious material - protein carbohydrate etc. present in vegetables is inclosed in cells surrounded by cellulose walls and before the digestive juices can act, these cellulose walls must be dissolved. This is done by the agency of bacteria.

In the horse and rabbit the chief part of the digestion of cellulose as well as the contents of the vegetable cells set free by the solution of the cell walls are gradually absorbed by the walls of the large gut.

In carnivora the large intestine has very unimportant functions to discharge in digestion and absorption.

The proteins of meat are practically entirely absorbed by the time the food has arrived at the ileocolic valve and the same applies to fat.

In man the importance of the large intestine will vary with the nature of the food.

Under the conditions of civilised life the food material is almost entirely absorbed by the time it reaches the lower end of the ileum.

If however, a large quantity of vegetable food be taken such as fruit or green vegetables or cereals roughly prepared, or coarsely ground a considerable amount of material may reach the large intestine unabsorbed.

A certain proportion of this may undergo absorption in the large intestine while the large part is merely evacuated giving bulk to the faeces.

The indigestible cellulose in the food is not without value. If the food were entirely digested and absorbed the amount of faeces would be limited to that produced by the intestinal wall itself.

The small bulk would exercise very little stimulating effect on the intestine and the movements of the latter would tend to become sluggish. The presence of a certain amount of cellulose in the diet of the normal individual is therefore very useful, giving bulk to the faeces and by the resulting stimulation to contraction, ensuring the regular evacuation. It is probable that the constipation which is so common a disorder in civilised communities is due as much to the refinement in the prepar-

ation of the food as to the prevalence of sedentary modes of life.

The production of mucin in cases of mucous colitis is abnormally increased.

What is the cause of this abnormal increase?

A study of pathological anatomy gives us very little help in solving this problem.

Autopsies are rare in this disease. Most observers are agreed that the lesion of the large intestine is inflammatory in character. They are superficial, consisting of simple irritation and exaggerated desquamation. Other inflammatory lesions including superficial erosions, and even ulceration occur in the later stages.

In addition to the type of Colitis just described which commences as a slight-non-ulcerative form and occasionally gradually increases in severity until it becomes a true ulcerative colitis, there are types which show the features of ulcerative colitis from the start. This group of cases has been termed Primary Ulcerative Colitis or Idiopathic Ulcerative Colitis.

The study of primary ulcerative colitis may be said to have originated with the writings of Sir Samuel Wilks, who in 1859 first suggested that this

condition was distinct from epidemic tropical dysentery and held the view that the cases as they occurred in England were due to a non-specific ulceration of the colon. This opinion was later supported by Hale-White, who, in 1888 published a report on eight cases that had died from this disease, and who is accredited with first having adequately described its pathology and symptomatology.

Shiga in 1897 discovered the Bacillus dysenteriae during an investigation of epidemic diarrhoea in Japan. This was soon followed by similar observations in other countries. Vedder and Duval in America (1902), and Eyre in England (1904), demonstrated the presence of this specific organism in epidemics of asylum dysentery. The first reference that I have been able to find reporting the isolation of this organism in a sporadic case is that of Saundby in 1906. The bacteriological study carried out in this case by Hewetson revealed a "bacillus which was almost purely and identical with that of Shiga". The patient had never been away from England.

Hawkins in 1909 reported eighty-five cases of ulcerative colitis of varying types. He held the view that all these cases were primarily caused by

the Bacillus dysenteriae of Shiga.

With the introduction of the sigmoidoscope and ante-mortem study colon pathology entered upon a new phase. In 1910 Lockhart Mummery published the results of his observations with this instrument in ulcerative colitis and since then sigmoidoscopy has done more to advance our knowledge than any other single procedure.

Lately the assistance of the X-Ray in determining the extent and severity of the lesions has been of great value.

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Bacteriology.

Rolleston states that "bacteriologically ulcerative colitis is not a specific disease for numerous organisms, Bacillus coli, coliform organisms, Bacillus pyocyaneus, pneumococci, and streptococci may appear to be the predominating and causal agents", and furthermore, "that ulcerative colitis is not a disease in the strict sense of the word any more than rhinitis or bronchitis is, but it is a syndrome with fairly constant clinical manifestations and anatomical changes".

While this is a good clinical dictum for practical purposes, there is a good deal of bacteriological evidence pointing to a specific causal agent.

The Bacillus dysenteriae is almost universal in distribution and there is the very obvious possibility that sporadic cases overlooked in their acute or early stage may subsequently develop a chronic ulcerative condition due to the invasion of secondary pyogenic organisms. The primary infection serves to lower the local and general resistance of the patient, the Bacilli dysenteriae eventually becoming outnumbered by the more common intestinal bacteria, which render their recognition extremely difficult, There is the statement of writers on

tropical diseases that the symptoms and pathological changes of amoebic and bacillary dysentery may recur and persist for a long time after the specific organisms have disappeared from the stool. Saundby and Miller have reported a case of amoebic dysentery with typical ulcerations of the colon and a large single abscess of the liver in a case that had never been away from the Birmingham district. These cases of sporadic amoebic dysentery must be exceedingly rare and can only be ascribed to the presence of chronic carriers. Apart from these rare cases of amoebic infection, I believe there is sufficient evidence to warrant the opinion that all cases are primarily bacillary dysentery.

Judging from the bacteriological reports received the secondary infective organisms are chiefly the Bacillus coli, the Streptococcus fecalis and the Diplococcus pneumoniae. Lockhart Mummery has reported two such cases.

In dealing with ulcerations of the colon no matter whether the underlying cause is tuberculosis or the condition under consideration, we must conclude that we are ultimately dealing with a "mixed infection".

The symptoms and pathological changes are to a great extent due to this secondary infection, and to the toxaemia caused by it.

Ulcerative colitis usually occurs during early adult life. Yeomans series shows the age incidence varying from sixteen to sixty-six. Helmholt in a paper delivered at the annual meeting of the American Pediatric Society in 1924 reported a series of five cases of primary ulcerative colitis of unknown etiology in children of eight to ten years of age. Females seemed more frequently affected than males.

In these cases of true primary ulcerative colitis, I have not been able to elicit anything from their history, which could be rightly called a predisposing factor. They invariably state that they have been enjoying good health until stricken with this malady. However, in spite of their statements to the contrary, I am inclined to believe that some at least, due to stress of circumstances were living in conditions of prolonged under-nourishment.

McCarrison has shown that healthy monkeys, carriers of Entamoeba Histolytica when fed on a diet low in the vitamine content developed amoebic dysentery.

This illustrates very well how a diet deficient in these accessory food factors can lower the resistance of the body and predispose to disease.

At a discussion on Ulcerative Colitis which tool place at the Royal Society of Medicine in 1923 it was generally agreed that the disease was one occurring shiefly in early adult life, that it affected both sexes almost equally, and that the diagnosis could only be made satisfactory by means of the sigmoidoscope. The etiology is quite uncertain. There are some who regard the disease as simply sporadic bacillary dysentery in which secondary infection by various types of intestinal organisms has occurred. The majority of observers, however were of opinion that it is not a specific disease, for numerous organisms - B. Coli, B. pyocyaneus, pneumococci, and streptococci - may appear to be the predominating and causal agents. All are agreed that it is in no way related to amoebic dysentery. Dudgeon, in six cases in which scrapings had been examined from the ulcers, found in one a Flexner bacillus, in four a haemolytic colon bacillus in almost pure culture, and in one case haemolytic streptococci and a Staphylococcus

aureus. He is opposed to the view that ulcerative colitis and true dysentery are always caused by the same organisms, and suggests the following:-

1. Examination of the bowels by the sigmoidoscope.
2. If ulceration is present, material from the floor of the ulcer should be submitted to detailed bacteriological and protozoological investigation.
3. Repeated examination of the faeces.
4. Examination of the blood, more especially in relation to the presence of immune substances in the serum.

A specially interesting form of the desease is produced by pneumococcal infection. In two cases of this sort described by Lockhart Mummery there was excessive haemorrhage and a high temperature. Neither patients had had pneumonia.

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Diagnosis.

The history in these cases usually dates back for months, and in some instances for many years. The onset may be sudden and acute, or very gradual but persistent. In others there have been variable periods of freedom from symptoms only to be followed by a more severe attack of diarrhoea.

There is one characteristic feature in the physical examination of the abdomen in chronic progressive cases, which I believe is important. It is the retracted or scaphoid abdomen. This absence of distension is due to the fact that the ileum is so rarely affected. Marked abdominal tenderness and rigidity suggests perforation with localized peritonitis. The chest should always be examined to exclude pulmonary tuberculosis, as this has a direct bearing on the differential diagnosis.

The examination of the faeces should be undertaken as early in the disease as possible. The outstanding feature in ulcerative colitis is the passage of pus, blood, and mucus in every stool. Where the lower part of the sigmoid and rectum are alone involved, some of the stools may consist entirely of these elements.

During the periods of remissions, these may not be obvious to the naked eye, but can always be detected on microscopic examination. The blood is bright red in colour. The amount of pus varies with the extent and severity of the ulcerative process. The stool is usually fluid, but not watery in character.

In addition to the naked eye and microscopic examination of the faeces, a careful bacteriological investigation should always be instituted. The stool should be sent to the laboratory in as fresh a state as possible. Still better, is the examination of scraping made directly from the ulcer surfaces, through the rectal speculum or sigmoidoscope. We must not be satisfied with one bacteriological report.

By far the most important evidence in arriving at a correct diagnosis in these cases is obtained by the use of the sigmoidoscope. Where the anal canal is greatly irritated a pledget of cotton impregnated with 5% cocaine solution can be left in the canal for a few minutes. The examination should be conducted without pain. When gently and carefully done there is no danger of injury to the bowel by its manipulation. With the sigmoidoscope we can exclude malignant ulceration, tuberculosis, polyposis, and other conditions, which might be confused

with ulcerative colitis. The characteristic features as noted under the section of pathology should be looked for.

The X-Ray gives very definite and valuable information. Following administration of a barium meal the twenty-four hour picture usually shows all barium expelled. In acute cases, the colon is occasionally represented by a mere streak of barium. The earlier plates are usually negative, but there may be a five hour gastric residue. Completion of the roentgeno-logical examination by a barium enema shows a narrowing of the lumen and absence of haustral markings. It also shows the extent of involvement and the presence of strictures. The lungs should always be X-Rayed to exclude early pulmonary tuberculosis.

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Differential Diagnosis.

Cases of primary ulcerative colitis are to be differentiated from the auto-toxic enterocolitis of uraemia and diabetes, and other chronic conditions. The history and co-existing manifestations will as a rule determine the diagnosis in these cases. Other conditions which have to be excluded, are malignant ulceration, diverticulitis of the sigmoid, gastrogenic diarrhoea and tuberculosis. Of these tuberculous colitis alone requires special consideration. The points of differentiation may be anumerated as follows:-

		Tuberculosis of the Intestines.	Chronic Ulcerative Colitis.
1.	Anorexia.	An early symptom.	A late symptom.
2.	Loss of weight.	Gradual.	Rapid.
3.	Lungs.	Associated with Pulmonary Tuberculosis.	Lungs negative.
4.	Abdomen.	Slight distension.	Retracted or scaphoid.
5.	Localization.	Ileo-caecal sigment, first involved.	Sigmoid and rectum chiefly affected.
6.	Blood in stools.	Absent or slight.	Marked.
7.	X-Ray.	Irregular moth-eaten appearance.	Colon narrow and ribbon-like in outline.
8.	Tubercule. Bacillus.	Present.	Absent.
9.	Sigmoidoscopic.	Usually negative.	Characteristic ulceration seen.
10.	Bowels.	Alternating constipation and diarrhoea or unaffected.	Continuous and increasing diarrhoea.

11. Ileum. Often involved. Rarely involved.

In place of anorexia patients with intestinal tuberculosis often complain that after the first few mouthfuls of food, they have a sensation of fullness in the epigastrium and no desire to eat more (Pritchard). The slight abdominal distension is due to involvement of the ileum and interference with the proper digestion of food. Even in the terminal cases of tuberculous colitis the abdomen is sometimes flat but never scaphoid in appearance, as in ulcerative colitis.

In the series of autopsies reported by Schwatt and Steinbach, the rectum showed tuberculous ulceration in seven per cent of the cases, and usually associated with marked involvement of the other segments.

Haemorrhage from a tuberculous colon is rare because of the obliterative endarteritis characteristic of the tuberculous process. Blood in the stool is therefore not a feature, whereas in chronic ulcerative colitis, it is one of the outstanding clinical findings.

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Prophylaxis.

Believing as I do that constipation is the essential predisposing factor in bringing about mucous colitis, it is obvious that the treatment of early cases before permanent organic changes have occurred in the Colon, resolves itself into the treatment of the underlying constipation. These remarks do not of course, apply to cases of dysenteric or other specific types of the disease. I can only deal with this vast subject very briefly. Constipation should be dealt with on hygienic or common sense lines.

The immediate recourse to laxatives and purgatives cannot be too strongly condemned. The numberless proprietary and patent medicines which are used for the treatment of Constipation produce a vast amount of mischief.

The host of preparations in the official pharmacopoeias of different countries for the treatment of Constipation is productive of perhaps, even greater mischief. On the one hand, it is a testimony to the faulty nature of our diet and mode of life, and on the other hand it bears witness to the lack of proper advice as to diet and prevention of consti-

pation which should be the province of the Medical profession to give. Another great blot on scientific medicine is the indiscriminate administration of Mist. Alba etc. in our Hospital O.P. departments and to our private patients.

The modern practitioner whilst scoffing at the blunderbus prescriptions of our predecessors will prescribe with equanimity the elegant laxative pastilles of the enterprising manufacturing chemist - and probably the matter are equally harmful. One of the best laxatives in my opinion is Liquid Paraffin. We have a preparation here which passes through the alimentary canal unchanged. It does not cause irritation of the intestinal tract which other purgatives do, and in certain cases it is a help to hygienic and dietetic methods. But even that remedy is abused and we find patients drinking gallons of liquid paraffin without taking any steps to find a suitable dietary to prevent their constipation.

Early cases of mucous colitis will probably be completely cured by hygienic and dietetic measures.

A stay at one of the hydropathic resorts may be useful in inculcating a proper hygienic and dietetic regimen.

In advanced cases where organic changes have occurred in the colon, interfering with its functions, these measures will not suffice, and local or surgical treatment is indicated. In this brief sketch I will not attempt to deal with the various local applications which have been advocated by different physicians.

The method I adopt in my cases is fully described in the notes of cases. These consist of injections of various kinds to promote healing of the colon mucosa, applied either by rectal injection or through an appendicostomy opening. Where however, disorganisation of the colon has gone further and the organ is unable to function properly, and the attacks are so grave as to endanger life - the rational treatment is Colectomy. Surgical treatment of Mucous Colitis is however, beyond the scope of the present thesis.

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Treatment:

The keynote in the successful treatment of ulcerative colitis is the protection of the ulcerated mucosa from irritation by the faeces and septic discharge. The application of this principle must be carried out by whatever method or combination of methods may be indicated in each individual case. In the earlier stages, active medical treatment is indicated; but this should not be persisted in too long where improvement does not occur. No case can be considered purely and always medical, or purely surgical. One must be supplemental to the other. The various forms of treatment may be tabulated as follows:-

Medical:

1. Diet.
2. Drugs.
3. Vaccines.
4. Bowel irrigation:
   a. Duodenal tube.
   b. Jointed intestinal tube of Einhorn.
   c. Enemas.
5. Anti-dysenteric serum of Flexner.
6. Heat.

Surgical:

1. Caecostomy.
2. Appendicostomy.
3. Ileostomy.
   a. Brown operation.
   b. Halsted method.
   c. Mikuliez method.
4. Colectomy.
5. Blood transfusion.

The diet should be light but highly nutritious and should be so planned as to leave a minimal faecal residue. The drugs that have been recommended are many, the chief of which are: bismuth, iodine, and the opium derivatives.

Irrigations with enemas are of no value in this condition. We are dealing with a contracted, irritable colon, and to attempt irrigation from below can only do harm.

The use of polyvalent anti-dysenteric serum has several advocates. Hurst reports ten cases treated with gratifying results. This is therapeutic evidence, favouring a specific infection by the Bacillus dysenteriae.

There are two objects sought in the operative treatment of ulcerative colitis: (1) to provide irrigation of the colon from above, and (2) to divert the faeces and place the colon in a state of physiological rest.

I would enumerate the indications for operation as follows: (1) Where active medical treatment has failed; (2) Recurrent exacerbations of attacks of diarrhoea; (3) In cases which are rapidly losing ground.

The first object of treatment is carried out most successfully by caecostomy or appendicostomy, preferably the former.

The advantage of caecostomy lies in the fact that the fistula will close spontaneously soon after the catheter is withdrawn. The fistula formed by the appendix may have to be closed at a subsequent operation, as it is lined with mucosa. The caecostomy is performed by sewing a catheter into the caecum by a combined Kader-Senn and Witzel method, under local anaesthesia if necessary. The catheter can later be attached by tubing to the irrigating can. A rectal tube is inserted and this connected by tubing to a large container under the bed. The irrigation of the colon with warm solutions may be commenced at once, continuously at first, later intermittently. First hot normal saline is used and later alternated with boracic acid. The advantages of the rectal tube are obvious. The bed-pan becomes unnecessary and, what is most important, the patient sleeps throughout the night undisturbed, the treatment causing no inconvenience. The patients whose bowels have moved seven or eight times during the night previous to operation become

exceedingly grateful for this rest. Solutions that have been used by others are: calcium carbonate 0.5% mercurochrome (220) 1½ sodium bicarbonate, potassium permanganate, hydrogen peroxide 2% silver nitrate 1:10,000, protargol, albargin.

The accomplishment of the second object if considered necessary is best carried out by means of a transverse ileostomy. Brown's technique is that most commonly employed.

A muscle splitting incision is made and the caecum identified. The ileum is drawn up into the wound and division effected about eight inches above the ileo-caecal junction. A catheter is purse-stringed into the distal segment to provide for irrigation and a larger tube into the proximal end. The bowel ends are sutured to the parietal peritoneum, the wound lightly stitched and packed with gauze.

Colectomy is rarely necessary, but in selected cases, where the colon has become transformed into a narrow, strictured, suppurating tube, with no hope of healing taking place, this procedure becomes justifiable.

Blood transfusions have proved of distinct value in the cases which have become markedly exsanguinated

and exhausted. Young's case shows how profound this anaemia may become. This case was finally diagnosed as bacillary dysentery after having been treated for some time as one of pernicious anaemia.

I do not agree with Jones when he says that "before any operation is done it is important to explore the whole colon to determine the extent and severity of the disease, provided the patient is not too ill." By careful consideration of the history and physical findings, corroborated by sigmoidoscopic and X Ray examination, one can usually estimate the severity of the condition beforehand, and decide the proper procedure in each individual case, and thus obviate a more prolonged operation. A McBurney incision is all that is necessary in the large majority of these cases.

Before re-establishing the continuity of the intestinal canal we must first satisfy ourselves that the colon has healed. The following points can be mentioned as evidence of cure:-

1. A normal appearance of the mucosa on proctoscopic examination.

2. The disappearance of all symptoms.

3. The absence of pus and blood microscopically in centrifuged salt solution that has been

run through the colon from the artificial opening to the anus (Stone).

These findings are to be checked, and if maintained for two months a re-anastomosis is indicated, provided the colon has not in the meantime become transformed into a small fibrous inelastic and useless tube.

During the course of any given treatment it is essential to check up the progress by direct visualisation of the mucosa of the bowel through the sigmoidoscope.

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Summary of Treatment:

The general management of the patient consists of Rest, preferably in the open air, with an abundant but Unirritating Diet.

Drugs are of little use. The effect of Vaccines is very uncertain. If used at all a mixed autogenous vaccine is best. Hurst claims very good results from the intravenous injection of the Polyvalent Antidysenteric Serum of the Lister Institute: 40 c.c. should be given the first day, 60 the next, 80 the third, and 100 the fourth. The four injections are often sufficient but it may be necessary to repeat the maximum dose two or three times. There may be a considerable reaction, with rise of temperature, a rash, and joint pains, but these effects are transient. They are less likely to occur if 15 gr. of calcium lactate are given three times daily during the treatment and four a day preceding it.

Hurst regards the benefit of this treatment as specific and not merely due to the horse serum. If confirmed, his results would be strong evidence in favour of the dysenteric origin of many cases.

Local treatment consists in Lavage of the Colon. This may be done per rectum with the patient in the

knee-elbow position. There is no need to introduce the tube more than four inches. The injection should be at body temperature. An immense number of different solutions have been employed, but it is probable that benefit is derived chiefly from the mechanical effect of the washing. The fluid should be rather hypotonic so as to prevent any dehydration of the patient; and if an antiseptic is added, weak permanganate (1:5,000), 2% iodine, flavine, or 2% peroxide are as good as any. If one desires to use astringent solution, one of the organic salts of liver is best. Ordinary silver nitrate should be avoided, as it has been known to produce argyria.

If the patient does not rapidly improve under these measures, an opening should be made in the colon so as to permit of more thorough irrigation. Appendicostomy seems to be the operation of choice. A catheter should not be tied into the stoma, but passed as required, and the opening should not be allowed to close for at least a year, owing to the tendency of the disease to relapse.

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Results of the Treatment:

It would appear that, largely owing to the adoption of earlier operation, the prognosis of the disease has considerably improved. Ten years ago the mortality was about 50 per cent. Now it is 13 per cent (Lockhart Mummery).

Stricture never results from healing of the ulcers, but it may follow caecostomy. Relapses, even after an interval of years, are, however, a common experience.

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Notes of Cases:

Case 1. In 1915

Female, age 39: Chronic persistent diarrhoea since 1911 when she had an acute attack. Average of six stools daily which would improve slightly on a low light diet. Also intermittent attacks of Gall Stone Colic and a very severe dyspepsia. Her appendix was removed, also operated on for ventral suspension of uterus and excision of gastric ulcer in 1913 in Johannesburg.

There was a history of treatment by eight different medical men before I saw her, but the diarrhoea persisted. Motions were semisolid with copious viscid mucus and occasionally very watery stools with membranous shreds. No blood or pus in stools. No amoebae.

Sigmoidoscopy showed an oedematous and injected mucus membrane and no signs of ulcer or other abnormality.

Treatment: Confinement to bed.

Diet: Half and half Milk and Barley-water for four days.

Then pure milk for four days.

Then Arrowroot and Maizena for four days.

Then add toast and biscuits next two days.

Then add fish and chicken minced next two days. Then grated biltong and minced meat thereafter. Vegetables as well.

Drugs: Emetine ½ gr. t.i.d. hypodermically.

Increased to 1 gr. t.i.d. hypodermically. Morning and evening.

Colon lavage with 2 pints lukewarm solution of Sod. Bic. 3i to pint followed 10 minutes after by Ac Boric Solution also 3i to 1 pint. This latter to be retained for 10 minutes.

After the third day the motions improved ~~and~~ to only three a day, and the mucus diminished as well. The treatment was continued for 10 days, when all mucus had disappeared and only action with the washout. After that, one lavage per day and ½ gr. emetine twice daily for the next eight days.

Patient was discharged and had no return of the trouble. Unfortunately this colitis caused secondary retro-duodenal abscess for which I had to resect the Duodenum and Pylorus and at the same operation I removed 40 gall stones and did a gastrojejunostomy.

Three years afterwards cholecystenterostomy was performed with removal of more gall stones.

She is at present quite well and has had no return of the Colitis.

Case 2:

Male, age 41 - married. Consulted me in 1916 complaining of Diarrhoea for the last 13 years. This began with an acute attack which lasted two months. His motions then were from 20 to 30 a day, pure blood and mucus with severe pain. The acute attack passed off but his motions were never less than 3 to 4 a day, sometimes up to 7 or 8 a day. He was treated by various medical men who all prescribed medicine by mouth and diet. He had no pain, no blood in stools, only a fair amount of membranous mucus mixed in a soft stool.

Sigmoidoscopy showed a greyish muco-membranous covering in patches. No carcinoma or pressure tumor or obstruction to lumen of colon. Microscopically no Entamoebae.

Treatment:

Diet: Same as Case 1.

Drugs: Same as Case 1.

After fourteen days all symptoms disappeared. I treated his family up to date and know that he has had no return of the Colitis.

Case 3:

Male, age 43. I was called in consultation to see an old standing case of Dysentery treated by a colleague for six months. I examined and found he had a large liver abscess in the right lobe of the liver. This was successfully opened and drained. The chocolate-coloured discharge from the abscess showed no sign of changing to yellow colour. As the stools were only two or three a day the primary condition was really ignored and I trusted to the ultimate drying up of the discharge with proper tonics and nourishing diet. When the case dragged on I began the administration of Emetine hypodermically and colon lavages. Within 3 days the discharge changed to a yellow colour and dried up within a week. At the same time his diarrhoea ceased and stools were normal when the abscess discharge stopped. This Case had Amoebae in the discharge as well as in the stools and was obviously a case of Amoebic Dysentery.

Case 4:

Male, age 63. History of acute diarrhoea four years ago, and ever since a chronic diarrhoea. About four stools fairly well digested, no blood, but a moderate amount of mucus daily.

Sigmoidoscopy showed nothing except mucus membrane covered with tape-like strings of mucus. Microscopically no amoebae.

Treatment: Same as Case 1 and 2, and all symptoms disappeared in 14 days. A return of the same condition after 9 months resisted treatment until I found a deficiency of HCl, which was made good by 5-15 mins. HCl t.d.s. p.c.

The symptoms were soon relieved and there has been no return of diarrhoea up to the present.

Case 5:

Male, age 26. Complaining of diarrhoea after acute Dysentery contracted in Kenya Colony in 1920. Previous Emetine treatment only had a partial effect. Colon lavage was not given at first. His stools were watery and slightly muco-purulent. Very offensive; no blood and shreds of membrane.

Sigmoidoscopy - small depressions covered with mucus membrane injected and oedematous. Also small necrosed areas. Microscopically Amoebae were present.

Treatment: Emetine and colon lavage as before relieved all symptoms within 16 days and he has had no return of the condition up to the time of writing (early 1926).

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