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 any value to you, and your President has confirmed to me 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 the disease was described more clearly by emphasizing the fact that there must be mucus as well as blood in the stools, and that the symptoms of tenesmus and tenesmus must be present. This definition still holds good, and I went to India this year. We were taught that the association of frequent defaecations 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 received mucus in the stools in that tropics, he is the 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 fear you may have a patient who is really suffering from that disease. There are numerous "pseudo-dysenteries," and the symptoms in question may be merely manifestations of tuberculosis or malignant ulceration of the bowel, of ulcer or tumours 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 rectum and abdominal and digital exploration of the rectum, by microscopic examination of the faeces, anced, in the case of kala-azar, by an exploratory puncture of the enlarged spleen and search for the Leishmania-Donovani parasite.

BACILLARY AND AMOEBOIC 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—bacillary or amoeboic?"

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 the German in Japan and Kruze in Germany during the years 1898-1900. They proved that the causal organism of the disease is a short bacillus, belonging to the typhoid group, which is Gram-negative, non-motile, and which does not either outgrow or produce gas in any sugar media. Further investigations by Flexner, His and Russell, and Strong have shown that there are various strains of bacilli which resemble the original Shiga-Kruze bacillus in 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-Kruze bacillus, however, in two 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-Kruze bacillus is the only true type, and this view receives some support from the results obtained by serum therapy. These different forms of dysentery 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 forms 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 abscesses of the liver. This type is associated with the constant presence of amoebeae in the stools and in the lesions, especially in the walls of liver abscesses, and the pathological changes found in the liver after death are entirely different from those of bacterial dysentery. As regards the relationship of the amoebeae to this disease, there are three views: (1) That the presence of the amoebeae 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 amoebeae—namely, Entamoeba histolytica and E. tetragona; (3) that, under certain conditions, all amoebeae may become pathogenic. For reasons which it is not necessary for me now to explain, I shall give later on my own view of this 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 amoebeae. Also 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. In bacillary dysentery is always acute, and its onset, and it runs a rapid course in nearly all cases. Ameobie 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 ameobie dysentery it is rare.

3. In bacillary dysentery toxemia is a marked symptom; in ameobie 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 ameobic 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 ameobie dysentery.

DIAGNOSIS OF ABSCESSES OF THE LIVER.

Its onset is always insidious, but no difficulty occurs in a typical case in which you have the classical symptoms 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 fulness 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 in the absence of the liver disease the liver does not contain serum, 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 sterile, 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 aspiration. In ameobie dysentery there is an absence of toxemia, except when a liver abscess is forming. If, therefore, a patient who has suffered from ameobie 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 furry, 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 told by a patient within 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 peritonieum. Moreover, if which you move the liver you find a small fluctuation 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 bichloride of lime (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 the patient whose case is to be seen in this way. 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.

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 acute immunity; (2) that 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.c.; second dose, 0.2 c.c.; third dose, 0.3 c.c.; fourth dose, 0.4 c.c. 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 "case." 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 method. In some 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 (lysin, agglutinins, etc.) as the result of exposure to the action of an antibody. Such bacteria are "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 I am prepared to modifying my views as regards the unsuitability of vaccine treatment in the acute stages of disease.

2. Antidysenteric Serums.—These may be either monovalent or polyvalent, and are divided into the highest dose of human serum that is confounded 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 proven bacilli, for they may act almost immediately, whereas the bichloride of lime solution is not so prompt in its action.

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.c.m. 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 urti
cariah rash and other anaphylactic phenomena such as bronchial asthmatic attacks, etc. Text-books tell us that these complications may be avoided by the injection of a solution 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 doses is unequal, and 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 second 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 dracons 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 symptoms become fasiculant, after which it is given every four hours for another twenty-four hours. If this is 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 ipe
cacuanha. This treatment, if persevered in, was undoubtedly efficacious, but the nausea caused by the drug was so severe that the patient was unable to take the patients to take it. To remedy this a de-emethizined 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 amoebaie 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 hydro
cloride and the hydrobromide of emetine are equally useful, but the former is more soluble, the latter requiring 2 c.c.m. of sterile water or saline for the solution of one dose. Rogers began with ⅛ grain, which is equal to 30 grains of ipecacuanha, but he now gives 1 grain of emetine each day, in two hypodermic doses of ½ grain each, and he claims that by this method the amoeba in the body can be killed in from two to four days, the average for a number of cases being 2.5 days. He has also shown that one or two ¼ grain tablets may be given by the mouth on an emesis stimulant, although this is not a 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 successful when for any reason therefore 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, and which does not manifest 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 is often with its mesaline 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.

by T. LISTER LLEWELLYN, M.D., B.S.LOND.,
TYNDALL RESEARCH STUDENT OF THE ROYAL SOCIETY.

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, Grafé, and Snell. Romée 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 Mueller 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 work, and the other that it is due to deficient illumination. The first view was very sly and strongly advocated by Dransart, Nieden, and Snell, and the last named had several discussions with Dr. Court, who had 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 stopping, inability to see at night time, and dread of objects, 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>
<thead>
<tr>
<th>Symptom</th>
<th>Number</th>
<th>Percentage</th>
<th>Marked</th>
<th>Very Marked</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>507</td>
<td>81.5%</td>
<td>51</td>
<td>16</td>
</tr>
<tr>
<td>Giddiness</td>
<td>490</td>
<td>81.6%</td>
<td>52</td>
<td>8</td>
</tr>
<tr>
<td>Night blindness</td>
<td>459</td>
<td>76.5%</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Dread of light</td>
<td>284</td>
<td>47.3%</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

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 of the head with dropping 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>
<thead>
<tr>
<th>Year</th>
<th>Number of Certified Cases</th>
<th>Percentage to Underground Workers</th>
<th>Cost of All Industrial Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1908</td>
<td>460</td>
<td>0.05%</td>
<td>£1,782</td>
</tr>
<tr>
<td>1909</td>
<td>1,011</td>
<td>0.33%</td>
<td>£26,759</td>
</tr>
<tr>
<td>1910</td>
<td>1,518</td>
<td>0.19%</td>
<td>£42,507</td>
</tr>
<tr>
<td>1911</td>
<td>2,519</td>
<td>0.29%</td>
<td>£68,017</td>
</tr>
</tbody>
</table>

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 was taken from returns, extending over four years, from five large colliery companies employing over 28,000 men:

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Cost of All Industrial Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Back at old work</td>
<td>£152 or 45 %</td>
</tr>
<tr>
<td>Left employ or commuted</td>
<td>£90</td>
</tr>
<tr>
<td>At surface work</td>
<td>£105</td>
</tr>
<tr>
<td>Idle</td>
<td>£73</td>
</tr>
</tbody>
</table>

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. Many men are sent out of the pits by managers from fear of his danger, but this is with the object of keeping the man out of the way of the accident. 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 personnel 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.