45 ± 8 kg/m^2; 21 males, BMI 44 ± 11 kg/m^2) underwent nocturnal oxymetry recording, pulmonary functional testing (PFT) and diurnal arterial blood gas measurements. Their waist circumference (WC), waist/hip ratio (W/H) and neck circumference (NC) were measured. The NC was positively correlated with the WC ($p < 0.001$), independently of BMI. Group 1 (15 F/17 M) consisted of OB with NOD > 15% of recorded sleep time spent with oxygen saturation (OS) < 4% of the diurnal OS. Group B (30 F/4 M) consisted of OB without NOD. As compared with group B, subjects in group A had significantly higher BMI ($p < 0.003$) and W/H ($p < 0.01$) but had no difference in NC. In women with NOD, W/H and WC were significantly higher than in women without NOD ($p < 0.01$). Such a difference was not observed in men. In group A, the NC was positively correlated to the W/H ratio ($p < 0.005$) and $paCO_2$ ($p < 0.005$) and negatively to the residual volume (RV) ($p < 0.05$), functional residual capacity (FRC) ($p < 0.01$) and total lung capacity (TLC) ($p < 0.05$) independently of BMI. The WC was positively correlated with FRC ($p < 0.03$). The subjects in group A were classified according to the polysomnography recording. As compared with patients without OSAS ($n = 8$), patients with OSAS ($n = 21$) had significantly higher NC ($p < 0.04$), similar WC and lower vital capacity (VC) ($p < 0.05$) and TLC ($p < 0.05$). In subjects with OSAS, NC was positively correlated to $paCO_2$ ($p < 0.03$) and negatively to RV ($p < 0.03$), FRC ($p < 0.01$) and VC ($p < 0.01$). It was concluded that: i) NOD is associated with abdominal obesity, especially in women; and ii) patients with SAS differ significantly from patients without SAS in terms of NC and PFT.

Left ventricular function (LVEF) in patients with obstructive sleep apnea syndrome (OSAS) and massive obesity.

JP Laaban [1], D Cassuto [2], S Pascal [1], E Orvoën-Frija [1], A Basdevant [2], B Guy-Grand [2] *([1] Dept of Pneumology; [2] Dept of Nutrition, Hôtel-Dieu, 75181 Paris cedex, France)*

Obstructive apneas may induce acute left ventricular dysfunction mainly *via* an increase of after-load due to a negative intrathoracic pressure and a release of catecholamine. However, conflicting results have been reported concerning the chronic effects of OSA on LVEF. Previous studies did not take into account the cardiac diseases associated with obesity, and no data are available on the LVEF of massively obese patients with OSAS. In 59 consecu-

Table I. Data for patients with and without OSAS (Laaban *et al*).

	OSAS (n = *25*)	*No OSAS* (n = *34*)	p
LVEF (%)	57 (± 10)	60 (± 8)	NS
Apnea index (/h)	32 (± 23)	1 (± 0.5)	< 0.001
Hypertension (% pat)	37%	28%	NS
Diabetes (% pat)	37%	28%	NS
Myocardial ischemia (% pat)	27%	20%	NS
BMI (kg/m^2)	51 (± 9)	50 (± 10)	NS
WHR female	0.95 ± 0.16	0.94 ± 0.11	NS
WHR male	1.04 ± 0.16	0.94 ± 0.3	NS

tive patients with massive obesity (body mass index (BMI) > 40 kg/m^2), we prospectively performed polysomnography, measurements of LVEF using gated equilibrium radionuclide angiography and detection of myocardial ischemia using dipyridamol thallium-201 scintigraphy. A decrease in the LVEF (< 55%) was observed in 36% (9/25) of patients with OSAS and in 26% (9/34) of patients without OSAS (NS) (table I).

These almost negative results suggest that OSAS *per se* is not responsible for chronic left ventricular dysfunction and that the moderate decrease in LVEF frequently observed in patients with OSA and massive obesity is probably related to obesity or cardiac diseases frequently associated with massive obesity, such as coronary artery disease or hypertension.

Thyroid hormone and thyrotropin variations during long-term overfeeding. JM Oppert [1,4], JH Dussault [2], A Tremblay [1], JP Després [1,3], G Thériault [1], C Bouchard [1*] *([1] Physical Activity Sciences Laboratory; [2] Medical and Molecular Genetic Research Unit; [3] Lipid Research Center, Laval University, Sainte-Foy, PQ, G1K7P4, Canada; [4] Hôtel-Dieu, Paris, France)*

Thyroid hormone physiology and action are modulated by the energy intake. Thyroid hormones are also well-known determinants of metabolic rate. In the long-term overfeeding studies conducted by EAH Sims in Vermont 2 decades ago, it was shown that overnutrition was associated with increased serum T3 concentrations while the serum T4 concentrations remained unchanged (Danforth *et al* (1979) *J Clin Invest* 64, 1336-1347). The energy expenditure, however, was not measured during these experiments. There is also no report concerning the TSH concentrations or responsiveness during a prolonged positive energy balance period. The aims of the present study were, therefore, to evaluate the plasma thyroid hormone levels and the TSH variations during a standardized long-term overfeeding protocol (4.2 MJ = 1 000 kcal/d over basal energy requirements, 5 d/week, during a 100 d period) in 24 lean adults (12 pairs of monozygotic twins), and to assess their relationships with body composition and resting metabolic rate (RMR) changes. The body composition and energy expenditure changes brought about by this protocol were previously reported (Bouchard *et al* (1990) *N Engl J Med* 322, 1477-1482; Tremblay *et al* (1992) *Am J Clin Nutr* 56, 857-862). Compared to the pre-overfeeding values, the basal plasma T3 concentrations increased at day 25 but not later, the basal plasma T4 and free T4 concentrations were unchanged, the basal plasma rT3 concentrations decreased persistently throughout the entire protocol, and the TSH response to TRH stimulation was persistently enhanced. The TSH response to TRH before overfeeding was negatively correlated with the changes in RMR with overfeeding ($r = -0.53$, $p < 0.01$). No association was found between the changes in basal plasma T3 concentrations and the changes in RMR. The changes in basal T3, however, were positively related to changes in body weight ($r = 0.46$, $p < 0.05$). A significant within-pair similarity was found for the changes in T4 and free T4 with overfeeding ($p < 0.05$). We conclude that: 1) during overfeeding, the early increase in T3 concentrations is a transitory phenomenon whereas the decrease in rT3 concentrations and the increased TSH response to TRH are more sustained; 2) the TSH responsiveness to TRH stimulation could be a predictor of the changes in RMR in times of increased energy intake; 3) there is no evidence for a direct role of T3 in the adaptation of resting energy expenditure during a long-term overfeeding protocol; and 4) the genotype could be involved in the changes in T4 and free T4 during a prolonged positive energy balance period.