MESTRADO INTEGRADO EM MEDICINA

Impact of basal metabolic rate on the results of bariatric surgery

José Miguel de Almeida Gonçalves

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ARTIGO ORIGINAL

Dissertação de Mestrado Integrado em Medicina

Instituto de Ciências Biomédicas Abel Salazar

Universidade do Porto

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Dedicatória

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Resumo

Introdução: A cirurgia bariátrica é o tratamento de eleição para obesidade severa e suas comorbilidades, porém os seus resultados em termos de perda ponderal e remissão de patologias associadas são heterogéneos e difíceis de prever. As taxas metabólicas basais (TMB) pré-operatória e pós-operatória poderão influenciar estes desfechos, mas a sua relação ainda não está bem estabelecida.

Objetivos: Avaliar se a TMB pré-operatória influencia os resultados clínicos no primeiro ano após a cirurgia, em termos de perda de peso e melhoria das comorbilidades relacionadas com a obesidade, bem como avaliar se a TMB 1 ano após a cirurgia bariátrica se correlaciona com os resultados alcançados.

Métodos: Doentes com obesidade candidatos a cirurgia de bypass gástrico (N=88) foram recrutados para este estudo e seguidos durante 12 meses. O gasto energético em repouso (GER), como medida substituta da TMB, foi avaliado por calorimetria indireta (ReeVue) antes (n=88) e 12 meses após a cirurgia (n=14). Foram recolhidos dados antropométricos, clínicos e analíticos antes da cirurgia e aos 6 e 12 meses após a cirurgia.

Resultados: A TMB medida antes da cirurgia não previu a perda de peso total (PPT) ou a remissão de comorbilidades um ano após a cirurgia. A TMB medida foi superior à TMB estimada, embora só tenha sido estatisticamente significativa após a cirurgia (6,82 ± 0,40 kJoule/dia vs. 5,73 ± 0,11 kJoule/dia, p = 0,024). Apesar da redução observada da TMB medida aos 12 meses após a cirurgia, esta não foi estatisticamente significativa. Os doentes que apresentaram uma diminuição da TMB apresentaram uma PPT superior aos que apresentaram um aumento (39,96 ± 1,87% vs. 33,25 ± 1,37%; p = 0,0451).

Conclusão: O GER medido no pré-operatório não tem impacto na perda de peso ou na remissão das comorbilidades relacionadas com a obesidade após o procedimento. A observação de que os pacientes com uma diminuição pós-operatória do REE tinham um TWL maior do que aqueles com um aumento suporta que as alterações no REE são mais provavelmente uma consequência da perda de peso do que a sua causa. Além disso, o GER medido foi superior ao estimado pela equação de Harris-Benedict, sugerindo que as equações preditivas podem subestimar o gasto energético em indivíduos com obesidade submetidos a cirurgia bariátrica e sobrestimar a extensão da redução do GER devido à termogénese adaptativa (TA).

Palavras-chave: Obesidade, Cirurgia bariátrica, Bypass gástrico em Y de Roux, Taxa metabólica basal, Gasto energético em repouso, Perda de peso, Comorbilidades da obesidade.

Abstract

Introduction: Bariatric surgery is the treatment of choice for severe obesity and its comorbidities, but its results in weight loss and remission of metabolic conditions are heterogeneous and difficult to predict. Preoperative and postoperative basal metabolic rate (BMR) may influence these outcomes, but their relationship is not yet well established.

Aim: To assess whether pre-operative BMR influences clinical outcomes in the first year after surgery, in terms of weight loss and improvement in obesity-related comorbidities, as well as to evaluate whether the BMR 1 year after bariatric surgery correlates with the outcomes achieved.

Methods: Patients with obesity who were candidates for gastric bypass surgery (N=88) were recruited to this study and followed up for 12 months. Resting expenditure energy (REE), as a surrogate measure of BMR was assessed by indirect calorimetry (ReeVue) before (n=88) and 12 months after surgery (n=14). Anthropometric, clinical, and analytical data were collected before surgery and at 6 and 12 months after surgery.

Results: REE measured before surgery did not predict total weight loss (TWL) or remission of comorbidities one year after surgery. Measured REE was higher than the Estimated REE, although this was only statistically significant after surgery (6.82 ± 0.40 kJoule/day vs. 5.73 ± 0.11 kJoule/day, p=0.024). Despite the reduction in Measured REE observed 12 months after surgery, this reduction was not statistically significant. Patients who exhibited a decrease in REE had a greater TWL than those who exhibited an increase (39.96 ± 1.87% vs. 33.25 ± 1.37%; p=0.0451).

Conclusion: Pre-operative measured REE does not impact on weight loss or remission of obesity-related comorbidities after the procedure. The observation that patients with a postoperative decrease in REE had greater TWL than those with an increase supports that changes in REE are more likely a consequence of weight loss rather than its cause. Additionally, measured REE was higher than that estimated by the Harris-Benedict equation, suggesting that predictive equations may underestimate energy expenditure in individuals with obesity submitted to bariatric surgery, and overstate the extent of REE reduction due to adaptive thermogenesis (AT).

Keywords: Obesity, Bariatric surgery, Roux-en-Y gastric bypass, Basal metabolic rate, Resting energy expenditure, Weight loss, Obesity comorbidities.

List of Abbreviations

AT: adaptive thermogenesis
ALT: alanine transaminase
AST: aspartate aminotransferase
BMR: basal metabolic rate
BMI: body mass index
EBMIL: excess BMI loss
FeO_2: fraction of expired oxygen
GGT: gamma-glutamyl transferase
Hb: hemoglobin
HbA1c: glycated hemoglobin
HDL-C: high density lipoprotein cholesterol
HT: hypertension
Htc: hematocrit
LDL-C: low density lipoprotein cholesterol
MCHC: mean cellular hemoglobin concentration
MS: metabolic syndrome
NA: not applicable
O_2: oxygen
PDW: platelet distribution width
PTH: parathyroid hormone
RDW: red cell distribution width
REE: resting energy expenditure
T2D: type 2 diabetes mellitus
T4L: thyroxine
TEE: total energy expenditure
TG: triglycerides
TSH: thyroid-stimulating hormone
TWL: total weight loss
VLDL-C: very-low density lipoprotein
VitB12: vitamin B12
VitD: vitamin D
VO_2: volume of oxygen
NLR: neutrophil-lymphocyte ratio
PLR: platelet-lymphocyte ratio

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I. Introduction

Obesity is a chronic, complex, and multifactorial disease that represents one of the main public health problems in most societies nowadays (1,2). There has been a growing increase in the prevalence of obesity, accompanied by a significant increase in obesity-related comorbidities, such as type 2 diabetes mellitus (T2D), hypertension (HT), and metabolic syndrome (MS), as well as the socio-economic impact they have on health systems (2–4).

Bariatric surgery is a safe and effective intervention for the treatment of obesity and its comorbidities (5–8). It is currently the most effective therapeutic approach for inducing sustained weight loss in individuals with severe obesity (5,7). In addition to a significant reduction in body weight, bariatric surgical interventions have been shown to lead to considerable improvements of obesity-related comorbidities, especially the resolution or improvement of T2D (5–7).

However, despite the high efficacy rate of bariatric surgery, the long-term clinical outcomes concerning weight loss and improvement of obesity-related comorbidities can be very heterogeneous and difficult to foresee, therefore, there is an unmet need to identify factors to enable the improvement of the prediction scores currently available (9–13). This would enable the optimization of the selection of candidates for bariatric surgery, allow for a better management of patient and physician expectations, and a more personalized approach in the pursuit of precision medicine (10,13).

The basal metabolic rate (BMR) is an indicator of the daily energy expenditure required to maintain vital functions at rest and may therefore play an important role in the weight loss outcomes of bariatric surgery, either by promoting or hindering weight loss (14). Resting energy expenditure (REE) and BMR are often used interchangeably, though they are slightly different. REE is measured under less stringent conditions than BMR, which typically requires a highly controlled environment like metabolic chambers, and therefore is usually 10% higher (15,16). Indeed, some studies suggest that REE can directly influence the magnitude of weight loss after surgery, impacting energy balance and, consequently, clinical outcomes (17,18). Thus, individuals with a higher preoperatory REE have been hypothesized to have a more favorable weight loss response to bariatric surgery, in contrast to those with a lower REE (19). It should be noted that the negative energy balance inherent in weight loss leads to a physiological and adaptive reduction in REE, which could make it difficult to continue the weight loss process and even contribute to weight regain (20). However, previous studies have shown that the adaptive variation in REE observed after bariatric surgery, often does not reflect the reduction expected for the weight loss achieved, nor does it seem to be directly explained by the fat and lean mass reductions, which suggests that other factors may be involved and contribute to the weight loss mechanisms underlying the surgical intervention, although the available data is scarce (21–23).

To contribute to filling the current knowledge gaps, this study had two main objectives: firstly, to assess whether pre-operative REE influences clinical outcomes in the first year after surgery, in terms of weight loss and improvement in obesity-related comorbidities, secondly, to evaluate whether the REE 1 year after bariatric surgery correlated with the outcomes achieved.

II. Methods

2.1 Study population

The study participants were recruited among patients undergoing gastric bypass bariatric surgery at a single public academic center who met the criteria for bariatric surgery treatment. These included having a body mass index (BMI) higher than 40 kg/m^2 or a BMI higher than 35 kg/m^2 in the presence of obesity-related comorbidities, having documented failure to non-invasive weight loss interventions, and no contraindications for surgical intervention. Patients undergoing bariatric surgery procedures other than gastric bypass were excluded from this study.

Patients who agreed to take part in the study (n=88) underwent a REE assessment preoperatively and 1 year after the intervention (n=14) in addition to the standard practice pre- and post-operative anthropometric and analytic evaluations, which included a complete blood count, fasting blood glucose, glycated hemoglobin (HbA1c), lipid profile, creatinine, urea, albumin and total proteins serum levels.

This study was approved by the institutional ethics review board (approval number: CES_90/2024), and no study procedure was conducted before its approval, nor without the participant's signed consent.

2.2 Data collection

Demographic and clinical data were recorded, including gender, date of birth, date of study visits, height, weight, obesity comorbidities, current medications, date of surgery, type of bariatric surgery, size of the gastric bypass loop, and short- and long-term complications after the surgery.

Anthropometric data was used to calculate BMI, percentage of excess BMI loss (EBMIL) and percentage of total weight loss (TWL) using the mathematical formula presented in **Supplementary Table I**.

Laboratory tests included hemoglobin (Hb), hematocrit (Htc), mean cellular hemoglobin concentration (MCHC), red cell distribution width (RDW), platelet distribution width (PDW), leukocytes, neutrophils, lymphocytes, leukocytes, neutrophils, lymphocytes, iron, ferritin, transferrin saturation rate, vitamin B12 (VitB12), folic acid, glucose, HbA1c, urea, creatinine, uric acid, total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), triglycerides (TG), bilirubin, alanine transaminase (AST), aspartate aminotransferase (ALT), gamma-glutamyl transferase (GGT), alkaline phosphatase, total proteins, transferrin, albumin, thyroid-stimulating hormone (TSH), thyroxine (T4), parathyroid hormone (PTH), insulin, C-peptide and vitamin D (vitD).

The REE was assessed as a surrogate measure of BMR using the “ReeVue Metabolic Analyzer” device, which provides “Measured REE (KJoule/day and Kcal/day)”, “Estimated REE (KJoule/day and Kcal/day)”, “Volume of oxygen (VO2) (ml/min)”, “VO2 (ml/kg/min)”, “Fraction of expired oxygen (FeO_2) (%)”, ‘Minute Volume (L/min)’, “Tidal Volume (ml)”, “Respiratory Rate (breaths/min)”.

The “ReeVue Metabolic Analyzer” assessment is conducted in a controlled setting, with the patient seated at rest, breathing normally through a disposable mouthpiece attached to a sampling tube with a filter, while wearing a nasal clip. The device performs automatic calibration before testing, followed by a 1-minute stabilization phase, and then respiratory data are collected for approximately 10 minutes.

The “Tidal Volume (ml)” parameter represents the tidal volume and is calculated by the airflow sensor. The “Respiratory Rate (breaths/min)” parameter corresponds to the count of respiratory cycles per minute. The “Minute Volume (L/min)” parameter represents the total volume of air per minute. The “FeO_2 (%)” parameter represents the fraction of O_2 in the exhaled air and is measured directly by the device's sensors. The “VO_2 (ml/min)” parameter represents the oxygen consumed per minute. The “VO_2 (ml/kg/min)” parameter corresponds to normalization according to body weight. The “Measured REE” parameter represents the resting energy expenditure value measured by the device using a formula developed by the manufacturers, which was adapted from the modified Weir equation (24). This algorithm is not public, but the official technical documentation from KORR Medical Technologies reveals that the “Constant” will vary according to a correction for the respective FeO_2 (%) measured(25). The “Estimated REE (Kcal/day)” parameter represents the resting energy expenditure calculated using the Harris-Benedict formula, which considers weight, height, age, and gender, with a small correction to adjust for the current population (26). The mathematical equations used to calculate these parameters are presented in Supplementary Table I.

After bariatric surgery, the patients were followed up by a multidisciplinary team comprising surgeons, endocrinologists, psychologists, and nutritionists. At 6 and 12 months after surgery, the patients were reassessed for anthropometric and laboratory parameters during routine appointments. Additionally, REE assessment was planned for the 12-month postoperative timepoint; however, at the time of data collection, it was still being performed, and data were only available for analysis for a reduced number of patients (n=14).

2.3 Statistical analysis

Patients were included in an anonymized database and characterized according to demographic and clinical variables.

Continuous variables are presented as mean ± standard error of the mean, while categorical variables are presented as absolute (n) and relative (%) frequencies. Comparisons of variables in the same patients before and after surgery were performed using paired analyses, specifically the paired t-test or the Wilcoxon signed-rank test, depending on the results of the Shapiro–Wilk test for normality.

For comparisons between independent groups, either the Independent Samples t-test or the Mann–Whitney U test was applied, based on the data distribution. Categorical variables were compared using the Chi-square (χ^2) test. Correlations between numerical variables were assessed using Pearson's or Spearman's bivariate correlation, as appropriate. Linear regression and binary logistic regression were used to evaluate associations with continuous and binary outcomes, respectively.

A $p < 0.05$ was considered statistically significant. All statistical analyses were conducted using IBM® SPSS® Statistics, version 30.0, and GraphPad Prism, version 10.4.1.

III. Results

3.1 Patient characteristics

The study participants (n=88) were predominantly females (n=75; 66%) vs. males (n=22; 25%), in parallel to the sex proportion attending the obesity clinic. At the time of surgery, patients' age was on average 44.28 ± 1.12 years, with a mean BMI of 39.28 ± 0.30 kg/m^2. Detailed patients´ characteristics are depicted in **Table I.**

3.2 Postoperative anthropometric, analytical, characteristics, and comorbidity outcomes

After bariatric surgery, there was a statistically significant reduction in body weight and BMI during the follow-up period (39.28 ± 0.30 kg/m^2 pre-operatively, 27.81 ± 0.30 kg/m^2 at 6 months, 24.92 ± 0.33 kg/m^2 at 12 months, p<0.001), yielding a TWL of 29.35 ± 0.57 % and 36.56 ± 0.75 % and EBMIL of 82.39 ± 1.97 % and 102.66 ± 2.49 % at 6 and 12 months after surgery, respectively. It should also be noted that over 97% and more than 98% of the participants achieved a TWL above 20% at 6 and 12 months after surgery, respectively. There was a reduction of obesity-related comorbidities, although not statistically significant. Since the patients underwent surgery over 25 months, patient evaluation is ongoing. Therefore, four patients have not yet reached the 6-month follow-up visit after surgery (n=4), and 22 patients have not reached the 12-month follow-up (n=84 at 6 months and n=66 at 12 months). Detailed data are depicted in **Table I**.

Twelve months after bariatric surgery, despite being within the normal ranges statistically significant reductions were observed in Hb (13.92 ± 0.13 g/dL vs 13.22 ± 0.14 g/dL; p<0.001), Htc (42.25 ± 0.33 % vs 39.88 ± 0.38 %; p<0.001), platelet counts (263.10 ± 5.32 ×10^3/µL vs 231.15 ± 5.85 ×10^3/µL; p<0.001), leucocytes (7.19 ± 0.18 10^9/L vs 5.91±0.18 10^9/L; p<<0.001), neutrophils (56.39 ± 0.77 % vs 52.10 ± 1.15 %; p<0.003), while there was a significant increase in lymphocyte percentage (34.05 ± 0.71% vs 37.85 ± 1.04; p=0.003), yielding a statistically significant decrease in neutrophil-lymphocyte ratio (NLR) (1.76 ± 0.06 vs 1.50 ± 0.08; p=0.002). There was a improvement in glucose, lipid and metabolic parameters, fasting glucose levels (95.05 ± 2.00 mg/dL vs 83.93 ± 0.83 mg/dL; p<0.001), TG (112.79 ± 5.23 mg/dL vs 69.70 ± 3.39 mg/dL; p<0.001), total cholesterol (180.13 ± 3.26 mg/dL vs 165.64 ± 4.23 mg/dL; p=0.009), HDL-C (48.13 ± 1.21 mg/dL vs 55.66 ± 2.49 mg/dL; p=0.002), calculated LDL-C (109.44 ± 2.98 mg/dL vs 96.04 ± 3.24 mg/dL; p=0.004), creatinine (0.74 ± 0.02 mg/dL vs 0.67 ± 0.02 mg/dL; p=0.004), uric acid (5.53 ± 0.15 mg/dL vs 4.34 ± 0.17 mg/dL; p<0.001), GGT (34.12 ± 2.09 U/L vs 21.27 ± 2.54 U/L; p<0.001), total protein (7.13 ± 0.04 vs 6.71 ± 0.05; p<0.001). Additionally, significant increases were noted in AST (24.24 ± 1.04 U/L vs 29.05 ± 1.34 U/L; p=0.001), iron (56.50 ± 9.50 µg/dL vs 97.08 ± 3.92 µg/dL, p=0.031, transferrin saturation (19.60 ± 4.70 % vs 35.60 ± 1.46 %;

p=0.027), and a borderline significant increase in VitB12 (280.50 ± 43.50 pg/mL vs525.67 ± 27.92 pg/mL; p=0.050) (**Table II**).

3.3 Preoperative and post-operative resting energy expenditure data

Before surgery, mean Measured REE numerically higher than the Estimated REE, although this difference was not statistically significant (8.07 ± 0.21 vs. 7.82 ± 0.14 kJ/day, p=0.285). Additionally, measured REE showed greater variability than estimated REE.

After surgery, only 14 patients have undergone REE reassessment so far. Measured REE was not significantly different when compared to the preoperative values (7.86 ± 0.45 kJoule/day preoperatively vs. 6.82 ± 0.40 kJoule/day at 12 months, p = 0.108), although Estimated REE was significantly reduced (7.27 ± 0.21 kJoule/day preoperatively vs. 5.73 ± 0.11 kJoule/day at 12 months after surgery, p < 0.001). Furthermore, different trajectories for Measured REE were noticed, as some patients depicted a decrease of Measured REE (n=8), while it increased in others (n=6) (Figure 1). Additionally, after surgery, Measured REE was significantly higher than Estimated REE (6.82 ± 0.40 kJoule/day vs. 5.73 ± 0.11 kJoule/day, p=0.024) (**Figure 2**). Additionally, there was a significant increase in VO2 (2.67 ±0.13 ml/kg/min vs 3.58 ±0.21 ml/kg/min, p=0.003). The entire dataset is presented in **Table III**.

3.4 Resting energy expenditure as a predictor of weight loss

A statistically significant association was observed between pre-operative FeO_2 and TWL at 6 months after surgery, with a regression coefficient of 1.581 (95% CI: 0.145–3.018; p = 0.031). This result suggests that higher FeO_2 values before surgery are associated with a greater weight loss at this time point after surgery. No statistically significant associations were identified between the remaining parameters, measured or estimated REE, and weight loss six or twelve months after surgery (**Table IV)**.

Twelve months after surgery, TWL was significantly greater in patients who experienced a reduction in Measured REE than those in whom an increase in Measured REE was noticed (39.96 ± 1.87% vs. 33.25 ± 1.37%; p=0.0451) (**Figure 3**).

3.5 Resting energy expenditure and obesity-related comorbidities

Before bariatric surgery, patients with T2D Estimated REE was significantly lower than that of patients without the condition (7.07 ± 0.29 kJ/day in T2D vs. 7.90 ± 0.15 kJ/day in non-T2D, p = 0.045). This group also showed significant differences in tidal volume (431.00 ± 56.24 ml in T2D vs. 656.14 ± 32.75 ml in non-T2D, p = 0.008) and respiratory rate (18.76 ± 1.33 breaths/min in T2D vs. 14.85 ± 0.51

breaths/min in non-T2D, p=0.012). No other statistically significant differences were found in the remaining groups or parameters. Data are presented in **Table V**.

Among the 14 patients who underwent REE assessment 12 months after surgery, 3 out of 4 patients with T2D experienced remission, 2 out of 6 patients with HT showed remission, while none of the 6 patients with dyslipidemia had disease remission. To evaluate whether pre-operative REE influenced the remission of obesity-related comorbidities 12 months after surgery a logistic regression was made. However, no statistically significant associations were found **Table VI**.

Discussion

The primary objective of this study was to assess whether the basal metabolic rate before bariatric surgery influences its short-term outcomes, in terms of weight loss and improvement in obesity-related comorbidities.

Bariatric surgery provides significant and sustained weight loss over the long term in patients with severe obesity. Weight loss typically reaches its maximum 1 or 2 years after bariatric surgery, followed by a non-significant weight gain and maintenance over time. The average TWL reported up to 12 months after gastric bypass surgery varies between 25% and 34%, and the TWL within the first year after surgery was shown to be a good predictor of long-term weight loss maintenance (27–31). So the vast majority of the patients in this study achieved an anticipated TWL above 20%, defined as optimal (32). It should be mentioned that the weight loss attained after bariatric interventions is not only the result of the procedure, but also influenced by various factors, such as patient characteristics, nutritional choices, physical activity, mental health, and multidisciplinary team experience at addressing their individual needs (33–37).

Besides weight loss, bariatric surgery was also shown to be very effective at inducing considerable improvement or even remission of obesity-related comorbidities, such as T2D, HT, and dyslipidemia (38–41) as we were able to witness in this study. Moreover, subtle shifts in circulating parameters illustrate the complex readjustments of biological processes that accompany bariatric surgery-induced weight loss, including the decrease of the inflammatory state, improvement of insulin resistance, and adaptive thermogenesis (AT) to weight loss. Indeed, obesity is known for being associated with a chronic low-grade inflammatory state, with occasional secondary thrombocytosis (42). There is also evidence that weight loss after bariatric surgery improves systemic inflammation (43). So, the decrease in platelet number after surgery observed in our study suggests a reduction in systemic inflammation and prothrombotic state, as previously advocated by other studies (44,45). The decrease in NLR ratios, which serve as surrogates of systemic inflammation, further reinforces this hypothesis (46–48). Additionally, weight loss and particularly loss of visceral fat decreases insulin resistance, and glucose and triglyceride levels are also expected to decrease, as a consequence of enhanced insulin sensitivity and reduced hepatic lipogenesis, as observed and extensively previously reported (49–51).

Additionally, a significant increase in AST levels within the normal limits was observed. Although this has not been detailed by previous studies, given the transaminases' roles in amino-acid synthesis and catabolism, the observed increase is likely to reflect an upregulation of transaminase activity, which is consistent with increased amino acid catabolism following postoperative changes in body composition(52).

Twelve months after bariatric surgery, patients showed significant hematological changes despite adherence to supplementation, likely related to altered nutrient absorption and reduced gastric acid production. In contrast, improvements in biochemical markers such as iron, transferrin saturation, and vit B12 indicate effective postoperative micronutrient supplementation (53). Furthermore, patients exhibited marked improvements in metabolic and hepatic function, as well as reductions in creatinine and uric acid. Favorable shifts in lipid profiles, namely lower total and LDL-C calculated and increased HDL-C, reflecting improved cardiovascular risk profile (54,55), while a slight reduction in total protein may indicate shifts in nutritional status or protein metabolism, common in the postoperative setting (56,57).

REE plays an important role in body weight regulation, which needs to be taken into account when interpreting conditions associated with shifts in energy balance. The assessment of REE in routine clinical practice often relies on prediction formulas, which do not require equipment that is unavailable in most settings, rather than its actual measurement. However, in people with severe obesity, significant differences between estimated and measured REE values have been reported, with a tendency for predictive equations, especially Harris-Benedict equation, to overestimate REE, although the opposite has also been shown (58–64). Therefore, it is not surprising that in our study, patients before bariatric surgery had a measured REE numerically higher and more heterogeneous than the estimated REE. Despite the difference between measured and estimated REE in people with obesity not being statistically significant, this difference is clinically relevant, since estimated REE is used to calculate the individual caloric needs for dietary plans. Thus, underestimating REE can have important implications, as this results in the prescription of exceedingly restricted diets, which can be responsible for low adherence and greater loss of lean mass, with a negative impact on the efficacy of the intervention (65).

In our study, we were unable to document any significant association between pre-operative measured REE and TWL at 6 and 12 months after surgery, suggesting that pre-operative REE is not useful at predicting TWL, as previously suggested (66,67).

Although short-term significant reductions in both measured and estimated REE after gastric bypass have been widely reported (68–72) and despite estimated REE being significantly lower, we found no significant difference between measured REE before and 12 months after surgery. Furthermore, the difference between measured and estimated REE became statistically significant, indicating that REE did not decline as predicted, which reinforces the hypothesis of AT being modified after bariatric surgery. Moreover, it should be noted that a greater dispersion in the measured REE values was also observed after surgery, suggesting an individual variability that is not captured by the predictive equation (21,73). Additionally, a significant increase in VO2 (ml/kg/min) was observed after surgery, which could be related to the improvement in functional capacity relative to body weight, as well as the alleviation of obesity-related mechanical restrictions on diaphragmatic movement described in respiratory function studies (74–76).

In this study, we evaluated whether the REE variation influenced TWL, given the different trajectories observed over time. After surgery, patients who exhibited a decrease in REE achieved greater TWL compared to those whose REE increased. This suggests that REE is more likely to reflect TWL than contribute to it. REE is predominantly conditioned by AT that accompanies weight loss. As such, it is not entirely surprising that those patients who lose more weight present a greater AT and therefore lower REE in line with what was shown for non-surgical weight loss at “The Biggest Loser” study (73).

This study also evaluated whether preoperative REE correlated with the presence of obesity-related comorbidities, namely T2D, HT, and dyslipidemia. No significant correlations were found between Measured REE, before surgery, and the abovementioned obesity-comorbidities. Although individuals with T2D presented significantly lower Estimated REE, there was no difference in Measured REE. The relationship between preoperative REE and comorbidities remission 12 months after surgery was also sought; however, no significant associations were found, although this could have been hampered by the number of individuals available at each subgroup for the analysis.

This study has several limitations that should be acknowledged. Firstly, the small sample size reduces the generalizability of the findings and limits the statistical power of the analysis. Moreover, not all participants completed the scheduled follow-ups, as four patients did not reach the 6-month follow-up point and 22 did not complete the 12-month evaluation, which resulted in incomplete longitudinal data for analysis. Consequently, the number of patients who underwent REE assessment was limited to 14 individuals, which restricts the strength of metabolic inferences. Another important methodological limitation was the fact that body composition, lifestyle, and physical activity, including exercise levels, were not directly evaluated, which limits our ability to assess their contribution to weight loss and REE variation. Moreover, it was not possible to conduct subgroup analysis for the type of gastric bypass technique performed, namely according to the length of the biliopancreatic limb, which may have introduced additional variability in the outcomes observed.

Despite the abovementioned limitations, the present study revealed relevant findings that justify and encourage future in-depth investigations. As a first step, it would be valuable to explore the outcomes observed in this preliminary study by completing the longitudinal study to increase the sample size, implementing strategies to minimize loss to follow-up, and continuing to assess all relevant parameters. In parallel, it would be of interest to integrate emerging technologies, such as smartwatches and advanced wearables, to enable continuous and personalized monitoring of energy expenditure, particularly in contexts of exercise, physical activity, and occupational settings~~.~~ Additionally, incorporating body composition data could allow for correlations with lean and fat mass to be explored, along with analytical parameters, which may provide further insight into individual metabolic responses. In this context, this research will enhance our understanding of the role of REE in determining the outcomes of bariatric surgery, which could support the development of predictive outcome models to enhance the personalization, individualization, and optimization of obesity treatment and its related comorbidities. Ultimately, the inclusion of machine learning algorithms to model these variables may lead to the development of robust predictive tools, capable of guiding personalized clinical decision-making and optimizing the bariatric surgery long-term outcomes.

Conclusion

Our study showed that measured REE in patients with obesity before bariatric surgery does not impact on weight loss or remission of obesity-related comorbidities after the procedure. Additionally, the observation that patients with a postoperative decrease in REE had greater TWL than those with an increase supports that changes in REE are more likely a consequence of weight loss rather than its cause. Moreover, the results obtained in this study suggest that classical formulas Harris Benedict equation may underestimate energy expenditure in individuals with obesity, as well as after weight loss induced by bariatric surgery, and overstate the extent of REE reduction due to AT.

Appendix

Table I- Anthropometric features and obesity-related comorbidities before and after surgery

	0M	6 Months	12 Months	p#
n	88	84	66	NA
Weight, kg	105.66 ± 1.44	74.68 ± 1.22	66.48 ± 1.27	p<0.001
BMI, kg/m^2	39.28 ± 0.30	27.81 ± 0.30	24.92 ± 0.33	p<0.001
EBMIL (%)	NA	82.39 ± 1.97	102.66 ± 2.49	p<0.001
TWL (%)	NA	29.35 ± 0.57	36.56 ± 0.75	p<0.001
TWL >20%, n (%)	NA	82 (97.6)	65 (98.5)	-
T2D n. (%)	9 (10.2)	7 (8.3)	4 (6.1)	0.671
Dyslipidemia n. (%)	30 (34.1)	28 (33.3)	18 (27.3)	0.619
HT n. (%)	37 (42.1)	35 (41.7)	20 (30.8)	0.270
MS n. (%)	6 (6.8)	5 (6.0)	2 (3.0)	0.635

Variables are presented in mean ± standard error of the mean.

P values refer to all possible comparisons between the three different follow-up stages. For values without pre-op data, the comparison is limited to the 6- and 12-month follow-up only.

Abbreviations: BMI, body mass index; EBMIL, excess BMI loss, TWL, total weight loss; T2D, type 2 diabetes mellitus; HT, hypertension; MS, metabolic syndrome; NA, not applicable.

Table II- Analytical parameters pre-operative and 12 months post-operatively

	Pre-operative	12 months post-operative	p
n	88	66	NA
Hb g/dL	**13.92 ± 0.13**	**13.22 ± 0.14**	**<0.001**
Htc %	**42.25 ± 0.33**	**39.88 ± 0.38**	**<0.001**
CHCM g/dL	32.94 ± 0.11	33.13 ± 0.11	0.179
RDW %	13.12 ± 0.12	12.79 ± 0.09	0.091
Platelets 10^9/L	**263.10 ± 5.32**	**231.15 ± 5.85**	**<0.001**
PDW %	12.54 ± 0.22	13.13 ± 0.26	0.059
Leucocytes 10^9/L	**7.19 ± 0.18**	**5.91 ± 0.18**	**<0.001**
Neutrophils %	**56.39 ± 0.77**	**52.10 ± 1.15**	**0.003**
Lymphocytes %	**34.05 ± 0.71**	**37.85 ± 1.04**	**0.003**
Iron µg/dL	**56.50 ± 9.50**	**97.08 ± 3.92**	**0.031**
Ferritin ng/mL	83.05 ± 40.19	148.07 ± 16.32	0.473
Transferrin saturation %	**19.60 ± 4.70**	**35.60 ± 1.46**	**0.027**
VitB12 pg/mL	280.50 ± 43.50	525.67 ± 27.92	0.050
Folic acid ng/mL	6.90	7.81 ± 0.56	0.951
Glucose mg/dL	**95.05 ± 2.00**	**83.93 ± 0.83**	**<0.001**
Hb1Ac %	5.44 ± 0.08	5.30 ± 0.05	0.553
Urea mg/dL	30.53 ± 0.86	30.90 ± 0.91	0.770
Creatinine mg/dL	**0.74 ± 0.02**	**0.67 ± 0.02**	**0.004**
Uric acid mg/dL	**5.53 ± 0.15**	**4.34 ± 0.17**	**<0.001**
Total Cholesterol mg/dL	**180.13 ± 3.26**	**165.64 ± 4.23**	**0.009**
HDL-C mg/dL	**48.13 ± 1.21**	**55.66 ± 2.49**	**0.002**
LDL-C mg/dL	122.29 ± 3.47	NA	-
Calculated LDL-C mg/dL	**109.44 ± 2.98**	**96.04 ± 3.24**	**0.004**
VLDL-C mg/dL	22.98 ± 1.09	NA	-
TG mg/dL	**112.79 ± 5.23**	**69.70 ± 3.39**	**<0.001**
Total bilirubin mg/dL	0.61 ± 0.02	0.67 ± 0.02	0.054
AST U/L	**24.24 ± 1.04**	**29.05 ± 1.34**	**0.001**
ALT U/L	31.96 ± 3.58	32.19 ± 2.19	0.258
GGT U/L	**34.12 ± 2.09**	**21.27 ± 2.54**	**<0.001**
Alkaline phosphataseU/L	85.00 ± 3.92	83.82 ± 3.16	0.841
Total protein g/dL	**7.13 ± 0.04**	**6.71 ± 0.05**	**<0.001**
Transferrin mg/dL	NA	237.60 ± 11.85	-
Albumin g/dL	4.41 ± 0.04	4.34 ± 0.03	0.198
TSH µUI/mL	1.62 ± 0.08	1.53 ± 0.14	0.672
T4L pmol/L	11.23 ± 0.19	11.90 ± 0.50	0.404
PTH pg/mL	NA	7.31 ± 0.51	-
Insulin µU/mL	NA	4.46 ± 0.54	-
PeptideC ng/mL	NA	412.00 ± 45.27	-
VitD ng/mL	21.30 ± 4.32	28.50 ± 1.45	0.253
Cortisol urine24h µg/24h	123.31 ± 7.25	NA	-
NLR	**1.76 ± 0.06**	**1.50 ± 0.08**	**0.002**
PLR	115.40 ± 3.65	111.20 ± 4.21	0.419

Significant differences are highlighted in bold.

Variables are presented in mean ± standard error of the mean.

Abbreviations: Hb, hemoglobin; Htc, hematocrit; MCHC, mean cellular hemoglobin concentration; RDW, red cell distribution width; PDW, platelet distribution width; VitB12, vitamin B12; HB1Ac, glycated hemoglobin; HDL-C, high density lipoprotein cholesterol; LDL-C low density lipoprotein cholesterol; VLDL-C, very-low density lipoprotein cholesterol; TG, triglycerides; AST, aspartate aminotransferase; ALT, alanine transaminase; GGT, gamma-glutamyl transferase; TSH, thyroid-stimulating hormone, T4L, thyroxine; PTH, parathyroid hormone; vitD, vitamin D; NLR, neutrophil-lymphocyte ratio; PLR, platelet-lymphocyte ratio; NA, not available.

Table III- Comparison of pre and 12-month post-operative resting energy expenditure data

	Pre-op	**Pre-Op**	**12 Months**	**p**
n	88	14	14	
Measured REE kJoule/day	8.07 ± 0.21	7.86 ±0.45	6.82 ±0.40	0.108
Measured REE kcal/day	1881.75 ± 53.78	1877.21 ±108.00	1629.29 ±94.71	0.109
Estimated REE kJoule/day	7.82 ± 0.14	**7.27 ±0.21**	**5.73 ±0.11**	**<0.001**
Estimated REE kcal/day	1824.11 ± 44.60	**1735.64 ±50.20**	**1367.64 ±26.79**	**<0.001**
VO_2 mL/min	278.70 ± 6.98	272.00 ±15.60	236.57 ±13.68	0.112
VO_2 mL/kg/min	2.62 ± 0.05	**2.67 ±0.13**	**3.58 ±0.21**	**0.003**
FeO_2 %	16.43 ± 0.08	16.64 ±0.19	16.92 ±0.23	0.198
Minute Volume L/min	8.99 ± 0.36	9.26 ±1.04	8.73 ±0.90	0.639
Tidal Volume mL	633.11 ± 30.77	605.14 ±63.02	624.55 ±103.70	0.850
Respiratory Rate breaths/min	15.25 ± 0.49	16.36 ±1.55	15.10 ±1.01	0.146

Variables are presented in mean ± standard error of the mean.

Significant differences are highlighted in bold.

Abbreviations: REE, resting energy expenditure; TEE, total energy expenditure; O_2, oxygen; VO_2, volume of oxygen; FeO_2, Fraction of expired oxygen.

Table IV- Linear regression of preoperative resting energy expenditure data with total weight loss at 6 and 12 months

	6 Months **β (95% CI)**	**12 Months** **β (95% CI)**
Measured REE kJoule/day	0.111 (-0.473; 0.696) p=0.706	-0.182 (-1.007; 0.644) p=0.662
Measured REE kcal/day	0.002 (-0.001; 0.004) p=0.157	-0.001 (-0.004; 0.002) p=0.592
Estimated REE kJoule/day	-0.206 (-1.049; 0.637) p=0.628	0.079 (-1.129; 1.287) p=0.896
Estimated REE kcal/day	0.001 (-0.001; 0.004) p=0.358	0.000 (-0.004; 0.004) p=0.878
VO_2 mL/min	0.003 (-0.014; 0.021) p=0.695	-0.008 (-0.033; 0.017) p=0.525
VO_2 mL/kg/min	0.694 (-1.671; 3.059) p=0.561	-2.213 (-5.418; 0.992) p=0.173
FeO_2 %	**1.581 (0.145; 3.018)** **p=0.031**	-0.114 (-2.017; 1.790) p=0.905
Minute Volume L/min	0.286 (-0.051; 0.624) p=0.096	-0.154 (-0.596; 0.287) p=0.488
Tidal Volume mL	0.001 (-0.003; 0.005) p=0.605	-0.003 (-0.008; 0.002) p=0.226
Respiratory Rate breaths/min	0.135 (-0.109; 0.379) p=0.274	0.163 (-0.158; 0.485) p=0.314

Significant differences are highlighted in bold.

Variables are presented in β regression coefficient and 95% confidence interval.

Abbreviations: REE, resting energy expenditure; O_2, oxygen; VO_2, volume of oxygen; FeO_2, Fraction of expired oxygen.

Table V- Comparison of preoperative resting energy expenditure data with the presence or absence of comorbidities (type 2 diabetes mellitus, hypertension and dyslipidemia)

	T2D		HT		Dyslipidemia	
	No	Yes	No	Yes	No	Yes
Measured REE kJoule/day	8.20 ± 0.21	6.96 ± 0.74	8.13 ± 0.25	7.99 ± 0.36	8.27 ± 0.26	7.69 ± 0.34
Measured REE kcal/day	1922.78 ± 52.05	1521.56 ± 239.98	1897.00 ± 64.50	1860.73 ± 92.93	1927.00 ± 63.74	1794.27 ± 98.03
Estimated REE kJoule/day	**7.90** ± **0.15**	**7.07** ± **0.29**	7.88 ± 0.19	7.73 ± 0.22	7.97 ± 0.18	7.52 ± 0.23
Estimated REE kcal/day	1858.39 ± 43.03	1523.16 ± 201.23	1836.57 ± 56.92	1806.93 ± 72.24	1864.02 ± 52.76	1746.95 ± 81.36
VO_2 ml/min	283.00 ± 7.11	241.00 ± 25.64	281.78 ± 8.57	274.46 ± 11.78	285.17 ± 8.57	266.20 ± 11.92
VO_2 mL/kg/min	2.63 ± 0.05	2.48 ± 0.25	2.63 ± 0.06	2.59 ± 0.09	2.64 ± 0.06	2.56 ± 0.10
FeO_2	16.42 ± 0.09	16.52 ± 0.27	16.44 ± 0.11	16.42 ± 0.13	16.42 ± 0.09	16.46 ± 0.17
Minute Volume L/min	9.09 ± 0.37	8.10 ± 1.51	9.07 ± 0.42	8.87 ± 0.65	9.04 ± 0.39	8.90 ± 0.77
Tidal Volume mL	**656.14** ± **32.75**	**431.00** ± **56.24**	660.21 ± 39.03	595.76 ± 49.58	650.03 ± 34.89	600.40 ± 60.42
Respiratory Rate breaths/min	**14.85** ± **0.51**	**18.76** ± **1.33**	14.81 ± 0.67	15.85 ± 0.71	14.98 ± 0.62	15.76 ± 0.80

Significant differences are highlighted in bold.

Variables are presented in mean ± standard error of the mean.

Abbreviations: REE, resting energy expenditure; O_2, oxygen; VO_2, volume of oxygen; FeO_2, Fraction of expired oxygen; TD2, type 2 diabetes mellitus; HT, hypertension

Table VI- Logistic regression of preoperative resting energy expenditure data with remission of comorbidities at 12 months

	T2D remission HR (95% CI)	HT remission HR (95% CI)	DL remission HR (95% CI)
Measured REE kJoule/day	0.885 (0.466; 1.680) p=0.709	1.214 (0.844; 1.747) p=0.295	1.205 (0.708; 2.053) p=0.491
Measured REE kcal/day	0.999 (0.997; 1.001) p= 0.422	1.001 (0.999; 1.004) p=0.305	1.000 (0.998; 1.001) p=0.863
Estimated REE kJoule/day	0.205 (0.020; 2.080) p=0.180	1.459 (0.813; 2.618) p=0.206	0.871 (0.400; 1.896) p=0.727
Estimated REE kcal/day	0.994 (0.985; 1.004) p=0.243	1.002 (0.999; 1.004) p=0.192	0.999 (0.996; 1.001) p=0.280
VO_2 ml/min	0.997 (0.978; 1.015) p=0.727	1.005 (0.994; 1.016) p=0.373	1.005 (0.990; 1.021) p=0.496
VO_2 ml/kg/min	1.077 (0.174; 6.680) p=0.936	1.612 (0.376; 6.908) p=0.502	3.501 (0.391; 31.377) p=0.262
FeO_2 %	0 (0; 219.997) p=0.234	1.216 (0.502; 2.941) p=0.665	1.196 (0.501; 2.855) p=0.687
Minute Volume L/min	0.755 (0.428; 1.331) p=0.331	1.103 (0.920; 1.323) p=0.228	1.104 (0.912; 1.335) p=0.311
Tidal Volume mL	0.999 (0.991; 1.008) p=0.86	1.004 (1.000; 1.008) p=0.083	1.001 (0.999; 1.004) p=0.363
Respiratory Rate breaths/min	0 (0; NA) p=0.995	0.834 (0.679; 1.024) p=0.083	1.004 (0.834; 1.208) p=0.968

Variables are presented in HR regression coefficient and 95% confidence interval.

Abbreviations: REE, resting energy expenditure; O_2, oxygen; VO_2, volume of oxygen; FeO_2, Fraction of expired oxygen; TD2, type 2 diabetes mellitus; HT, hypertension; DL, dyslipidemia; NA, not available.

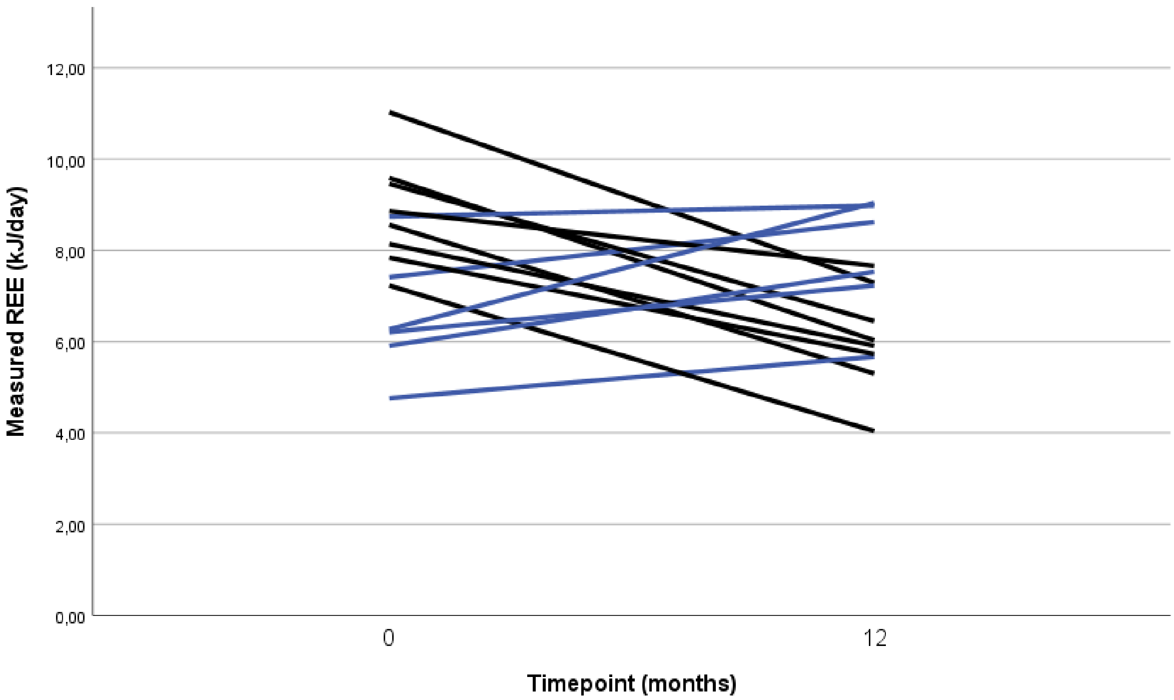
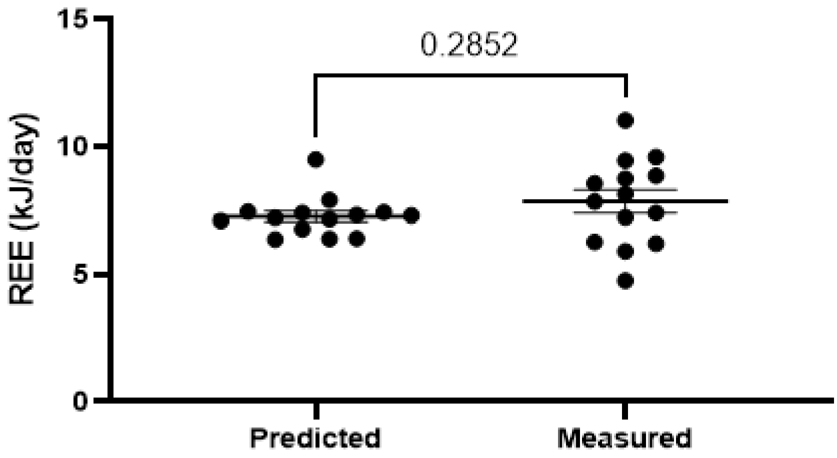


Figure 1 - Individual Trajectories of the measured Resting Energy Expenditure (REE). Blue lines indicate patients with increased REE at 12 months compared with preoperative values, while black lines indicate patients with decreased REE.

A



B

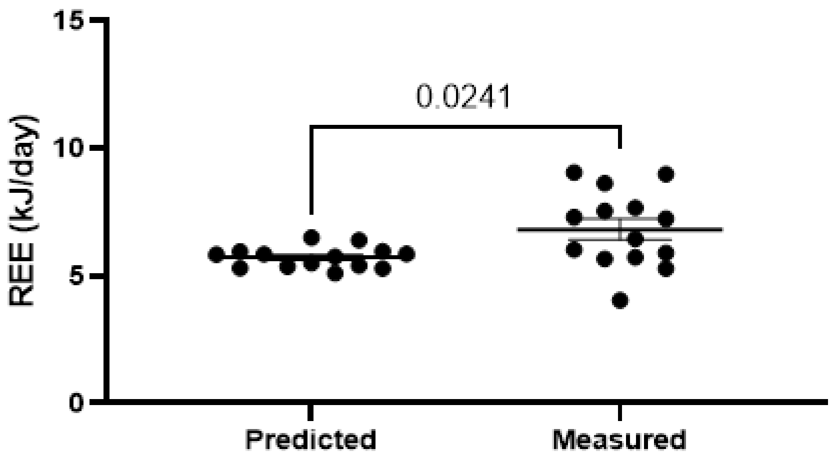


Figure 2 - Comparison of Estimated and Measured Resting Energy Expenditure (REE) values before (A) and 12 months after surgery (B)

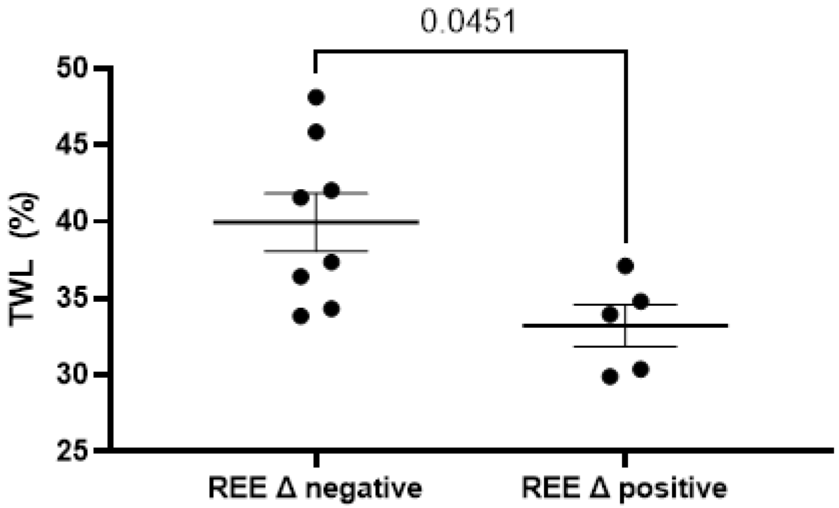


Figure 3 – Percentage of total weight loss (TWL) in patients with a decreased (Δ negative) or an increased (Δ positive) measured resting energy expenditure (REE) at 12 months compared with the pre-operatory analysis

Supplementary table 1 – Metrics and Equations and Parameters for Anthropometric and Resting Energy Expenditure Evaluation

Metric	Units	Formula
Body Mass Index (BMI)	kg/m^2	weight (kg) / height^2 (m^2)
Excess Body mass index loss (EBMIL)	%	preoperative BMI - postoperative BMI) / (preoperative BMI - 25) * 100]
Total Weight Loss (TWL)	%	(preoperative weight - postoperative weight) / preoperative weight * 100
Total volume of air per minute (Minute volume)	mL/min	Tidal Volume (mL) × “Rate (breaths/min)
Oxygen consumed per minute (VO2)	mL/min	Minute Volume (L/min)” × (0.21 - “FeO_2 (%)”/100) × 1000
Harris-Benedict Resting energy expenditure	kJoule/day or kcal/day	Male: (13.7516 × weight in kg) + (5.0033 × height in cm) – (6.755 × age in years) + 66.473 Female: (9.5634 × weight in kg) + (1.8496 × height in cm) – (4.6756 × age in years) + 655.0955
Estimated Resting energy expenditure	kJoule/day or kcal/day	Harris-Benedict REE, adjusted for current population
Measured Resting energy expenditure	kJoule/day or kcal/day	VO_2×1440×Constant
Weir Equation for Resting energy expenditure	kJoule/day or kcal/day	[3.941x(VO_2) +1.106x(VCO_2)] ×1440

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