pass water into it. If the urine is clear, the deep urethra is clear. If the urine is purulent, the deep urethra is purulent.

What could be more simple than this? It tells you if the patient is suffering from anterior urethritis alone, or if he is suffering also from posterior urethritis. This is the vital fact you want to know, as you want to explain matters to your patient.

RECTAL EXAMINATION.

Tactile Diagnosis of Inflammation of the Prostate and Vesiculæ Seminales.

In all cases, and especially in cases of acute posterior urethritis, examine the rectum with the right index finger encased in a rubber cot. The prostate may not be swollen at all. Do not, however, massage the prostate, as you will only irritate. (Only in exceptional circumstances is it wise to massage the prostate during the acute stage (five to eight weeks).)

The prostate may be swollen up to the size of a Tangerine orange on one or both sides. This means that there is a prostatic abscess containing several drachms of pus. There is seldom any need to open such an abscess. The exceptions will be considered later. Leave it alone to burst naturally into the deep urethra, unless there is concomitant retention of urine. In that case it is justifiable and often helpful to press gently on the abscess and partially empty it into the deep urethra, with relief of the retention. This is one exception to the rule given above.

In other cases the prostate is not swollen, but one or both vesicles can be felt to be swollen above and outside the prostate. These cases need great care in handling, and should be put to bed. They suggest the likelihood of an approaching epididymitis or of swollen joints and iritis.

I repeat, "Do not as a general rule begin to massage the prostate or the vesicles until at least five to eight weeks from the onset of the disease"—that is to say, only when the chronic and quiescent stage is approaching in the natural course of events.

A gonorrhoeal vesicle is sometimes mistaken for a tuberculous vesicle by the injudicious. The diagnosis is seldom in doubt if a proper history and examination has been made. Do not begin to think of tuberculosis unless there exists no history of exposure to infection or of urethral discharge, but a history of spontaneous epididymitis of a nodular type, the urine remaining clear of pus.

THE DIAGNOSIS OF EPIDIDYMITIS.

Do not forget that by far the commonest cause of epididymitis is gonococcal urethritis, but that at the onset of this complication the urethral discharge in the anterior urethra often suddenly dries up. Ask the man to pass water. You will find it full of pus in a gonorrhoeal case, and, if still in doubt, the urine can be centrifugized, and specimens of gonococci and pus obtained.

In the absence of venereal history and with a clear urine a sudden epididymitis is probably tuberculous. Suspend and rest the testicle a few days, when the acute hydrocele will subside, and it will be possible to distinguish the nodular craggy epididymis, with perhaps isolated nodules or beadings in the vas, an unfailing sign of tuberculosis. If necessary apply the tuberculin tests, and search the urine or the prostatic fluid for tubercle bacilli.

There is, however, another trap for the unwary not mentioned in the textbooks. It is not uncommon for men who are run down by exposure and fatigue to develop suddenly an acute epididymitis with fever, pyuria, pain at the end of micturition, increased frequency of micturition, possibly pain in the back, and even a slight urethral discharge. The pathology is as follows: Being run down, such persons absorb colon bacilli from their bowel into the blood and throw it out through the kidney and prostate. The bacilli lodge and set up inflammation, and spread to the epididymis. Such cases often suppurate; gonorrhoeal epididymitis never suppurates.

The diagnosis is made by cultural examination of the urine, which reveals the colon bacillus in pure growth. I have many instances in my notes of this trouble. Such patients often have a concomitant stricture or senile enlargement of the prostate.

Finally, if a testicle has suppurated and you detect a sinus then you may be certain you are dealing with the tubercle bacillus or the colon bacillus.

DIFFERENTIAL DIAGNOSIS.

Be on your guard against making a rash diagnosis of urethritis because you see pus issuing from a long foreskin. Many a case of balanitis is wrongfully labelled as urethritis. If there is phimosis and pus is present under the foreskin—

1. Make a film and stain. In balanitis no specific diplococci are seen, only mixed cocci or bacilli.

2. Wash away all the pus from under the foreskin and then ask the patient to pass water. If the water is clear you have proof that there is an absence of acute urethritis.

3. Slit up the foreskin if it cannot be retracted and inspect the parts beneath. Usually soft sores, or a chancre, or septic warts will be found, and occasionally even a cancerous or phagedaenic ulcer. Do not, however, forget that specific urethritis may be present as well as balanitis.

4. Another common lesion of the foreskin or glans penis is herpes. Herpetic sores are usually multiple, a little larger than a pin's head, with well-defined punched-out edges, a yellow or grey floor, and painful and tender to the touch. Once a man has developed herpes he is constantly liable to relapse of the trouble. The exact pathology of herpes of the penis is not known, but I believe these little ulcers are caused by some specific germ which, once contracted, lives on the penis, and from time to time works up its virulence and causes ulceration. Probably mechanical excoriation during coitus plays a part in each relapse. A little urethral discharge with pain on passing water is quite common in herpes, and is due to ulceration inside the meatus. The urine is clear, and the microscope proves the absence of gonococci.

5. Do not forget how common it is to find the burrows of scabies not only in the foreskin, but on the surface of the glans itself.

6. Remember also that ulcers about the fraenum may be simple mechanical tears, not infected by any specific organism.

THE CLINICAL ASPECT AND TREATMENT OF ACUTE BACILLARY DYSENTERY.

BY

CAPTAIN J. B. FISHER, R.A.M.C.

BACILLARY DYSENTERY may exist in an acute or chronic form, but I intend to confine my remarks entirely to the acute variety, and in connexion with it shall omit all reference to the bacteriological and pathological aspect of the disease, as my own experience is purely clinical.

Since January 1st, 1916, I have treated 183 cases of acute bacillary dysentery. They have been drawn from the camps in the vicinity of this hospital, and almost all have been admitted within forty-eight or seventy-two hours of the onset of symptoms. Some, indeed, are admitted during the first five or six hours of their illness, and occasionally I have been fortunate in obtaining the case before blood and mucus have had time to appear in the stools. The cases seem to fall into six groups, which clinically merge insensibly one into another, but all can be recognized as true dysentery by the isolation of dysentery bacilli from the motions.

1. Catarrhal Cases.

The onset is sudden, with perhaps ten or more motions in the first twenty-four hours. Afterwards, if untreated, these patients continue to pass frequent diarrhoeic stools containing possibly a trace of clear mucus. Clinically, the cases are distinguishable from simple gastro-enteritis by absence of vomiting and colic, a clean tongue, and preservation of a good appetite. There is also a marked tendency for the bowels to be freely open in the early morning and after a meal. The only absolute proof of the diagnosis is isolation of one of the dysentery bacilli, and this has been done in a number of cases. These patients present no serious symptoms, and generally are easily amenable to dieting and medicinal treatment, though in some of the cases diarrhoea persists until serum is administered.

2. The Ordinary Ulcerative Type.

This class is much commoner, and includes the majority of the patients treated. A common history is that of slight diarrhoea a week before admission, apparent cure, and then a sudden onset of true dysentery, but more often the more serious disease has directly followed a little ordinary diarrhoea.

These patients are generally admitted with a temperature varying from 100° F. to 103° F. They are somewhat toxæmic and drowsy, often having a bluish-red flush upon the cheeks. The tongue is furred, and there is a certain amount of abdominal pain. Examination of the abdomen will only reveal tenderness over the colon. The stools may vary from two or three to twenty or thirty in twenty-four hours.

The seriousness of the case must be judged more by the quality than the quantity of the motions, for a patient with extensive infection of the caecum and ascending colon will not have as much diarrhoea or present the same aspect of discomfort as another patient in whom a much less virulent infection is situated near the lower end of the rectum. The bluish, unhealthy flush of the cheeks just mentioned must not be confounded with the healthy red, sunburnt appearance of many patients. In a few cases it is not present, and in others, as in a patient also suffering from nephritis, the anaemia of the latter disease may entirely mask it.

If the condition be untreated, the temperature is usually remittent and the pulse slightly more rapid than normal. In these patients vomiting is extremely rare. If this symptom is present in a patient admitted with a provisional diagnosis of dysentery, it usually means that either he is only suffering from gastro-enteritis, or that his attack of dysentery is going to be of the fulminating type, and is an indication for the immediate injection of a large dose of serum.

I think the first stage of the disease is a toxæmia emanating from the bacilli multiplying in the mucosa of the large intestine. The first effect of this is to inhibit peristalsis, and it is only when the local irritation becomes sufficiently intense that this stage gives place to the passage of blood and mucus. Consequently, except in the fulminating type, the toxæmia is often in inverse proportion to the diarrhoea, and as the latter increases the former becomes less marked. In the majority of patients this stage of inhibition of peristalsis is transient, but sometimes it is so marked that it is necessary to administer aperients. At first the stools are simply diarrhoeic, but within twelve or twenty-four hours the faecal matter gradually gives place to clear and blood-stained mucus; sometimes also there is a fairly copious admixture of pure blood.

Only in the worst cases do the patients pass much blood-stained serous fluid. As the intensity of the symptoms abates the fresh blood becomes less, the mucus thicker and less watery, until finally the stools may consist of greenish and opaque mucus, which in turn is gradually replaced by faecal material. Sometimes sloughs are also present, but if serum is administered early this is exceptional, so I have purposely omitted to describe them as an ordinary constituent of these motions. In some of the cases the mucus is mixed with pus and broken-down blood, so that the stool is rather like the contents of a hepatic abscess. When the infection includes the lower end of the rectum the stools may be small and frequent, as in amoebic dysentery, but I have always found them less tenacious and more copious and watery than in the amoebic disease.

3. The Toxæmic Type.

Though in this type the patients may have had slight diarrhoea for one or two days, it is not a prominent symptom on admission, and is quite secondary in importance to the toxæmia, which may be so intense that it is only with difficulty that an accurate history can be obtained from the patient. They have, in addition to the characteristic flushed appearance, a moist furred tongue, a high temperature, and a pulse more rapid than normal. They complain of headache and slight abdominal pain, and, if not disturbed, may sleep continuously for twenty-four or forty-eight hours.

During this stage there may be constipation, and it is not until two or three days later, when blood and mucus appear in the stools, that the true condition is revealed.

The local damage is small, and saline aperients are generally all that is required, though, if the case be diagnosed, it is safer to give serum.

One of the cases in this class is interesting in that the first stool, passed forty-eight hours after admission, showed no evidence of dysentery, and it was not till another twenty-four hours had elapsed that blood and mucus began to appear.

4. The Fulminating Type.

In this type the patients are generally admitted within twelve or twenty-four hours of the onset of symptoms. The temperature may be raised, but they are often collapsed, toxic, and give a history of vomiting. The diarrhoea is intense, often passing on to incontinence, and there may be either retention or incontinence of urine. The tongue is dry, and covered with fine and sticky fur. Abdominal pain is very severe, the abdomen so tender that they dread being touched; there is no appetite but great thirst is present. The straining and tenesmus cause insomnia, and such restlessness that morphine may be required. There is, in fact, a combination of severe toxæmia and severe local symptoms.

As the case progresses the loss of fluid causes the patient to shrink and appear smaller, his bones become prominent, his eyes sunken, his skin inelastic, and the tongue so dry that there is difficulty in protruding it. Hiccough may also add to his discomfort, and abdominal pain may remain as severe as on admission. The toxic appearance gives place to an ashen, bluish-grey colour, though even when this stage is reached the patient may recover.

When first admitted the stools are frequent and copious, often one or two pints in volume. They consist of blood-stained serous fluid, sometimes looking like the urine of a case of acute nephritis, and at other times like a weak solution of potassium permanganate with lumps of clotted blood and fibrin floating about. At first there is no mucus, and it would seem that the irritation of the mucosa is so severe that there is no opportunity for its secretion. I am told that if a patient dies in this stage *post-mortem* examination reveals only an intense hyperaemia of the mucous membrane of the intestine.

As the body fluids are excreted the stools become less liquid and blood-stained mucus begins to appear. Soon sloughs are also present.

In spite of the administration of serum the patient will continue to pass necrotic products, blood-stained mucus and pus, and it may be three or more weeks before blood entirely disappears and the mucus takes on that greenish, opaque appearance which is generally a precursor of ultimate recovery.

The progress of the case is to be judged by the diminution of the blood rather than the sloughs, for those portions of mucosa which have been irrevocably damaged in the first week of the illness must take some time to be completely separated. The only treatment of any avail in these cases is early administration of large doses of anti-dysentery serum, and a delay of twelve hours in deciding whether the case is amoebic or bacillary dysentery may mean the ultimate death of the patient. This is why I lay so much stress on the naked-eye appearance of the stools and the bluish-red unhealthy flushed cheeks of early bacillary cases as diagnostic features. I have never seen this toxic appearance in a pure amoebic case.

The temperature cannot be relied upon, as there may be a rise at the onset of acute amoebic dysentery, and in the worst bacillary cases it may be subnormal; the patient may be so ill that administration of a large dose of serum may not cause any reaction.

It must, however, never be forgotten that acute bacillary dysentery may be superimposed upon a milder amoebic infection. In these "mixed cases" the bacillary element may obscure the amoebic, and it is not until four or five days later, when the serum has taken effect, that the case presents the characteristics of amoebic dysentery. This holds good when both emetine and serum have been given from the first, as the action of emetine on the amoebic element, though just as certain, is not so rapid as that of serum on the bacillary.

5. The Choleraic Type.

The onset in these cases is sudden, with collapse, cramps in the arms, legs, and abdomen, and the passage

of rice-water stools. The collapse may necessitate intravenous saline infusion. Lieutenant Willmore tells me that this type was quite common at the quarantine station at El Tor.

I have only had one case, which closely resembled ptomaine poisoning, but was confirmed as dysentery by the bacteriological findings.

The patient had had slight diarrhoea on the Gallipoli Peninsula, but had not been excused duty on account of it. At the time the attack commenced he had been three weeks under treatment for myalgia. He was allowed out of bed, had had no intestinal symptoms, and was upon an ordinary diet. No other case occurred in the same ward. The diagnosis was made upon the following data:

(a) Very acute onset, with rice-water stools containing mucus and mucous sloughs, the latter a dirty white colour.

(b) Absence of cholera vibrios.

(c) Rapid cure following a large dose of antidyenteric serum given within six hours of the onset of symptoms.

(d) Three weeks later the blood strongly agglutinated Flexner Manila dysentery bacilli. It did not agglutinate Shiga-Kruse bacilli.

(e) The patient was isolated, and the sister looking after him developed a similar but slighter attack, and in her case Flexner's bacillus was isolated.

(f) His previous diarrhoea could be accounted for by the presence of *Lambia intestinalis* and *Trichomonas intestinalis*, which were found in abundance in the stools.

6. Localized Attack.

The sixth and last type, in which patients in good health are found to be passing formed stools coated with a little blood and mucus, demonstrates how mild and localized an attack of bacillary dysentery may be, and these cases are interesting as they offer such a contrast to the two preceding types. They sometimes resolve in one or two days, but one patient who refused antidyenteric serum was still passing blood and mucus after two months. This particular patient objected to all forms of treatment except emetine, and of this he had 18 grains, from which he received no benefit.

TREATMENT.

Dietetic.

The aim in deciding upon a diet is to find one which will sustain the strength of the patient without leaving much residue to pass into the large bowel. In accordance with this principle, cases admitted to my lines are only allowed albumin water, barley water, and lemon water for the first forty-eight hours. This may seem rather drastic, but I am sure it is the correct treatment. Also this period of abstinence from food combined with an aperient is generally sufficient to cure the simple gastro-enteritis, and such cases can then be discharged to duty two or three days later. Coincidentally with the improvement of the general conditions of the patient with true dysentery, his diet is gradually increased somewhat upon the following lines:

Breakfast (7 a.m.): Two or three eggs and tea.

Lunch (11 a.m.): Jelly or arrowroot and a piece of chocolate.

Dinner (1 p.m.): Beef-tea and milk pudding.

Tea (4 p.m.): Custard and tea.

Supper (7 p.m.): Cocoa or biscuits.

During the night bovril or a little Horlick's malted milk.

Milk is not allowed except in the form of pudding or custard, as it is much too bulky for an acute or chronic dysentery. In addition it passes through the intestines, and by giving the stools an intense green colour tends to mask their character.

Upon this diet many cases increase in weight. No further increase is allowed until the patient has been ten consecutive days without passing more than two stools in any twenty-four hours, after which bread-and-butter is added, and chicken follows in two or three days more. It is very important that this period of abstinence be enforced in order to give the inflamed and damaged intestine time to recover.

I believe that the irritation and secondary infection caused by increasing the diet too soon after cessation of acute symptoms is one of the chief causes of the cases of post-dysenteric colitis which are so resistant to treatment.

The fulminating cases need further consideration. On account of the subsequent griping, beef-tea, bovril, and similar tasty foods are not indicated, but stimulants may be required. I have found the latter to do good, though in most textbooks they are stated to be contraindicated. In these cases also the mucus may persist so long, the patient becomes so emaciated, and the bowels remain so weak

and irritable, that it may be advisable, for the purpose of increasing the strength and general resisting powers of the patient, to add to the diet before all signs of active dysentery have disappeared.

Medicinal Treatment.

In view of the frequent complaints that the constant desire to go to stool robs patients of sleep, it may seem at first sight that some theoretically sedative mixture such as bismuth or chlorodyne is indicated in order to ease pain, diminish the diarrhoea, and thus allow of sleep.

In a few of my earlier cases I adopted this line of treatment, in spite of the fact that by so doing I was locking up, in intimate contact with the bowel wall, the noxious matter and organisms which the intestine had been doing its best to expel. I found that the effect on the pain was transitory, and that what little sleep was obtained did not refresh the patient, who, on the contrary, began to complain in addition of headaches, flatulence, and a nasty taste in the mouth. I found further that the diminution of the diarrhoea was only apparent for two or three days, and afterwards became as bad as, if not worse than, before, and the stools from being odourless became foul. It is to be presumed, also, that the damage to the intestinal mucosa was greater in proportion to the length of time during which this pseudo-sedative effect lasted.

The drugs above all others which ease pain and allow of sleep are those which aid rather than oppose the effort of nature to free the host of the poison which is invading him, or to neutralize it *in situ*.

Reliance must therefore be placed on specific treatment—namely, multivalent antidyenteric serum. This receives material assistance from such aperients as the sulphates, castor-oil, and liquid paraffin. The former in particular are said to be absorbed in the first part of the alimentary canal, and to be excreted through the mucosa of the large gut, where they exercise a marked exosmotic effect. They thus conduce to lavage from within outwards of the diseased mucosa, and wash out the morbid material in the process.

My treatment has therefore been magnesium sulphate $\frac{1}{2}$ thrice a day for the first few days, and then, as the stools diminished in frequency and improved in appearance, the amount was gradually reduced. This applies to practically all cases, and seems to me of such importance that I would again emphasize the fact that these drugs instead of increasing the diarrhoea diminish it, for by washing out noxious material and cleansing the bowel they get rid of much of the irritation. Larger doses than those which I have mentioned are not indicated, as they may nauseate the patient, increase his discomfort, and perhaps, by scouring too completely the intestinal wall, open the door to bacterial infection of the blood by secondary infecting organisms.

Sometimes in bad cases morphine is needed, and for very severe tenesmus a suppository of cocaine gr. $\frac{1}{2}$ and iodoform gr. iiij will be found most useful. A hot-water bottle will in the early stages add to a patient's comfort. With the above medicinal treatment, combined with the requisite specific treatment, there is a marked improvement in the majority of cases within forty-eight hours. For those patients, however, in whom a dry, furred tongue persists indefinitely I sometimes prescribe pure water one pint thrice a day, and, so far, have found this very satisfactory. Rectal lavage with 1 in 1,000 solution of copper sulphate and mist. simaruba often benefit more chronic cases.

Specific Treatment.

With regard to multivalent antidyenteric serum, no hard-and-fast rule can be laid down as to the quantity it may be necessary to inject. Each individual case must be judged upon its merits, with the proviso that cases coming under treatment late in the disease require larger doses than those of equal or even greater severity seen at the onset. In the more severe cases I inject 80 c.cm. into the flank within a few hours of admission, but generally leave the milder ones until the following morning, and then may give 40 or 60 c.cm. Some of the latter do not require serum, but I have noticed that a severe case given serum immediately after admission often recovers more rapidly than a mild one treated without it.

One large dose of serum is sufficient to cure some cases;

others, though much improved, require more, and in these patients, after waiting forty-eight hours, I give a further 60 or 80 c.cm.

This amount has been sufficient to cure some very severe cases, but a few of the worst may need a third or even a fourth dose. Some cases of the fulminating variety have been given 140 or 160 c.cm. serum within twenty-four hours of admission, but in these patients the first injection should, if possible, be given intravenously, for every moment saved is of vital importance.

In the majority of patients antidysentery serum acts like magic, but there are a very few in whom it seems only to have a slight beneficial effect. If four doses of 80 c.cm. each, spread over a period of six or seven days, have not brought about the desired result, I do not think further injections are indicated; consequently, I have never yet given more than 320 c.cm.

As a general rule there is a distinct rise of temperature within twelve hours of the injection of the serum, and sometimes this may last forty-eight or seventy-two hours. I look upon this phenomenon as a good prognostic sign, for in all cases in which it has been well marked the patients have recovered rapidly. It is on account of this reaction, which may be rather severe, that I generally allow forty-eight hours to elapse between each injection of serum.

The onset of true serum disease has been most frequent on the seventh and eighth days after the first injection. The severity of the symptoms has varied with the stock of serum in use. The patients have complained of urticarial rashes, headaches, sore throats, arthritis, and oedema of the scrotum. They always have a rise of temperature, and five of them showed signs of cardiac dilatation. These symptoms have occurred in varying degrees of severity in 70 per cent. of the cases to whom serum has been given.

RESULTS.

The results obtained in the cases I have treated along the lines above indicated may now be summarized. I have had altogether 456 definitely diagnosed cases. They are made up as follows:

One Hundred and Sixty Cases between August 30th and December 31st.—During this period the pressure of work was so great that it was impossible to keep notes of them all, and in consequence, although an accurate diagnosis of the type of dysentery present was made at the time, I am not now able to give exact figures.

Two Hundred and Ninety-six Cases admitted from January 1st to May 31st.—Of these 183 were acute bacillary cases and the remaining 113 were either chronic amoebic or bacillary. In this period only one case of acute amoebic dysentery was admitted.

In the first 160 cases two deaths occurred. Both patients came from Gallipoli; one a bacillary case, died the night of admission, and the other, who was a "mixed case," died of a perforated amoebic ulcer.

There were no deaths amongst the 113 chronic cases, but five were invalided to England and one to Cairo on account of persistence of symptoms. Possibly others may have been invalided from the convalescent homes to which they were sent.

The 183 acute cases to which this paper particularly refers were all bacillary, but in eight of these *Eutamoeba histolytica* was also present. In this series 69 mild cases did not have serum, 114 had serum. The mixed cases had emetine in addition.

One death only occurred. This was in an emaciated patient aged 39, but who looked twenty years older. The attack was of the fulminating variety, and although he improved a little after large doses of serum, hiccough set in, and he died after being eighteen days in hospital. The diagnosis was verified *post mortem*.

After apparent recovery, 7 cases had a return of diarrhoea before they left the hospital. This, however, cleared up without specific treatment. One case definitely relapsed and required further injections of serum. One other case discharged to duty has since been readmitted. One case who refused serum and continued to pass blood and mucus indefinitely was sent to England. Eight other cases were also sent to England, but they were all convalescent.

They had all been severe cases, and it was thought inadvisable to subject them to an Egyptian summer. Of a total of 183 cases, it will thus be seen that in 173 the recovery was so complete that they were considered fit for duty in Egypt. Apart from serum disease, the symptoms

of which have already been described, there have been very few complications worth mentioning. Hiccough has occurred in 2 cases, intense tenesmus in 1, retention of urine in 2, persistent vomiting in 4, herpes in 1, tachycardia in 4, pleurisy in 2, acute rheumatism in 1, enlargement of the liver in 2.

The offending germ has generally been the mannite-fermenting type of dysentery bacillus.

The length of time in hospital varies with the severity of the disease. Mild cases may be discharged at the end of one month, others may require six to eight weeks before being fit for duty, and, finally, patients of the fulminating class may be three or four months, and can then only be considered convalescents.

The length of persistence of the acute symptoms is in inverse proportion to early diagnosis and early administration of antidysentery serum. If an ordinary case be diagnosed soon after admission the injection of serum will generally limit the acute stage to a period of four to nine days. I have had numerous cases in which one dose of 60 c.cm. of serum has reduced, within a period of twenty-four hours after the injection, the number of stools from about twenty to about four. Even although all blood has disappeared from the stools and the case appears convalescent within a few days of his admission, his subsequent complete recovery cannot be hurried, as the diet can only be very gradually increased.

When the patient comes under treatment in the stage in which the bacilli are attacking the intestine, the virulence and intensity of the symptoms are not of much moment, provided that they are accurately gauged and a correspondingly large dose of serum be given.

If, however, the severity of the case is underestimated or the patient be unfavourably situated for early treatment, serious damage may be done to the mucosa before an adequate dose of serum is given.

In these cases, when serum is finally injected, it cannot be expected to give such striking results. By killing all remaining active dysentery bacilli it can prevent further sloughing, but the symptoms due to the ulceration already present will naturally only gradually abate.

ON THE TREATMENT OF DYSENTERY.

BY

G. A. FINLAYSON, M.B.ABERD., M.R.C.P.LOND.,
GOVERNMENT PATHOLOGIST, SINGAPORE; LATE CAPTAIN R.A.M.C.(T.).

THOUGH stationed for the past year at a base hospital where the facilities afforded for the treatment of dysentery were excellent, I am fully alive to the difficulties which have to be faced at a field ambulance or casualty clearing station, but I am still of opinion that a more active and vigorous treatment of cases of acute bacillary dysentery is demanded in the early stages of the disease, more particularly by the exhibition of larger doses of antidysenteric serum than have usually been administered in the transferred cases. In the great majority "Mulford's" serum was employed, and it seemed unfortunate that more attention was paid to the dosage prescribed in a "booklet" widely distributed than to the directions printed on the wrapper. A point naturally of great importance in early diagnosis is whether the patient has been infected by the specific amoeba or bacillus. A microscopical examination of the faeces should be made as soon as possible. There is no valid reason why a medical officer attached to a field ambulance and stationed where dysentery is likely to occur should not be supplied with a portable microscope provided with a $\frac{1}{4}$ in. objective and No. 2 ocular; a brief demonstration of the more common intestinal protozoa would, it is needless to add, prove of immense service.

Immediate treatment rests mainly on the microscopical finding, bearing in mind that one is dealing with the early stage of a primary attack. During the past six months only six cases of primary amoebic dysentery were admitted within two to three days of onset; all were discharged cured in less than three weeks. Emetine gr. j hypodermically daily for ten days, or gr. j hypodermically for six days in the morning, with gr. $\frac{1}{2}$ in a keratine-coated pill in the evening. Another case, transferred from Mesopotamia, had been given injections of tannic acid twice daily for seven weeks (the amount of tannic