tomy showed a marked decrease, averaging about 25 per cent. less than the normal value. The feeding of 6½ grams of glucose per kilo in 40 per cent. solution before the operation to fasting animals showed the normal hyperglycemia to last three hours. After thyroidectomy, glucose fed under the identical experimental conditions, failed to raise the blood sugar to the level attained before the operation. The increase persisted however for an average of five hours. The urine remained free from glucose both before and after the operation with a single exception.

We have observed hypoglycemia in cretinism. It is present in myxedema, in Addison's disease, and after removal of the suprarenal bodies. Evidence is accumulating showing that a persistent low sugar content is a sign of insufficient internal secretion.

Quite aside from the doubtful question of whether increased alimentary tolerance to glucose can be demonstrated by an examination of the urine, these experiments show that there is less tendency to hyperglycemia on carbohydrate ingestion after thyroidectomy. It seems therefore, that the removal of thyroid tissue might lower the blood sugar also in diabetes mellitus, possibly with beneficial results.

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Further observations on the influence of diet on the toxicity of sodium tartrate.

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In a previous publication from this laboratory[1] it was stated that the toxicity of sodium tartrate might be modified by diet. Rabbits that were fed carrots resisted larger doses of the tartrate than those which received oats and cabbage. The investigation of the effect of diet on the toxicity of this substance was resumed recently. The observations were made on rabbits and on cats with a large number of diets which were given some time previous to the subcutaneous injection of tartrate. Striking differences in toxicity were observed. When young carrots were fed four days

[1] Salant and Smith, *Am. J. Physiol.*, 1914, *35*, 239.

before the tartrate was given, the dose survived was 3.0 grams per kilo. One rabbit only developed albuminuria when this dose was given. In one series of four rabbits three survived such a dose. In another series with 3.5 grams per kilo, one survived and three died, two in 12 and 36 hours and one in 6 days. The resistance was likewise very marked when carrot leaves were fed 4 to 11 days before injecting tartrate, but was less than in the case of young carrots, the minimum fatal dose being about 2.5 grams per kilo. The duration of life in this case was 2 to 5 days, the rabbit dying, however, without developing albuminuria. The toxicity when mature or winter carrots were fed was on the contrary considerably greater, the fatal dose being 1.25 to 1.5 grams per kilo.

Exactly the same results were obtained with sweet potatoes as with carrot leaves, as 2.5 grams per kilo likewise proved to be fatal, while a dose of 2.0 per kilo failed to produce any nervous symptoms or renal irritation. Sodium tartrate proved to be most toxic when the diet consisted of oats and cane sugar, glucose, or levulose. When 0.5 gram per kilo was injected subcutaneously on this diet, seven out of 8 rabbits died after 2 to 13 days. Symptoms of renal irritation and nervous disturbances were noticed in these experiments. The toxicity on hay, cabbage or oats was about the same in each case and was approximately twice that on oats and sugar, or about one fourth that on young carrots. The resistance to sodium tartrate on a diet of sugar beets was about half that on young carrots. Experiments with tartrate on cats that received different diets failed to show any marked difference, but when starved for eight days the toxicity was increased about 40 per cent.

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A note on the parenteral administration of starch.

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Very little work has been done on the question of the production of a protective amylase after the parenteral administration of starch. Most observers agree that the blood normally contains