Mechanisms of Weight Loss After Surgery for Clinically Severe Obesity

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Obesity is the most common chronic disease in the United States, affecting more than one-third of adult Americans. About 5% of obese individuals are considered to have clinically severe or morbid obesity, defined as a body mass index (BMI) ≥40 kg/m². When considered in aggregate, the effect of obesity on the nation's health is enormous.

Obesity is an independent risk factor for all-cause mortality, diabetes mellitus, hypertension, coronary artery disease, stroke, dyslipidemia, gallstones, and certain malignancies, with risk increasing in proportion to the degree of overweight. There are also negative social and psychologic effects associated with obesity and the well-being of its victims. So widespread are individual and societal concerns about obesity that surveys indicate that at any given time, 33% to 40% of adult women and 20% to 24% of men are trying to lose weight, with an additional 28% involved in weight maintenance. The cost of commercial weight loss programs alone is estimated at more than $30 billion annually, and the economic impact of the treatment of obesity and its related conditions is enormous, accounting for more than 8% of annual health-care costs.

Treatment of obesity is generally divided into medical and surgical treatment. Medical treatment usually consists of dietary and/or drug therapy in combination with behavior modification and exercise, while surgery is reserved for patients with clinically severe or morbid obesity. The extreme difficulty associated with obesity treatment is reflected by its high rates of failure and recidivism. Little scientific data is available documenting the effectiveness and safety of most commercial weight loss regimens. Medically supervised weight loss programs, usually consisting of very low calorie diets in combination with behavior modification and exercise, have met with modest success, but suffer from a high degree of weight regain. Drug treatment has also produced limited success and is being subjected to intense scrutiny due to public and regulatory concerns over the safety of long-term treatment. In the morbidly obese, the efficacy of these modes of treatment is even less. Regarding the efficacy of nonsurgical approaches to weight loss on severely overweight individuals, the National Institutes of Health's Technology Assessment of Methods of Voluntary Weight Loss (1992) stated: "Although acceptable weight reduction may be achieved, a major drawback to the nonsurgical approach is failure to maintain reduced body weight..."
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In the vast majority of patients. The possibility should not be excluded that the highly motivated patient can achieve sustained weight reduction by a combination of supervised low-calorie diet and prolonged, intensive behavior modification.13

In contrast to medical treatment, surgical treatment of severe obesity has met with considerable success. In evaluating the role of surgery in the treatment of clinically severe obesity, a National Institute of Health sponsored Consensus Conference panel evaluating Surgical Treatment of Obesity (1991) concluded:” The surgical procedures in use [gastric bypass and vertical banded gastroplasty] can induce substantial weight loss [in severely obese patients], and this, in turn, may ameliorate most of the comorbid conditions studied.”14

To better understand the results and benefits of surgical treatment of obesity, it is important to have a working knowledge of the mechanisms of weight homeostasis and weight loss associated with different treatment modalities.

Control of Body Weight

The etiology of obesity is exceedingly complex, with varying contributions from hereditary and environmental factors. Studies of twins and adopted children support a strong genetic component in some cases of obesity.15,16 Similarly, variations in eating patterns and taste preferences among obese patients may be partly hereditary.17,18 However, despite the presence of many seemingly uncontrollable influences, body weight is ultimately governed by the first law of thermodynamics—namely, that energy consumed must equal energy expended. Thus, obesity results when a positive energy balance exists due to increased intake, decreased expenditure, or both.

In general, body weight is tightly controlled, with most adults fluctuating very little from year to year. The defense of body weight has been well demonstrated in numerous human and animal studies.19 In the 1940s, men who were fed a semi-starvation diet and lost 25% of their weight returned to baseline when allowed to resume a normal eating pattern. Conversely, in the 1960s, prisoner volunteers who were overfed to the point where they gained weight also returned to their baseline weights when permitted to eat ad lib. These data have led to the suggestion that there is a “set-point” for weight, similar to that for body temperature, so that the body maintains compensatory mechanisms designed to restore or protect weight in the event of change19 or a buff-

Body Composition and Energy Metabolism

Energy expenditure is composed of three parts: basal metabolic rate (BMR) or resting energy expenditure (REE), thermic effect of food (TEF), also known as dietary-induced thermogenesis (DIT), and the thermic effect of exercise or activity (TEE).22

BMR is the minimal energy expenditure of an organism necessary for life, normally constituting about two-thirds of daily energy expenditure, with the heart, liver, kidney, and brain accounting for approximately 60% of REE.23 In practice, because of the difficulty in measuring BMR, the MREE, which is the energy expenditure of an organism measured at rest in a postabsorptive state, is used. If not measured by calorimetry or double-labeled water, RMR is usually estimated using the Harris-Benedict equation and is a function of height, weight, gender, and age. BMR and REE are predictably lower in women and the aged, because these two populations have a greater percentage of body fat than lean body mass (e.g., skeletal muscle, bone, visceral protein). However, obese individuals have a greater metabolic rate than lean individuals, largely due to the fact that obese individuals, in addition to having significantly greater fat mass, have more fat-free mass (FFM) or lead body tissue, which is more active metabolically. FFM is responsible for about 75% of REE.24,25

The TEF, which is the amount of energy consumed due to eating and digestion, is responsible for approximately 5–10% of energy expenditure. For this reason it is more energy efficient to eat several smaller meals over the course of a day than one or two large meals. Whether TEF in obese patients is lower26 or the same as27 it is in lean counterparts is controversial. However, it is clear that TEF represents the smallest component contributing to daily energy expenditure.

TEE is the most variable component of 24-hour energy expenditure. Obese and lean subjects are equally efficient and expend equivalent amounts of energy for the same activity.28 However, for any given activity in which obese individuals need to support their own weight, such as walking or run-