Thus it would appear that the beta oxybutyric acid and the aceto-acetic acid produced in the course of the disease are intermediate products leading ultimately to acetone, providing the conditions are favourable. This conclusion is supported by the observations on the fermentation, where it is noted that the end of the reaction is determined by the fall in the acidity of the mash.

The fact that the alcohols produced in the fermentation are not detected in the excreta of diabetic patients can be explained to some extent by their absorption and possible oxidation in the liver to products which would not be easily detected. Stepp⁷ claims that he has detected aldehydes (or substances which react in every way like aldehydes) in the blood of diabetic patients. Aldehydes would be the first products of the oxidation of normal alcohols, and complete oxidation would lead to CO₂ and water. In endeavouring to obtain good yields of acetone the inhibitory effects of certain bacteria came to be recognized. Thus Thaysen states that the organism which causes most damage to a fermentation is the *B. volutans*. Others have described the *B. acidi lactici*, and we have found the presence of a diplococcus and of a large thick bacillus, of unknown identity as yet, to be responsible for much inhibition. It appears probable that these facts, combined with a diet so composed as to allow digestion and absorption by the patient of foods which the bacteria cannot assimilate, will prove an important part of treatment, combined with the administration of certain antiseptics. In this respect the authors⁸ would call attention to their work on the subject of antiseptic action amongst the coal-tar dyestuffs, which gives useful data as to the relative value of dyes as antiseptics in regard to different organisms.

SUMMARY.

1. From the stools of diabetics a new organism (*B. amyloclasticus intestinalis*) has been isolated which splits up starchy foods, forming oxybutyric acid, diacetic acid, butyl alcohol, and acetone. Sugar is also formed during this fermentation.

2. Acetone has been found in quantities capable of estimation in the stools of diabetics.

3. In diabetes carbohydrate fermentation occurs in the alimentary canal, forming abnormal products which probably so affect the glycogenic function of the liver as to lead to improper storage therein of the glucose from the alimentary canal during digestion.

4. Definite lines of treatment include elimination or suppression of this organism.

We wish to express our indebtedness for access to diabetic cases to Dr. J. Gray Clegg, Dr. Wm. Stirling, and Dr. Langley; and to Mr. A. Gill, B.Sc., for his technical assistance with the various processes herein detailed.

REFERENCES.

- ¹ Schürdinger: *Centralbl. f. Bakteriol.*, 1905, 14, Abt. ii, 772. ² Brede-mann: *Centralbl. f. Bakteriol.*, 1909, 23, Abt. ii. ³ Fernbach and Strange (Eng. Pat. 1912, No. 21,073). ⁴ Northup, Ashe, and Senior: *Journ. Biol. Chem.*, 1919, 39, 1-28; *Journ. Ind. Eng. Chem.*, 1919, 11, 723-27. ⁵ Amos Gill: *Journ. of Society of Chemical Industry*, July 31st, 1919, vol. xxxviii, pp. 273-80. ⁶ Thaysen: *Journ. of Institute of Brewing*, vol. xvii, No. 11, pp. 529-542. ⁷ Stepp: *Biochem. Zeit.*, 107, 60-80 (1920). ⁸ Fairbrother and Renshaw: *Journ. of Path. and Bact.*, vol. xxv, 1922, pp. 145-151.

FRAGILITAS OSSIIUM ASSOCIATED WITH BLUE SCLEROTICS IN FOUR GENERATIONS.

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INSTANCES of fragilitas ossium associated with blue sclerotics have from time to time appeared in medical literature, and as recently as December 24th, 1921, Blegvad and Haxthausen of Copenhagen reported in the BRITISH MEDICAL JOURNAL cases showing, not only blue sclerotics and brittle bones, but also associated with macular atrophy of the skin and zonular cataract. Such cases are, however, sufficiently rare to warrant putting on record the following history:

The patient, F. F., aged 7½ years, presented himself at my out-patient department about a year ago. The history was that he suffered from repeated fractures—the most recent having been one of the lower end of the right tibia. Some deformity was present in the right leg and the right thigh, and it was ascertained that both the tibia and the femur had been previously fractured—the latter having occurred at birth. In addition a history was obtained of past fractures of the left humerus and the tibia and fibula of the left leg. These had healed with apparent perfect union and minimal

amount of callus. The most striking feature which one observed when the boy entered the consulting room was the marked blueness of both sclerotics. The child was somewhat pale and debilitated, the forehead more prominent than normal, but no evidence of rickets was apparent. In other respects the mother stated that the boy had always been healthy. The mother, to whom the boy bore a strong resemblance, showed very deep blue sclerotics. She had, however, no fractures. Her hearing was defective, and she informed me that she had suffered from deafness for many years. The patient's two sisters, whom I saw later, also presented blue sclerotics, with, however, no history of fractures. On inquiring further into the history of the family I found that the aunt of the patient (the patient's mother's sister) had sustained altogether seven fractures during her life and had, as in the other members of the family, deep blue sclerotics. The grandmother and great-grandmother had both been the subjects of fragile bones and blue sclerotics. No further history of these phenomena was forthcoming in previous generations to the ones already mentioned; it is quite possible, however, that a careful study of this family's genealogy would have revealed the same condition in former generations.

This remarkable association of brittle bones and blue sclerotics was first described by Eddowes in 1900, who suggested that "the transparency of the sclerotics indicated a want of quantity or quality of fibrous tissue forming the framework of the various organs of the body, and probably explained the want of spring or toughness in the bones of these individuals." The importance of heredity in these cases was most clearly indicated by Stephenson and Harman in 1910, who published accounts of the same family of blue sclerotics—altogether 55 individuals in five generations, 31 of whom showed typical sclerotics. No mention was then made of fragile bones; but in 1915 Stephenson showed at the London Ophthalmological Society a mother and two daughters with fractures, belonging to this family. Rolleston, in 1911, reported 4 cases of blue sclerotics in three generations, with fragile bones occurring only in the third generation. Peters, in 1908, published the account of blue sclerotics in a family during three generations, and five years later further reported the occurrence of fractures in these cases. The interesting case reported by Ostheimer in the *Journal of the American Medical Association* of December, 1914, had blue sclerotics and nine fractures between the ages of 1½ and 4 years. In this case there was no history of the condition occurring in other members of the family. The literature of the subject is further enhanced by the publication of similar cases by Burrows, in 1911 (a family of 29 individuals—13 with blue sclerotics, and 9 of the 13 with brittle bones); Poynton, in 1913 (1 case); Hoffmann, in 1915 (3 cases); E. Bronson, in 1917 (description of two families); and Olaf Blegvad and Holger Haxthausen, *BRITISH MEDICAL JOURNAL*, December 24th, 1921.

There seems to be little doubt that the blueness of the sclerotics is due to the increased transmission of the colour of the choroidal pigment and not to any inherent colouring of the sclerotics themselves. Some think, however, that there is an actual thinning of the sclerotics. Fridenberg has suggested that the blue sclerotics are due to a transparency depending upon the absence of lime salts in the connective tissue of the sclera. The long-standing deafness in the case of the mother described is worthy of notice, and might possibly be explained, as Bronson suggested, by a deposition of calcium salts in the middle ear, relating to the abnormal osteogenetic process.

THE RECOGNITION AND TREATMENT OF MINOR DEGREES OF FIBROSITIS.

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KNOWING the danger of missing an internal lesion which may be associated with superficial pain we naturally in such cases look for some objective sign. Failure to realize that the cause of many pains can be definitely detected in the soft tissues may explain why their examination is neglected. A patient who complains of pain in the region of the right scapula may be taken as an example. There may be some tenderness on pressure over a particular area, but otherwise nothing definite is noted; there is no swelling, no grating in the joints, and the lungs and heart are normal. The teeth may be surveyed and search made for some source of infection or irritation, without finding anything to confirm the patient's statement. If, however, we now devote a little