Editorial

POSTPRANDIAL HYPOGLYCAEMIA

Postprandial hypoglycaemia was first described in 1924 (Harris 1924). Neuroglycopenic symptoms were observed several hours after eating, either in the late morning or late afternoon. Ingestion of food brings about a rise in blood glucose and amino acid level, which in turn stimulates insulin production. This insulin deposits the glucose in the liver (Ensinck and Williams, 1974). As glucose absorption from the gut stops, the plasma glucose falls, insulin secretion diminishes and glucagon, growth hormone and cortisol are released (Gerich et al., 1974). These hormones cause the liver to give out glucose. This change over from the rising to the falling plasma glucose level is the period when hypoglycaemic symptoms can occur.

Patients having undergone gastric surgery as gastrectomy or gostrojejunostomy, show early hypoglycaemia occurring 2 to 3 hours after glucose ingestion (Cameron et al., 1969; Breuer et al., 1972; Wiznitzer et al., 1974). Post-operative gastric emptying is rapid, producing hyperglycaemia half to one hour after glucose ingestion, which leads to an excessive insulin release followed by hypoglycaemia two to three hours later (Roth and Meade, 1965). The glucose absorption may be related with an excessive release of some gut factor which potentiates glucose stimulated insulin release (Rehfeld et al., 1973).

Postprandial hypoglycaemia has been denoted as an early sign of Diabetes Mellitus (Harris 1924). An oral glucose tolerance test performed on a number of subjects showed an early hyperglycaemia followed four to five hours later by hypoglycaemia. The cause was postulated as a defect in the early release of insulin (Seltzer et al., 1956).

Nervous individuals exhibit a postprandial hypoglycaemia (Conn 1955; Fabrykant 1955). These persons are emotionally labile, tense and anxious and show a fall in blood sugar after meals which has been attributed to excessive vagal tone and is labelled as vagotonic hypoglycaemia.

Idiopathic postprandial hypoglycaemia has been found in individuals who are not obese and have a peak of plasma insulin 60 minutes after oral glucose (Luyckx 1971). The etiology is speculated to be either a relative increase in insulin sensitivity or impaired gluconeogenic mechanism (Hofeldt 1975).

Postprandial hypoglycaemia in hormonal conditions has been observed in hypopituitary children (Brasel et al., 1965), and growth hormone deficient children (Fabrykant 1955). Patients with cortisol deficiency and with hypothyroidism have also been reported to show a postprandial hypoglycaemia (Hofeldt et al., 1974).

Regarding therapy for postprandial hypoglycaemia, carbohydrate restricted diets with high protein contents have been tried giving no incidence of hypoglycaemia (Conn 1936). It has also been suggested that diabetic types of carbohydrate diet with frequent feedings would alleviate the attack (Anderson and Herman, 1975). The effect of anticholinergics on preventing postprandial hypoglycaemia is not yet fully established. The beneficial effect may be through delayed gastric emptying and slowing of gastro-intestinal motility.

Emotional disturbances have been correlated with postprandial hypoglycaemia. There is abundant evidence that catecholamine secretion impairs carbohydrate tolerance (Wahren et al., 1975). The use of mild sedatives and tranquilizers has proved to be beneficial (Ensinck and Williams, 1974; Smelo, 1966). Psychotherapy is necessary where more serious psychiatric conditions co-exist.

Surgery in the form of reversal of a 10 cm jejunal segment in patients having undergone gastric surgery and experiencing disabling hypoglycaemia show symptomatic improvement. More controlled studies are required to yield more definite information on postprandial hypoglycaemia.

References

Anderson, J.W. and Herman, R.H. (1975) Effects of carbohydrate restriction on glucose tolerance of normal men and reactive hypoglycaemic patients. Am. J. Clin. Nutr., 28:748.

Brasel, J.A., Wright, J.C., Wilkins, L. and Blizzard, R.M. (1965) An evaluation of seventy-five patients with hypopituitarism beginning in childhood. Am. J. Med., 38:484.

Breuer, R.I., Moses, H., Hagen, T.C. and Zuckerman, L. (1972) Gastric operations and glucose homeostasis. Gastroenterology, 62:1109.

Cameron, A.J., Ellis, J.P., McGill, J.I. and LeQuesne, L.P. (1969) Insulin response to carbohydrate ingestion after gastric surgery with special reference to hypoglycaemia. Gut, 10:825.

Conn, J.W. (1936) The advantage of a high protein diet in the treatment of spontaneous hypoglycaemia. J. Clin. Invest., 15:673.

Ensinck, J.W. and Williams, R.H. Disorders causing hypoglycaemia. In textbook of endocrinology, edited by Robert H. Williams. 5th ed. Philadelphia, Saunders, 1974. p. 627.

Fabrykant, M. (1955) The problem of functional hyperinsulinism or functional hypoglycaemia attributed to nervous cases. Laboratory and Clinical correlations. Metabolism, 4:469.

Gerich, J.E., Schneider, V., Dippe, S.E., Langlois, M., Noacco, C., Karam, J.H. and Forsham, P.H. (1974) Characterization of the glucagon response to hypoglycaemia in man. J. Clin. Endocrin. Metab., 38:77.

Harris, S. (1924) Hyperinsulinism and dysinsulinism. JAMA, 83:729.

Hofeldt, F.D. (1975) Reactive hypoglycaemia. Metabolism, 24: 1193.

Hofeldt, F.D., Lufkin, E.G., Hagler, L., Block, M.B., Dippe, S.E., Davis, H.W., Levin, S.R., Forsham, P.H. and Herman, R.H. (1974) Are abnormalities in insulin secretion responsible for reactive hypoglycaemia? Diabetes, 23:589.

Luyckx, A.S. and Lefebvre, P.J. (1971) Plasma insulin in reactive hypoglycaemia. Diabetes, 20:435.

Rehfeld, J.F., Heding, L.D. and Holst, J.J. (1973) Increased gut glucagon as a pathogenetic factor in reactive hypoglycaemia. Lancet, 1:116.

Roth, D.A. and Meade, R.C. (1965) Hyperinsulinism-hypoglycaemia in postgastrectomy patients. Diabetes, 14:526.

Seltzer, H.S., Fajans, S.S. and Conn, J.W. (1956) Spontaneous hypoglycaemia as an early manifestation of diabetes mellitus. Diabetes, 5:437.

Smelo, L.S. (1966) Treatment of functional hypoglycaemia — an early manifestation of diabetes mellitus. Mod. Treat., 3:342.

Wahren, Jr., Felig, P., Ahlborg, G. and Jorfeldt, L. (1971) Glucose metabolism during leg exercise in man. J. Clin. Invest., 50:2715.

Wiznitzer, T., Shapira, N., Stadler, J., Ayalon, D. and Harell, A. (1974) Late hypoglycaemia in patients following vagotomy and pyloroplasty. Int. Surg., 59:229.