

sparse chains of streptococci. After sowing again, the cultures were obtained in a state of purity. The microbe in question caused the fermentation of the milk which it coagulated and separated into a serous lower layer and a frothy upper layer bearing large, firm bubbles of an alveolar appearance; the culture gave out a butyro-cheesy odor which was not at all foetid. The medium was strongly acid. On sheets the presence of a large bacillus was recognized; it was of a variable length, sometimes short, sometimes a little longer, with rounded extremities. This microbe colored well with the different reagents, and tolerated Gram's stain. Inoculated in doses of from two to three cubic centimetres in the muscular mass of the thigh of a guinea-pig, the cultures caused death in from twenty-four to thirty hours, and gave rise to the formation of a large sero-sanguineous collection in the fold of the groin. The microbe was met with again in a state of purity in this serous liquid. The morphological characteristics and, better still, the reaction of the cultures in the anaerobic sterilized milk, and also the results of the intramuscular inoculation in the guinea-pig, form a mass of details which absolutely corresponded to that which Thiroloix had described several times in regard to bacteriological investigations made with the blood of living rheumatic subjects. It was shown from that, by the authors, that the microbe isolated by them thirty-six hours after death should not be considered as a common microbe of putrefaction. The observation presented, aside from the ascertaining of a possible specific, pathogenic microbe, was open to considerations of another order. The child had presented, during its life, evident symptoms of chorea. The cultures of a segment of the spinal cord having given pure cultures of the microbe in question, it was allowable, the authors thought, to suppose

that the presence of even this microbe in the nervous centres was probably the exciting agent of the abnormal movements.—*N. Y. Med. Jour.*

DIABETIC ALBUMINURIA AND ITS TREATMENT.—Goudart (*Journ. de Méd.*, August 25th, 1897) has recently devoted much attention to this subject: first, the frequency of albuminuria in diabetes is variable and may occur in two forms, functional and that due to grave nephritic disease. In the first form it may be extremely slight, or else may constitute a very marked feature in the case. When slight, proper dieting and small doses of antipyrin combined with a little bicarbonate of soda in the form of a powder may be given every one and a half hour before each meal. This treatment should not be continued more than three or four days, beyond which time the antipyrin will become injurious. It is well to prescribe some quinine wine and Vichy water at meals. After this treatment the sugar decreases considerably, in other cases it remains unaffected. In the first instance anti-diabetic treatment may be set aside and attention devoted to the albuminuria; in the second instance it is advisable to order small doses arseniate of soda combined with codeia and carbonate of lithia. Most usually the glycosuria diminishes under this treatment, and the albuminuria is then treated in the same manner as above. This line of treatment is usually followed by extremely satisfactory results. After a fortnight or so it is recommended to give phosphates with nux vomica, or later hypophosphites of lime potash or soda with quinine, etc. Should the quantity of albumen eliminated in twenty-four hours reach 2 to 3 g the case is practically one of Bright's disease, and the patient is put on milk diet. The author now recommends lactate of strontium in small doses.—*Brit. Med. Jour.*