($P < 0.001$ vs 6 months), and 85 ± 5 kg at 24 months (NS vs 12 months). The body weight loss was greatest at the beginning of the experimental period: 21 ± 1 kg during the first 3 months, then 12 ± 2 between 3 and 6 months ($P < 0.001$) and 9 ± 2 kg between 6 and 12 months ($P < 0.001$). BW was then stabilized (2.6 ± 2.8 kg between 12 and 24 months). RM was initially at 2 149 ± 47 kcal/day and then decreased: 1 750 ± 54 kcal/day at 3 months ($P < 0.001$), 1 726 ± 56 kcal/day at 6 months (NS vs 3 months), 1 582 ± 50 kcal/day at 12 months ($P < 0.05$ vs 6 m), 1 553 ± 64 kcal/day at 24 months (NS vs 12 m). RM reduction was massive between 0 and 3 months (–20 ± 1%), the later changes in RM were smaller (–3 ± 2% between 3 and 6 months, –5 ± 1% between 6 and 12 months, 1 ± 2% between 12 and 24 months). RM/BW ratio was initially 17.1 ± 0.3 kcal/day/kg and dropped at 3 months (16.5 ± 0.3 kcal/day/kg; $P < 0.002$) to increase at 6 months (17.7 ± 0.4 kcal/day/kg; $P < 0.05$ vs 3 months), at 12 months (18.6 ± 0.4 kcal/day/kg; $P < 0.05$ vs 3 and 6 months), and at 24 months (19.1 ± 0.5 kcal/day/kg; NS vs 12 m).

We observed in the initial period (0 to 3 months) a massive decrease in RM more than would have been predicted by BW loss. The RM/BW ratio increase was probably linked to a body weight composition modification and a diminution of the fat mass/fat free mass ratio. In conclusion, during VBG we observed a RM stabilization between 3 to 24 months after an initial phase of RM reduction. BW and RM stability after 1 year showed that the calorie level of the diet was probably over 1 500 kcal/day.

Stabilization of energy expenditure 3 months after vertical banded gastroplasty treatment for morbid obesity.
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Weight loss in response to a vertical banded gastroplasty (VBG) is due to a reduction in calorie intake. This will continue as long as food intake is less than total energy expenditure (TEE). Changes in energy expenditure and food intake are the major determinants of weight loss. Estimation of dietary intake after VBG is very difficult because of vomiting. We studied energy expenditure before and 3 and 6 months after surgery in four morbidly obese patients, aged 32 ± 4 years, weight 131 ± 2 kg, body mass index (BMI) 49 ± 2 kg/m^2 (mean ± SEM). Total energy expenditure and body composition (lean mass, fat mass) were determined with doubly labeled water ($^2H_2{}^{18}O$). After the oral absorption of $^2H_2{}^{18}O$, the evolution of ^2H and ^{18}O isotopic enrichment of total body water was measured in urine samples at 0 h, 4 h and 14 days with an isotopic ratio mass spectrometer OPTIMA (Fisons). Resting metabolic rate (RMR) was measured by indirect calorimetry (Deltatrac, Datex). Energy expenditure due to physical activity and thermogenesis was calculated: EP = TEE–RMR. Weight loss was maximal during the first 3 months after VBG: –21 ± 4 kg, then –10 ± 3 kg between 3 and 6 months. Fat mass loss was large: –14 ± 3 kg after 3 months and –7 ± 2 kg between 3 and 6 months. There is a decrease of fat mass when expressed as a percent of body weight from 52 ± 4% before, to 46 ± 4% 6 months after VBG. Lean mass was better preserved: –6 ± 3 kg at 3 months and –2 ± 1 kg at 6 months. TEE decreased during the first 3 months from 3 227 ± 187 to 2 557 ± 110 kcal/day and was stable between 3 and 6 months at 2 642 ± 72 kcal/day. RMR followed the same kinetic from 1 975 ± 107 to 1 626 ± 58 kcal/day at 3 months and 1 659 ± 72 kcal/day at 6 months. EP changed little during body weight evolution (1 265 ± 206, 931 ± 167, 923 ± 224 kcal/day) before, 3 and 6 months after VBG respectively.

In summary, 3 months after VBG, we observed a stabilization of TEE and RMR

which was probably related to the maintenance of lean mass. This stabilization contributed to the weight loss between 3 and 6 months. A determination of TEE when the body weight stabilizes will allow the quantification of the energy intake.

Modification of the β-adrenergic lipolytic response in obese human adipocytes.
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The mobilization of stored triglycerides is under the control of different subtypes of β-adrenergic receptors, which can be modified under several physiopathological situations, such as obesity (Hellmer et al [1992] *J Clin Endocrinol Metabol* 75, 15). This investigation focused on the study of the lipolytic response induced by β-adrenergic agonists in adipocytes from obese and nonobese patients.

Omental adipose tissues were obtained during different surgical situations and the adipocytes were isolated by incubation with collagenase in a KRBA buffer (pH 7.4). A control (body mass index [BMI] < 25; n = 17) and an obese (BMI ≥ 25; n = 21) group were formed. The in vitro lipolytic response was induced by dobutamine (Dob; β_1), clenbuterol (Clen; β_2), metaproterenol (Met; β_1 and β_2) in a range of concentrations of 10^{-8} to 10^{-4} M.

The adipocytes from the obese group showed a higher basal lipolysis (0.18 ± 0.01 µmol glycerol/100 mg lipids) than controls (0.16 ± 0.06 µmol glycerol/100 mg lipids; $P < 0.05$). The minimum concentrations of the β-agonists needed to produced a significant lipolytic effect were higher in adipocytes from obese subjets than controls (Dob: 5×10^{-5} vs 10^{-5} M; Clen: ND vs 5×10^{-7} M; Met: 10^{-6} vs 10^{-7} M). In relation to maximal effect, a statistically significant reduction was only observed for the lipolytic response to Met in the obese group (139.5 vs 220.6% of basal lipolysis).

It can be concluded that obesity alters the metabolic pathways relating to the lipolytic capacity controlled by β-adrenergic agonists.

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