An Address
ON
THE DIAGNOSIS AND TREATMENT OF DYSENTERY.
DELIVERED BEFORE THE SOUTH MIDLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION
BY
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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 not be put forward by you in your practical knowledge. I remembered that a large Anglo-Indian colony exists in Bedford, it appeared to me that the choice of a subject for any occasion placed upon me that was more clearly an explanation of the fact that there must be mucous as well as blood in the stools, and that the symptoms of the disease are present. This definition still hold good when I went to India this year. We were taught that the association of frequent defecation and straining with blood and mucus in the stools was sufficient for the disease to be diagnosed. And any patient exhibiting symptoms of dysentery, and any disease, was once placed upon numerous 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 this is a disease which does not exist in the tropics, that it is being used necessarily a victim to some form of tropical disease. If, therefore, an Anglo-Indian colony exists in connexion with the tropical symptoms of dysentery, do not forget to consider the possibility that he is 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, or the result of the excess of infected cases, or the presence of helminthiasis, or may be a terminal condition of an acute disease, or a terminal condition of that very fatal disease, kala-azar. All these may, however, be excluded by careful examination of the faeces and of the stools, by digital exploration of the bowel, and by microscopic examination of the faeces, and, in the case of kala-azar, by 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—is it bacillary or amoebic?" This is of paramount importance, because the treatment suitable in 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 of Kruse in Germany during the years 1898-1900. They proved that the causative 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 multiply 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 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 particular cases— 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 true type, and this view receives some support from the results obtained by the diphtherin therapy. These bacilli are responsible for the production of three distinct types of disease—(1) epidemic dysentery, as seen in India; (2) asyly 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 is in a large proportion of cases complicated by abscess. This type is associated with the constant presence of amoebae in the stools and in the lesions, especially in the walls of the lesions, and the pathological changes found in the rectum after death are distinctly 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 amoeba, Entamoeba histolytica, is a harmless saprophyte, but that there are two pathogenic amoebae—namely, Entamoeba histolytica and E. tenagena; (3) that, under certain conditions, all amoebae may become pathogenic. For reasons which I will give later on I am of the opinion that there are two forms of amoebic dysentery, the one being associated with parasite in 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 pathogenesis, symptomatology, prognosis, and its reaction to treatment. As regards the symptomaticology, the points of distinction between the two types of dysentery as follows: 1. In bacillary dysentery the onset is rapid, 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 of interest to inquire how 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 toxaemia 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 purulent, with large cellular exudate and small in bulk; in amoebic dysentery they are purulent, with large cellular exudate and small in bulk; in amoebic dysentery they are amorphous, like those of serious collitis, 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 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 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 abscess of the liver does not contain septic matter, so that quite a large abscess may form deep in the substance of the 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 the history. In amoebic dysentery there is an absence of toxaemia, 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 told by a small and mean one day to find your patient coughing up pus from an unsuspected abscess of his liver which has burst into the lungs, or whirling in agony from its rupture into the peritoneum. Moreover, if white bacilli streak the liver you have a grain in your hand and especially if the absence of fever leads you to think that 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 mercury (0.3 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 lose, and all shreds of muscle should be marked off 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 1 in 5000 solution of neutral saline. A cover-glass is then placed on top, gently pressed down and “ringed” with vaseline, and the preparation is examined under a 3 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.**

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 ippecacuanha or emetine.

There are two methods of treating bacteriologically a case of bacillary dysentery: (1) The use of a vaccine, thus producing a specific immunity; (2) that used in the intramuscular injection of a toxic 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-Krusse 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.m.; second dose, 0.2 c.c.m.; third dose, 0.3 c.c.m.; fourth dose, 0.4 c.c.m. Females and children are given proportionately smaller doses. According to Forster these doses produce a 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 saline method. Some authors contended 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 saline method. Some authors contended 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. Some authors contended 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 saline method. Some authors contended 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 saline method. Some authors contended 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 saline method. Some authors contended 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 saline method.

**Amoebic Dysentery.**

Up till recently our only method of treatment of this disease was by the administration of huge doses of ippecacuanha. This treatment, if persevered in, was undoubtedly efficacious, but the nausea caused by the drug was so severe that it was found difficult to carry the patients to take it. To remedy this a de-emetinized ippecacuanha 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.c.m. of sterile water or saline for the solution of one dose. Rogers began with 1 grain, which is equal to 30 grains of ippecacuanha, but he now gives 1 grain of emetine daily, in two hypodermic doses of ½ 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 2.5 days. He has also shown that one or two ½ grain tablets may he given by the mouth on an empty stomach in any great nausea, especially if they are keratin-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 connection 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 conform to the points mentioned above 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 meals. In halcyon 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., TYNDALE 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, Grafe, 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 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 work, and the other that it is due to defective illumination. The first view was very silly 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 stopping, inability to see at night time, and dread of 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.

<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>3</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 image 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 the country. The figures are taken from the Blue Books on statistics of compensation.

Table II.—Frequency of Disableness.

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of Certified Cases</th>
<th>Percentage to Under-ground Workers</th>
<th>Cost of All Industrial Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1908</td>
<td>460</td>
<td>0.057</td>
<td>£41,782</td>
</tr>
<tr>
<td>1929</td>
<td>1,011</td>
<td>0.33</td>
<td>£28,759</td>
</tr>
<tr>
<td>1980</td>
<td>1,618</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 table was taken from returns, extending over four years, from five large colliery companies employing over 28,000 men:

Table III.—Late Results.

<table>
<thead>
<tr>
<th>Class</th>
<th>Number</th>
<th>Percentage</th>
<th>Cost of All Industrial Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Back at old work</td>
<td>......</td>
<td>152 or 45 %</td>
<td>£42,507</td>
</tr>
<tr>
<td>Left employ or commuted</td>
<td>......</td>
<td>......</td>
<td>£105</td>
</tr>
<tr>
<td>Idle</td>
<td>......</td>
<td>73</td>
<td>£68,017</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. Men are often sent out of the pits by managers from fear of this danger, but this is without any 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.

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*Delivered at the Mining Machinery Exhibition, London, on May 13th.