THE ERYTHROCYTE SEDIMENTATION TEST IN LEPROSY.

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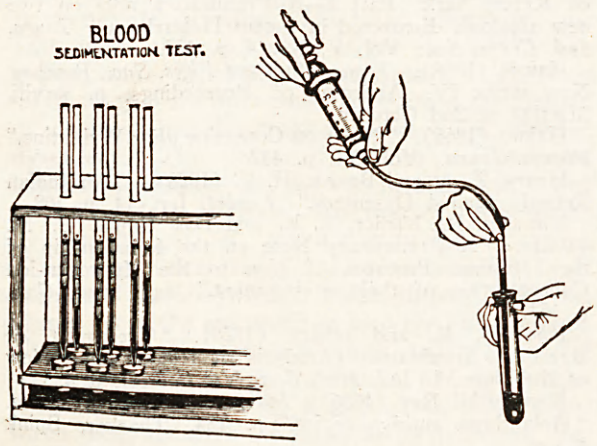
EXPERIENCE has shown that the rate of sedimentation of the red blood corpuscles varies in the different types, stages and phases of leprosy and that the use of the sedimentation test in leprosy provides a valuable guide in treatment, as well as giving information of diagnostic and prognostic value. The classification of leprosy used below is that adopted by the Leprosy Research Department of the Calcutta School of Tropical Medicine & Hygiene.*

The technique advised is an adaptation of that used by other workers and is chosen because it makes it possible to test a large number of bloods at once with fair accuracy and with the expenditure of a minimum of time. 0.3 c.c. of a 5 per cent. solution of sodium citrate in distilled water is drawn into an all-glass 2 c.c. syringe; 1.2 c.c. of blood is drawn from the patient's vein into the same syringe, and a small quantity of air having been taken into the syringe barrel, the blood and citrate solution are thoroughly mixed by reversing the syringe several times, and the mixture is evacuated into a clean test tube. If several patients are to be tested, their bloods are taken in a similar manner and placed in labelled test tubes in a rack. Sedimentation is carried out in 300 mm. pipettes graduated from above downwards from zero to 100, there being a space of 3 mm. between each mark. The content of the pipettes when filled up to zero is approximately 1 c.c., but a variation of 0.05 c.c. is allowed as such a variation makes no appreciable difference in the results. The pipettes are placed upright in a rack, with their points inserted in small holes bored in rubber corks (as in the illustration).



One of these pipettes is taken from the rack and its upper end is attached to a 10 c.c. syringe by means of a rubber tube. The point of the pipette is inserted in one of the test tubes and, suction being applied by pulling on the piston of the syringe, the blood-citrate mixture is drawn up into the pipette to the zero mark. The pipette is then replaced in the rack, the point again being inserted in the rubber cork which prevents the mixture escaping, and the rubber tube is then disconnected from the pipette. In this way the other pipettes are filled up to the zero mark from the other test tubes.

The top level of the erythrocytes is read off after 1½ hours and again after 2½ hours and the average of these two readings is taken as the sedimentation index (hereafter called "S. I."). Thus if the level of the top of the blood cells falls to 10 (30 mm.) after 1½ hours and to 20 (60 mm.) after 2½ hours, the S. I. will be the average of 10 and 20, i.e. 15. The maximum reading is about 80 (240 mm.). Generally speaking the S. I. in the different stages of leprosy increases in direct proportion to the number of bacilli in the body. In the early (A 1) stage, it is practically the same as in uninfected persons, i.e. between 10 and 20. In B 1 cases it may be slightly increased; in B 2 cases it is 30 to 40; and in B 3 cases 40 to 60.

The S. I. is also a criterion of the reaction phase in direct proportion to the degree and extent of the reaction. Thus the sedimentation index in a B 2 case may increase from 30 to 60 or 70 during a reaction, returning to the former level as the reaction passes off.

The S. I. is also a criterion of the reaction level. Thus a non-reacting patient, who shows for a considerable time more rapid sedimentation (high S. I.) than would be expected from the clinical stage of the disease, will react to a smaller dose of potassium iodide or any other reaction-producing drug than a patient with a comparatively low S. I. The more easily reaction is produced, the lower is the reaction level. This is important in the regulation of treatment, which can be pressed when there is a comparatively low S. I. (and therefore high reaction level), but must be proceeded with carefully when the S. I. is high, and the reaction level low; otherwise a severe reaction may be produced which will lower still further the resistance of the patient and cause an increase of the disease.

It must be remembered, however, that the sedimentation test is not in any sense specific in leprosy. A rise of the S. I. is caused by malaria, syphilis, septic processes, and in fact by everything which causes unusually rapid metabolism or breaking down of the tissues of the body and consequent absorption of waste materials. This must always be kept in mind in interpreting the S. I. These diseases and other causes also lower the

* *Leprosy, Diagnosis, Treatment and Prevention*, British Empire Leprosy Relief Association (Indian Council), Delhi.

patient's reaction level and lower his resistance to leprosy. In treating such cases special leprosy treatment must be delayed or given only in small doses, while predisposing and complicating diseases, etc., must be sought for and remedied. When this is accomplished the S. I. may be expected to fall, whereupon the special treatment may be pressed with safety.

The effect of a given dose of medicine (such as potassium iodide given orally, or injections of hydnocarpus preparations) upon the sedimentation rate, serves to indicate whether the next dose should be increased or diminished and after what interval it should be given. If the S. I. is low or only moderately high considering the type of case, and if there is no change produced in the rate of sedimentation by the given dose, there is a clear indication that the treatment may be pressed. If, on the other hand, there is a marked rise in the sedimentation index following the dose, the next dose should be delayed till the former level is reached and the dosage should not be increased but should possibly be lowered.

If the S. I. is high or shows further signs of rising, then special treatment (hydnocarpus, potassium iodide, etc.), must be stopped and drugs which have an anti-reaction effect should be used. One of the most valuable of these is potassium antimony tartrate given intravenously in 0.02 gramme doses every second day; alkalies given orally or intravenously are also useful for this purpose.

The gradual lowering of the S. I. which takes place under treatment is of considerable prognostic value as an indication that the disease is gradually being eliminated from the body.

An unusually low S. I. (3 to 8) which sometimes occurs even in B 2 cases, may generally be counted as a good sign. Such patients, as a rule, improve rapidly under treatment.

When a former B 2 or B 3 case has become bacteriologically negative, and if he can stand large doses of potassium iodide (up to 240 grains) without an increase in S. I., it is a good prognostic sign.

The sedimentation test is useful in testing the efficiency of any drug in the treatment of leprosy. If the remedy does not possess the power of raising the S. I., it is not likely to be effective in eliminating the leprous infection from the body. There are however certain very useful reaction-reducing drugs, the effect of which is to lower the S. I., such as those mentioned above.

A NOTE ON CHOLERA IN INFANTS.

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CHOLERA is seldom seen in infants, but during this past epidemic in Calcutta, I had a number of cases in the fourth week of May and the first week of June while attending the infants at the "Babies Home." The first two cases ended fatally within a few hours of the attack and I could not diagnose the condition on account of the very peculiar symptoms which each of these babies presented and which I shall relate later. These cases were also seen by other physicians whom I called to my help. When the third case came, my suspicion arose about cholera, though the symptoms were abnormal, but there were a large number of cases in the immediate neighbourhood. It was only when I took a sample of stool from the third case, cultured it and performed the agglutination test with high titre cholera serum that I came to a definite diagnosis. Four other cases occurred after this in quick succession, and in each of these, excepting one, the stools were cultured and agglutination tests were performed and found to be positive. Fever was present in all these cases excepting one. The ages of these babies ranged from 3 months to 1½ years. As regards the source of infection, I tried to find it out, but there were so many difficulties in the way that I had to give up the idea; but when the nursing staff was entirely changed and the new staff and all the babies were inoculated with cholera vaccine and the utmost hygienic precautions were taken, the epidemic stopped without any further fresh cases. That the first two cases passed undiagnosed but to me appeared as cases of cholera will be evident from the following reports:—

Case No. 1.—Baby Sushila, aged 4 months. Passed the first stool in the morning on 29th May, 1929, white, curdy in character; reaction acid when I saw the stool; no urination after the first stool. The child had another similar characteristic stool at 4 p.m. and after this it began to sink very rapidly; tympanites set in; the pulse became very weak; at about 9-30 a.m. the child began to show a peculiar continuous twitching of the face and legs, the knee jerks were exaggerated. The baby had received no injury. On 30th May, 1929, the baby had no stool, but the condition remained the same. Bromides and chloral completely failed to check the convulsions. The pulse was almost imperceptible. In the evening the temperature rose to 108°F., but it came down to 97°F. in two hours after cold sponging. The baby died the same evening. A provisional diagnosis of gastric tetany was given in the death certificate.

Case No. 2.—A Belgachia baby one year old; it was a healthy baby the previous night, i.e., on 30th May, 1929, and so no special notice was taken of it. On the morning of 31st May, 1929, the baby presented a quite changed appearance, eyes sunken, pulse very bad, toxæmic, with excessive tympanites; according to the nurse the baby had had two stools in the previous night and these were curdy white in appearance. Twitching of the face and legs set in the early hours of the morning; urination was absent after the first night stool until death. Rectal saline, atropine, adrenalin and a rectal wash were given, but the baby became uræmic so rapidly and collapsed that I had no time to give subcutaneous saline. The baby died the same morning.

These two cases aroused my suspicion that either they might be cases of food (milk) poisoning or cholera. I stopped the existing supply of milk and the utmost precaution was taken in feeding the babies. Still a third case developed.