betes mellitus or thyroid dysfunction; none was taking any drug or had taken an oral contraceptive during the previous 3 months.

Plasma samples taken after an overnight fast were used to evaluate PAI-1 activity (Spectrolyse pL, BioPool), insulin, cholesterol and triglycerides (TG). BMI and waist-to-hip girth ratio (WHR) were calculated for each patient. Daily dietary intake was evaluated from food diaries kept during 1 week by the patients and analyzed with the REGAL programme (Inra, 1991) for calorie intake, carbohydrates, lipids (L), proteins (P), animal (AP) and vegetal proteins, saturated, monounsaturated (MFA), polyunsaturated fatty acids, saccharose and cholesterol.

Eight patients had cholesterol levels above 2.5 g/L, and seven had TG levels above 1.5 g/L. In comparison with the 41 patients with elevated PAI-1 levels, the 23 patients with normal PAI-1 levels (< 10 U/mL) had lower TG levels (0.75 ± 0.08 vs 1.10 ± 0.08 g/L, $P < 0.01$), lower daily intake of P (71.4 ± 3.3 vs 82.1 ± 3.3 g, $P < 0.05$), L (63.3 ± 4.4 vs 77.8 ± 3.8 g, $P < 0.02$), AP (50.0 ± 3.3 vs 60.0 ± 2.7 g, $P < 0.05$), MFA (19.7 ± 1.6 vs 24.4 ± 1.2 g, $P < 0.05$). No significant difference was found between the two groups regarding BMI (28.4 ± 0.7 vs 29.6 ± 0.5 kg/m^2), WHR (0.793 ± 0.020 vs 0.846 ± 0.016), fasting insulin levels (11.9 ± 1.9 vs 15.9 ± 1.2 mIU/L), plasma cholesterol (1.92 ± 0.10 vs 2.13 ± 0.08 g/L) and the other results of the dietary intake evaluation.

In the 64 patients, PAI-1 levels were significantly correlated with fasting insulin levels ($r = +0.290$, $P < 0.05$), TG ($r = +0.317$, $P < 0.02$), intake of P ($r = +0.370$, $P < 0.01$) and AP ($r = +0.389$, $P < 0.01$), and not with the other parameters evaluated in this study; stepwise regression showed that PAI-1 levels were dependent both on TG levels and intake of AP ($P < 0.05$).

In conclusion, in overweight premenopausal women, PAI-1 levels were less dependent on the degree of obesity or WHR than on the dietary and metabolic characteristics of the patients. Further studies should determine if nutritional counseling aimed at reducing TG levels and taking into account the intake of AP, could lead to a normalization of PAI-1 levels in patients who remain overweight.

The nature of changes in cardiovascular vagal and sympathetic functions is different in obese and non-insulin-dependent diabetic patients. P Valensi, NT Nguyen, S Idriss, G Karam, J Pariès, P Miossec, JR Attali *(Department of Endocrinology-Diabetology-Nutrition, Jean-Verdier Hospital, Paris-Nord University, Bondy, France)*

We have previously shown a high prevalence of alterations in the heart rate (HR) variability in obese and diabetic patients. The aim of this study was to investigate cardiovascular vagal tone and sympathetic response in these diseases. Sixty-two non-diabetic obese and 54 non-insulin-dependent diabetic (NIDD) patients and 35 healthy controls were investigated. Heart rate variations were analysed during three standardized tests (deep-breathing, lying-to-standing, Valsalva) and the hemodynamic response was studied during a handgrip test sustained during 5 min. The standardized tests showed a parasympathetic dysfunction in 55.7% of the obese subjects and 48.9% of the NIDD. During the handgrip test the HR increase at 1 min which results from vagal withdrawal correlated negatively with age in the controls and obese subjects taken together and correlated positively with HR variations during the lying-to-standing test. In the obese subjects without parasympathetic dysfunction, it was significantly higher than in controls, suggesting vagal hypertony at rest whereas the later increase in HR and blood pressure, which results from sympathetic activation, was particularly

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,*