Changes in Resting Energy Expenditure and Body Composition after Weight Loss following Roux-en-Y Gastric Bypass

Fernando Carrasco, MD¹; Karin Papapietro, MD²; Attila Csendes MD²; Gabriela Salazar, PhD³; Constanza Echenique, MD¹; Cecilia Lisboa, MD¹; Emma Díaz, MSc²; Jorge Rojas, MD²

¹Department of Nutrition, Faculty of Medicine, ²Department of Surgery, Clinical Hospital, ³Institute of Nutrition and Food Technology, University of Chile, Santiago, Chile

Background: The objective of this study was to evaluate changes in resting energy expenditure (REE), body composition and metabolic parameters, and to investigate predictors of the results in seriously obese patients after Roux-en-Y gastric bypass (RYGBP).

Methods: 31 patients (BMI 44.4 \pm 4.8 kg/m²; 27 female, 4 male; 37.3 \pm 11.1 y) were evaluated at baseline and 6 months after RYGBP. Weight, REE, waist circumference (WC), fat mass (FM) and fat-free mass (FFM), physical activity, food intake, fasting glucose (GLU), insulin (INS), HOMA-IR and lipid concentrations were measured.

Results: At 6 months, percentage of weight loss (%WL) was 29.0 \pm 4.4% and percentage of excess weight loss was (%EWL) 59.7 \pm 12.3%. FM loss corresponded to 77.1 \pm 12.2% of the weight loss. REE decreased from 33.4 \pm 4.1 to 30.1 \pm 2.6 kcal/kg FFM (*P*<0.05). Significant decreases (*P*<0.001) were observed in GLU, INS, HOMA-IR, LDL-cholesterol and triglycerides. %EWL was correlated with baseline INS (r=0.44; *P*=0.014), baseline HOMA (r=0.43; *P*=0.017), change in %FM (r=0.67; *P*<0.001) and change in WC (r=0.5; *P*<0.01). Decrease in REE/FFM (%) was positively correlated with baseline REE/FFM% (r=0.51; *P*<0.005) and change in %FM (r=0.69; *P*<0.001). Initial REE/FFM, baseline energy balance and FM change explain 90% of REE/FFM decrease.

Conclusion: RYGBP was an effective procedure to induce significant weight loss, fat mass loss and improvement in metabolic parameters in the short term. Metabolic adaptation was not related to FFM wasting but to a higher baseline REE. Fasting hyperinsulinemia was the best single predictor of weight loss after RYGBP.

Key words: Resting energy expenditure, body composition, gastric bypass, morbid obesity, cardiovascular risk factors, body fat loss

Introduction

Obesity is an illness of growing prevalence, which is associated with an increase in morbidity and mortality.^{1,2} The prevalence of morbid obesity (body mass index, BMI, \geq 40 kg/m²) in adult Chileans, according to the data in the first national health survey, are 2.3% in women (8.4% of total obese women) and 0.2% in men (1% of total obese men).³ In these patients, bariatric surgery is considered the most effective method to induce significant weight loss (~70% of excess weight) that is maintained in the long term, where the Roux-en-Y gastric bypass (RYGBP) is the most recommended technique currently used. This operation is associated with major improvement in comorbidities, psycho-social factors, eating behavior, the level of physical activity and physical capacity.^{4,6}

Some studies have investigated the potential factors that could predict better results in patients submitted to bariatric surgery, especially after RYGBP. Among these, the initial excess weight and baseline BMI, energy expenditure, presence of type 2 dia-

Correspondence to: Fernando Carrasco, MD, Professor, Department of Nutrition, Faculty of Medicine, University of Chile, Independencia 1027, Postal Code 838-0453, Santiago, Chile. Fax: (562) 737-8778; e-mail: fcarrasc@med.uchile.cl

Supported by a grant from University of Chile (DID-SAL 01/04-2).

betes or insulin resistance, postoperative nutritional intake, level of physical activity, psychological illness and eating disorders have been mentioned.⁷⁻¹⁴

On the other hand, the maintenance of the longterm weight loss depends on favorable changes in eating habits, physical activity and nutritional behavior,¹⁴⁻¹⁶ and on the absence of metabolic factors that predispose to regain of weight.^{15,17,18}

The purpose of the present investigation was to evaluate the magnitude of the changes in resting energy expenditure (REE), nutritional intake, body composition, level of physical activity and cardiovascular risk factors in patients submitted to a resectional RYGBP. Also, we intended to detect metabolic or behavioral parameters that could predict the reduction in weight, the loss in body fat and the improvement in cardiovascular risk factors.

Subjects and Methods

Subjects

We prospectively studied 38 adult patients (34 women and 4 men; age 36.3 ± 10.5 y; age range 17-58 y), with BMI \geq 40 kg/m² or BMI \geq 35 kg/m² with co-morbidities, who underwent RYGBP in the Department of Surgery of the University Hospital. The average BMI was 44.0 ± 4.5 kg/m² (range 36.1-59.4): 4 had a BMI between 35 and 39.9 kg/m², 31 patients (81.6%) had a BMI between 40 and 49.9, and 3 subjects presented a BMI \geq 50 kg/m².

All patients who agreed to enter the study signed an authorized consent form. The study was approved by the Ethics Committee for Human Investigation of the University Hospital.

The surgical technique consisted of a 95% distal gastrectomy, with resection of the bypassed stomach, leaving a small gastric pouch of 15-20 ml.¹⁹ Then, an end-to-side gastro-jejunostomy with circular stapler No. 25 was performed. The length of the Roux-loop was 125-150 cm.

All patients underwent complete preoperative evaluation. Prior to and 6 months after surgery, the following evaluations were carried out in all patients: anthropometry, food and physical activity survey, REE, body composition analysis and evaluation of cardiovascular risk parameters.

Anthropometric and Food Intake Evaluations

Weight was measured to the nearest 0.1 kg on a digital scale (Detecto), and height (m) was measured to the nearest 0.1 cm with a scale-mounted stadiometer. BMI (kg/m²) was calculated. Waist (WC) and hip (HC) circumferences (cm) were registered according to the procedures recommended by the Anthropometric Standardization Reference Manual.²⁰ Patients were interviewed by a dietitian who conducted a food frequency questionnaire, and 3-day records were filled. The recorded data were analyzed using a computer program (Food Processor II) to calculate energy, protein, lipid and carbohydrate intake.

Resting Energy Expenditure and Fasting Respiratory Quotient

Resting energy expenditure (REE) was measured by open circuit indirect calorimetry using a ventilated chamber system (Deltatrac, Datex Inst. Corp., Helsinki, Finland). Subjects were evaluated in the morning, after a 12-h overnight fast with at least 30 minutes of supine rest in a thermo-regulated environment (21-22°C). After a resting period, O_2 consumption, CO₂ production and respiratory quotient (RQ) were measured during 15-20 minutes, and the last 5-10 minutes were used to calculate REE, RQ, and percentage of lipid oxidation.

Body Composition

Total body water was measured by means of isotopic dilution with deuterium oxide $(H_2^{18}O)$.²¹ Saliva samples were analyzed in the Stable Isotope Laboratory of the Nutrition Institute and Food Technology of the University of Chile, for the measurement of deuterium content by means of mass spectrometry (HYDRA, Europe Scientific, Crewe, UK). Applying the dilution method and calculating the saliva isotope enrichment, after 3 hours of a dose of 0.7 g/kg of 99.9% deuterium oxide, total body water (liters) was calculated. Fat free mass (FFM, kg), was calculated assuming a hydration coefficient of 0.756 and 0.747, before and after surgery, respectively, according to data obtained by Das et al²² in morbidly obese patients using 3-compartment analysis.²² Body fat (BF, kg) was obtained using the difference between total body weight and FFM.

Carrasco et al

Physical Activity

We applied a survey with regards to physical activity during leisure time and hours of viewed TV per week.²³ In 12 patients, heart rate was measured at 1min intervals during 24 hours in a free-living situation with a cardio-frequency monitor (Polar Vantage NV, Polar Electro, Finland).²⁴ The activity factor was calculated as the average of activity heart rate divided by the average of resting heart rate.

Parameters of Cardiovascular Risk

Systolic and diastolic blood pressure were measured in supine position after a resting period. Fasting venous blood samples were obtained for the measurements of glucose and lipid concentrations. Plasma insulin was measured with a radioimmuneassay kit specific for human insulin. Insulin resistance index was calculated using glucose and insulin fasting values according to the HOMA method (Homeostasis Model Assessment).

Statistical Analysis

Results were expressed as mean \pm standard deviation. Paired *t*-tests were used for the comparison between determinations before and after surgery. Wilcoxon Signed Rank test was applied when variables did not show normal distribution. Single correlations among variables were evaluated with the Spearman coefficient of correlation. We developed multivariate models by means of regression analysis (Best Subsets Regression) between dependent and independent variables for the purpose of finding the best predictors of changes after surgery. All analyses were developed with the statistical program, Jandel Sigma Stat, version 2.0 (Copyright 1995, Jandel Corp.) with a 5% (*P*<0.05) significance level.

Results

The general characteristics and measurements of the 38 initial patients (34 women and 4 men) are summarized in Table 1. In this sample, we found hypertension in 15 patients, type 2 diabetes in 7 patients, glucose intolerance in 5 patients, dyslipidemia in 19 patients,

Table 1. Baseline demographics, anthropometrics,
body composition and energy metabolism charac-
teristics of patients before bariatric surgery (n= 38)

Variable	Mean ± SD	Range
Age (years) Body weight (kg) Excess weight (kg) BMI (kg/m ²) Waist (cm) Hip (cm) Waist to Hip ratio Body fat (%)*	36.3 ± 10.5 114.9 ± 16.1 57.2 ± 12.9 44.0 ± 4.5 121.4 ± 12.0 134.0 ± 9.8 0.91 ± 0.08 52.0 ± 5.3	17 - 58 87.0 - 166.8 33.9 - 95.2 36.1 - 59.4 94 - 147 114 - 169 0.73 - 1.13 41.2 - 60.8
TBW (L)* TBW (% of body weight)* REE (kcal/d) npRQ Fat oxidation (%)	$41.9 \pm 7.1 \\36.3 \pm 4.0 \\1830.1 \pm 295.9 \\0.86 \pm 0.07 \\43.9 \pm 19.6$	32.2 - 63.8 32.2 - 44.5 1380 - 2750 0.74 - 1.05 0.0 - 81.4

BMI = body mass index; TBW = total body water measured by deuterium dilution; REE = resting energy expenditure measured by indirect calorimetry; npRQ = non-protein respiratory quotient measured by indirect calorimetry; *Body fat and TBW were measured in 37 patients.

and arthropathy in 21 patients. In 31 of the 38 patients, we re-evaluated the REE, and in 30 of them we measured the body composition 6 months after the RYGBP.

Body Composition

The average weight loss was 33.4 ± 7.6 kg (59.7 \pm 12.3% EWL), with a decrease of 26.0 ± 6.9 kg of BF and 7.8 ± 4.8 kg of FFM. Body fat corresponded to $77.1 \pm 12.2\%$ of total weight loss. In Table 2, preoperative and 6th month anthropometric and body composition parameters are shown. In the simple regression analysis, percentage of initial weight lost (%WL) had a significant positive correlation with the initial fasting insulinemia (r=0.437, P <0.05) and with HOMA index (r=0.427, P<0.05). There was no significant correlation among %WL and baseline parameters such as age, BMI, %BF, FFM, WC, WC/HC ratio, REE and fasting lipid oxidation. Weight loss had a significant correlation with %BF reduction (r=0.674, P < 0.001) and with WC change (r=0.5, P < 0.01), but not with FFM loss.

Changes in Energy Metabolism

REE decreased from 1845 \pm 302 kcal/d to 1449 \pm 215 kcal/d, with a mean decrease of 396 \pm 157 kcal/d

Variable	Baseline	6th month	Change	Change (%)	P-value
Weight (kg)	115.4 ± 17.5	82.0 ± 12.3	-33.4 ± 7.6	-29.0 ± 4.4	<0.001
BMI (kg/m ²)	44.4 ± 4.8	31.6 ± 4.1	-12.9 ± 2.3	-29.2 ± 4.7	<0.001
Body fat (%)*	51.6 ± 5.4	41.2 ± 6.2	-10.6 ± 4.6	-20.4 ± 8.8	<0.001
Body fat (kg)*	60.0 ± 11.3	34.0 ± 8.6	-26.0 ± 7.0	-43.4 ± 8.3	<0.001
Fat-free mass (kg)*	56.1 ± 10.2	48.0 ± 7.3	-7.8 ± 4.8	-13.4 ± 6.7	<0.001
Waist (cm)	121.2 ± 12.7	92.6 ± 10.6	-28.6 ± 8.1	-23.4 ± 5.8	<0.001
Hip (cm)	134.2 ± 10.2	110.3 ± 8.9	-27.5 ± 18.2	-18.2 ± 3.5	<0.001
Waist to Hip ratio	0.90 ± 0.08	0.84 ± 0.07	-0.06 ± 0.06	-6.8 ± 6.3	<0.001

Table 2. Changes in weight and body composition parameters (n=31)

BMI = body mass index; *body fat and fat-free mass were evaluated in 30 patients.

(*P* <0.001). Ratio of REE/FFM was reduced from 33.4 ± 4.1 to 30.1 ± 2.6 kcal/kg FFM/d (*P* <0.05), with a mean reduction of 3.25 ± 3.63 kcal/kg FFM/d (8.9 ± 9.8% of initial value). RQ decreased from 0.86 ± 0.06 to 0.82 ± 0.05 (*P* <0.05), and fasting lipid oxidation increased from 43.9 ± 19.4% to 55.3 ± 16.4% (*P* <0.05). Reduction in REE/FFM ratio did not show significant correlation with %WL (r=0.173, *P*=0.37), but showed a significant positive correlation with the percentage of BF reduction (r=0.522, *P*<0.01), and a negative correlation with the percentage of FFM loss (r=-0.653, *P* <0.001).

The REE measured at 6 months postoperatively was compared with those values obtained applying the regression equation among REE and FFM before surgery, in order to observe if REE was found to be less than the expected value. The regression equation for REE and FFM before surgery was the following: REE (kcal/d) = 926.1 + 17.2 x FFM (kg)

(r=0.66, standard error estimation = 224.9 kcal, P<0.001).

In Figure 1, all values of REE 6 months after surgery (except in one patient) were found below the regression line: therefore, lower than expected for the FFM.

In the regression analysis, variability in preoperative REE was best explained when fat mass and age were included, along with FFM. The following regression equation was obtained:

REE (kcal/d) = 654.1 + 20.6 x FFM (kg)+ 7.3 x BF (kg)-10.3 x age (y) (r =0.80, standard estimation error = 185.6 kcal, *P*<0.001)

This equation was applied to estimate REE of patients 6 months after surgery and to compare it to the measured REE, observing that the measured value was 83.1 \pm 123.6 kcal/d less than the value calculated by formula (*P* <0.005), with a 95% confidence interval for the difference estimation minus

measurement of -159.2 to 325.3 kcal/d. In Figure 2, we observe a great dispersion of the difference between estimated REE and measured REE, with an increasing negative value as the measured REE value increased (r=-0.37, P < 0.05). In Figure 3, we compared REE measured at 6 months after surgery with the estimated REE using the regression equation developed in the preoperative state (with FFM, FM and age as variables). In most patients, the measured value was lower than that estimated by the equation (r=0.806, P < 0.001). The decrease of REE/FFM as a percentage of baseline value, had a significant positive correlation with the initial REE/FFM (r=0.6, P < 0.001), and with the percentage of body fat loss (r=0.69; P < 0.0001). There was no correlation in decrease of REE/FFM with age, %WL, reduction of WC, changes in energy or protein intake, or variation in physical activity level.



Figure 1. Values of resting energy expenditure (REE) in relation to fat-free mass (FFM) at 6 months after RYGBP compared with the regression line of REE and FFM before surgery [REE (kcal/d) = 926.1 + 17.2 x FFM (kg)].



Figure 2. Difference of calculated REE by regression* against measured REE at 6th month (mean in solid line), with 95% limits of agreement (broken lines) and regression line (A). *Baseline predictive regression: REE (kcal/d) = $654.1 + 20.6 \times FFM$ (kg) + 7.3 x FM (kg) - 10.3 x age (y)

FFM = fat-free mass; FM = fat mass.

Variations in Physical Activity

An increase in scoring of the leisure time physical activity questionnaire was found between the mean value (\pm SD) of 10.0 \pm 14.4 before surgery (range 0 - 72), and 23.5 \pm 22.9 at 6 months postoperatively (*P* <0.001). Also, TV hours per-week showed a decrease from 19.1 \pm 17.8 to 13.7 \pm 10.5 (*P* <0.01). In 12 patients, the activity factor increased significantly from 1.25 \pm 0.06 to 1.33 \pm 0.11 (+ 6.5 \pm 10.3%, *P*=0.041).



Figure 3. Comparison between REE measured 6 months after RYGBP (plot) and REE calculated by linear regression including baseline fat-free mass, fat mass and age (dashed line).

Changes in Food Intake

In Table 3, variations in energy and macronutrient intake between preoperative and the 6th postoperative month are shown. At 6 months after RYGBP the patients have maintained a significant deficit in their energy intake with regards to the calculated energy requirements.

Variation in Cardiovascular Risk Parameters

We observed a significant improvement in the values for blood pressure and metabolic parameters of cardiovascular risk, except for HDL-cholesterol whose increase did not reach statistical significance (Table 4). Variation in all parameters (except HDL-cholesterol), showed no correlation with %WL, %BF reduction or WC reduction. The increase in HDL-cholesterol showed a significant positive correlation with the decrease in WC (r=0.43, P=0.02).

Discussion

Changes in Weight and Body Composition

RYGBP is the most extensively performed operation for morbid obesity. This procedure induces substantial weight loss, essentially by reducing energy intake and decreasing absorption of nutrients. In comparison with a purely restrictive method, RYGBP induces a greater and long-lasting reduction of weight.^{4,26,27} In the present study, at 6 months postoperatively, we observed a mean weight loss of 28.7% of the initial weight, which corresponds to a 59.2% EWL. This weight reduction is similar or greater than that observed in other studies with short-term followup.^{9,28} In the present study, the mean body fat loss was of 26 kg, corresponding to 77% of the total weight lost at 6 months after surgery; similar results have been reported in studies of bariatic surgery with body composition analysis,⁴ and in obese patients submitted to non-surgical treatment.^{29,30}

In Bobionni-Harsch's study,⁹ it was found that the weight loss induced by RYGBP was influenced by the postoperative caloric intake, and by variations of the surgical technique, such as size of gastric pouch, alimentary limb-length, and gastro-jejunostomy diameter, among other factors. In our study, because

Food intake	Baseline (range)	6th month (range)	P-value
Energy			
kcal/d	3585 ± 1666 (1654 to 9745)	1000 ± 166 (790 to 1418)	<0.001
kcal/kg/d	30.4 ± 10.6 (16.3 to 58.4)	11.2 ± 1.7 (7.7 to 15.9)	<0.001
Carbohydrates			
g/d	447.9 ± 215.4 (148 to 1180)	113.9 ± 25.8 (85 to 172)	<0.001
% energy intake	49.8 ± 6.8 (35.9 to 62.9)	45.3 ± 4.8 (38.9 to 55.9)	NS
Fats			
g/d	142.5 ± 77.9 (67 to 424.5)	37.2 ± 7.2 (26.2 to 57.5)	<0.001
% energy intake	35.5 ± 6.9 (23.5 to 48.6)	33.4 ± 3.2 (26.4 to 38.9)	NS
Proteins			
g/d	134.3 ± 71.8 (59.7 to 420.4)	54.1 ± 6.3 (40.1 to 65.2)	<0.001
g/kg/d	1.14 ± 0.5 (0.5 to 2.5)	0.67 ± 0.1 (0.4 to 0.9)	<0.001
% energy intake	14.7 ± 4.5 (7.4 to 26.0)	21.3 ± 3.5 (12.3 to 26.1)	<0.001
Energy balance*			
kcal/d	1177 ± 1443 (-816 to 6170)	-891 ± 197 (-1389 to -529)	<0.001

Table 3. Variation in food intake between pre- and post-RYGBP periods

*Energy balance was calculated as follows: EB (kcal/d) = energy intake (kcal/d) – [REE (kcal/d) x 1.3†]. REE = resting energy expenditure measured by indirect calorimetry. †Activity factor proposed by Bray G. in sedentary subjects.²⁵

Table 4. Changes in cardiovascular risk parameters between pre- and post-nitobr

Variable	Baseline	6th month	Change	Change (%)	<i>P</i> -value
SBP (mmHg)	135.6 ± 16.4	121.7 ± 10.1	-13.9 ± 17.2	-9.2 ± 12.7	0.001
DBP (mmHg)	82.4 ± 12.3	72.1 ± 8.2	-10.3 ±12.2	-11.1 ± 14.4	0.001
Fasting glucose (mg/dl)	102.5 ± 16.6	88.2 ± 8.2	-14.3 ± 16.1	-12.3 ± 13.1	0.001
Insulin (μU/ml)	21.3 ± 12.7	6.6 ± 3.0	-14.7 ± 12.9	-62.3 ± 19.2	0.001
HOMA-IR	5.43 ± 3.62	1.44 ± 0.71	-4.0 ± 3.7	-66.2 ± 19.2	0.001
Total cholesterol (mg/dl)	210.2 ± 45.4	155.6 ± 30.4	-54.6 ± 36.7	-24.7 ± 13.4	0.001
LDL cholesterol (mg/dl)	131.1 ± 39.7	93.8 ± 21.5	-39.9 ± 32.9	-27.0 ± 17.3	0.001
HDL cholesterol (mg/dl)	43.3 ± 10.9	46.8 ± 13.1	+ 3.5 ± 10.7	+ 9.9 ± 21.4	NS
Triglycerides (mg/dl)	170.1 ± 82.9	91.3 ± 22.7	-78.1 ± 77.5	-37.8 ± 20.5	0.001

SBP = systolic blood pressure; DBP = diastolic blood pressure; HOMA-IR = insulin resistance index calculated by Homeostasis Model Assessment.

the operative technique utilized was similar in all patients, the inter-individual variability of weight loss after surgery could be due to energy expenditure prior to surgery, postoperative reduction of REE, reduction of energy intake following surgery, or to a combination of these factors.

Predictors of Weight Loss

In contradiction to what was reported in other studies, in our series the initial parameters, weight, BMI, REE and RQ, were not predictors of weight loss in the simple correlation analysis. On the other hand, baseline fasting insulin and HOMA index were indeed predictors. However, in the multivariate analysis model, the baseline parameters, weight, waist circumference and fasting insulin level were the best predictors of weight reduction, explaining a combined 45% of the weight loss. The impact of hyperinsulinemia as a predictor for the best results, in our series, could be attributed to the drastic anatomical changes induced by the surgical technique, with modification of the levels of intestinal mediators that would result in lower insulin resistance. Therefore,

Carrasco et al

those patients with higher insulin resistance could obtain greater benefit from this surgical procedure.³¹ When incorporating the baseline REE/FFM ratio in the multivariate analysis, we can explain 54% of the variability in weight loss, without obtaining better predicting results by adding other variables.

In our patients, the physical activity variables were not considered predictors for weight loss, similar to what has been reported by other authors.^{7,32} Energy intake, whose reduction in the postoperative period has been reported as a fundamental factor that explains the weight loss after RYGBP,⁹ was not found to be a good predictor of results after RYGBP in our study. This scarce correlation cannot be explained by a low inter-individual variability in the energy intake after RYGBP, because of the great dispersion of data of the nutritional intake at 6 months postoperatively (Table 3).

Changes in Resting Energy Expenditure and Lipid Oxidation

Many studies have reported that weight loss in obese subjects is associated to a reduction in REE, which can limit weight loss in the long-term.³³ However, a 440 kcal/day decrease in REE 6 months after RYGBP, while significant, was found by Carey et al³⁴ not to be greater than expected due to the decrease in fat-free mass. In the present study, the reduction of weight was associated with a significant decrease in REE/FFM ratio, showing a greater decrease in patients with a high baseline REE. In this matter, Das et al⁷ postulated that a greater energy intake in subjects with higher values of weight, would behave in a compensatory form with a higher energy expenditure, and upon having a restricted nutritional intake in the postoperative state, this compensation would disappear, observing a greater reduction in REE in these patients. The latter agrees with the results obtained in our series, because the initial REE showed a significant correlation with the preoperative nutritional intake.

Metabolic adaptation in the patients studied, in which the REE expressed by fat-free mass at 6 months after RYGBP was 83 kcal/day less than the expected value, could be attributed also to changes in the levels of mediators such as thyroid hormones, leptin, sympathetic stimuli, among other factors that were not evaluated in our study. The decrease in the nutritional intake that allows for a greater energy deficit could be related to the level of metabolic adaptation. However, in our series, the correlation between changes in energy intake and changes in REE was not found to be statistically significant. Neither did we observe a relation between the level of physical activity surveyed 6 months after the operation and the changes in REE.

The positive correlation between the changes in REE and the reduction in body fat suggests that this metabolic adaptation is probably influenced by the reduction in adipose tissue and variations in plasma levels of mediators derived from this tissue, such as leptin.

Changes in Cardiovascular Risk Factors

In the present study, the diabetic patients normalized their fasting glycemia 6 months after RYGBP, with cessation of the antidiabetic agents and a significant improvement in the levels of fasting insulin and HOMA index. These results are similar to those observed in other studies,^{4,35} and it has been postulated that the results of the diabetic group showed certain dependence on the surgical procedure, and that although the weight loss is important after RYGBP, the recovery from diabetes could be related to other changes in intestinal peptides (GLP-1, PYY₃₋₃₆, etc.) related to the gastric bypass that precede the massive reduction in weight.^{36,37} However, the study of Ballantyne et al³¹ did not find a different response in the short-term between gastric banding and RYGBP.

With respect to the dyslipidemia, in the present study we observed a remission in all patients, concerning the levels of total cholesterol, LDL-cholesterol and trigly-cerides, as reported by Buchwald et al.⁴ However, the HDL-cholesterol increased, without statistical significance. In the latter meta-analysis,⁴ a more frequent improvement of dyslipidemia was noted in those studies of malabsorptive procedures and combined restrictive-malabsorptive procedures.

With regard to hypertension, in our series there was a high percentage of remission in those patients who had this diagnosis at baseline, and a noticeable reduction in blood pressure values in the remainder of them. As opposed to what occurs with type 2 diabetes and dyslipidemia, the reduction in blood pressure appears to be independent of the surgical procedure employed.⁴ This finding suggests that improvement in metabolic conditions is proportionally dependant on weight loss and on the reversal of insulin resistance that also improves in these co-morbidities. This could be confirmed by the correlation obtained among percentage of loss of the initial weight and changes in insulinemia and HOMA index. On the other hand, the absence of correlation between cardiovascular risk parameters and changes in body fat, could be explained by the considerable weight loss that occurred in all patients, where we observed a maximal improvement in co-morbidities, without having additional benefits with a greater weight loss.

Changes in Nutritional Intake and Physical Activity

As discussed previously, caloric intake significantly influences weight loss,9 and in this matter there exists great variability among individuals, suggesting the presence of influencing factors prior to the surgery that could modify this intake. In the present study, we emphasize the significant decrease of daily energy intake in the patients. It is important to note that the postoperative evaluation of these patients was carried out at 6 months after surgery; therefore, the patients still kept a low calorie diet, and were not fulfilling their estimated requirements in most cases (61% and 82% of the energy and protein requirements, respectively). Although a decrease of lipid and carbohydrate intakes (g/d) was observed compared to preoperative, which was statistically significant (not like the change in the proportion of these macronutrients in the total energy intake), it would be interesting to observe patients who underwent surgery compared to medical treatment in future studies. In this aspect, Klem et al¹⁶ have reported that in morbidly obese patients who achieved a reduction in weight with medical treatment, there was a lower fat intake and a higher carbohydrate intake than patients treated surgically. The decrease in carbohydrate intake after RYGBP is related to the risk of dumping syndrome and the discomfort that this implies after intake of simple sugars.

With respect to physical activity, patients in our series presented low levels of physical activity prior to treatment and a significant increase after surgery, which probably could contribute to the maintenance of weight loss in the long term. Another study reported no change in physical activity level after surgery.³² Surprisingly, there was a significant positive correla-

tion between the increase in the activity factor and the changes in REE, which would be discordant with that expected; however, it is necessary to note that both variables are related to a third variable: weight loss.

Conclusion

In our study of morbidly obese patients who underwent a RYGBP, we conclude that the operation induces a significant weight loss in a short time period, provided fundamentally by body fat loss and determined by a smaller energy intake especially of carbohydrates and fats. In this series, the metabolic adaptation that accompanies the weight loss, was related mostly to loss of fat mass rather than to decrease in fat-free mass. In accordance with other studies, RYGBP induces a significant improvement in cardiovascular risk factors, and results in an increase in physical activity and a decrease in sedentary behavior. According to our results, the presence of baseline hyperinsulinemia and initially high levels of HOMA index, were the best single predictors for the results of the weight loss after RYGBP.

References

- 1. WHO. Obesity: preventing and management the global epidemic. Report of a WHO Consultation on Obesity. Geneva: WHO, 1997.
- 2. Drenick EJ, Bale GS, Seltzer F et al. Excessive mortality and causes of death in morbidity obese men. JAMA 1980; 243: 443-5.
- 3. Ministerio de Salud. Encuesta Nacional de Salud, 2003. http://epi.minsal.cl /epi/html/ invest/ ENS/ENS_mayo 2004.pdf. Minsal, Chile.
- Buchwald H, Avidor Y, Braunwald E et al. Bariatric Surgery: a systematic review and Meta-analysis. JAMA 2004; 292: 1724-37.
- 5. Maggard MA, Shugarman LR, Suttorp M et al. Meta-Analysis: surgical treatment of obesity. Ann Intern Med. 2005; 142: 547-59.
- Bocchieri-Ricciardi LE, Chen EY, Munoz D et al. Presurgery binge eating status: effect on eating behavior and weight outcome after gastric bypass. Obes Surg 2006; 16: 1198-204.
- 7. Das SK, Roberts SB, McCrory MA et al. Long-term changes in energy expenditure and body composition

after massive weight loss induced by gastric bypass surgery. Am J Clin Nutr 2003; 78: 22-30.

- Bloomston M, Zervos EE, Camps MA et al. Outcome following bariatric surgery in super versus morbidly obese patients: does weight matter? Obes Surg 1997; 7: 414-9.
- 9. Bobbioni-Harsch E, Huber O, Morel P et al. Factors influencing energy intake and body weight loss after gastric bypass. Eur J Clin Nutr 2002; 56: 551-6.
- Pekkarinen T, Koskela K, Huikuri K et al. Long-term results of gastroplasty for morbid obesity: Binge eating as a predictor of poor outcome. Obes Surg 1994; 4: 248-55.
- 11. Guisado JA, Vaz FJ, Alarcón J et al. Psychopathological status and interpersonal functioning following weight loss in morbidly obese patients undergoing bariatric surgery. Obes Surg 2002; 12: 835-40.
- 12. Clark MM, Balsiger BM, Sletten CD et al. Psychosocial factors and 2 years outcome following bariatric surgery for weight loss. Obes Surg 2003; 13: 739-45.
- Ma Y, Pagoto SL, Olendzki BC et al. Predictors of weight status following laparoscopic gastric bypass. Obes Surg 2006; 16: 1227-31.
- 14. Silver HJ, Torquati A, Jensen GL et al. Weight, dietary and physical activity behaviors two years after gastric bypass. Obes Surg 2006; 16: 859-64.
- Pasman WJ, Saris WHM, Westerterp-Plantenga MS. Predictors of weight maintenance. Obes Res 1999; 7: 43-50.
- 16. Klem ML, Wing RR, Chang CH et al. A case-control study of successful maintenance of a substantial weight loss: individuals who lost weight through surgery versus those who lost weight through non-surgical means. Int J Obes 2000; 24: 573-9.
- Shah M, Miller DS, Geissler CA. Lower metabolic rates of post-obese versus lean woman: thermogenesis, basal metabolic rate and genetics. Eur J Clin Nutr 1988; 42: 741-52.
- Zurlo F, Lillioja S, Esposito-Del Puente A et al. Low ratio of fat to carbohydrate oxidation as predictor of weight gain: study of 24-h RQ. Am J Physiol 1990; 259: E650-E657.
- Csendes A, Burdiles P, Papapietro K et al. Results of gastric bypass plus resection of the distal excluded gastric segment in patients with morbid obesity. J Gastrointest Surg 2005; 9:121-31.
- 20. Lohman TG: Anthropometry and body composition. In: Lohman TG, Roche AF, Martorell R, eds. Anthropometric Standardization Reference Manual. Champaign, IL: Human Kinetics, 1988: 125-9.
- Schoeller DA. Hydrometry. In: Roche AF, Heymsfield SB, Lohman TG, eds. Human Body Composition, Champaign, IL: Human Kinetics, 1996: 25-44.
- 22. Das SK, Roberts SB, Kehayias JJ et al. Body composition assessment in extreme obesity and after massive weight loss induced by gastric bypass surgery. Am J Physiol Endocrinol Metab 2003; 284: E1080-E1088.

- 23. Godin G, Shephard RJ. A simple method to assess exercise behavior in the community. Can J Appl Sport Sci 1985; 10: 141-6.
- 24. Livingstone MB, Prentice AM, Coward WA et al. Simultaneous measurement of free-living energy expenditure by doubly labelled water method and heart rate monitoring. Am J Clin Nutr 1990; 52: 59-65.
- 25. Bray GA. Nutrition, diet, and treatment of overweight. In: Contemporary Diagnosis and Management of Obesity. Newton, Pennsylvania: Handbooks in Health Care Co. 1998: 192-224.
- 26. Pories WJ, Flickinger EG, Meehleim D et al. The effectiveness of gastric bypass over gastric partition in morbid obesity: consequence of distal gastric and duodenal exclusion. Ann Surg 1982; 196: 389-99.
- Hall JC, Watts JM, O'Brien PE et al. Gastric surgery for morbid obesity: the Adelaide study. Ann Surg 1990; 211: 419-27.
- 28. Flancbaum L, Choban P, Bradley L et al. Changes in measured resting energy expenditure after Roux en Y gastric bypass for clinically severe obesity. Surgery 1997; 122: 943-9.
- 29. Donnely JE, Jacobsen DJ, Whatley JE. Influence of degree of obesity on loss of fat-free mass during very-low-energy diets. Am J Clin Nutr 1994; 60: 874-8.
- Foster GD, Wadden TA, Swain RM et al. Changes in resting energy expenditure after weight loss in obese African and white women. Am J Clin Nutr 1999; 69: 13-7.
- 31. Ballantyne GH, Farkas D, Laker S et al. Short-term changes in insulin resistance following weight loss surgery for morbid obesity: laparoscopic adjustable gastric banding versus laparoscopic Roux-en-Y gastric bypass. Obes Surg 2006; 16: 1189-97.
- 32. van Gemert WG, Westerterp KR, van Acker BA et al. Energy substrate and protein metabolism in morbid obesity before, during and after massive weight loss. Int J Obes 2000; 24: 711-8.
- Astrup A, Gotzsche PC, van de Werken K et al. Metaanalysis of resting metabolic rate in formerly obese subjects. Am J Clin Nutr 1999; 69: 1117-22.
- 34. Carey DG, Pliego GJ, Raymond RL et al. Body composition and metabolic changes following bariatric surgery: effects on fat mass, lean mass and basal metabolic rate. Obes Surg 2006; 16: 469-77.
- 35. Schauer PR, Burguera B, Ikramuddin S et al. Effect of laparoscopic Roux en Y gastric bypass on type 2 diabetes mellitus. Ann Surg 2003; 238: 467-84.
- 36. Patriti A, Facchiano E, Sanna A et al. The enteroinsular axis and the recovery from type 2 diabetes after bariatric surgery. Obes Surg 2004; 14: 840-8.
- Wickremesekera K, Miller G, De Silva T et al. Loss of insulin resistance after Roux-en-Y gastric bypass surgery: A time course study. Obes Surg 2005; 15: 474-81.

(Received September 20, 2006; accepted November 7, 2006)