ABSTRACT

Intravenous glucose tolerance tests with early repeated sampling were done on twenty children nutritionally recovered from protein energy malnutrition (PEM). Three (15%) of the children had fasting plasma glucose levels which were below normal. All showed maximal plasma glucose levels one minute after intravenous load, and five (25%) showed delayed removal of glucose. Rate of removal of plasma glucose, K_G, was positively correlated with age. All fasting plasma insulin levels were normal. Maximum insulin response peaks were normal and occurred between 2 and 5 minutes in seventeen (85%) of the babies. Three (15%) children had maximal insulin peaks at 1, 7, and 10 minutes respectively. Suppressibility of growth hormone by glucose was shown by 6 out of 15 (38%) of the children for whom GH levels were determined.

Protein deprived and protein-energy deprived rat models were developed for in vivo and in vitro studies of insulin release. In vivo, these models showed lowered fasting insulin levels. PED models showed reduced insulin release in response to intravenous glucose and impaired glucose tolerance.

Insulin content of the pancreas was not reduced in the malnourished rat models. In vitro, there was no difference in insulin responsiveness to increasing concentrations of glucose in malnourished and control rats. Caffeine (2mM) was an effective potentiator of insulin release in the control rat pancreas. However caffeine inhibited insulin release from the

malnourished rat pancreas. Theophylline did not potentiate insulin relea in either control or malnourished rats. PEM led to significant reduction in leucine-induced insulin release. This defect was corrected after refeeding malnourished rats for 21 days. Arginine alone stimulated insulin release *in vitro* from control and malnourished pancreas. Mannoheptulose inhibited insulin release *in vitro* to the same extent in both control and protein-energy deprived rats, suggesting that the ability to phosphorylate glucose had not been impaired by PEM. Epinephrine inhibited the *in vitro* response to both basal and stimulatory levels of glucose to the same degree in control and malnourished rats. This suggested that responsiveness of alpha-receptors had not been altered by PEM.