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The Mechanism of the Glycosuria of Acute Uranium Nephritis.

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A glycosuria occurring early in the course of severe acute uranium nephritis is a matter of common observation, but its mechanism is by no means clearly understood. The appearance of the urinary sugar has almost universally been attributed to the high blood sugar levels that are frequently observed in this type of nephritis, although Frank[1] believed that glycosuria might occur while the blood sugar was normal.

In our series of experiments 11 fasting female dogs were each given a single subcutaneous injection of 15 mg. uranium nitrate per kilogram of body weight. Four of the animals were killed with chloroform when the glycosuria was estimated to be at its greatest height, while the remaining 7 were permitted to live until they died with uremic manifestations. The blood sugar, blood non-protein nitrogen, total blood solids, urinary sugar and nitrogen, water ingestion, and urinary secretion were accurately determined at frequent intervals both before the administration of the uranium and throughout the subsequent life of the animals.

It was found that the glycosuria accompanied by a polyuria made its appearance 6 to 10 hours after the injection of uranium, reached its greatest height in about 28 hours, and lasted 9 to 44 hours. Throughout the course of the glycosuria the blood sugar remained normal or fell to a level slightly below that of the pre-uranium period. None of the blood sugar values observed during the glycosuria were suggestive of a hyperglycemia.

However, during the period of anuria, partial or complete, which closely followed the cessation of the glycosuria and persisted until the death of the animals, blood sugar values distinctly above the normal were found, but in no instance was the hyperglycemia accompanied by any glycosuria.

It seems probable that the glycosuria, which unquestionably is of renal origin, is due to the involvement of the kidney tubules, whereby the glucose normally present in the glomerular urine is prevented from being completely reabsorbed. Histological sections of the kidneys from the four animals killed during the height of glycosuria showed extensive tubular damage but no demonstrable pathological changes in the glomeruli while the kidneys of those

animals which died in uremia showed extreme degeneration of both the glomeruli and the tubules. Calcification of the tubules which has been considered pathognomonic of bichloride poisoning occurred in the kidneys of one animal which had been totally anuric for six days prior to death.

[1] Frank, E., *Arch. exp. Path. u. Pharm.*, 1913, lxxii, 387.

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Availability of Carbon Compounds for the Streptococcus.

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The synthetic media described in our earlier papers[1, 2] have been used for testing the value of various substances as nutrients. The studies in this paper are concerned with the effect on the growth and viability of streptococci, of the addition of various carbon compounds to the media. These compounds include dextrose, galactose, mannite, lactose, maltose, sucrose, raffinose, glycerol, lactic, tartaric, malic and citric acids.

The experiments show: (1) In most cases dextrose shortens the period of viability markedly. Apparent increases in viability due to the addition of dextrose have been obtained but they were too small to be taken into account. (2) A comparison of dextrose with other sugars in reference to their availability for growth gives series such as sucrose > lactose, maltose > raffinose > mannite, dextrose.

There are several references in the literature to the growth value of dextrose. Samkow[3] and Waksman and Joffe[4] found that although dextrose did no harm, it did not aid materially. Frankel and Pyle[5] showed that dextrose retarded growth slightly. Koser and Rettger[6] reported glycerol better for growth than dextrose because less acid is formed and the organism is therefore "freer to develop". None of these investigations refer specifically to the streptococci. The only definite allusion to these organisms with respect to their utilization of dextrose was found in the communication made by Gordon.[7] He states that dextrose is not utilized by streptococci. Our results corroborate this.