Resting Energy Expenditure in Morbid Obesity

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Resting energy expenditure (REE) was measured in 112 morbidly obese adults prior to elective gastric bypass surgery. The patients studied ranged from 157 to 327% of ideal body weight. Standard nutritional assessment indices (serum total protein, albumin, total iron binding capacity, hematocrit, and white blood cell count) were within normal limits. REE was estimated by the Harris–Benedict formula using both current weight and ideal weight. Measured REE was significantly less than expected ($p < 0.01$) using current weight and significantly greater than expected ($p < 0.01$) when ideal weight was used as the standard. Linear regression analysis between standard indices that reflect resting metabolic rate in normal adults and measured REE in study patients did not demonstrate sufficient correlation to be clinically useful in this patient population. Standard surgical therapy may result in highly variable weight loss in this population if the wide range of resting energy expenditure and the consequential variability in individual caloric deficits is not considered. Standard predictors do not identify those patients likely to be unsuccessful with a given weight loss regimen.

THE PREVALENCE of obesity in the United States varies between 6 and 22% for males and 11 and 46% for females, depending upon age and income.[1-3] The prognosis for successful weight reduction is poor, particularly for persons who become obese during childhood.[4] Treatment techniques have included fasting, pharmacologic treatment, behavior modification, surgery, physical activity, and group self-help.[4]

Since the identification of metabolic and hepatic complications[5-9] and the development of protein malnutrition[10] in some patients following intestinal bypass procedures, the gastric bypass has become the preferred surgical procedure for the treatment of morbid obesity. Weight loss following gastric bypass surgery is variable.[6,7,11-13] Most explanations of this phenomenon have focused on the technical perfection of the operative procedure.[6,7,11] The hypothesis is that a second factor contributing to this variable weight loss may be individual alterations in energy expenditure. Garrow[14] has noted reduced metabolic rate in some obese individuals and Danforth[15] has described the inability of some obese patients to respond to over feeding with the normal increase in resting energy expenditure (REE). Morbidly obese persons may have marked individual variations in energy expenditure. When gastric bypass surgery imposes a standard therapy for every patient, this may result in widely different exogenous caloric deficits and subsequently variable weight loss.

Regardless of the treatment modality employed, successful weight loss requires sustained negative energy balance, or the maintenance of a daily dietary intake with fewer calories than are expended over that same 24-hour period. Therefore, the determination of the optimum caloric intake required to produce a given degree of weight loss over time is dependent upon an accurate appraisal of an individual's total daily energy expenditure.

A popular method for estimating total energy expenditure is to calculate resting energy expenditure from standard predictor equations and then add an additional 20% increment to account for physical activity. Alternatively, resting energy expenditure may be measured by indirect calorimetry.

This study investigates the hypothesis that variable weight loss following gastric bypass surgery may be due to great variability in caloric deficits generated by the therapy. Measured resting energy expenditure in morbidly obese adults is examined prior to surgery and com-

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pared with the best available standards for estimating REE.

Patients and Methods

Resting energy expenditure was measured by a portable indirect calorimeter (Metabolic Measurement Cart, Beckman Instruments, Anaheim, CA)[16] in 112 morbidly obese adults (18 males and 94 females) as part of a routine nutritional assessment prior to elective gastric bypass surgery. All patients were either 45 kg or 100% above their ideal body weight (IBW), had failed at other approaches to weight reduction, and were evaluated by a psychiatrist prior to surgery.

All indirect calorimetric measurements of resting energy expenditure were made by a single investigator (IF). Oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) were determined continuously from expired air by a polarographic oxygen sensor and infrared carbon dioxide sensor which are integrated with a programmable calculator. The instrument has been evaluated during exercise testing[17] and during a wide variety of circumstances simulating physiologic conditions,[18] and its measurements were found to be within ±1.5% of known values for respiratory quotient.[18]

Gas exchange was measured with the patient resting supine in bed, greater than two hours following a meal, in the morning or afternoon. Expired air was collected using a noseclip and nonrebreathing valve. Continuous measurements of $\dot{V}O_2$ and $\dot{V}CO_2$ were computed at one-minute intervals. These measurements were then applied to the abbreviated Weir formula[19] to determine resting energy expenditure in kcal/min.

Abbreviated Weir Formula

$$3.9(\dot{V}O_2) + 1.1(\dot{V}CO_2) = \text{kcal/min}$$

$\dot{V}O_2$ = oxygen consumption in L/min

$\dot{V}CO_2$ = carbon dioxide production in L/min

Measurement of gas exchange continued for a period of five to 15 minutes until evidence of equilibration or "steady state" was demonstrated by five consecutive stable determinations of $\dot{V}O_2$ and $\dot{V}CO_2$. Only the mean values for the equilibrated data points were used in the final calculation of REE. The reproducibility of such measurements has been demonstrated at this institution. Resting energy expenditure was measured twice within one hour in 26 healthy subjects (nine males and 17 females). The mean per cent change from the first to second measurement was 1 ± 2% in the males and 2 ± 2% in the females. When REE was measured in ten clinically stable patients at mid-morning and mid-afternoon, the mean per cent difference between measurements was found to be 1 ± 2%.

Predicted resting energy expenditure was calculated from the Harris–Benedict formula[20] using the patient's current weight and ideal weight.

Harris–Benedict Formula

$$\text{Males} = 5H + 13.7W + 66 - 6.8A = \text{kcal/day}$$

$$\text{Females} = 1.8H + 9.6W + 655 - 4.7A = \text{kcal/day}$$

H = height in cm

W = weight in kg (current and ideal)

A = age in years

Measured REE was expressed as a per cent of predicted REE for each patient. Differences between measured and expected REE were analyzed by the paired Student's t-test.[21]

Linear regression analysis was performed to describe the relationship between estimated and measured REE. In a further attempt to include an obesity factor, multiple regressions with estimated REE and per cent ideal body weight (% IBW) as the independent variables were also performed. Finally, linear regressions with height, weight, age, and per cent IBW as the independent variables and measured REE the dependent variable were calculated in an attempt to describe a predictor of resting energy expenditure for morbidly obese patients.

Results

The patients studied were in generally good health, but markedly obese. The mean age for the males was 37 ± 10 years and 40 ± 10 years for the females. The mean body weight was 146.0 ± 25.6 kg for the males and 125.2 ± 23.6 kg for the females, representing a range of 157 to 326% IBW (Table 1). Standard nutritional indices such as serum albumin, total protein, total iron binding capacity, total lymphocyte count, hematocrit, and white blood cell count were within normal ranges.

Measured resting energy expenditure was significantly different from expected values ($p < 0.01$) for the males and females when predicted REE was calculated using either current weight or ideal weight (Table 2). When REE was calculated from current weight, measured rest-

TABLE 1. *Patients Studied*

Sex	N	Age* (years)	Height* (cm)	Weight* (kg)	IBW* (%)
Males	18	36.5 ± 10.4	175.4 ± 7.0	146.0 ± 25.6	211 ± 35
Females	94	39.8 ± 9.6	162.5 ± 12.33	125.2 ± 23.6	221 ± 43

* Mean ± SD.

Patients ranged from 157 to 326% of ideal body weight.

TABLE 2. *Measured and Predicted Resting Energy Expenditure*

	Expected (kcal/day)*		Measured*	Measured (% Expected)*	
Sex	Current Wt.	Ideal Wt.	(kcal/day)	Current Wt.	Ideal Wt.
Males	2610 ± 421†	1666 ± 130†	2274 ± 301	88.4 ± 15.0	120.0 ± 39.9
Females	1953 ± 254†	1289 ± 89†	1751 ± 291	89.5 ± 16.9	138.6 ± 22.3

* Mean ± SD

† $p < 0.01$ *vs.* measured.

Measured REE was significantly less than expected with current weight as the standard and significantly greater than expected with ideal weight as the standard.

ing energy expenditure was 88.4 ± 15.0% and 89.5 ± 16.9% of expected for the males and females, respectively. When resting energy expenditure was calculated using ideal weight, measured REE was 120.0 ± 39.9% and 138.6 ± 22.3% of expected for the males and females. Thus, actual resting energy expenditure was significantly less ($p < 0.01$) than predicted when current weight was used with the Harris–Benedict formula and significantly greater ($p < 0.01$) than predicted by the Harris–Benedict formula when ideal weight was used in the calculation. Individual variation in resting energy expenditure ranged from 57% to 135% of expected when the standard was calculated from current weight, with only 39% of the patients being within ±10% of expected REE (Fig. 1). Resting energy expenditure ranged from 95% to 194% of expected when the standard was calculated from ideal weight with only 13% of the sample being within ±10% of expected REE (Fig. 2).

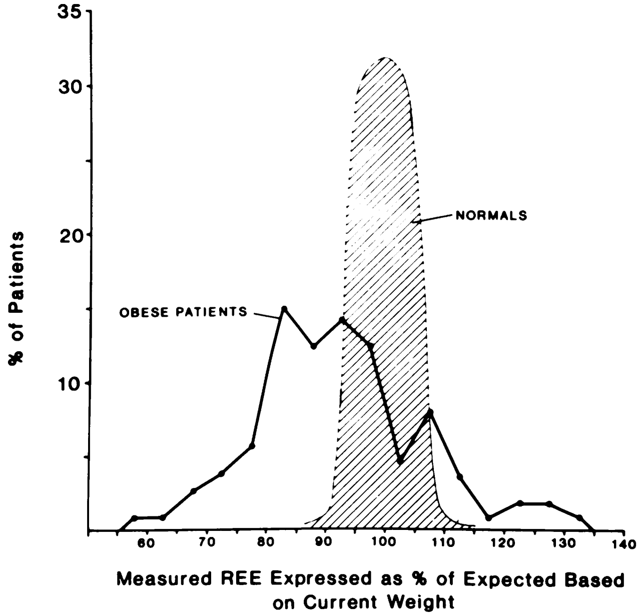

FIG. 1. Distribution of Measured Resting Energy Expenditure. Only 39% of the morbidly obese patients studied had measured resting energy expenditures that were within ±10% of expected when the standard was calculated from current weight. This contrasts markedly with Boothby's data (DuBois EF. Basal Metabolism in Health and Disease. 3rd Ed. Philadelphia:Lea and Febiger, 1936), which showed 92% of the normal, healthy volunteers studied to have metabolic rates within ±10% of expected values.

Linear regression analysis (Table 3) showed statistically significant correlations in some instances, but a sufficient amount of the variance in measured REE could not be attributed to the independent variables for the equations to be clinically useful. Multiple regression analysis with height, weight, age, and per cent ideal weight as the independent variables showed little linear relationship between those indices and measured REE ($r = +0.22$, N.S.).

Comments

Resting energy expenditure reflects the metabolic rate of the metabolically active cells of the body. This body compartment has been described by Moore as the body cell mass: the oxygen-exchanging, potassium-rich, glucose-oxidizing work-performing tissue.[22] The body cell mass equals total weight minus adipose tissue, extracellular fluid, skeleton, and connective tissue. Traditionally, a constant relationship is thought to exist between resting energy expenditure and a unit of body cell mass.[22] More recently, however, investigators have shown alterations in energy expenditure per unit body cell mass in malnourished patients[23] and in patients with gynecologic malignancies.[24]

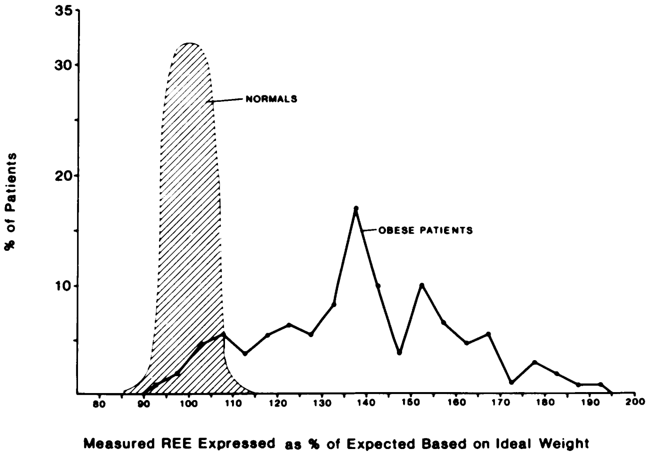

FIG. 2. Distribution of Measured Resting Energy Expenditure. Only 13% of the obese patients studied had measured resting metabolic rates within ±10% of expected values when the standard was calculated from ideal weight and the Harris–Benedict formula. In contrast, 92% of Boothby's normal adults were within ±10% of predicted resting energy expenditure.

TABLE 3. *Expected Vs. Measured REE*

Sex	Regression Equation	r	p
Males	REE = 1487 + 0.30(REEp)	0.42	N.S.
Females	REE = 817 + 0.48(REEp)	0.42	<0.01
Males	REE = 1332 + 2.12(%IBW) + 0.19(REEp)	0.46	N.S.
Females	REE = 824 + 1.09(%IBW) + 0.35(REEp)	0.43	<0.01

REE = Measured resting energy expenditure (kcal/day).

REEp = Estimated resting energy expenditure from Harris–Benedict using current weight (kcal/day).

% IBW = % ideal body weight.

Linear regression analysis showed statistically significant relationships between standard indices and measured REE for the females, however a sufficient amount of the variance in REE could not be accounted for by the independent variables for the equations to be clinically useful. Relationships between the same indices and measured REE were not significant for the male sample.

Predictive equations estimate resting energy expenditure from indices (height, weight, age, and sex) that reflect body cell mass size in healthy individuals with normal body composition. If these indices do not truly reflect the amount of metabolically active tissue in a given group of patients, or a linear relationship between REE and the amount of metabolically active tissues does not exist, the predictive equation will not be accurate and appropriate for that group of patients.

In addition, the conditions under which a predictor was developed should be considered when it is applied. The Harris–Benedict formula was developed on 136 healthy normal males and 103 healthy normal females. The subjects fasted for 12 hours, traveled to the monitoring center, and rested for a half hour before measurements of gas exchange were made. Multiple linear regression analyses using Harris–Benedict's original data showed correlation coefficients of +0.77 for the male subjects and +0.54 for the females. Although this predictor is frequently considered to be an estimator of basal energy expenditure, it was not developed under the measurement conditions for basal energy expenditure described by Wilmore,[25] *i.e.*, measurement within a half hour of waking, in a darkened room, following a 12-hour fast. It more closely adheres to the conditions of resting energy expenditure (which may be measured at any time of day). It is appropriate to compare measured REE with the predicted REE (Harris–Benedict formula) from the standpoint of the similarity of measurement conditions employed. However, its applicability to significantly overweight patients is questionable as there were no such subjects in the Harris–Benedict developmental population.

Moore describes the body composition of a normal healthy individual to be 12 to 20% total body fat and 35 to 45% body cell mass.[22] For a 70-kg man, this represents a total body fat compartment of 8.4 to 14.0 kg and a body cell mass of 24.5 to 31.5 kg. Shizgal has found morbidly obese patients to have a total body fat compartment comprising 50% (79.7 kg) of total weight (148.6 kg) and a body cell mass compartment that comprises 22% (33.0 kg) of body weight.[10] These values are significantly different from his normal controls[10] and from the values described by Moore.[22] Morbidly obese persons clearly have abnormal body composition.

These data indicate that the resting energy expenditure of morbidly obese persons cannot be estimated accurately by the Harris–Benedict formula. In addition, linear regression analyses indicate that there is no simple factor which adjusts for these observed differences, nor does the inclusion of an index describing degree of obesity (% IBW) provide a correction with sufficient sensitivity and specificity for clinical use. The fact that no statistically significant linear relationship was observed between height, weight, age, per cent IBW, and measured REE indicates that these indices alone are not adequate reflectors of the metabolically active compartment in morbidly obese individuals. In general, the Harris–Benedict predictors do not reflect the resting energy expenditures of morbidly obese adults and, therefore, cannot serve as the basis for estimating total daily energy expenditure in this population.

Investigators have described significant but variable weight reduction in the first year following gastric bypass surgery.[6,7,11–13] There may be a plateau in weight loss,[11] or a gradual increase in weight following the first postsurgical year.[6] Even though a standard surgical intervention with its accompanying decreased caloric intake was employed, weight loss is variable. The wide range of measured resting energy expenditure observed in this study may contribute dramatically to the eventual magnitude of weight loss obtained by obese persons via any standard weight loss regimen.

The variability of weight loss after gastric bypass is often attributed to technical imperfections of the operative procedure: the pouch was too big, the anastamosis too large, etc. In fact, such differences in weight loss can readily be explained by the variability of metabolic rates in this population. A caloric deficit of approximately 1100 kcal/day would be required to produce a 45 kg

TABLE 4. *Hypothetical Range of Weight Loss*

	Patient A	Patient B	Patient C
Estimated energy expenditure (kcal/day)	1,900	1,900	1,900
Measured energy expenditure (kcal/day)	1,900	2,550	1,150
Total caloric intake (kcal/day)	700	700	700
Daily caloric deficit (kcal)	1,200	1,850	450
Annnual caloric deficit (kcal)	438,000	675,250	164,250
Annual fat weight lost (kg)	48.7	75	18.3

Three patients receiving identical therapy (gastric bypass surgery followed by 700 kcal/day intake) could demonstrate drastically different degrees of weight loss after one year due solely to individual differences in REE.

(100 lb) weight loss in one year, if all weight were lost from the adipose tissue compartment; (9000 kcal = 1 kg fat). This 45-kg adipose tissue loss requires a 405,000 kcal/year deficit (1110 kcal deficit/day). Preliminary data from this institution indicate that these patients ingest approximately 700 kcal/day after the initial postoperative convalescence and adaptation following gastric bypass surgery.

Assuming metabolic rate to be constant over time, an individual with a measured energy expenditure of 1900 kcal/day would generate a caloric deficit of 1200 kcal/day or 438,000 kcal/year. This translates into a 48.7 kg (107 lb) weight loss in a year if it is assumed only fat is lost. These calculations are summarized for patient A in Table 4. Patient A had a measured REE within ±10% of expected as did 39% of patients in this study.

As can be seen in Figure 1, 8.9% of patients had measured resting energy expenditures >10% above expected. Patient B is in this category with an expected REE of 1900 kcal/day but an actual measured REE of 2550 kcal/day. Assuming the same constancy of metabolic rate and caloric intake, patient B will lose 75 kg (165 lb) of fat in a year. Patient C (Table 4) represents the reverse situation. This patient had an actual measured energy expenditure <90% of expected (1150 *vs.* 1900 kcal/day) as did 51.8% of patients in this study. With only a 450 kcal daily deficit, patient C will only lose 18.3 kg (40.2 lbs) of fat in a year.

These three patients illustrate that if one estimates energy expenditure (1900 kcal/day), applies a standard therapy (gastric bypass) that results in a standard caloric intake (700 kcal/day) and a standard caloric deficit (1200 kcal/day), the resultant fat weight loss may vary fourfold (18.3–75 kg) merely because of differences in actual energy expenditure. Morbidly obese individuals of the same age, sex, height, and weight will have identical predicted energy expenditures (Harris-Benedict equation), but their actual metabolic rate may vary from 55 to 135% of expected rate.

Clinical application of a standard therapy designed to reduce caloric intake and increase the caloric deficit will result in considerably variable weight loss in morbidly obese adults unless the wide range of actual resting energy expenditure in these patients is considered. Available predictive equations do not accurately estimate true resting energy expenditure in this population and cannot be used to rationally design a weight loss regimen. In view of the inability to generate a valid predictive equation for the morbidly obese, measuring energy expenditure is the only viable alternative.

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References

1. Goldblatt PB, Moore ME, Stunkard AJ. Social factors in obesity. JAMA 1965; 192:1039–1044.
2. U.S. Department of Health, Education and Welfare. The Ten State Nutrition Survey. DHEW Publication HSM, 72–8134, 1972.
3. Health and Nutrition Examination Survey. Height and Weight of Adults 17–74 Years of Age, United States, 1971–1974. Advance Data 1977; 14:1–11.
4. Stunkard A. Obesity. In: Kaplan HI, Freedman AM, Sadock BG, eds. The Comprehensive Textbook of Psychiatry, Vol II, 3rd Ed. Baltimore: Williams and Wilkins, 1980.
5. Brown RG, O'Leary JP, Woodward ER. Hepatic effects of jejunoileal bypass for morbid obesity. Am J Surg 1974; 127:53–56.
6. Buckwalter JA. Clinical trial of jejunoileal and gastric bypass for the treatment of morbid obesity: four-year progress report. Am Surg 1980; 46:377–381.
7. Griffen WO, Young VL, Stevenson CC. A prospective comparison of gastric and jejunoileal bypass procedures for morbid obesity. Ann Surg 1977; 186:500–507.
8. Halverson JD, Gentry K, Wise L, et al. Reanastomosis after jejunoileal bypass. Surgery 1978; 84:241–246.
9. MacLean L. Intestinal bypass operations for obesity: a review. Can J Surg 1976; 19:387–390.
10. Shizgal HM, Forse RA, Spanier AH, et al. Protein malnutrition following intestinal bypass for morbid obesity. Surgery 1979; 86:60–69.
11. Mason EE, Printen KJ, Hartford CE, et al. Optimizing results of gastric bypass. Ann Surg 1975; 182:405–413.
12. Mason EE. Surgical Treatment of Obesity. Philadelphia: WB Saunders, 1981.
13. Palombo JD, Hayward E, Reinhold R. Composition of weight loss after gastric bypass operation: long-term analysis. Surg Forum 1981; 32:114–115.
14. Garrow JS. Energy balance in obesity. In: Kinney J, ed. Assessment of Energy Metabolism in Health and Disease, Report of the First Ross Conference on Medical Research. Columbus, Ohio: Ross Laboratories, 1980; 134–138.
15. Danforth E Jr. Nutritionally induced alterations in metabolism. In: Kinney J, ed. Assessment of Energy Metabolism in Health and Disease, Report of the First Ross Conference on Medical Research. Columbus, Ohio: Ross Laboratories, 1980; 139–141.
16. Norton AC. Portable equipment for gas exchange. In: Kinney, J, ed. Assessment of Energy Metabolism in Health and Disease, Report of the First Ross Conference on Medical Research. Columbus, Ohio: Ross Laboratories, 1980; 36–41.
17. Wilmore JH, Davis JA, Norton AC. An automated system for assessing metabolic rate and respiratory function during exercise. J Appl Physiol 1976; 40:619–624.
18. Damask MC, Weissman C, Askanazi J, et al. Validation of gas exchange measurements (abstr). JPEN 1981; 5:366.
19. Weir JB deV. New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 1949; 109:1–9.
20. Harris JA, Benedict FG. Biometric studies of basal metabolism in man. Carnegie Institute of Washington, publication number 297, Washington, DC, 1919.
21. Steel R, Torrie J. Principles and Procedures of Statistics. New York: McGraw Hill, 1960.
22. Moore FD, Olesen KH, McMurray JD, et al. The Body Cell Mass and its Supporting Environment, Body Composition in Health and Disease. Philadelphia: WB Saunders, 1963.
23. Forse RA, Coughlin M, Shizgal HM. Effect of malnutrition on oxygen consumption. Surg Forum 1981; 32:99–102.
24. Knox L, Crosby L, Feurer I, et al. Energy expenditure and gynecologic cancer (Abstr). Clin Res 1980; 28:631A.
25. Wilmore DW. The Metabolic Management of the Critically Ill. New York: Plenum: 1977.