Body Composition and Energy Expenditure: Relationship and Changes in Obese Subjects Before and After Biliopancreatic Diversion

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Changes in total and segmental body composition were studied in 101 obese women before and 2, 6, 12, and 24 months after biliopancreatic diversion (BPD) and data 24 months after surgery were matched to 53 control subjects. The patients were studied by anthropometry, indirect calorimetry, and double-emission x-ray absorptiometry (DXA). The combination of calorimetry and body composition analysis allowed estimation of visceral and muscle lean mass. We observed a significant (analysis of variance [ANOVA]: $P < .05$) progressive reduction of fat and lean body mass (LBM) following BPD, with stabilization of both parameters between 12 and 24 months at levels not different from controls. Fat loss was significant in the arms, legs, and trunk segments. After 24 months, there was no significant difference in segmental fat mass between post-BPD patients and controls. Calorimetric data seem to confirm lean body mass (LBM) reduction. Visceral lean mass (kg) was significantly reduced from 8.1 ± 2.2 in obese subjects to 6.5 ± 1.8 in post-BPD patients at 24 months ($P < .05$); the control value was 7.2 ± 1.8. Muscular lean mass (kg) was also significantly reduced, from 50.2 ± 5.8 to 39.8 ± 5.7 in the same subjects ($P < .05$), with a control value of 42.5 ± 5.9. The decrease in muscle and visceral LBM reached control values without significant differences. Viscera/muscle ratio in pre-BPD patients was preserved in post-BPD patients at 24 months, but it was reduced during weight loss. Body composition studies showed a logarithmic relationship between fat and lean mass and a physiological contribution of lean mass to weight loss in the BPD patients. In conclusion, weight loss after BPD was achieved with an appropriate decline of LBM and with all parameters reaching, at stable weight, values similar to weight-matched controls.

OBESITY is a very common condition in Western society affecting both sexes and all ages. Although there are ethnic or sex variations in prevalence, obesity occurs mainly in communities where there is an abundant food supply: it is a behavioral as well as nutritional disease. More recent studies attribute a participating role to genetic factors in the etiology of obesity, but the energy balance between food intake and metabolic consumption is still key for weight accumulation or loss.[1] It has been known for a long time that obese subjects have an elevated basal metabolic rate (BMR), when compared to subjects of the same age,[2] but it is not generally appreciated that they must also have an increase in lean body mass (LBM).[3] This increase in LBM may explain the increased BMR, the increased glomerular filtration rate, and the greater urinary excretion of creatinine and steroids in these patients.[4]

Only 2 conditions of human obesity are associated with a decreased LBM: Cushing's syndrome and hypothalamic obesity,[5] such as in Prader-Willi syndrome. Thus an increase in LBM is usually associated with the increased weight in obesity. Several investigators have established a relationship between body fat and LBM.[6,7] However, the reasons for increased LBM are not known. It is evident[7] that certain organs, such as the heart, kidney, liver, and muscle, participate. Possible explanations include hypertrophy of skeletal muscle in response to the greater weight, the increased number of adipocytes, each with its component of intracellular fluids, and hypertrophy of the gastrointestinal tract as a consequence of increased food intake. Hormonal modifications might play a role. Insulin and adrenal steroids are increased and both are anabolic.[4]

The objective of the current study was to evaluate in a longitudinal way the quantitative relationship between LBM and body fat over a wide range of weight, and across the intervention of surgically induced weight loss.

MATERIALS AND METHODS

We studied sequential body composition changes in a group of obese subjects before and after biliopancreatic diversion (BPD). This surgical procedure, first described by Scopinaro[8] in 1978, consists of distal gastrectomy with a Roux-en-Y reconstruction. The residual gastric volume is 200 to 400 mL. The Roux-en-Y is constructed with a common tract of 50 cm from the ileocecal valve, 200 cm of alimentary tract, and a variable length, according to small bowel length, of the biliary tract. The rationale of this procedure is to avoid the contact and mixing of food with biliary and pancreatic secretions beyond the stomach. All patients are allowed to ingest food ad libitum, as there is a fixed length of ileum where the absorption of fat and polysaccharides is allowed. The malabsorptive mechanism due to biliary and pancreatic juices diversion continues to operate and is responsible for maintenance of weight loss.[9]

Patients

We studied 101 obese women before and after BPD and 53 female controls matched to the subjects at 24 months after surgery. All operations were performed in the period from 1990 to 1997. The age (mean ± SD) of the presurgical patients was 41 ± 8 years (range, 18 to 58), height (cm) 161.2 ± 8.9 (range, 144 to 177), weight (kg) 123.3 ± 24.1 (range 79-203), body mass index (BMI; kg/m^2) 45.5 ± 7.7, and excess body weight (%) 113.7 ± 35.2. The age of the control subjects was 40 ± 5 years (range, 22 to 55), height (cm) 162.7 ± 9.0 (range, 146 to 188), weight (kg) 82.3 ± 9.8 (range, 51 to 101), and BMI (kg/m^2) 30.1 ± 3.5.

We performed a metabolic evaluation in our patients before surgery and at 2, 6, 12, and 24 months after BPD. All data were collected during standard follow-up control visits.

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Submitted March 1, 2002; accepted December 30, 2002.

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0026-0495/03/5205-0021$30.00/0
doi:10.1053/meta.2003.50109

Table 1. Weight, BMI, Body Composition, and REE in Our Patients

	Weight (kg)	BMI (kg/m^2)	REE (kJ/d)	LBM (kg)	FAT (kg)
Obese	123.3 ± 24.1	45.5 ± 7.7	9,229 ± 75	58.0 ± 6.6	63.9 ± 20.1
Post-BPD					
2 mo	113.0 ± 24.8*	42.2 ± 7.8*	8,354 ± 193	57.8 ± 10.6	52.3 ± 15.6*
6 mo	91.6 ± 20.0*	34.1 ± 6.0*	6,889 ± 255*	51.9 ± 8.7*	37.0 ± 12.6*
12 mo	80.0 ± 15.3*	30.4 ± 4.5*	6,789 ± 301*	49.3 ± 6.6*	27.9 ± 11.4*
24 mo	77.3 ± 15.7*	29.6 ± 5.6*	6,986 ± 1,666*	47.9 ± 10.6*	29.4 ± 14.0*
Controls	82.3 ± 9.8*	30.1 ± 3.5*	7,680 ± 1,506*	47.9 ± 10.6*	33.5 ± 11.5*

Note stabilization of values between 12 and 24 months at levels similar to controls.
*$P < .05$ *v* obese.

Statistics

Means and standard deviations of the mean are presented. Analysis of variance (ANOVA) was used to determine the statistical significance of differences. Tukey's method was used for simultaneous post-hoc comparisons of means. The software used was Statistica for Windows 4.5 StatSoft (Tulsa, OK).

Body Composition Study

All patients were evaluated by anthropometry (weight, height, and BMI), hematochemical parameters, calorimetric studies, and dual-photon emission x-ray absorptiometry (DXA).

In the morning, after an overnight fast, the subjects were brought from the general surgery ward to the metabolic unit in light clothing, to determine barefoot heights and weights. They were familiarized with the indirect calorimetric equipment and then assumed a supine position at complete bed rest. Room temperature was 22 to 24°C and humidity was about 40 %.

Respiratory gas exchange was measured with a Deltatrac Metabolic Monitor (Datex, Helsinki, Finland). The machine was calibrated before each measurement.

DXA (LUNAR DPX, Madison WI) with photon emission of 40 and 70 KeV was used for a whole-body rectilinear scan to estimate segmental (left and right arm, leg, and trunk) and total LBM and body fat.[10,11] The DXA system was calibrated weekly with 7 bags of ground beef in which the fat content ranged between 3% and 83% based on chemical analysis.[12] The reproducibility of percent fat, bone mineral content, and bone density by DXA ranged between 0.97 and 0.99 in human subjects measured 5 times, and was 0.99 in phantom studies.[12] The total radiation dose EDE (energy dose equivalent) was less than 0.03 milliSievert.

Despite the high degree of adiposity in very obese patients, DXA can be a reliable method to estimate body composition in such individuals.[13,14]

LBM consists of several components: muscle, brain, liver, lung, etc (ie, muscle and viscera); each component has its own specific resting energy expenditure (REE/kg). Specific organ energy consumption (organ metabolic rate per killogram) does not appear to be different among individuals.[15] Animal and human studies have determined the specific energy consumption of different tissues.[15-18] Adding up the individual organ oxygen consumption and derived REE, according to de Weir, the REE of visceral mass, muscle, and fat can be calculated as follows: muscle REE = 45.45 kJ/kg/d, visceral REE = 675.67 kJ/kg/d, and fat REE = 20.93 kJ/kg/d. We included in viscera the sum of brain, heart, lungs, liver, kidneys, and intestines, ie, the high-energy-consumption organs; the muscular mass comprised the sum of muscle, skin, and connective tissues, because of their similar low specific REE. Bone mass, as measured by DXA, was not included in the model because of its very low specific REE (9.6 kJ/kg).[19] Combining data derived from body composition analysis and indirect calorimetry, it is possible to solve a system equation in order to infer muscular and visceral lean mass. So,

$$\mathrm{mLBM} = \frac{\mathrm{tREE} - \mathrm{FAT} \times \mathrm{fREE} - \mathrm{LBM} \times \mathrm{vREE}}{\mathrm{mREE} - \mathrm{vREE}}$$

$$\mathrm{vLBM} = \frac{\mathrm{tREE} - \mathrm{FAT} \times \mathrm{fREE} - \mathrm{LBM} \times \mathrm{mREE}}{\mathrm{vREE} - \mathrm{mREE}}$$

where: fREE = specific fat REE (20.93 kJ/kg/d); mLBM = muscle LBM (kg); mREE = specific muscle REE (45.45 kJ/kg/d); vLBM = visceral LBM (kg); vREE = specific visceral REE (675.67 kJ/kg/d); FAT = fat mass (kg); and tREE = total REE (kJ/d) (see Appendix 1).

Blood samples were collected at 24 months after operation in order to value plasmatic albumin, total proteins, and hemoglobin as nutritional parameters.

RESULTS

Sequential body composition changes in the post-BPD patients are shown in Table 1. We observed a progressive decrease in BMI with stabilization after 12 months. REE showed a clear reduction at 6 months with stabilization in the following months at values not different from controls. We obtained a significant decrease both in FAT and LBM at 12 months without further significant variations at 24 months.

Segmental variations in body composition are shown in Table 2. Arms, legs, and trunk participated in FAT decrease, but only trunk segment reached statistical significance in LBM decrease. Visceral lean mass (kg) was significantly reduced from 8.1 ± 2.2 in obese subjects to 6.5 ± 1.8 in post-BPD patients at 24 months ($P < .05$), with a mean decrease of 1.6 kg (19.8%); the control values was 7.2 ± 1.8. Also muscular lean mass (kg) was significantly reduced from 50.2 ± 5.8 to 39.8 ± 5.7 in the same subjects ($P < .05$), with a mean decrease of 10.4 kg (20.7 %). The control value was 42.5 ± 5.9. The decrease in muscle and visceral LBM reached control values without significant differences.

Viscera/muscle ratio in pre-BPD patients was preserved also in post-BPD patients at 24 months (0.171 *v* 0.167, respectively; P = not significant [NS]), but it was reduced during weight loss (0.141 at 6 months, $P < .05$ *v* pre-BPD patients and post-BPD patients at 24 months).

The REE/total weight ratio increased progressively from 76.2 ± 11.7 kJ/kg in presurgical patients to 97.5 ± 21.3 kJ/kg in postsurgical subjects at 24 months ($P < .05$), while the REE/LBM ratio showed an initial reduction from 158.6 ± 28.0 to 139.4 ± 20.5 kJ/kg at 6 months after operation ($P < .05$) and

Table 2. Segmental Changes in Body Composition in Our Patients

	FAT (kg)			LBM (kg)		
	Trunk	Legs	Arms	Trunk	Legs	Arms
Obese	26.9 ± 7.1	17.4 ± 4.6	5.9 ± 4.0	29.0 ± 6.7	16.9 ± 3.5	5.2 ± 3.2
BPD 24 mo	12.8 ± 6.4*	10.9 ± 4.1*	2.9 ± 1.3*	22.4 ± 5.5*	15.3 ± 2.3	4.0 ± 1.3
% Change	−52%	−37%	−51%	−23%	−9.5%	−23%
Controls	17.8 ± 8.1*	13.5 ± 7.5*	3.2 ± 1.7*	27.4 ± 8.2	16.9 ± 3.5	4.3 ± 1.6

NOTE. % Change indicates percent segmental composition changes after 24 months. Leg, trunk, and arm fat reveals significant segmental variation after BPD. Lean mass loss is evident only in trunk segment.

*$P < .05$ *v* obese.

a subsequent increase to 149.8 ± 28.9 at 24 months, reaching control values of 154.4 ± 25.1 kJ/kg (P = NS between presurgical, postsurgical at 24 months, and control values). It is notable that the REE/LBM ratio followed the trend of the viscera/muscle ratio.

The initial content of fat is in relationship with initial LBM. Figure 1 shows this relationship ($R = 0.60$; $P < .001$) in obese patients and a group of control subjects.

The LBM/FAT relationship is logarithmic: LBM = 26.55 × $\log_{10}$ (FAT) + 10.56. Figure 2 shows how the initial fat content influences the relative changes in LBM. The dLBM/dW ratio represents the LBM loss for every unit of weight loss (see Appendix 2).

In pre-BPD patients the hematochemical parameters were: glycemia 101.44 ± 35.21 mg/dL, total proteins 6.63 ± 2.16 mg/dL, albumin 3.87 ± 1.41 mg/dL, and hemoglobin 13.40 ± 1.89 g/dL.

Hematochemical evaluation of post-BPD patients showed good nutritional status: glycemia was 91.25 ± 24.59 mg/dL, total proteins 6.77 ± 0.74 mg/dL, albumin 3.76 ± 0.49 mg/dlL, and hemoglobin 12.40 ± 1.62 g/dL.

DISCUSSION

Body composition studies in obesity clearly show that the increase in body mass is due to accumulation of both fat and LBM in variable but measurable proportions. Significant weight gain both in animals and humans, whether induced by overfeeding, observed during nutritional rehabilitation, or occurring under natural circumstances, almost always consists of a mixture of lean mass and fat mass.[7,20] The relative contributions of these 2 components to the total weight gain depends on the initial status of body composition and the age of the subjects, given that protein intake is adequate.[21] Also during weight loss, fat mass and lean mass participate in the process. It has been known for many years that people who fast continue to excrete nitrogen in the urine, and so must be losing lean mass as well as fat mass. This has always been considered a detrimental effect of weight-loss programs. The construction of special diets claiming "protein-sparing" effects has revitalized the application of body composition studies in weight loss. The evidence shows that the loss of LBM is more marked in thin than in fat individuals, ranging from 50% to 60% of total weight loss in thin to 20% to 30% in fat subjects, demonstrating a clear relationship between initial body fat content and the relative composition of weight loss. All of the data support the idea that, similarly to what happens during weight gain, whenever significant amounts of body weight are lost, both LBM and fat participate in the process.[7,20,21]

We can consider 3 aspects of the problem: the sequential changes in absolute and percent body composition, the absolute changes in visceral and muscular body mass, and the influence of the initial patient status on the final results.

Most of weight loss is due to fat. Patients do lose LBM, on the average 11 kg, but their preoperative values were above

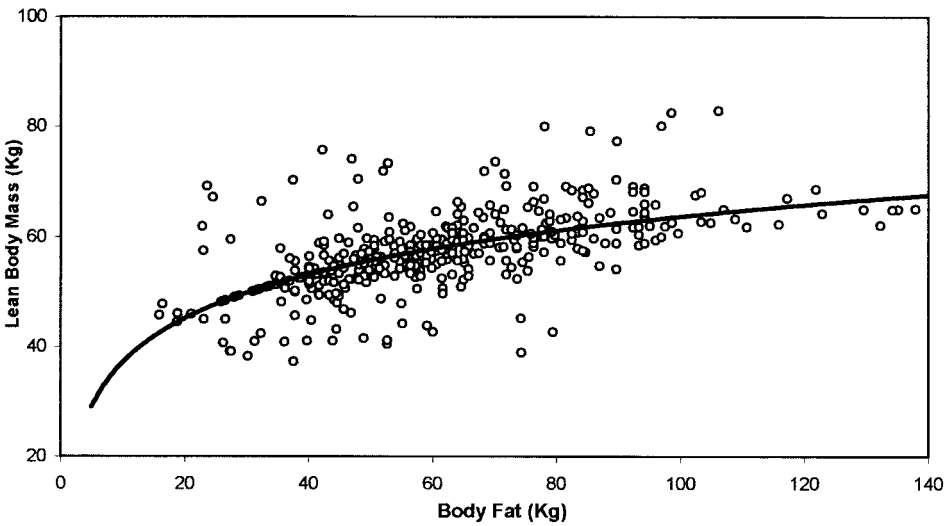

Fig 1. Logarithmic relationship between lean mass and fat mass found in subjects of a wide weight range (101 obese subjects and 53 controls). The regression equation indicated by the line is: $Y = 26.55 \times \log_{10}X + 10.56$ ($R = 0.60$; $R^2 = 0.37$; $F(1, 152) = 260$; $P < .001$).

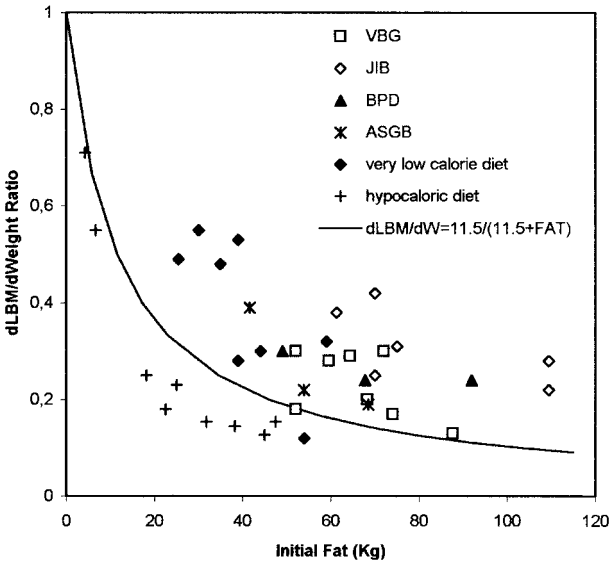

Fig 2. Relative LBM loss in hypocaloric diet subjects and after different surgical procedures, in relationship to initial body fat. VBG indicates patients after vertical banded gastroplasty. JIB indicates patients after jejunum-ileal bypass. BPD indicates our patients after biliopancreatic diversion, divided into 3 groups based on the initial FAT value: <60 kg, 60 to 80 kg, and >80 kg. Very-low-calorie diet patients ingested ≤900 kcal/d (215.03 kJ/d) diet; hypocaloric diet patients, 1,000 to 1,900 kcal/d (238.92-453.95 kJ/d). Symbols represent groups of patients from the literature.[21,29-51] The line represents the differential equation dLBM/dWeight = 11.5/(11.5 + FAT). See text for details.

normal (58.0 kg *v* an expected value of 50.1), as typical of obese patients; their final LBM is still slightly above normal and not significantly lower than the estimated LBM for their height, weight, and age. This means that in a patient who initially has a supranormal LBM, becoming normal after 1 year, the weight loss is primarily due to fat, (mean) 34 kg (see Table 1). The percent fat decreased markedly and, even if the final fat is slightly above normal, the patient has a body composition much closer to physiological values. The patients, in summary, lose a physiological mixture of fat and lean mass.

There is growing interest in studying body segment composition and, in particular, limb skeletal muscle mass in physiological and pathological processes.[22,23] The introduction of DXA makes this aim easier due to its lower cost and less radiation exposure compared, for example, with computerized axial tomography. A sequential assessment of regional body composition can be useful in monitoring weight loss in postsurgical patients. Here, we have shown a good preservation of extremity lean mass in post-BPD patients.

Most investigators have generally considered a 2-compartment model of body composition, consisting of fat mass and fat-free mass (lean mass). It is known that most of the REE is derived from the metabolic activity of the principal internal organs (brain, heart, lungs, kidneys, liver, and intestine) and there is no interindividual difference in specific metabolic rates of various tissues.[15-17]

To date, several studies have investigated the REE-LBM relationship and have predicted REE from tissue-organ mass measurements.[19,24-28] All are in agreement about the correlation of a lower REE-LBM ratio with a greater body mass. This is explained by a nonenergetic homogeneity of LBM: the greater the LBM, the lower the increasing rate of high-metabolism tissues versus low-energy consumption tissues.[19]

A simple mathematical elaboration of body composition and calorimetric data distinguishes between the high-energy-consumption component of LBM, the visceral LBM, and the low-energy-consumption component, the muscular LBM. It is possible, therefore, to elaborate a physiologic 3-compartment model of body composition: fat, muscle, and viscera, each of which is characterized by quite different, quite consistent, REEs. We assume that LBM composition in these patients is normal, ie, it has a normal water content, and, therefore, specific consumptions are those reported in literature. The patients, in fact, were well-nourished, as indicated by clinical examination and hematochemical analysis. So, we postulate that variations in REE per kilogram of LBM are expression of proportional changes in LBM components, ie, viscera and muscle, and not expression of changes in specific muscle or visceral energy consumption; for example, an increase in REE per kilogram of LBM is attributed to increased percentage of visceral versus muscular mass.

We have found that REE is strictly dependent on viscera and muscle mass and on the viscera/muscle ratio, whereas LBM is not in relationship with viscera/muscle ratio: thus, for the same LBM more combinations of muscle and viscera percentage and more values of REE are possible. Subjects with the same LBM but whose visceral mass is higher have a more active metabolism with a higher energy consumption and thus they reach a lower weight of stabilization. Thus, the percentage composition of LBM and, therefore, the metabolic status of the patient is a major factor influencing weight loss after BPD.

It is not predictable, therefore, what is the value of REE on the basis of LBM and its water content, whereas the composition of weight gain or loss from LBM or FAT percentage is predictable.

Viscera/muscle ratio is reduced during weight loss and settles 24 months after operation at values similar to the presurgical ones. We postulate that more visceral than muscular mass is lost in the first months and the viscera/muscle ratio is not constant in the same subject during weight loss, but it is preserved in the steady state. Furthermore, the REE/LBM ratio showed a temporal behavior similar to viscera/muscle ratio: this confirms the importance of viscera/muscle composition of LBM in determining the metabolic status of the subject.

The third aspect is how the final results are influenced by the initial status of the patient. As shown in Fig 1, the preoperative fat content and LBM are highly correlated; this relationship is maintained during weight loss. This means that, in each subject, a change in fat content is accompanied by a proportional change in LBM, a change that is dependent on the initial composition. This equation holds for all weight and fat ranges. The regression equation of Fig 1 can also be differentiated to give:

$$\frac{dLBM}{dFAT} = \frac{11.5}{FAT}$$

If we look at the data of Fig 1, we are convinced that the obese patients indeed have an increased body fat content, but the relationship with LBM holds in the whole range of weights from normal values to extremely overweight. There cannot be

an increase in fat without an increase in LBM. Thinner subjects acquire relatively more lean mass during weight gain than obese subjects, or, inversely, lose more lean mass during weight loss, while obese subjects modify fat mass during weight changes. The reason why this happens is not entirely clear, as mentioned earlier, but obese subjecta have supranormal LBM.

Figure 2 shows LBM changes per unit change of weight in relationship to initial fat content. The higher the dLBM/dW ratio, the higher the contribution of LBM to weight loss. The data show that the contribution of LBM is strongly determined by the initial body composition. The higher the initial fat content, the lower the percentage loss of LBM. The line shown is obtained by graphing the differential equation

$$\frac{dLBM}{dW} = \frac{11.5}{11.5 + FAT}$$

We can see that it very well describes the patient pattern, meaning that fat and LBM are strictly related to each other, both in preoperative obese and normal subjects, as well as in operated patients. The physiological relationship is preserved. In this respect the most obese subjects are privileged compared to thinner patients: as mentioned earlier, they in fact lose more fat mass and less lean mass.

Another contributing factor might be the dietary deficit. The higher the dietary deficit, the higher the dLBM/dW ratio and the lean mass waste for the same initial fat content.

If we compare our results with those derived from previous studies in the literature,[21,29-51] we note that there is a significant difference with jejuno-ileal bypass (JIB) patients: these patients lose significantly more LBM, probably because of the induced malabsorption. The gastric stapling and BPD patients are very similar. The line drawn represents the ideal change of LBM per unit change of weight if the patient preserves the physiological LBM/FAT ratio: patients lying above this line lose an excess LBM, those below preserve it. It is interesting how data derived from studies on hypocaloric diets of 1,000 kcal are very close to ideal, almost as are the BPD patients.

These 2 body components, LBM and fat mass, are so tightly linked that a change in one is accompanied by a change in the other; they are not completely independent entities.[18] This relationship between LBM and fat predicts reasonably well the relative changes in body composition when body weight is gained or lost. An increase in weight is always accompanied by an increase in LBM.[7,18] Both muscle and viscera contribute to gains in LBM, although muscle represents quantitatively the major factor.

In conclusion, we can state that the patient after BPD has a body composition close to physiological values. Despite the large amount of weight loss, LBM decreased to values comparable to control values, meaning that the composition of lost weight is that of the excess weight.

APPENDIX 1

Indicating with:
- r = total REE (kJ/d) (tREE);
- a = specific REE of LBM (kJ/kg/d);
- b = specific fat REE (20.93 kJ/kg/d);
- c = specific muscle REE (45.45 kJ/kg/d) (mREE);
- d = specific visceral REE (675.67 kJ/kg/d) (vREE),
- L = lean body mass (kg) (LBM);
- M = muscle LBM (kg) (mLBM);
- V = visceral LBM (kg) (vLBM);
- F = fat mass (kg) (FAT)

we can write the following system equation:

(1) $L \times a + F \times b = r$

(2) $L = M + V$

(3) $L \times a = M \times c + V \times d$

We can solve for M substituting $L \times a$ in (1):

(4) $M \times c + V \times d + F \times b = r$

(5) $M \times c + V \times d = r - F \times b$

Substituting V with $(L - M)$ from 2) we obtain:

(6) $M \times c + (L - M) \times d = r - F \times b$

(7) $M \times c = r - F \times b - (L - M) \times d$

(8) $M \times c = r - F \times b - L \times d + M \times d$

(9) $M \times c - M \times d = r - F \times b - L \times d$

(10) $M \times (c - d) = r - F \times b - L \times d$

$$(11)\ M = \frac{r - F \times b - L \times d}{c - d}$$

Solving for V yields:

$$(12)\ V = \frac{r - F \times b - L \times c}{d - c}$$

Using text abbreviations we can write:

$$(13)\ mLBM = \frac{tREE - FAT \times fREE - LBM \times vREE}{mREE - vREE}$$

$$(14)\ vLBM = \frac{tREE - FAT \times fREE - LBM \times mREE}{vREE - mREE}$$

For example, putting the mean control values from Table 1 in the equation 13 yields:

$$mLBM = \frac{7680 - 33.5 \times 20.93 - 47.93 \times 675.67}{45.45 - 675.67}$$

$$= \frac{7680 - 701.155 - 32364.593}{45.45 - 675.67}$$

$$mLBM = \frac{-25385.748}{-630.22} = 40.28$$

This is a result comparable with the reported mean of 42.5 kg.

APPENDIX 2

Indicating with "W" total weight and with prefix "d" differential value, and using text abbreviations, we can write:

(1) W = FAT + LBM

(2) LBM = $26.55 \times \log_{10}$ (FAT) + 10.56 (see text)

Differentiating for LBM yields:

$$(3)\ dLBM = \frac{11.5}{FAT} \times dFAT$$

Substituting LBM in (1) we obtain:

(4) W = FAT + LBM = FAT + $26.55 \times \log_{10}$ (FAT) + 10.56

Differentiating for W yields:

$$(5)\ dW = \frac{1 + 11.5}{FAT} \times dFAT$$

Thus, the dLBM/dW ratio may be calculated from the equations (3) and (5):

$$(6)\ \frac{dLBM}{dW} = \frac{11.5}{FAT} \times dFAT \Big/ \frac{1 + 11.5}{FAT} \times dFAT$$

$$= \frac{11.5}{11.5 + FAT}$$

REFERENCES

1. Stunkard AJ, Sorensen TIA, Hanis C, et al: An adoption study of human obesity. N Engl J Med 3114:193-201, 1986
2. Prentice AM, Black AE, Coward WA, et al: High levels of energy expenditure in obese women. Br Med J 292:983-989, 1986
3. Forbes GB, Welle SL: Lean body mass in obesity. Int J Obesity 7:99-106, 1983
4. Stokholm KH, Brochner-Mortensen J, Hoilund-Carlsen PF: Increased glomerular filtration rate and adrenocortical function in obese women. Int J Obesity 4:57-66, 1980
5. Bray GA, Gallagher TF Jr: Manifestation of hypothalamic obesity in man. Medicine 54:301-306, 1975
6. Pitts GC: Density composition of lean body compartment and its relationships to fatness. Am J Physiol 202:445-458, 1962
7. Forbes GB: Human Body Composition: Growth, Aging, Nutrition and Activity. New York, NY, Springer Verlag, 1987
8. Scopinaro N, Gianetta E, Civalleri D, et al: Bilio-pancreatic bypass, in Griffen WO, Printen KJ (eds): Surgical Treatment of Morbid Obesity, vol 12. New York, NY, Dekker, 1987, p 93
9. Tacchino RM, Castagneto M, Marino F, et al: Calorimetric evaluation of weight loss and maintenance mechanism after biliopancreatic diversion for morbid obesity. Int J Obesity 13:600-609, 1989
10. Mazess RB, Howard SB, Bisek JP, et al: Dual energy x-ray absorptiometry for total-body and regional bone mineral and soft-tissue composition. Am J Clin Nutr 51:1106-1112, 1990
11. Wang J, Heymsfield SB, Aulet M, et al: Body fat from body density: Underwater weighing vs dual photon absorptiometry. Am J Physiol 256:E829-839, 1989
12. Milliken LA, Going SB, Lohman TG: Effects of variations in regional composition on soft tissue measurements by dual-energy x-ray absorptiometry. Int J Obesity 20:677-682, 1996
13. Tataranni PA, Ravussin E: Use of x-ray absorptiometry in obese individuals. Am J Clin Nutr 62:730-734, 1995
14. Bertin E, Marcus C, Ruiz JC, et al: Measurement of visceral adipose tissue by DXA combined with anthropometry in obese humans. Int J Obesity 24:263-270, 2000
15. Holliday MA, Potter D, Jarrah A, et al: The relation of metabolic rate to body weight and organ size. Pediatr Res 11:185-195, 1967
16. Grande F: El consumo de oxigeno del cerebro. Rev Clin Espanola 39:1-17, 1950
17. Brozek J, Grande F: Body composition and basal metabolism in man. Correlation analysis versus physiological approach. Hum Biol 27:22-31, 1955
18. Nelson KM, Weinsier RL, Long CL, et al: Prediction of resting energy expenditure from fat-free mass and fat mass. Am J Clin Nutr 56:848-856, 1992
19. Heymsfield SB, Gallagher D, Kotler DP, et al: Body-size dependence of resting energy expenditure can be attributed to nonenergetic homogeneity of fat-free mass. Am J Physiol Endocrinol Metab 282:E132-E138, 2002
20. Benedict FG: A Study of Prolonged Fasting. Washington, DC, Carnegie Institute, 1915
21. Forbes GB: Lean body mass-body fat interrelationships in humans. Nutr Rev 45:225-231, 1987
22. Gallagher D, Visser M, De Meersman RE, et al: Appendicular skeletal muscle mass: Effects of age, gender and ethnicity. J Appl Physiol 82:229-239, 1997
23. Wang W, Wang Z, Faith MS, et al: Regional skeletal muscle measurement: Evaluation of new dual-energy x-ray absorptiometry model. J Appl Physiol 87:1163-1171, 1999
24. Holliday MA: Metabolic rate and organ size during growth from infancy to maturity and during late gestation and early infancy. Pediatrics 47:169-179, 1971
25. Gallagher D, Belmonte D, Deurenberg P, et al: Organ-tissue mass measurement allows modeling of REE and metabolically active tissue mass. Am J Physiol Endocrinol Metab 275:E249-E258, 1998
26. Heymsfield SB, Gallagher D, Wang Z: Body composition modeling: application to exploration of the resting energy expenditure fat-free mass relationship. Ann NY Acad Sci 904:290-297, 2000
27. Illner K, Brinkmann G, Heller M, et al: Metabolically active components of fat free mass and resting energy expenditure in nonobese adults. Am J Physiol Endocrinol Metab 278:E308-E315, 2000
28. Wang Z, Heshka S, Gallagher D, et al: Resting energy expenditure-fat-free mass relationship: New insights provided by body composition modeling. Am J Physiol Endocrinol Metab 279:E539-E545, 2000
29. Jacobson G, Seltzer CC, Bondy PK, et al: Importance of body characteristics in the excretion of 17-ketosteroids and 17 ketogenic steroids in obesity. N Eng J Med 271:651-656, 1964
30. Keys A, Brozek J, Henschel A, et al: The Biology of Human Starvation. Minneapolis, MN, University of Minnesota Press, 1950
31. Young CM, Di Giacomo MM: Protein utilization and changes in body composition during weight reduction. Metabolism 14:1084-1094, 1965
32. Parizkova J: Body Fat and Physical Fitness. The Hague, The Netherlands, Martinus Nijhoff , 1977
33. van Seters AP, Harinck HIJ, Moolenaar AJ, et al: Changes in body composition on Modifast, and electrolyte retention during carbohydrate refeeding. 4th International Congress on Obesity, New York, NY, October 5-8, 1983 (abstr)
34. Belko AZ, Meredith MP, Kalkwarf HJ, et al: Effects of exercise on riboflavin requirements: biological validation in weight reducing women. Am J Clin Nutr 41:270-277, 1985
35. Bogardus C, LaGrange BM, Horton ES, et al: Comparison of carbohydrate-containing and carbohydrate-restricted hypocaloric diets in the treatment of obesity. Endurance and metabolic fuel homeostasis during strenuous exercise. J Clin Invest 68:399-404, 1981
36. Young CM, Scanlan SS, Im HS, et al: Effect of body composition and other parameters in obese young men of carbohydrate level of reduction diet. Am J Clin Nutr 24:290-296, 1971
37. Phinney SD, Horton ES, Sims EA, et al: Capacity for moderate exercise in obese subjects after adaptation to a hypocaloric, ketogenic diet. J Clin Invest 66:1152-1161, 1980
38. Ditschuneit H, Wechsler JG, Ditschuneit HH: Clinical experience with very low calorie diet, in Blackburn GL, Bray GA (eds): Management of Obesity by Severe Calorie Restriction. Littleton, MA, PSG Publishing, 1985, pp 319-334
39. Brown MR, Klish WJ, Hollander J, et al: A high protein, low calorie liquid diet in the treatment of very obese adolescents: long-term effect on lean body mass. Am J Clin Nutr 38: 20-31, 1983
40. Warnold I, Carlgren G, Krotkiewski M: Energy expenditure and body composition during weight reduction in hyperplastic obese women. Am J Clin Nutr 31:750-763, 1978
41. Lantigua RA, Amatruda JM, Biddle TL, et al: Cardiac arrhythmias associated with a liquid protein diet for the treatment of obesity. N Engl J Med 303:735-738, 1980
42. Archibald EH, Harrison JE, Pencharz PB: Effect of a weight-reducing high-protein diet on the body composition of obese adolescents. Am J Dis Child 137:658-662, 1983
43. Dore C, Hesp R, Wilkins D, Garrow JS: Prediction of energy requirements of obese patients after massive weight loss. Hum Nutr Clin Nutr 36C:41-48, 1982
44. Vaswani AN, Vartsky D, Ellis KJ, et al: Effects of caloric restriction on body composition and total body nitrogen as measured by neutron activation. Metabolism 32:185-188, 1983
45. Bjorntorp P, Carlgren G, Isaksson B, et al: Effect of an energy-reduced dietary regimen in relation to adipose tissue cellularity in obese women. Am J Clin Nutr 28:445-452, 1975

46. Woo R, Garrow JS, Pi-Sunyer FX: Voluntary food intake during prolonged exercise in obese women. Am J Clin Nutr 36:478-484, 1982

47. Busetto L, Perini P, Giantin V, et al: Relationship between energy expenditure and visceral fat accumulation in obese women submitted to adjustable silicone gastric banding. Int J Obesity 19:227-233, 1995

48. van Gemert WG, Westerterp KR, et al: Reduction of sleeping metabolic rate after vertical banded gastroplasty. Int J Obesity 22:343-348, 1998

49. Busetto L, Tregnaghi A, Bussolotto M, et al: Visceral fat loss evaluated by total body magnetic resonance imaging in obese women operated with laparoscopic adjustable silicone gastric banding. Int J Obesity 24:60-69, 2000

50. van Gemert WG, Westerterp KR, van Acker BAC, et al: Energy, substrate and protein metabolism in morbid obesity before, during and after massive weight loss. Int J Obesity 24:711-718, 2000

51. Laimer M, Ebenbichler CF, Kaser S, et al: Markers of chronic inflammation and obesity: A prospective study on the reversibility of this association in middle-aged women undergoing weight loss by surgical intervention. Int J Obesity 26:659-662, 2002