From the data thus obtained he calculates that a man weighing seventy kilo, resting in bed, can receive and utilize sixty-three grams of glucose an hour without glycosuria. This represents an energy intake of 252 calories an hour or 6,048 calories a day. When the injection rate is increased and hyperglycemia maintained there are evidences that when present in excess in the blood, sugar may exert toxic effects. At least in those with restricted tolerance a continued forcing of the sugar handling machinery is found to result in its breakdown, with further lowering of tolerance. Whether a persistent forcing of the glycogenic powers may lead to a breakdown in a healthy person is not known. It is stated that the increase in the use of sweets which has followed the passage of the Volstead act is accomplished by an increased number of cases of diabetes. If this is true, it may be due to the tendency of those with lowered tolerance to become actual diabetics under such conditions.

Experimental glycosuria is of interest because of the light it throws on diabetes. We may produce glycosuria in the following way:

1. *Alimentary glycosuria.* — This form of glycosuria is produced by exceeding the assimilation limit as described above. Its interest to clinicians lies only in the testing of tolerance, and in the possibility that its frequent appearance may lead to fatigue and the establishment of tolerance at still lower levels.

2. *Phloridzin glycosuria.*—This follows the administration of phloridzin. At least primarily, its action is limited to the kidney which is stimulated to excrete the blood sugar. This results in a lowering of the blood sugar, a hypoglycemia. The liver glycogen supply is then drawn on, and by repeated doses of phloridzin the animal can be made to excrete both the carbohydrate of the food and that which is stored in the form of glycogen. The sugar from protein sources is also involved and a completely diabetic dextrose nitrogen ratio obtains. Acidosis secondarily results and further complicates the picture. Although this type of glycosuria has been of much experimental value it is doubtful if it has any relation to diabetes except to the rather uncertain type of renal diabetes.

3. *Glycogenolytic glycosuria.*—Glycosuria may result from such procedures as cause a sudden mobilization of liver glycogen. Claude Bernard's diabetic puncture of the floor of the fourth ventricle, the injection of epinephrine, asphyxia, anesthesia, fright, and acidosis all produce glycosuria by causing the liver to suddenly discharge its glycogen as dextrose. Many consider that there is a definite center in the medulla which controls the process of glycogenolysis. It has been pointed out that as normally operative it constitutes or governs an adaptive mechanism through which in time of stress or sudden demand for energy, the liver mobilizes its reserves for the use of the tissues. The above procedures result in glycosuria if the liver contains glycogen but are ineffective in its absence. The glycosuria which follows such procedures is transient. It apparently finds its parallel in such cases of diabetes as are associated with some involvement of the nervous system.

4. *Pancreatic glycosuria.*—The removal of the whole or the major part of the pancreas causes a condition which most closely parallels diabetes mellitus. We do not seem to be much nearer a solution of the question of how the pancreas functions in this respect than we were when Minkowski discovered the relation. It is general-