

An Address ON THE DIAGNOSIS AND TREATMENT OF DYSENTERY.

DELIVERED BEFORE THE SOUTH MIDLAND BRANCH OF THE
BRITISH MEDICAL ASSOCIATION

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GENTLEMEN,—In the first place let me say how greatly I appreciate the honour which your President has conferred upon me by asking me to address you to-day. When I first received his invitation I was doubtful as to my ability to tell you anything in connexion with tropical diseases that would be of use to you in your practice; but when I remembered that a large Anglo-Indian colony exists in Bedford, it appeared to me that the choice of a subject lay between malaria and dysentery, and I finally decided upon the latter.

The term "dysentery" was first employed by Hippocrates to distinguish a disease of the bowels characterized by the presence of blood in the stools, but later on the disease was differentiated more clearly by emphasizing the fact that there must be mucus as well as blood in the stools, and that the symptoms of tormina and tenesmus must be present. This definition still held good when I went to India thirty-four years ago. We were taught that the association of frequent dejections and straining with blood and mucus in the stools was sufficient for the diagnosis of clinical dysentery, and any patient exhibiting this syndrome was at once placed upon enormous doses of ipecacuanha, elaborate precautions being taken to prevent it from acting as an emetic.

Here I wish to warn you against the danger of thinking that because a patient who comes to you for advice has resided for many years in the tropics, he is on that account necessarily a victim to some form of tropical disease. If, therefore, an Anglo-Indian comes to you complaining of the classical symptoms of dysentery, do not at once jump to the conclusion that he is really suffering from that disease. There are numerous "pseudo-dysenteries," and the symptoms in question may be merely manifestations of tuberculous or malignant ulceration of the bowel, of stricture or gumma of the rectum, of inflamed piles, of helminthic infections, or they may be a terminal condition of that very fatal disease, kala-azar. All these may, however, be excluded by careful examination of the thorax and abdomen, by digital exploration of the rectum, by microscopic examination of the faeces, and, in the case of kala-azar, by an exploratory puncture of the enlarged spleen and search for the *Leishmania-Donovani* parasite.

BACILLARY AND AMOEBIC DYSENTERY.

Having by these means satisfied yourselves that you are dealing with a true dysentery, the next point for consideration is "What type is it—is it bacillary or amoebic?" This is of paramount importance, because the treatment suitable in the one form of the disease is useless, or even harmful, in the other. We owe our knowledge of the etiology of bacillary dysentery to the investigations of Shiga in Japan and Kruse in Germany during the years 1898-1900. They proved that the causal organism of the disease is a short bacillus, belonging to the *typho-coli* group, which is Gram-negative, non-motile, and which does not either clot milk or produce gas in any sugar media. Further investigations by Flexner, His and Russell, and Strong have shown that there are various strains of dysentery bacilli which resemble the original Shiga-Kruse bacillus, in that all of them have the common property of producing acid in the monosaccharides, glucose and galactose, whilst they do not ferment either lactose, dulcitol, or sorbitol. They differ from the Shiga-Kruse bacillus, however, in two important particulars—they all ferment mannite and they produce indol. Considerable divergence of opinion exists as to the exact significance of bacilli belonging to the latter subgroup.

American writers regard all of them as being of equal etiological importance, whilst the German school hold that the Shiga-Kruse bacillus is the only true type, and this view receives some support from the results obtained by serum therapy. These various forms of *B. dysenteriae* are responsible for the production of three distinct types of disease—(1) epidemic dysentery, as seen in India; (2) asylum dysentery, as seen in England; and (3) certain types of infantile diarrhoea.

But there is another type of dysentery which is endemic or sporadic in its nature, which is most apt to run a chronic course, and which in a large proportion of cases is complicated by abscess of the liver. This type is associated with the constant presence of amoebae in the stools and in the lesions, especially in the walls of liver abscesses, and the pathological changes found in the bowel after death are entirely different from those of bacterial dysentery. As regards the relationship of the amoebae to this disease, there are three views: (1) That the presence of the amoebae is accidental, and they play no part in the causation of the disease; (2) that the ordinary *Amoeba coli* is a harmless saprophyte, but that there are two pathogenic amoebae—namely, *Entamoeba histolytica* and *E. tetragena*; (3) that, under certain conditions, all amoebae may become pathogenic. For reasons which I shall give later on we may, I think, reject the first view. The other two are subjects for academic discussion, and do not concern us as practical clinicians. It is sufficient for our purpose to remember that in one form of dysentery there is an absence from the stools of the various types of *B. dysenteriae*, and that their place is taken by certain forms of amoebae. Also that this disease differs from bacterial dysentery both in its pathology, symptomatology, prognosis, and its reaction to treatment.

As regards the symptomatology, the points of distinction between the two diseases are as follows:

1. Bacillary dysentery is always acute in onset, and it runs a rapid course in nearly all cases. Amoebic dysentery, on the other hand, even though it may begin with an acute attack, always runs a chronic course. It is therefore the form you are most likely to see in this country.

2. In bacillary dysentery pyrexia is common; in amoebic dysentery it is rare.

3. In bacillary dysentery toxæmia is a marked symptom; in amoebic dysentery it is absent except when a liver abscess is forming.

4. In bacillary dysentery the stools are muco-purulent, with large cellular exudate and small in bulk; in amoebic dysentery they are grumous, like those of serous colitis, they are copious, and they contain little or no cellular exudate.

5. Liver abscess never occurs in bacillary dysentery; it is a common complication in amoebic dysentery.

DIAGNOSIS OF ABSCESS OF THE LIVER.

Its onset is always insidious, but no difficulty occurs in a typical case in which you have the classical symptom of hectic temperature with night sweats, leucocytosis, diminished movement of the right side of the diaphragm, pain under the right shoulder blade, a dragging sensation on the right side associated with fullness and tenderness in the right hypochondrium, and rigidity of the right rectus. In many cases, however, all or many of these signs are wanting. You must remember (1) that the interior of the liver does not contain sensory nerves, and that quite a large abscess may form deep in the substance of that organ without causing any pain; (2) that a considerable portion of the liver is under the ribs, so that there may be no appreciable swelling, especially if the abscess is on the upper surface of the liver; and (3) that about 40 per cent. of liver abscesses are sterile, so that there may be a complete absence of fever or leucocytosis. Indeed, I have on several occasions diagnosed and evacuated abscesses of the liver in which all these signs and symptoms have been conspicuous by their absence.

How, then, is the diagnosis to be made in a case of this kind? The answer is simple—"By watching your patient." In amoebic dysentery there is an absence of toxæmia, except when a liver abscess is forming. If, therefore, a patient who has suffered from amoebic dysentery is obviously toxic and is losing flesh rapidly;

if his face is drawn and his expression listless, his complexion sallow, his conjunctivae dull, and his tongue thickly furred, lose no time in making an exploratory aspiration of his liver. The operation can do no harm even if you fail to find pus, and it may save you from the unpleasant experience of being sent for one day to find your patient coughing up pus from an unsuspected abscess of his liver which has burst into the lungs, or writhing in agony from its rupture into the peritoneum. Moreover, if when you aspirate the liver you find only a small abscess, and especially if the absence of fever leads you to think it is sterile, you may frequently cut short the disease and render subsequent operation unnecessary by repeated irrigation of the abscess cavity with a solution of bihydrochloride of quinine (3 to 5 grains to 1 oz.) by means of the flexible sheathed aspiration cannula which has been designed by Major Rogers for that purpose.

From the description I have given of the symptoms you will see that in the majority of cases no great difficulty arises in the diagnosis of amoebic dysentery from the bacillary form of the disease, but it is not always plain sailing, and in any doubtful case a complete microscopic and bacteriological examination of the faeces is advisable. The identification of the various forms of *B. dysenteriae* takes up more time than the busy practitioner can afford to give to it, and shreds of mucus should be put up in sterile tubes and sent to a pathological laboratory. But it is quite easy to detect the amoebae; for this purpose all that is necessary is to take a small piece of a freshly-passed stool and place it on a slide, adding to it one or two drops of a 1 in 10,000 solution of neutral red in normal saline. A cover-glass is then placed on top, gently pressed down and "ringed" with vaseline, and the preparation is examined under a $\frac{1}{2}$ objective and a 1 or 2 in. eyepiece. With this magnification the amoebae are easily detected, and it will be found that they have taken up the neutral red, all the other constituents of the faeces, even the leucocytes, remaining uncoloured.

TREATMENT.

It is impossible to deal with this very important subject fully in the short time at my disposal. I shall confine myself, therefore, to the specific treatment of the causal organisms.

Bacillary Dysentery.

In bacillary dysentery we may adopt either the saline or the bacteriological methods of treatment; but in the amoebic form of the disease we must rely upon ipecacuanha or emetine.

There are two methods of treating bacteriologically a case of bacillary dysentery: (1) The use of a vaccine, thus producing an active immunity; (2) the use of an antitoxic serum, thus raising passively the immunity of the patient.

1. *Vaccines.*—Much work on this subject has been done in India by Major Forster, Professor of Pathology in the Lahore Medical College. His vaccine is prepared with the Shiga-Kruse bacillus, which he uses for all types of bacillary dysentery. He uses it in both acute and chronic cases, but in acute cases it is contraindicated from the fourth to the twenty-first day. After this period inoculation may be commenced with an interval of ten days between each dose. In both acute and chronic cases the dose recommended for an adult male is: First dose, 0.1 c.cm.; second dose, 0.2 c.cm.; third dose, 0.3 c.cm.; fourth dose, 0.4 c.cm. Females and children are given proportionately smaller doses. According to Forster these doses produce little or no negative phase, and in fourteen days the immunity is considerably raised, whilst a course of four doses usually suffices to get rid of all bacilli and to prevent the patient from becoming a chronic "carrier." Most authorities consider that on account of the severity of the local reaction and the risk of producing a negative phase the vaccine method is best reserved for chronic cases, and that in acute cases it is inferior to the serum and saline treatments. Certain experiments, however, are now being made with "sensitized vaccines," which, as you are doubtless aware, consist of bacilli which have become coated with specific antibodies (lysins, agglutinins, etc.) as the result of exposure to the action of an immune serum. Such bacilli are now "sensitized" and do not lose this property when killed by heat. As

the result of this sensitization, they are more easily dealt with by the leucocytes, and the toxicity of the vaccine is decreased, whilst its immunizing power remains unaltered. For these reasons I am disposed to think that there is a great future before these sensitized vaccines, and that it may be necessary ere long to modify our views as regards the unsuitability of vaccine treatment in the acute stages of disease.

2. *Antidysenteric Serums.*—These may be either monovalent or polyvalent, but the highest degree of immunity is conferred when the Shiga-Kruse bacillus is used in their preparation. These serums are of the greatest value in the acute stages of the disease, especially if there be profound and prolonged toxæmia unaffected by other forms of treatment. The dosage depends upon the strength of the serum used, and is, as a rule, marked on the phial, but it is necessary to give it in large doses, the injections being made with the usual antiseptic precautions under the skin of the abdomen or flank. Thus the serum from the Lister Institute should be given in 20 c.cm. doses twice, or even, in severe cases, four times daily. The interval between the injections should be six hours. They need not, as a rule, be continued after the second or third day, and the only untoward effects likely to follow are urticarial rashes and other anaphylactic phenomena such as pain in the joints, etc. Textbooks tell us that these complications may be avoided by the administration of calcium chloride or lactate. Experience does not support this statement, but it may be taken as granted that grave anaphylactic phenomena only occur when the interval between the doses is long or irregular. The remedy, therefore, is regular and systematic administration of the serum.

Salines.—When the serum treatment cannot be carried out, or when you get the patient in the first day of the disease, the saline treatment is, in my experience, the most useful. Indeed in many early cases it suffices for a complete cure. Two drachms each of magnesium sulphate and sodium sulphate dissolved in 1 oz. of water should be administered and then half doses of a similar mixture should be given every hour or every two hours until the motions became faeculent, after which it is given every three or four hours for another twenty-four hours and then discontinued; but do not stop it too soon, otherwise the patient will still suffer from faecal retention, even though he be passing a large number of stools containing only blood and mucus.

Amoebic Dysentery.

Up till recently our only method of treatment of this disease was by the administration of huge doses of ipecacuanha. This treatment, if persevered in, was undoubtedly efficacious, but the nausea caused by the drug was so severe that it was extremely difficult to persuade patients to take it. To remedy this a de-emetinized ipecacuanha was put on the market some years ago, but this proved to be useless, and we were reluctantly compelled to return to our old methods. Recently, however, the treatment of amoebic dysentery has been revolutionized by the researches of Vedder in 1911 and by the brilliant discovery of Major Rogers that the amoebae can be quickly killed by the hypodermic injection of emetine, and that, when given in this way, the patient experiences no nausea or discomfort of any kind. Both the hydrochloride and the hydrobromide of emetine are equally useful, but the former is more soluble, the latter requiring 2 c.cm. of sterile water or saline for the solution of one dose. Rogers began with $\frac{1}{2}$ grain, which is equal to 30 grains of ipecacuanha, but he now gives 1 grain of emetine each day, in two hypodermic doses of $\frac{1}{2}$ grain each, and he claims that by this method all the amoebae in the body can be killed in from two to four days, the average for a number of cases being 2.35 days. He has also shown that one or two $\frac{1}{2}$ grain tablets may be given by the mouth on an empty stomach without exciting any great nausea, especially if they are keratine-coated. This method of administration does not effect a cure as speedily as when the drug is given hypodermically, but it may be useful when for any reason there are difficulties in connexion with the giving of the injections.

The hypodermic injection of emetine is also a valuable aid to diagnosis. If you have a case which you suspect to be amoebic dysentery, but which does not react to emetine within three days, you may be sure that your diagnosis is

wrong, and you need waste no further time in treating it as such. Moreover, the powerful effect of emetine on amoebic cases, compared with its uselessness in bacillary cases, may, I think, be taken as strong proof that amoebic dysentery is really a separate entity. That is why, in the earlier part of this address, I said that we might reject the view that intestinal amoebae play no part in the causation of this disease.

A Lecture ON MINERS' NYSTAGMUS.*

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Miners' nystagmus is an occupational disease of the nervous system, and is only found among workers in coal mines. The first case was described in Belgium, by Decondé, in 1861; and among the earliest observers were C. Bell Taylor, Nieden, Gräfe, and Snell. Romiée pointed out that the earliest recorded case was described in Belgium ten years after the safety lamp had been recommended for use in that country, and that it was only after the compulsory introduction of the Mueseler lamp in 1876 that he noticed an increase in the number of cases of the disease.

There have been two chief views as to the causation of the disease—the one that it is due to the position assumed by the miner at his work, and the other that it is due to deficient illumination. The first view was very ably and strongly advocated by Dransart, Nieden, and Snell, and the last named had several discussions with Dr. Court, who has the honour to be the first English observer to call attention to the importance of the light factor. Most English observers now hold that the chief factor is deficient light, but on the Continent there is still diversity of opinion.

SYMPTOMS.

The first symptom is failure of sight, especially at night time, or when the sufferer is called upon to perform the more skilled portion of his work. The man next complains that the lamps dazzle his eyes, and sooner or later that the lamps and all surrounding objects dance before him. Headache, varying from slight pain between the temples to attacks of extreme severity, giddiness on exertion and stooping, inability to see at night time, and dread of a bright light, are often present.

There are two distinct varieties of the disease. In the first the symptoms are absent or latent, and the man, suffering no disability, is unaware that he has nystagmus; in the second the disease is manifest, and the man is more or less incapacitated and aware that his eyes are affected. Among 750 consecutive cases 150 latent cases were observed.

The table shows the frequency of the various symptoms in the remaining 600 manifest cases.

TABLE I.—Symptoms in 600 Manifest Cases.

	Number.	Percentage.	Symptom Marked.	Very Marked.
Movements of objects ...	566	94.3	—	—
Headache	507	84.5	51	16
Giddiness	490	81.6	52	8
Night blindness	459	76.5	4	—
Dread of light	284	47.3	10	—

PHYSICAL SIGNS.

The signs of the disease are: Involuntary and irregular movements of the eyeballs, chiefly of a rotatory character, tremor of the eyelids, eyebrows, head, and, in some cases, even of the neck and shoulders. A backward inclination

* Delivered at the Mining Machinery Exhibition, London, on May 31st.

of the head with drooping eyelids is characteristic and common. An attempt has been made lately to describe a condition in which the disease miners' nystagmus exists without the presence of the nystagmus proper, and in which the oscillation of the eyeballs is replaced by blinking of the eyelids. The question is at present under consideration by a Departmental Committee, and there is no time this afternoon to discuss the matter.

THE CAUSE OF NYSTAGMUS.

Owing to the deficient light in a coal mine, the images formed in the eyes are indefinite and inexact; this leads to indecision on the part of the controlling mechanism in the brain, with the result that irregular inco-ordinate movements of the eyeballs ensue. These irregular movements of the eyeballs are known as nystagmus.

FREQUENCY OF THE DISEASE.

Foreign authors estimate the frequency of the disease as being from 5 to 25 per cent. of all workmen employed underground. The following table will show the number of men disabled by the disease in this country. The figures are taken from the Blue Books on statistics of compensation.

TABLE II.—Frequency of Disablement.

Year.	Number of Certified Cases.	Percentage to Underground Workers.	Cost of All Industrial Diseases.
1908	460	0.057	£13,782
1909	1,011	0.23	£26,759
1910	1,618	0.19	£42,507
1911	2,519	0.29	£68,017

I have estimated the cost of the disease to the country in 1910 to have been over £100,000, and on the same basis to have been over £155,000 in 1911.

INCAPACITY.

With regard to the incapacity caused by the disease, I hold the following views: Slight cases can soon return to work underground; ordinary cases can return after an interval of surface work of three to twelve months. Although one attack of nystagmus predisposes to another, I think it only fair to allow a man to try to regain his income by working underground again if possible. The exceptional cases should not return to work underground, and by exceptional cases I mean the following: Men who have failed to work several times before; very young lads; old men with commencing cataract; men with high degree of refractive error, and those cases which have been of exceptional severity. The following table was taken from returns, extending over four years, from five large colliery companies employing over 28,000 men:

TABLE III.—Late Results.

Back at old work	152 or 45 %
Left employ or commuted	9
At surface work	105
Idle	73

It is at the present time much more difficult to persuade men to attempt to work underground.

A further question may be asked: Is it dangerous to employ a man underground who has once suffered from nystagmus? In the first place, is the man more liable to accident? It is very difficult to answer this question. Men are often sent out of the pits by managers from fear of this danger, but this is when they are about to fail on account of the disease. I think it probable that many slight accidents are due to nystagmus, but I have only once had a complaint from a man that his accident was directly due to his nystagmus. There is, however, the possibility of a greater danger—namely, that a catastrophe may result from the failure of a fireman or collier suffering from the disease to detect the presence of gas. The table given below shows the results of tests made on all the firemen of five large collieries. The tests were made on the surface with the help of an Oldham gas-testing chamber. The men were first examined for the presence of nystagmus and then tested independently for their ability to detect the cap given in the presence of gas.