Alterations in vagosympathetic control and glucose-induced thermogenesis in obese patients

B Lormeau, G Karam, P Miossec, J Pariès, S Idriss, Jr Attali, P Valensi

reduced in the obese subjects with parasympathetic dysfunction. In NIDD the early increase in HR was normal; the later increase in HR and the blood pressure response were reduced in NIDD with parasympathetic dysfunction. This study suggests that i) in obese subjects, sympathetic activity is reduced, vagal hypertony, which might participate in hyperinsulinemia, would be replaced later by a vagal dysfunction; ii) in NIDD sympathetic activity is reduced only in the patients with vagal dysfunction; iii) obesity per se might be involved in vagosympathetic changes in NIDD.

Alterations in vagosympathetic control and glucose-induced thermogenesis in obese patients. B Lormeau, G Karam, P Miossec, J Pariès, S Idriss, JR Attali, P Valensi *(Department of Endocrinology-Diabetology-Nutrition, Jean-Verdier Hospital, Paris-Nord University, Bondy, France)*

Alterations in vagosympathetic control have been reported in animal models of obesity and obese patients. The aim of this study was to investigate the link between these alterations and glucose-induced thermogenesis (GIT) in non-diabetic subjects referred to our department for obesity. Thirty-three subjects were included. GIT was studied by continuous measurements by indirect calorimetry (Deltatrac Monitor) in the hour before and the 3 h after the oral consumption of a 75 g dose of glucose. O_2 consumption and CO_2 production were continuously monitored. Glucose and lipid oxidation rates were calculated from the respiratory quotient. Five standardized tests, three studying parasympathetic control (deep-breathing, lying-to-standing and Valsalva) and two depending on sympathetic activity (postural hypotension, blood pressure response to a handgrip test) were performed. Fat free mass was measured by impedancemetry method. The five standardized tests were normal in ten patients (group 1), whereas in the 23 other patients (group 2), one or several parasympathetic tests were altered (18 cases) or both parasympathetic and sympathetic tests were abnormal (five cases). Age, sex ratio, body mass index (BMI) and fat free mass did not differ between the two groups. During the oral glucose test, none of the patients met the criteria for diabetes mellitus. The plasma glucose response was very similar in both groups. The insulin response was also very similar. GIT was not significantly different. In the basal state before glucose ingestion, compared with group 1, group 2 had a lower respiratory quotient (0.83 ± 0.03 vs 0.88 ± 0.02, $P < 0.0001$), lower glucose oxidation (1.34 ± 0.56 vs 1.92 ± 0.32 mg/kg/min, $P = 0.004$) and a higher lipid oxidation (0.54 ± 0.14 vs 0.32 ± 0.16 mg/kg/min, $P = 0.001$). During the 3 h following glucose ingestion, the calculated cumulative oxidation of glucose was higher ($P = 0.05$), the cumulative oxidation of lipids was lower in group 2 ($P < 0.02$) than in group 1 and GIT was not significantly different. The increase in plasma noradrenalin was lower in group 2, the difference being only significant at 90 min (1.75 ± 0.70 vs 2.18 ± 0.60 nmol/L, $P = 0.05$). The changes in blood glucose, plasma insulin and adrenalin levels were very similar in the two groups. These results suggest that vagal dysfunction was associated with a change in substrate oxidation. The higher basal lipid oxidation in the patients with vagal dysfunction may be due to a relative increase in sympathetic tone. The lower increase in plasma noradrenalin, which suggested a lower sympathetic activation after glucose ingestion, might account for the stronger reduction in lipid oxidation.

Preliminary results of treatment of severe obesity by adjustable silicone gastric banding (ASGB). P Lecomte, JP Marmuse, G Benhamou *(Department of General Surgery, hôpital Bichat-Claude-Bernard, 46,*