period the blood sugar is either about normal or definitely hypoglycemic as an indication that the vago-insulin system has come into prominence. This shift in the balance is, however, not due to an alteration in the activity of the vago-insulin system, but is a result of a diminished excitability of the sympathetico-adrenal system as age increases since the hypoglycemic effect of anoxia on the vago-insulin system as shown by experiments on adreno-demedullated rats is not influenced by the age of the animals.

15103 P

Effect of Glucose Injections on Fasting Ketonemia After Low Protein Diets.

HERBERT C. TIDWELL AND C. R. TREADWELL. (Introduced by Donald Slaughter.)
From the Department of Biochemistry, Southwestern Medical College, Dallas.

The rapid onset of a marked fasting ketosis following low protein diets has been attributed by MacKay *et al.*[1,2] to a lack of "stored" protein available for the formation of antiketogenic material during fasting. Their conclusions were based on the higher nitrogen excretion observed during fasting after the high protein intakes. The difference in nitrogen excretion between groups on 5 and 25% protein diets averaged approximately 6 mg per square decimeter of body surface per day during the first 48 hours. In a comparable experiment we observed a difference in excretion of 8 mg of nitrogen. It seemed unlikely that 30 mg of glucose per square decimeter of body surface, the amount available from the metabolism of protein containing 8 mg of nitrogen, would have an appreciable effect on the metabolism of the rat. However, Deuel[3] has reported a marked lowering of ketonuria after giving 100 mg of glucose per square decimeter of body surface daily.

The present study was designed to determine the effect of glucose available from the additional protein metabolized after higher protein intakes upon fasting ketonemia in the rat. Two groups of 12 male rats, weighing approximately 224 g, were fed for 3 weeks a 5% protein, high fat diet similar to that used previously[4] and then fasted for 48 hours. Twice daily during the fasting period one group received intraperitoneally a 5% glucose solution containing 15 mg of glucose per square decimeter of body surface. The other group received similar volumes of normal saline.

The blood ketone levels at the end of the 48-hour fast were 25.6 $\pm$ 1.3* mg % of acetone in the glucose injected animals and 28.5 $\pm$ 1.0 mg % for the controls. There was a slight lowering of the ketonemia by even this small amount of glucose but not to the level (18.8 $\pm$ 1.7 mg %) obtained on animals receiving a 25% protein diet. These results do not support the suggestion that the greater protein catabolism in the animals with the larger protein stores can account for sufficient antiketogenic material to prevent the marked fasting ketosis following low protein diets.

The available carbohydrate from the excess protein metabolized by the animals on the high protein diets does not appear to be the major factor responsible for the markedly decreased fasting ketosis in such animals and the effect must at least in part be otherwise explained. Other data we are accumulating suggests that an increased rate of metabolism in the animals on the low protein diets plays a more important role than the lack of protein stores.

[1] MacKay, E. M., Carne, H. O., Wick, A. N., and Visscher, F. E., *J. Biol. Chem.*, 1941, **141**, 889.

[2] MacKay, E. M., Visscher, F. E., and Wick, A. N., PROC. SOC. EXP. BIOL. AND MED., 1942, **49**, 514.

[3] Deuel, H. J., Jr., Hallman, L. F., and Murray, S., *J. Biol. Chem.*, 1938, **124**, 385.

[4] Treadwell, C. R., King, W. C., Bebb, K. C., and Tidwell, H. C., *J. Biol. Chem.*, 1942, **143**, 203.

* Standard error of the mean included.