

Miss Te Winkel was for many years a member of the T. N. A. I. and always manifested a deep interest in its growth, as she had known the Association in its infancy.

She was a member of the Mission Examining Board in South India and its Secretary for a short period. As a member of a Committee to write a Nursing Text book for Indian Nurses she worked indefatigably writing some of the chapters herself and helping to edit the book. The first edition of this book was soon exhausted and again as a member of the revision Committee she helped prepare the book for its second edition as well as helping with the Telugu translation. This book has been of invaluable help to nurses not only in South India but in other parts of India as well.

In 1935 Miss Te Winkel retired from active service in India. She was then in poor health and prepared to go to U. S. A. On the way home she contracted pneumonia and was a patient for some time in a Liverpool hospital. When able to travel she continued her journey to U. S. A. and made her home in Florida. She was never very strong after her return being more or less of an invalid all the time. Always interested in the progress of Nursing in India she kept up her contact with her many nurse friends both Indian and Europe through correspondence.

To those of us who knew her well her patient perseverance in difficult situations, her loyal devotion to duty, her self-forgetfulness and her loving sympathy inspire us to follow in her footsteps so that India's young women may see the beauty of the ideal of service in the Nursing profession.

In Miss Te Winkel, we have lost a very great friend and one of the builders of the Association. Our deepest sympathy goes out to her relatives and friends and as we feel that those members who knew her would like to write to her sister, we are printing her address.

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W. NOORDYK.

THE DYSENTERIES OF INDIA

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(Being a lecture delivered to the members of the Calcutta branch of the Trained Nurses' Association for India.)

Having already previously spoken to you all on the subject of 'Cholera and its treatment', my subject for this evening is 'The Dysenteries of India'. This will, with perhaps for that single exception, Sprue, (which by the way, is after all another name for chronic, undiagnosed, untreated dysentery) cover the vast field of tropical Medicine which Sir Leonard Rogers so aptly described as being 'the bowel diseases of the tropics'.

The treatment of the dysenteries of India—an ever-present problem, very often a most difficult one at that, which daily faces the medical practitioner and whose solution cannot as yet be said completely satisfactory—is more random, and less standardised than the treatment of most other tropical diseases.

That this is so, is shown by the almost universal and erroneous use of emetine in cases which are so often obviously bacillary in origin, or in patients who are permitted during its use to be up and about doing their

work—a line of treatment which may inflict irreparable damage upon the patient's cardiac mechanism, but which cannot do his symptoms any good. The term dysentery, like those terms jaundice, and uræmia, is not the name of a disease, but of a condition-complex in which there are frequent stools containing blood and mucus, whose passage is accompanied by pain, and tenesmus.

'Mucus' and 'tenesmus' in the definition are important factors, since the mere presence of either blood or mucus alone is insufficient evidence to establish the diagnosis of dysentery. There have been cases of intestinal tuberculosis and even of internal hæmorrhoids which have been wrongly treated with emetine, simply because there happened to be blood present in the stools, whilst such other conditions as fistula-in-ano, rectal syphilis, and carcinoma have also, on account of the mucus they cause to be present in the stools, met with the same fate. On the other hand, in many cases of true dysentery, whilst the presence of mucus is visible to the naked eye, that of the blood may only be possible to detect microscopically.

Of the causative agents of dysentery in India, we can eliminate a good few as they happen not to be indigenous to the country. The dysenteries associated with Malaria and Kala-Azar have been now proved to be either of amoebic or bacillary origin. *Giardia intestinalis* which are not uncommonly met with in the stools of patients suspected of dysentery may cause a diarrhoea but they never do cause a true dysentery. To all intents and purposes therefore, the dysenteries of India are due to infection with either the *Entamoeba histolytica*, or with dysentery bacilli, or in some cases, with both.

In discussing their relative frequencies, we must turn to those who can speak on the subject with authority. In Egypt, it was found by Wenyon, and Connor that bacillary dysentery was sixteen times as common as amoebic, whereas in Mesopotamia, bacillary causes during the Great War of 1914-1918 constituted almost 90 per cent of the dysentery cases. In the jails in Eastern Bengal almost similar findings were arrived at, while in Calcutta according to the findings of the School of tropical Medicine, bacillary dysentery is at least five times as common as is amoebic dysentery. In looking up the returns of this hospital,* I find that they are about equally common—(this of course refers to only European and Anglo-Indian cases).

Not one of us can treat a case of dysentery effectively until we have correctly diagnosed the type of dysentery from which the patient is suffering. Hence the true diagnosis can only be established by laboratory examination, although pending the results of this most important examination or as it often happens, in its enforced absence, a fairly accurate forecast can often be given upon purely clinical grounds. For this purpose, let us take in turn each element in the differential diagnosis of dysentery.

Bacillary dysentery—particularly if of the Shiga type—is as a rule an acute and febrile disease with fairly sudden onset, and in some cases not very unlike a cholera infection. It is what we call the dysentery of in-patients, since such patients are at once or very soon prostrated by toxæmia and rushed to hospital. Essentially, bacillary dysentery is in the acute phase a febrile disease which sends its patient to bed, unable to cope with the degree of toxæmia present.

On the other hand, amoebic dysentery is characteristically 'walking dysentery', or 'out-patient dysentery'. It is only when secondary infection supervenes or when grave lesions of the colon are present, that the amoebic dysentery patient is compelled to take to bed. As a rule he tries to remain at work, however incommoded.

*The Presidency General Hospital, Calcutta.

In general, we may say then, that acute bacillary dysentery is a disease which at once sends the patient to bed with fever, prostration, and symptoms of toxæmia; whereas typical amœbic dysentery is a disease of more gradual onset and the patient is often able to carry on his work although his colon may be ulcerated.

The Stools. Naked-eye examination and characters.

The bacillary dysentery stool may vary much, but essentially it is an inoffensive stool mainly composed of bright red blood and mucus, with few other elements. In amœbic dysentery, the naked-eye appearances may also vary; it may be simply diarrhœic; it may be semi-formed with or without adherent traces of blood and mucus; and its colour may vary from dark-brown to greyish-green. It is most often a small stool dark and tarry in colour. As a rule the blood and mucus—the former of which may not be visible to the naked eye—tend to mingle more intimately with the fæcal matter than in the bacillary stool, and as a rule there is more fæcal matter present.

Taken all round, however, an inoffensive stool consisting of only bright red blood and mucus is usually from a case of bacillary dysentery; whilst an offensive, dark coloured stool with adherent or admixed blood and mucus, and much fæcal matter is usually from a case of amœbic dysentery. In amœbic dysentery the stool is usually acid to blue litmus paper, whereas in bacillary dysentery it is usually alkaline to red litmus paper. This very easily applied and most useful test deserves further trial than it usually does secure. The point is of considerable importance, since in the acid amœbic stool, the hæmoglobin of the red blood corpuscles is changed into acid hæmatin, which leads to the dark-brown or tarry colour of the blood in the amœbic stool. In the alkaline bacillary dysenteric stool the blood is bright red and the hæmoglobin remains unchanged. As will be seen later this point is not without its importance from the point of view of prognosis and treatment.

The Microscopical Characters of the stools

These are very important and interesting, but they hardly come within the nurse's knowledge of the disease, or what is expected of her. A brief, and general idea, however, should prove valuable and add interest to the study of the dysenteries.

In the stool of **amœbic** dysentery, the total cellular exudate is scanty. Clumps of broken-down red blood corpuscles with a few leucocytes, presenting a mouse-eaten appearance, and the characteristic *Entamoeba Histolytica* pushing their way through their surroundings like 'slugs moving at express speed' constitute the microscopical picture.

In the **bacillary** stool the whole field under the microscope will be seen to be full of cells, and inactivity. Of these 90 per cent., are leucocytes, which have appeared to have died *en masse*. The red blood cells appear alone and not in clumps as seen in the amœbic stool. Other cells known as 'ghost cells' are a prominent feature; they are cells which have lost their structure but not their outline, showing a clear definite cell wall almost devoid of cell content—mere shadows of their former selves.

A diagnosis of amœbic dysentery can be given, as soon as the *entamoeba histolytica* is found present in the slides made from the stool examined, but in the case of the bacillary type, the microscopical picture can only tell us that a bacillary exudate is present, but we have to wait till the stool is plated, cultured, and put through further examination before the actual causative organism—be it Shiga, Flexner, or Strong—can be given. Mixed dysentery is relatively uncommon. What is common on the other hand is a transitional phase associated with a rapidly changing pH; bacillary

dysentery occurring in a convalescent amoebic case and the stool becoming more and more alkaline; or amoebic dysentery supervening in a convalescent bacillary case and the stool becoming more and more acid.

To ensure that a correct diagnosis is arrived at, only fresh stools should be sent for examination. The examination of a stool more than two hours after it has been passed is useless. Stools passed when the laboratory is not open should be plated and put in the incubator. ONLY by such means can we expect correct diagnosis, upon which rests correct and effective treatment.

Treatment

Amoebic. The diagnosis being established, the patient is put straight to bed for at least ten days, and is TOLD to use the bed-pan.

A bland diet is given, and the course of emetine started. Emetine should be given per body weight. Usually for an adult of average weight, one grain per night is given for ten consecutive nights. As the stools clear up, the diet is brought up to normal standards. On the completion of the Emetine, a course of Stovarsol, or Carbarsone is given outside hospital (one tablet or capsule as the case may be for another ten nights). Emetine is a direct depressant to the myocardium and is cumulative. For these reasons, the patient must be in bed while receiving full doses of the drug.

Bacillary

Strict bed. Diet depends upon the organism: Carbohydrates for Shiga, and protein diet for Flexner type.

In Shiga, and severe Flexner type, give anti-dysenteric serum in large dose early in the attack. Under this treatment 'the number of stools are immediately reduced; the symptoms of toxæmia disappear; the temperature comes down and the patient passes from a critical condition to one of relative comfort'.

Bacteriophage 2 cc., 4-hourly by mouth in bacillary cases is also given, but the old-fashioned sodii. sulphate by mouth is hard to beat. Bowel washes early in the disease are soothing and cut short the toxæmia. Flavine 1-6,000 given for three nights as a bowel wash appears to do a lot of good.

Chronic Dysentery: Causes. Most often because the patient refuses to go to bed, gets up before the ulcers have healed, or because the patient is inadequately or improperly treated. These are the victims of chronic dysentery, a condition only too common, and very difficult to treat.

The middle-aged European female patient suffering from this complaint finally drifts or tends to drift into the 'chronic abdomen', so amusingly described by Hutchinson, 'the subject of the Surgeon, the Homeopath, and the Quack; presenting at last a confirmed neurasthenic picture with a history of appendicectomy, cholecystectomy, gastro-enterostomy, plication of the stomach wall for distension, unrecognised endocrine deficiency, introspective, morbid, a nuisance to all around her, and a misfortune to her medical attendants'.

In concluding, I acknowledge with thanks my indebtedness to that wonderful little book written by those two great men, late of the School of Tropical Medicine, Acton and Knowles—entitled 'The Dysenteries of India'.

After the lecture, thanks to Dr. R. Hayter, Asst. Director of the School of Tropical Medicine, Calcutta, who made it possible, cinematographic pictures depicting the life history of the *Entamoeba histolytica*, and relating to dysentery and water-borne diseases were shown and greatly appreciated by all present.