

DISCUSSION

MAXWELL (Los Angeles): Dr. Snyder, I do not really understand how you can say that these changes in carbohydrate tolerance are not due to changes in total body potassium or serum levels of potassium. In the first place, the work you cited and other work in the literature present convincing evidence, in animals and in uraemic man, that potassium can be a significant factor; total body potassium can be directly correlated with carbohydrate intolerance and the carbohydrate intolerance can be corrected by the administration of potassium.

Secondly, all studies on this type of diet, the so-called Giordano-Giovannetti diet, including our own study on 34 patients, which we published a few months ago, have uniformly shown that these patients become hyperpotassaemic and acidotic, and that the acidosis is a further factor in moving potassium from the intracellular to the extracellular compartment. In all of your slides you showed nothing on potassium; you simply mentioned that it did not look from your data as though this was a factor. I think when there is such strong evidence for it, it is your obligation to present your data and to rule this out before talking about other possible factors.

SNYDER (Brooklyn): You are quite right, of course. We were concerned about the problem of the lack of correlation between the rubidium levels and the K^{42} levels, and we had an insufficient number of patients to show it convincingly. We did not want to bring preliminary data to this forum.

You can, of course, see significant acidosis with the selected protein diet, particularly if you supplement it with methionine. However, I would like to point out that we made no effort whatever to replete potassium stores in these patients. Serum potassium may rise or fall but this bears very little correlation with total body exchangeable potassium, and it is not serum potassium, presumably, which is influencing carbohydrate metabolism, but rather total body potassium.