BACILLARY DYSENTERY.

A Thesis

for the Degree of M.D. (Glasgow)

by

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This thesis is wholly based on clinical work on Bacillary Dysentery done in Egypt and Palestine during the years 1915 - 1919. I regret that charts of cases intended to illustrate certain points discussed were lost at sea on the way home.

At the beginning of this period clinical knowledge of Bacillary Dysentery was difficult to obtain, due partly to the scarcity of men with previous experience, and partly to the diversity of opinions expressed in the few books available.

It was therefore necessary to form ones own opinion on actual clinical experience fortunately backed by the laboratory experience of competent Bacteriologists.

Having seen many hundreds of cases during all stages of disease and convalescence I have tried in this thesis to emphasize the points that have forced themselves on my attention, and have passed over briefly those points, common to all epidemics of the disease, which are common to all text-books.

No reference to Bacteriological work will be made save to state clinical comparisons between cases due to the bacillus of Shiga and those due to a bacillus of the Flexner type.

Early/

Early experience soon showed that the dysentery bacillus caused death in two ways.- (1) Death early in the disease from acute toxaemia. (2) Death late in the disease due to starvation, caused by loss of intestinal mucous membrane in large quantities, and chronic toxaemia.

Thus the lesson was learnt that the toxin should be inhibited and eliminated as early in the disease as possible, lest an incomplete victory in the early days should lead to complete defeat later on.

This early lesson is still, to me, the most important one because fuller experience showed that the majority of cases were so alike in their onset that it was impossible to be sure that anything short of the most vigorous treatment would be sufficient to cure any given case.

That the disease is not invariably dangerous is shown by the very large number of cases of diarrhoea occurring usually in troops soon after landing in Egypt.

I believe these slight cases form a very definite class of bacillary infection. Some of them may be caused by Flagellate protozoa, but the majority are the result of the dysentery bacilli.

In Egypt all English speaking people refer to these cases by the name "Gyppy Tummy". That this is in/

in reality a form of Bacillary Dysentery is proved on clinical grounds by examples such as the following.

On a Tuesday evening a ship containing, among the personnel, fifty Medical Officers, berthed in Alexandria harbour. A few of these men went ashore for a few hours, returning to the ship that night.

On Wednesday morning all officers below field rank were despatched to a certain camp. Their number was roughly forty.

On Thursday morning 10 or 12 had more or less severe diarrhoea, and on Friday the number increased further.

On Saturday morning nearly all were posted to units in various parts of Egypt. By that time nearly all had diarrhoea, two at least had been admitted to hospital, and fifty per cent were quite unfit for any duty which kept them far from a latrine.

In after months I met again many of these men and found that most of them had recurring diarrhoea sometimes with blood and frequently with mucus in the stools.

One had been invalided home without doing any duty in the country at all.

Some others had to be sent home after a few months on account of recurring diarrhoea and loss of weight.

One had died as the result of his original infection.

In hospital when questioning patients regarding previous attacks of diarrhoea the common reply was "I have had two or three attacks of "Gyppy Tummy" before, but none so severe as this."
These cases never come before the Bacteriologist for accurate diagnosis because the primary illness is slight and they are not sent to hospital but recover partially under treatment in camp.

This treatment was generally a dose of castor oil in the case of the men, with some form of alcohol added in the case of officers. Indeed I found that many officers messes each had their own pet treatment and frequently the attached Medical Officer was hardly consulted and less seldom obeyed.

Many Australians assured me that the only cure was to drink condensed milk out of the tin without dilution.

If this class of case is indeed, as it appears clinically to be, the result of B. dysenteriae infection, then the so-called "Gyppy Tummy" must be regarded seriously as one such patient may by carelessness determine a local outbreak of serious proportions.

Until these cases are proved to be cases of Flagellate dysentery (Lamblia, Trichomonas, etc.) clinical suspicion is justified in considering them as cases of mild bacillary infections.

Having/

Having stated my belief that these should be considered slight cases of bacillary dysentery, let me now outline some of the features of severe and undoubted cases.

Incubation. This period is usually short. Two days was quite a common period, estimating from the day of patient's disembarkation. The shortest period was noted in the case of a man who left his ship at 10 a.m., marched four miles to camp, and was admitted to hospital at 7 p.m. with severe tenesmus and stools composed entirely of blood and mucus. A dysentery bacillus was later isolated from his stools.

Onset. This is always sudden. Generally the abdominal symptoms precede the rise of temperature.

A feeling of fulness and unrest in the abdomen is closely followed by strong griping pains producing one or two large faecal stools. These are normal stools and mark the hurried evacuation of the whole of the large bowel. These are the last normal stools passed for, it may be, many days.

The temperature may rise to 104° F., but more usually to 102° F. and is accompanied by headache and nausea, sometimes vomiting and giddiness.

Course./

Course. Within a few hours of the first griping pains diarrhoea of varying intensity is established. Griping is intense, evacuation gives no lasting relief and indeed the patient may find it impossible to leave the latrine or part from the bed pan if he is in hospital. This is partly because desire for further evacuation is almost constant, and also because the slightest muscular movement may determine another spasm of intestinal contraction. Physical weakness becomes marked and there may be delirium generally in the form of dreams. The patient lies perfectly still and conscious, though he may be muttering to himself, and is easily and frequently aroused by the necessity of passing a stool.

In the early stages there may be apparent loss of control of the bowel, but this is not due to unconsciousness or paresis, but to the intestinal contractions, whose suddenness may defeat attempts to reach a bed pan.

As weakness becomes more marked in serious cases, it may be impossible for the patient to do other than lie still and pass stools into wool pads. Even here there is not loss of consciousness. The man knows that a stool has been passed and that he has been quite unable to prevent it for more than one or two seconds.

As/

As diarrhoea becomes established it is possible to recognise two distinct types depending on the main site of the bowel lesion. In the first the pain is chiefly in the upper abdomen or right side of abdomen. Patients generally describe it as "round about the navel and above it". In a few cases the greatest pain was felt close to the costal margin.

This class of case has its main lesion in the upper portion of the large bowel, and possibly in the last few feet of the small one. The second type is characterised by tenesmus of varying degree and has its main lesion low down in the large intestine.

However severe tenesmus may be, it cannot entirely mask griping pain when it is also present.

In Bacillary Dysentery the essence of tenesmus is obstruction to the passage of a stool. During the spasm practically nothing is passed though the effort is enormous. When the spasm relaxes a small amount of mucus and blood is passed, but the feeling that there is more to come persists in varying degree. It may even persist until the intestinal contractions from above force down another stool to again harass the rectum.

For it seems that it is in the upper part of the rectum that this obstructive spasm occurs. In some cases even it seems as though the condition may be

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more that of intussusception than merely spasm of the circular muscular fibres of the bowel.

At first I thought the tenesmus was the result of an actual lesion at the site of the obstruction. Further experience suggested that this was not necessarily the case.

Sigmoidoscopy is of course out of the question during the acute stage of the disease, but digital examination may sometimes be performed. This showed that as soon as tenesmus occurred a portion of the sigmoid or upper rectum was suddenly forced down to meet the finger and that no lumen could be felt until the spasm ceased. In some cases digital examination produced tenesmus at once. In others the determining factor came from above.

During convalescence a very few cases were examined by means of the sigmoidoscope and in none of these was a lesion found as low down as the point where obstruction occurred. In one case no lesion was found in the observed portion of bowel at all. In this case the sigmoidoscope was passed by an experienced surgeon who estimated that he had examined twenty-two inches of bowel length. In all the cases thus examined extreme tenesmus had been a constant feature.

What/

What then could be the cause of this tenesmus which was by far the most painful feature of the disease, and one of the most difficult to relieve?

Anatomically I know of nothing described that would suggest that some point in the rectal mucosa is specialised to give warning of an impending stool. At the time of writing (February 1919) it is impossible to get any text-book to consult. Yet if, as is known, a portion of the urethra is stimulated by a drop of urine to effect the emptying of the bladder by reflex action, then it is possible that some such device exists in the rectum.

The over-stimulation of this mechanism may have the effect of preventing the result which a normal stimulus should achieve. At all events the rectum remains empty, or practically so, as long as the occlusion of its upper portion lasts, despite the most frenzied efforts of the muscles of the colon to force the passage.

I have said that after the initial emptying of the large bowel of its faecal contents no faeces are passed for some time.

During the period of griping pains and tenesmus what then is the character of the stool? It consists entirely of mucus of varying viscosity which sooner

or/

or later is mixed with blood.

In some cases at the beginning of this second stage of the disease the stool may be perfectly clear mucus, for during this stage,which lasts usually 48 hours,there is usually not even a faecal odour from the stools. The mucus is generally white or grey, and is frequently of fairly firm consistence. So much so that when it later becomes streaked or mixed with blood the appearance in the bed pad is much like a small dead jelly-fish lying on the sea shore.

The appearance of blood indicates the point when some damage occurs to the structure of the mucosa.

Later still the appearance of yellow pus points to increased damage and ulceration. When this occurs shreds of mucous membrane may be passed, and later sloughs of varying size in all stages of decomposition.

The patient is usually first seen in this stage and therefore it is to this class of stool that one must look for help in diagnosis and prognosis.

Although the accurate diagnosis between Bacillary and Amoebic dysentery must rest with the microscopist and bacteriologist, yet a few points are present which may help the clinician.

1. As a rule I found that the stools of Amoebic dysentery showed more free blood comparatively early; the Bacillary stool being usually tinged or streaked with blood which was mixed with the mucus.

2. The Amoebic stool quite frequently showed at least traces of faecal matter. The Bacillary stool did not.

3. The Bacillary stool had not even a faecal odour but had instead a distinct odour of Spermin.

This was found to be very generally present and taken along with the absence of faecal odour is, I think, diagnostic of the bacillary type of the disease. This symptom was frequently checked by the laboratory findings on the cases, and I have never seen a stool of proved amoebic dysentery which had not some faecal odour.

In only one case giving an odour of spermin was the Amoebic histolytica afterwards found. In this case I saw only a small portion of the stool in the test tube. In it I could not detect a faecal odour, but the bacteriologist stated that there had been a small quantity of faeces in the bed-pan from which the specimen was taken.

I do not wish to emphasise this point too much, but in the absence of early laboratory diagnosis it may prove a guide to treatment. During this 48 hours the temperature has reached its height and is beginning to drop. This is the stage of intoxication.

At the end of this stage the opening of the third/

third and longer stage is marked by two points.-

1. The temperature falls to nearly normal. This may bring with it some slight improvement in the patient's general condition but does not necessarily bring any relief from pain and tenesmus.

2. In cases which are under treatment the fall of temperature is closely followed by the appearance of faeces in the stools. The mucus is now greyer or yellower in colour, there is still blood and probably pus, and there is now a small quantity of faeces added.

This shows that the small bowel is now once more passing its contents on. Frequently griping becomes less now, and there is some rest from tenesmus though the intervals may not be of long duration. The appearance of faeces is often marked by a small rise in the temperature, very evanescent in most cases. Probably this marks the absorption of some fresh toxin from the faeces now passing along the ulcerated colon.

I have learnt to watch for this rise for this reason. If it is of short duration followed by a fall to normal, it is fairly safe to predict a rapid recovery. If, on the other hand, the rise is maintained the probability is that recovery will be slow/

slow and it may sometimes presage a fatal result.

In these latter cases the patient shows signs of a deeper intoxication which may in a few hours lead to collapse and death.

Should this stage of the disease be passed satisfactorily it is certain that death from the immediate effect of the bacillary toxin will not occur.

It is after this point that the abdominal symptoms begin to abate. Pain ceases almost entirely and tenesmus is less severe and less frequent. Also the patient gains more courage as his feeling of well being increases with the respite from pain.

The number of stools, which may have reached almost any figure, now becomes much less, and it is not at all uncommon to find on the chart that no stool has been passed during the night.

While this is a good sign yet there is an element of danger in it. On no account should aperient treatment be withheld at this point. Constipation may occur suddenly which may have serious results on a thinned out and ulcerated bowel.

It may be taken as a fair general rule that a sudden decrease of purely mucus and blood stools before faeces have reappeared, is a sign of the gravest moment, calling for prompt treatment.
On the other hand a decrease of stools containing faeces is of favourable moment, so long as constipation

is avoided.

The progress of some of these favourable cases is so rapid and well maintained that the painfully slow progress of others is hard to understand. It is only in the postmortem room that the real effects of the infection can be accurately judged.

It has seemed to me that cases successfully treated with Antidysenteric serum progress more rapidly.

Whatever the rate of progress may be, the result is that pain, urgency of stool and tenesmus diminish and disappear, mucus ultimately disappears from most of the stools. The whole clinical picture changes from that of an acute toxaemia with bowel symptoms to that of a subacute enteritis or colitis.

I have seen no case die at this stage with re-appearance of toxaemia, although as previously stated cases do undoubtedly die in the later stages of the disease from chronic toxaemia and starvation due to insufficiency of normal mucosa.

When there is no longer danger of death from the primary toxaemia the question arises.- Has he lost so much mucous membrane that death must ensue from chronic toxaemia and starvation?
There can be no definite answer to this in the case of those whose infection has obviously been a severe one.

Everyone has seen at some time large sloughs passed just as in the postmortem room everyone has seen a gut so denuded of mucosa that it was hard to believe that the patient could have long survived even had he not succumbed to the early toxaemia.

It is known that when more than a certain area of skin is destroyed by a superficial burn the patient dies. It seems probable that destruction of a certain area of mucosa must also lead to death by reducing the power of absorption or lessening the resistance to pathogenic organisms.

I have seen two cases which, judged by comparison with other fatal cases, should have died late in the disease, and yet lived to go home on a hospital ship. Both men were well enough to walk a little in the ward before being sent home.

The chief sign of probable late death is emaciation which shows no sign of improvement on varying diets.

When toxaemia is at its worst many patients become very thin with sunk face and hollow abdomen, a picture of wasting more marked than the duration of the disease would suggest. This is often of short duration and equally surprising is the rapidity with which the picture changes for the better if the man

is going to get well. But if this emaciation persists, no matter what improvement takes place in the other symptoms, the prognosis is grave.

Another sign is the character of the stool in the wasting case. If the stool is faecal, with comparatively viscid mucus or mucopus, prognosis is hopeful. On the other hand if there is a thin mucus discharge with blood in nearly every stool, the prognosis is bad. The continuance of much haemorrhage is serious but not necessarily fatal as was seen in one of the two cases referred to above.

In this case the bleeding was usually free, sometimes pure blood recurring at intervals as though from haemorrhoids though none were present.

These then are the chief clinical points of the cases which may end fatally.

Of those cases which obviously will get better, it is very hard to forecast the length of convalescence. Is recovery to be rapid or will the patient drift into a condition of chronic colitis?

There is no definite answer to this question, as many cases seem to get to a certain point of recovery and then stay there indefinitely. Their general condition is fairly good, they can take moderate exercise but three disabilities remain.- (1) The passage of mucus in the stools: (2) The

absence/

absence of full muscular tone: (3) An abnormal number of stools. The mucus may be passed in every stool, but this is seldom the case. The stools number 4 - 6 in 24 hours and possibly only one may show mucus. This condition may be due to inefficient treatment or injudicious dieting. It is the most difficult point to deal with after the patient's life is safe.

Treatment. Whatever varieties of treatment I may have tried at the beginning, experience soon showed that there were only two possible methods from which to expect success.

1. Treatment by saline aperients. Sulphate of Soda or Sulphate of Magnesia, singly or in combination.
2. Treatment by Antidysenteric serum along with saline aperients.

Experience soon showed that the latter was the better for general use as it was safer, though in many cases the former did quite well. On this account a description of the complete treatment will be given as it necessarily includes the less complete treatment by salines alone. As a means of giving temporary rest from pain and gaining a few hours sleep, a dose of chlorodyne or other opiate is most useful and not, I believe, harmful if only given at night. It is seldom required after the second night.

Salines./

Salines. As a routine every case was given a morning dose of Mag. Sulph. and Sod. Sulph. one drachm of each. As a rule only this one dose was given in the twenty-four hours. In some cases a similar dose was given in the afternoon, but in only a few cases did I give repeated doses two hourly as is sometimes recommended. These frequent doses were soon given up as the cases showed no better results than those cases which only got one morning dose. In fact the only cases which frequent saline doses really benefit are, I think, those where the mucus stools are very small and there is almost constant tenesmus. Experience showed that when the mucus was passed in fair quantity there was no need of frequent salines. The effect of serum on these cases will be discussed later. It was also found that these cases got more ease from tenesmus and as great a mucus flow from a single dose of Castor oil (half an ounce). Any case that appeared to be developing constipation at once got more frequent doses of saline and this was latterly the only reason for departing from the routine morning dose.

How long should this morning dose be continued? Certainly until the stools become of normal character. There is nothing to be gained by stopping sooner, and much may easily be lost.

Antidysenteric serum. Having given polyvalent sera of different makes in doses ranging from 20 c.c. to 100 c.c., it is sufficient to give the conclusions arrived at as to size and time of dosage.

The best time to give the first dose is the earliest possible moment after it is definitely decided that the patient is passing blood and mucus. To be of use it must be given before the fourth day of illness. I have said above that after 24 hours the intoxication is severe, therefore the sooner it is given the better.

There seems to be no reason for giving less than 60 c.c. or more than 100 c.c. for the initial dose. I have seldom given this intravenously; usually it was given subcutaneously in right or left iliac region, or in the loose tissues of the thigh. One good reason against the intravenous method is that sudden pain or tenesmus may cause sudden movement on the patient's part and thus make administration difficult.

In many cases one dose of 60 c.c. given early brought about speedy cure. In most cases two subsequent smaller doses were given with one day's interval between each. Thus the routine was three doses of 60 c.c., 40 c.c., and 20 c.c., though severe cases might have 100 c.c., 80 c.c., and 60 c.c., sometimes on consecutive days. I have never given a case more than 260 c.c. in all.

The/

The main point is not so much the size of the dose as the period of disease at which the initial dose is given. 60 c.c. given when the patient is first seen, usually at the end of the first 24 hours, is of more value than a larger dose given later.

Whatever may be urged by some observers in favour of saline treatment alone, there are two strong points in favour of an early dose of serum.

I. It cuts short, in many cases, the toxaemia, and thus in my opinion diminishes the chance of extensive destruction of mucosa.

II. Many patients stated that it markedly decreased tenesmus in a few hours after administration.

At first I doubted this result but as cases multiplied so the patients' testimony to this effect increased. In certain camps this treatment became so well known that patients on admission sometimes asked to be given serum. As many patients were not seen as early as the second day it naturally happened that many got their first serum on the third or even fourth day. I have never seen such striking improvement result in these cases, though in many improvement certainly took place.

I have never seen any ultimate bad result from the administration of serum at any stage of the disease/

disease nor have I ever seen a case of anaphylaxis caused by it. Yet there are two transient disabilities of frequent occurrence.

I. Nearly every patient who got serum developed a very marked rash on the tenth or eleventh day after the injection. This began as an area of deep redness at the site of injection with sharply defined edges at first. Later it merged in an urticarial rash which spread all over the body in patches of varying shape and size. This rash was always very itchy and nothing seemed to relieve this annoying symptom.

II. About the same time as the rash developed some men developed pains in the joints without swelling. Any joint might be affected, but commonly if this occurred it was not limited to a single joint. It was not a very common occurrence but it was in some cases very severe. One case, the worst I have seen, had every joint affected and could not move in bed, having to be fed with small quantities of fluid on account of the temporary fixation of his jaw. These joint pains subsided in three or four days and left no loss of mobility of the joints however immobile they might have been. A very few cases developed fluid in one or more joints, generally the knee joints. None of these required/

required surgical interference and in no case did stiffness or fixation of the joint ultimately result. I cannot think that this latter affection is attributable to the serum as it occurred so infrequently. Yet if it is due to the dysentery bacillus I can only recall one case not treated with serum that developed slight pain and stiffness in both knee and ankle joints of both legs.

Sequelae. These were mainly of three kinds.-

I. General wasting. (Loss of weight)

II. Bladder symptoms.

III. Gastro-intestinal symptoms.

1. Wasting. Among several hundred convalescent dysenteries many were found in whom loss of weight and muscular wasting was marked many weeks after discharge from hospital. In these the general health was fairly good. They had normal stools and no undue frequency of stool (In Egypt 2 - 3 stools in 24 hours is not abnormal among men recently brought into the country) and they were fit for a certain amount of light work. Yet there was marked wasting of muscles and absence of superficial fat for many weeks. One man seen two years afterwards still showed apparent entire absence of superficial fat, though he was in good health and working/

working regularly on lines of communication.

These cases seem to me to represent those whose loss of mucosa has been almost but not quite sufficient to cause death late in the disease.

II. Bladder symptoms. Many cases when convalescent complained of frequency of micturition, some almost amounting to incontinence. These men did not have necessarily any incontinence or even frequency during the acute stage of the illness. Iron and Arsenic tonics generally brought about a great improvement. A more intractable symptom was Prostatorrhoea. Quite a number of cases show this in slight degree during convalescence, and a few had it to such an extent as to interfere with general health. One case, a previously healthy yeoman aged 21, developed this two months after discharge from hospital. Nearly every hight he had prostatic discharge and sometimes also during the day. No treatment made any difference and loss of weight became so great that he was readmitted to hospital and later invalided home.

This is the worst case that I have seen, but commonly men reported nocturnal emissions two nights out of three. Many of these got quite well on general tonics.

A less serious class of case was those who had copious/

copious prostatic discharge whenever they had a fairly solid stool. This condition in some lasted for many months.

Four men asked for treatment on the grounds that they believed themselves to have entirely lost sexual power after dysentery. All were married men, the youngest 29, the oldest 38 years of age. All stated that they were accustomed to nocturnal emissions at fairly regular intervals since enlistment, but that since their dysentery attack these had ceased. All reported the condition as they feared permanent impotence and none of them had any venereal history.

Gastro-intestinal symptoms.

1. Dyspepsia. This occurs chiefly as an inability to digest vegetables and sometimes bread. Early experience in dieting showed that these were the last articles to be added to a patient's diet. It must be admitted however that some samples of army bread might have taxed the digestive power of a normal digestive tract.

II. Alternating constipation and diarrhoea. This was probably the most common sequela. These patients had great difficulty in having a daily stool without medicine, though a very mild aperient usually was sufficient to produce a result./

result. If this was neglected a period of a few days diarrhoea set in when there was considerable griping and frequently a return of mucus in the stools but practically never any blood.

Morning diarrhoea is common for weeks after an attack. It takes the form of 1 - 3 fluid normal stools generally all passed before 9 a.m. The first is passed on waking, and is urgent. Apparently the first semiconscious movement in bed brings on peristalsis. There may be slight griping but there is no tenesmus. The urgency may be so great as to defeat attempts to reach a latrine. This was frequently noticed in convalescents recently returned to their units.

For the remainder of the 24 hours there is no diarrhoea, but frequently a more or less formed stool is passed later in the day.

Tympanitis. A few cases occurred where sudden abdominal distention appeared. This came on at any hour and was very intractable. Most usually it occurred at night during sleep, though the same patient might have another paroxysmal distention through the day. These cases tended to develop atonic constipation, but most of them were invalided home before this stage was reached.

When a case developed this symptom there was only slight pain or discomfort, but because of its suddenness and irregularity of onset it was one of the most disabling sequelae from the point of view of army work, though the general condition might be fairly good.

Diet in Dysentery. Experience in the wards early showed that careful dieting was second in importance only to prompt early treatment. The most complete scheme of diets has been worked out by Colonel John M. Cowan, A.M.S., and Captain Hugh Miller, R.A.M.C. (R.A.M.C. Journal, Oct. 1918). As it was my privilege to work under the guidance of Colonel Cowan during part of my early experience, his scheme was adopted and carried out in the course of my work in dysentery wards in different medical units in after years.

One point is worth emphasising. It is not advisable to keep a patient too long on a very low diet (Albumen water and Barley water). After 36 hours Beef tea at least may be added. The patient is weak enough without any unnecessary starvation added to his condition.

Dysentery/

Dysentery Bacilli. In spite of the numerous bacilli isolated by many bacteriologists from dysentery stools and therefore classed as pathogenic, I have in discussing dysentery only considered cases which showed bacilli of the Shiga or Flexner type. If the ward is near the laboratory the percentage of Shiga or Flexner positive infections is greater than if the converse is the case. It is important that as short a time as possible should elapse before the stool is examined. It is also important that an early stool should be examined. I am sure that later in the disease a greater variety of organisms is found. It is only in very early stools that I have had a report from the laboratory that an almost pure culture has been obtained. As regards the severity of these two infections, undoubtedly cases of Shiga infection are as a rule more severe and lead often to post-dysenteric debility and wasting. But a severe Flexner infection may kill just as surely or lead to as much debility although the Flexner organism usually causes disease of a milder type.