

are analogous to those of cholera, intoxication resulting from the liberation of endotoxins by the solvent action of the tissue fluids or cells on the bacilli. Dysenteric symptoms are not produced in animals by feeding the organisms.

The stools of the patient are the only known source of the organism and it continues to be excreted during convalescence.

Dissemination and Infection.

Latent or chronic cases are a source of danger to a community. Although the conditions outside the body are not favorable for the growth of the organism, it may remain living and virulent for several months. The methods of infection appear identical with those seen in typhoid. Water infection seems certain, and indirect transmission is readily accomplished by contact with the discharges. The best examples of contact infection are found in institutions, as insane asylums, or foundlings' homes, where the disease may become endemic.

The first essential for prophylaxis is correct diagnosis, for which the agglutination tests and bacteriologic examination of the stools are essential. Disinfection and other precautions should be practiced as rigidly as in typhoid. The patient should not be discharged until the stools are free from dysentery bacilli.

Prophylaxis and Susceptibility.

Poorly nourished individuals are particularly susceptible to infection, and among them the mortality is high. The disease is most common among young children, old people, and those who are confined in institutions. The conditions in Japan, however, where from June to December of one year nearly 90,000 were attacked, and in Germany, where severe epidemics occur in industrial communities, indicate that susceptibility is quite general. Digestive disturbances and enteritis from other causes are said to be predisposing factors. The normal serums of man and animals have very little bactericidal power for dysentery bacilli.

The subject of acquired immunity to dysentery is hardly on a satisfactory basis. The serum of convalescents shows a distinct bactericidal power for the organism, and there is good reason to believe that the acquired immunity persists for some time after the disappearance of the bactericidal amboceptors, an event which takes place rather early. As in typhoid, animals which through immunization have once been stimulated to produce antibodies, form them much more readily on the occasion of a subsequent inoculation. This acquired facility of producing antibodies may be a factor in acquired immunity. By immunizing horses, serums of rather high protective power have been obtained. Kruse prepared a serum of which 1/80000 gram would save a guinea-pig from a dose of the bacilli which killed a control in 20 hours. It is assumed that the protective power of this serum is due to its bactericidal action. The antitoxic serum which Rosenthal prepared, by immunizing with 30 days' old bouillon cultures, protected not only against the toxin, but also against the bacilli; and conversely an antibacterial serum protected against the toxin (cited by Lentz). Such results leave us very much in doubt as to the existence of a true antitoxic serum.

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The value and feasibility of protective inoculations are not well established. Shiga at one time practiced mixed active and passive immunization (bacilli plus immune serum) on 10,000 individuals. This did not decrease the number of infections, although a lower mortality resulted. Shiga claims that the therapeutic use of his serum reduces the mortality to one-third of the untreated. The serum of Kruse, and also that of Rosenthal, are said to be curative; the discharges rapidly decrease in number and the course of the disease is shortened. In the hands of the Rockefeller Institute antidyenteric serum proved of no distinct value.

Vaccination and Serum Therapy.

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The agglutination reaction with the serum of patients shows great variability. It is sometimes absent in spite of the presence of bacilli in the stools, and often disappears rapidly during convalescence (in two weeks occasionally). It is rarely as high as in typhoid, and this is true also of artificially prepared serums. In infantile diarrrheas it com-

Agglutination.

monly appears at about the end of the first week of illness (Duval and Bassett). Evidently mild cases in which the course of the disease is from four to eight days may not be recognized by means of the agglutination reaction before the period of convalescence. In chronic cases it may persist from three to four months. In a series of cases studied by Duval and Bassett, the reaction with the serums of the patients was obtained in dilutions varying from 1/16 to 1/25, twenty times; 1/40 to 1/60, twelve times; 1/100, twice; 1/200, three times 1/500, once; 1/1000 twice. No reaction was obtained with the typhoid bacillus. Kruse considers the reaction diagnostic when it occurs in a dilution of 1/50; Pfuhl, 1/30. Strong co-agglutinins for other organisms, i. e., above 1/50, have not been observed (Lentz). The tests should always be performed with both the "Shiga" and "Flexner" types, as the two have not identical agglutinable properties, and either organism may be the cause in a given instance. The absence of the reaction does not exclude dysenteric infection positively. Bacteriologic examination of the stools is important, often necessary, for early diagnosis.

Clinical Report

A CASE OF AINHUM.

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Seeing the article on ainhum by Dr. Brayton in *THE JOURNAL*, July 8, 1905, reminded me of a similar case which came under my care about six years ago and which was reported to the Newport Medical Society during the past year.

Previous and Family History.—M. R., negro, widow, aged 53, was born in Queen Anne County, Maryland. Her mother died in childbirth, aged 42; her father was part Indian, and died of rheumatism, aged 92; he lost both toes when about 23 years old.

The patient consulted me in December, 1899, at which time a band had formed around the base of the little toe of the left foot, and from a fissure formed at this point there came a

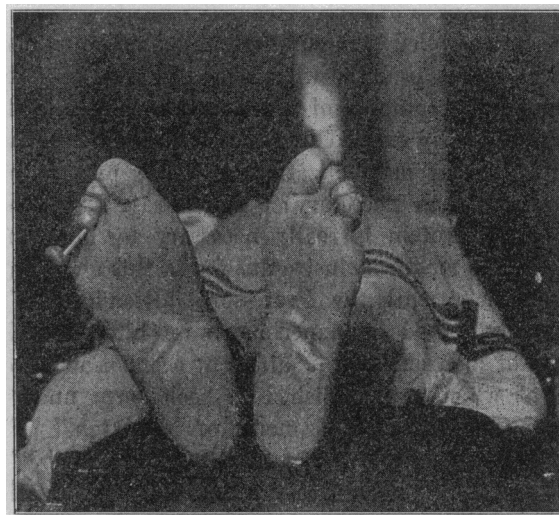


Fig. 1.—Little toe on left foot severed. Little toe on right foot constricted at base. Toe not much swollen.

discharge. The toe was swollen and painful. With cleanliness it improved and remained quiescent until 1903, when it again began to swell and to cause great pain. The patient stated that one evening while in great pain the toe parted from the foot as if shot from a pistol and larded about 8 feet across the room carrying with it a piece of cloth which was wrapped around it. The disease appeared in the right foot about two years previous to its appearance in the left, but the process in the former was never very active. She thinks the process in the left foot was completed in four years.