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Controversies in Plastic Surgery: Suction-Assisted Lipectomy (SAL) and the hCG (Human Chorionic Gonadotropin) Protocol for Obesity Treatment

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Abstract. The advent of SAL (suction-assisted lipectomy) has dramatically increased the number of obese patients coming to our consultation offices. Despite several articles suggesting a conservative approach to fat suction, some reports insinuate that SAL might be a useful tool for obesity treatment. This hypothesis is refuted by a vast body of evidence that concludes that the adipose tissue may regenerate in adult humans. Therefore, surgical procedures are not advised as the method of choice to manage the disease. On the other hand, the terms obesity and being overweight may not be interchangeable. Obesity may be a disease whereas being overweight is a *sign* of the disease. Consequently, proper preoperative selection of candidates for SAL becomes mandatory. The hCG (human chorionic gonadotropin) method for obesity treatment appears to be a complete program for the management of obesity. It contains pharmacologic, dietetic, and behavior modification aspects in a 40-day course of treatment. Some data suggest hCG to be lipolytic, thus explaining former clinical observations regarding body fat redistribution in treated patients. hCG commercial preparations contain β-endorphin, an opioid peptide linked to mood behavior. This article speculates on the possible actions of the complex hCG β-endorphin in the neuromodulation of mood and energy metabolism. The method comprises a behavior modification that helps in handling the patient better. There are some correlations between a current behavior modification program and the basic guidelines contained in the hCG protocol. Thus, the hCG method appears to be a reasonable alternative in the management of a long-standing, unsolved problem of human metabolism.

Key words: Adipose tissue — Metabolism — Endorphins — physiology — Obesity, treatment — Gonadotropin(s), chorionic, pharmacodynamics — Gonadotropin(s), chorionic, therapeutic use

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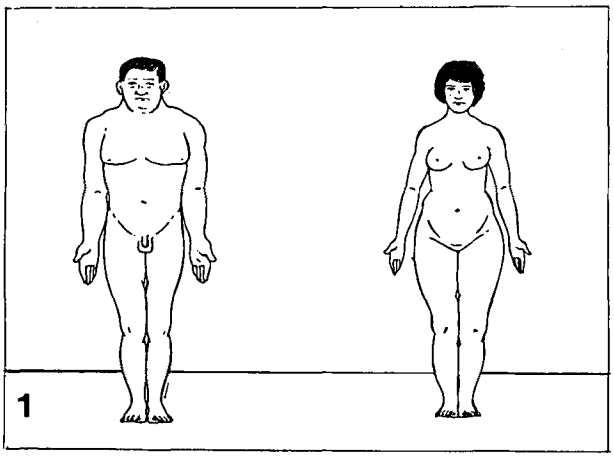
Introduction

SAL (Suction-Assisted Lipectomy) and Obesity

Few surgical procedures aroused as much interest from plastic surgeons and the lay press as SAL did. Pioneered by the early reports of Fischer and Fischer [103] and Schrudde [273], SAL reached its heyday after the publications of Illouz [159–162] and Kesselring [186–189]. Actually, SAL has become the "prima donna" in the surgical armamentarium of plastic surgeons, and nearly no part of the human anatomy is spared, including the thighs, knees, neck, buttocks, calves, arms, breasts, flaps, back, and abdomen. Patients under and over the age of 50 have, therefore, experienced the back-and-forth movement of a cannula connected to a suction pump [72–73b, 107, 128, 145–146, 186–189, 250, 308a–b, 326]. Cautious words about SAL are scarce [69, 124, 322] compared with the myriad of enthusiastic reports. The number of SAL performed surpasses any other aesthetic surgical procedure and this tendency is growing [4].

Nevertheless, because SAL is a novel surgical technique, its precise indications remain the center of dispute. Articles have been published that propose a conservative approach to fat suction, whereas diverse publications suggest SAL may be an useful tool in the therapy of obesity [241]. The differences between the criteria are not of mere academic interest. If SAL is the appropriate maneuver



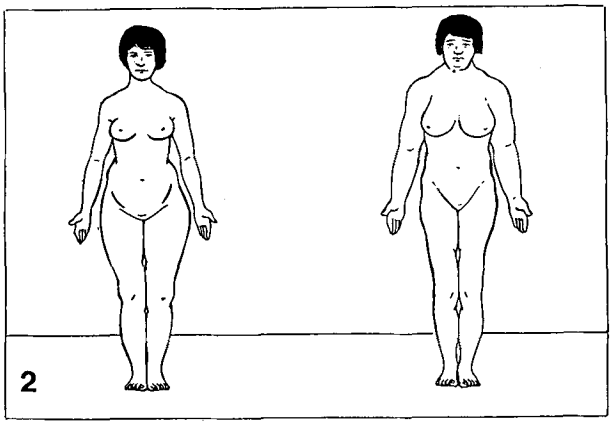


Fig. 1. Android **(left)** and gynoid **(right)** types of obesity

Fig. 2. The hypothetic long-term result from an overzealous SAL. Patient with a classical gynoid-type of obesity **(left)** showing waviness in thighs region and an android redistribution of fat after a SAL **(right)**. This "new" body fat distribution forewarns a higher incidence of metabolic complications

for the management of the obesity, then plastic surgeons would receive the merit of having developed an easy procedure that solves a disease that has been present for thousands of years. But if SAL *is not* the adequate method for obesity therapy, then preoperative patient *selection* becomes of utmost importance.

Body Fat Distribution and SAL

Fat suction might have adverse effects in obese patients: it is well known from Vague's report that there exist two types of obesity, depending on body fat distribution: a gynoid type and an android type [312, 314] (Fig. 1). The gynoid type of obesity tends to accumulate fat in the hips, buttocks, thighs, and lower abdomen. In the android type, adipose tissue largely localizes in the back, shoulders, and upper abdomen. Gynoid fatness is more resistant to dieting. The android type is easier to treat but shows an increased incidence of clinical complications, such as hyperlipemia, hypercolesterolemia, diabetes, hypertension, diabetes, and gout [313, 316].

Excessive fat removal by SAL may stimulate, in the long run, counterregulatory balances resulting in adipocyte hypertrophy and/or hyperplasia from the adipocytary pool. Faust and Kral concluded: ". . . rapid weight gain after a lipectomy cannot involve tissue that is no longer present, so it may require hypertrophy of the remaining tissues." [96]. In the case of gynoid obesity, this compensatory growth after a lipectomy (or an overzealous SAL) may occur in the upper part of the body. Thus, by force of surgical treatment a "cosmetic" obesity may evolve into a "medical" one [Fig. 2]. This modification of body fat distribution certainly does not benefit the patient. When compared with gynoid fatness, the android type of obesity shows an increased cardiovascular risk [313, 316].

Alternatively, counterregulatory mechanisms may generate adipocytary hypertrophy and/or hyperplasia in the liposuctioned area. Figure 3 shows an obese patient twice liposuctioned elsewhere, showing recurrence both of obesity and body contour deformity.

In our opinion, SAL has a definite place in body contour surgery provided that it is performed in small quantities, in conspicuous fat accumulations, and for an aesthetic improvement of the body contour (Fig. 4) [325]. Therefore, today's plastic surgeons should bear the responsibility for selecting those patients who would benefit from a preoperative weight reduction program. During the past seven years we have insisted that candidates for a body contour surgical procedure should correct their obese condition prior to the operation itself [319–325]. This is imperative, because of the increased numbers of moderately obese patients coming to our consultation offices.

In our experience, a most difficult issue is the selection of an adequate obesity therapy suitable to our plastic surgical requirements as well. Weight reduction programs offering an acceptable weight loss but poor skin tone are fairly common (Fig. 5).

After many trials, we concluded that the hCG (human chorionic gonadotropin) program suited our

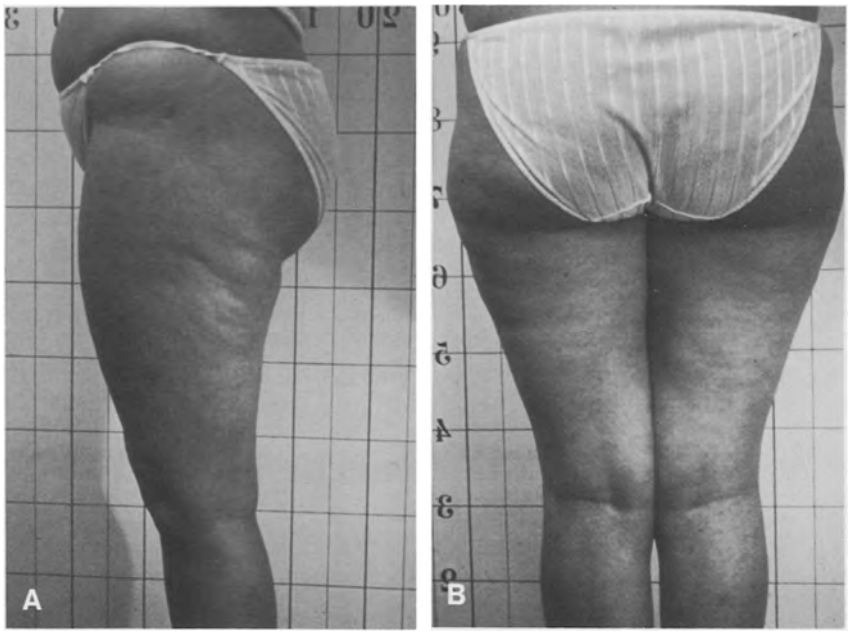


Fig. 3. Obese patient twice liposuctioned elsewhere showing recurrence of obesity and body contour deformity

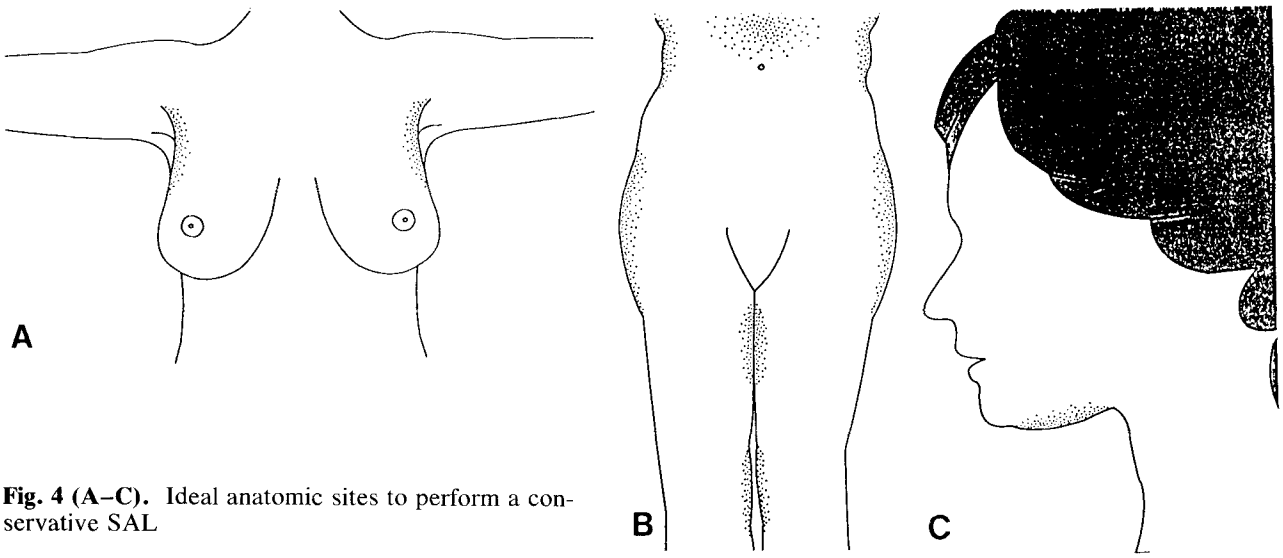


Fig. 4 (A–C). Ideal anatomic sites to perform a conservative SAL

needs and goals: rapid weight loss, excellent body contour, and good skin tone after treatment (Fig. 6). As is well known, this striking approach to obesity provoked several years ago a growing wave of criticism [6, 14, 19, 28, 42, 60, 61, 74, 108, 130, 140, 224, 247, 256, 276, 294, 337]. Nevertheless, because of recent data suggesting hCG might be lipolytic *in vivo,* we believe the whole subject deserves a new evaluation. For this objective we have mapped out a working hypothesis on the subject. Consequently, the purposes of this article are (1) briefly review some new trends on obesity classifications and summarize current concepts on obesity and adipose tissue metabolism and (2) discuss several lines of evidence that suggest that the hCG method is a complete pharmacologic, dietetic, and behavioral modification program for obesity treatment.

Part I: Overview of Obesity and Adipose Tissue

"Observe that the things which are considered to be right today are those which were considered to be impossible yesterday. The things which are thought wrong today are those which will be esteemed right tomorrow." *Hudhaifa*

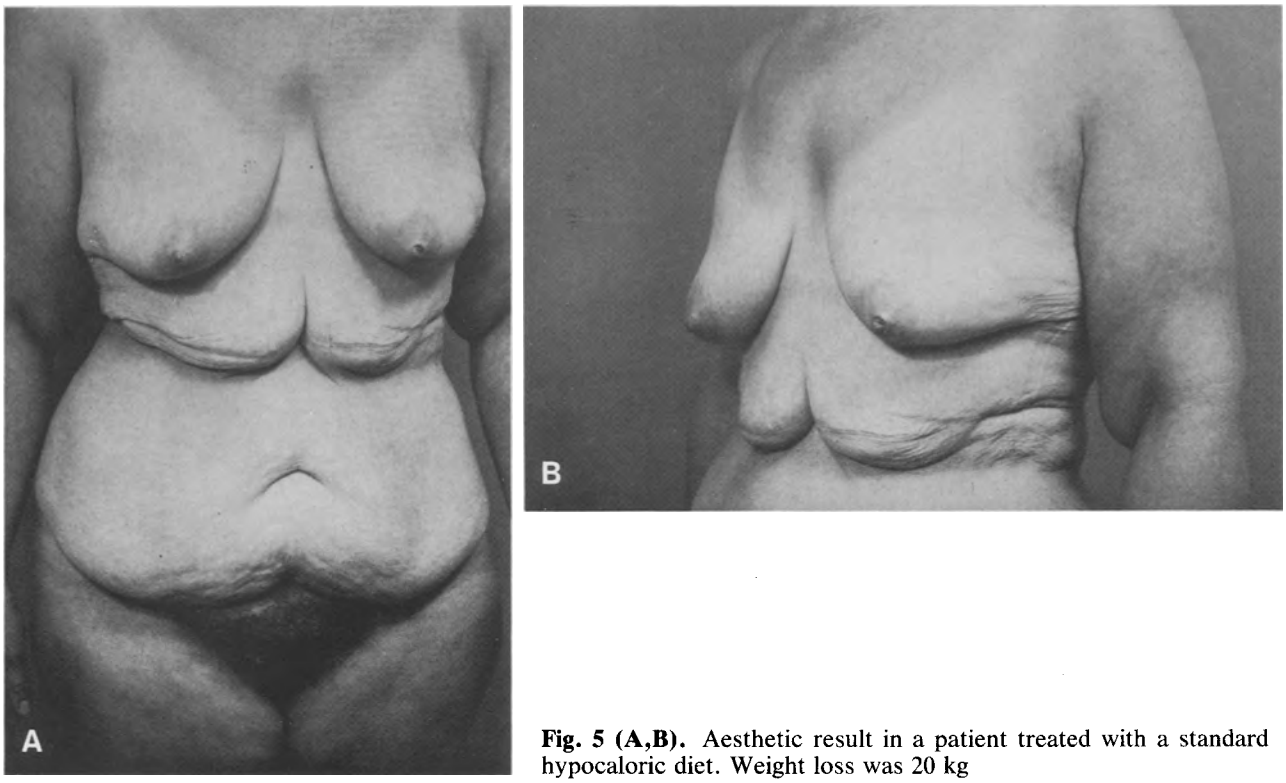


Fig. 5 (A,B). Aesthetic result in a patient treated with a standard hypocaloric diet. Weight loss was 20 kg

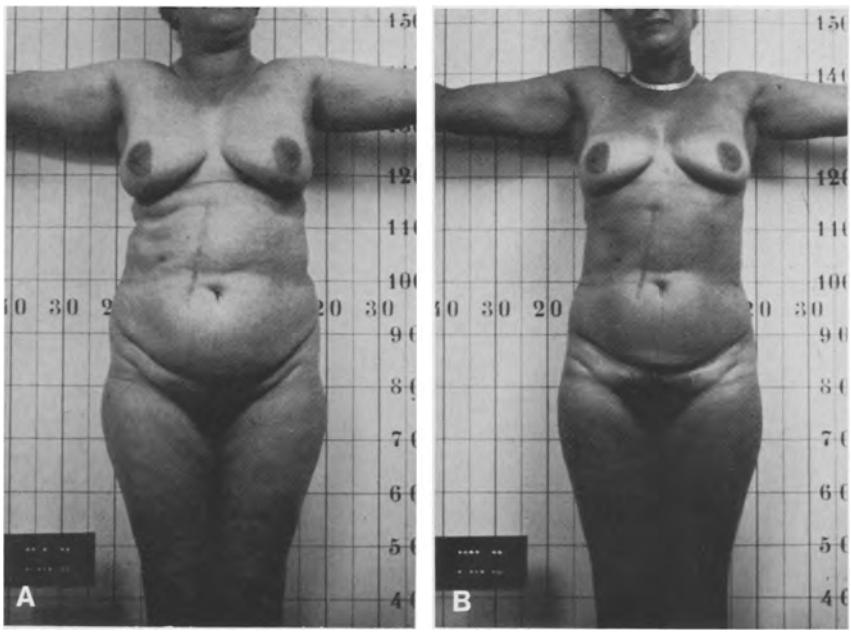


Fig. 6. Patient before **(A)** and after **(B)** a full course of treatment (40 days) under the hCG program. Weight loss was 16.3 kg

OBESITY

Classification of Obesity

Clinical signs other than weight and height are currently relevant in the assessment of the obese patient. A considerable body of evidence suggests that fat distribution plays a prognostic role in the evaluation of the disorder of obesity. As mentioned before, Vague's report was a major advance on the subject. Similar conclusions regarding the clinical importance of body fat topography were put forward by several authors. Kissebah's laboratory

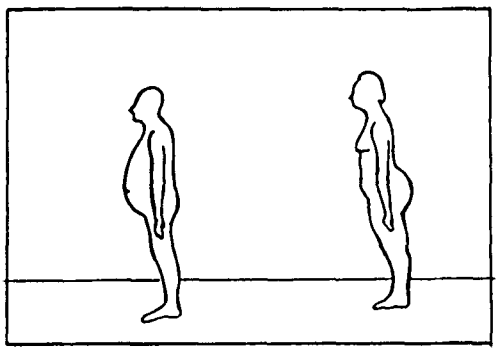


Fig. 7. Abdominal **(A)** and gluteofemora, **(B)** types of obesity. (Redrawn from [11])

classified the obesities in UBSO (upper body segmental obesity) and LBSO (lower body segmental obesity) [92]: UBSO is characterized by a WHR (waist to hip ratio, the relationship between waist and hip circumference) close to or above 1. LBSO shows a WHR below 1. A WHR close to or above 1 forewarns of clinical complications such as atherosclerosis, heart strokes, and infarcts. The Swedish school categorized obesity in abdominal and gluteofemoral types (Fig. 7) [31a–b, 201, 204]. The former is more prone to metabolic complications (diabetes, hypertension, heart strokes), but it is easier to treat than the latter.

The NHANES survey designed a classification based on the relationship between the BMI (body mass index) and the sum of skinfold thickness (166a–b). Thus, individuals may be classified as obese–overweight, obese–lean, overweight not obese, and lean not obese. The incidence of hypertension and hypercholesterolemia was higher in the obese-not-overweight group.

Recently, the National Institutes of Health proposed a major breakthrough on this subject. The panel concluded that a treatment for obesity was advisable even in discretely overweight patients, provided that a family history of obesity, obesity-related diseases, or personal antecedents of obesity-related diseases was present [235]. Consequently, obesity was declared a disease. Being overweight was not the single diagnostic tool used to characterize the disorder [231, 235].

"Not by Food Alone"

The overall state of obesity treatment remains a disappointing subject [22, 34, 58, 83, 84, 121, 123, 129, 163, 184, 212, 239, 292, 300]. Notwithstanding the social pressures to keep pounds off, statistics show success in the opposite direction [1, 53, 191, 235]. Interest in the disease and research on the topic are relatively new. Until the early 1940s, the adipose tissue was a neglected subject [119], and obese patients were generally blamed for gluttony, cheating, lack of will power, and greed [123, 127, 173, 184]. Many students of obesity adhered to the nihilistic attitude that obesity is caused simply by overeating, and that it can be cured only by undereating. Despite the patients' efforts, however, for many the disease remained "incurable" [121].

Fortunately, not everyone shared this gloomy opinion of the disease. A group of researchers felt obesity might be characterized by a basic disorder of energy metabolism. Direct and indirect data contributed to the researcher's conclusions.

Indirect Data

Neumann [232] conducted a study on himself. During a prolonged control period he varied his daily food intake significantly. His weight, however, remained stable. He concluded that somehow his body managed to get rid of the surplus caloric intake.

Similar conclusions were advanced by Gulick [138] and Passmore [244]. The term "luxusconsumption" was coined to describe these clinical observations. The next step in the research was to investigate the mechanisms whereby the organism maintained a relative constancy in body weight. Miller and Mumford [222] proposed that the extra ingested calories were dissipated by heat loss during exercise, a conclusion not unanimously agreed to by other investigators [48]. Despite contradictory evidence, it soon became apparent that there was no direct relationship between daily food intake and body weight.

Sims published a classic report on this topic. In his study, normal healthy individuals fed with a high caloric diet (up to 4000 kcal/day) maintained a fairly stable weight during the test period. In those cases where weight was increased, normalization was attained by restoring subjects to a normal daily food intake. Sims concluded: "A primary disturbance of the mechanisms which monitor energy balance of the body, and which regulate food intake, could secondarily lead to metabolic and endocrine changes; these in turn could contribute to perpetuate obesity [280, 281]." Interestingly, not all the volunteers for the study showed the same response to a hypercaloric diet.

Edholm [89], after a series of clinical experiments measuring caloric intake and energy consumption in soldiers, concluded that there were no significant

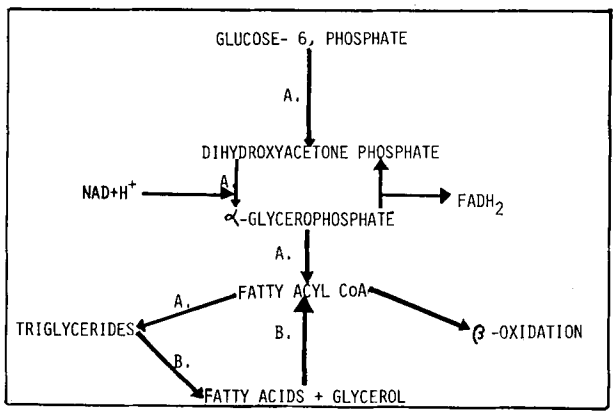


Fig. 8. Possible "wasteful" or "futiless cycles" in lipid metabolism. Letter "A" points to the diversion of carbohydrates into the fatty acid synthesis cycle; letter "B" to the hydrolysis–esterification cycle. Both metabolic pathways are less conservative from the bioenergetic viewpoint

correlations between body weight and daily food consumption.

Direct Data

Direct evidence from studies of obese patients were reported by several authors. Miller [223] demonstrated that under well-controlled conditions, a percentage of dieting obese patients failed to lose weight when compared with their counterparts. Since the study was performed on an in-patient basis, the failures could not be ascribed to a poor adherence to the diet. The reasons why these obese individuals were resistant to change remained unexplained.

Different reports reached the same conclusions: Obese subjects did not always overeat in the expected quantities. Some of them were, in fact, eating the same amount or less than their lean counterparts [17, 44, 170, 171a–b, 246, 293].

Theories on Obesity Genesis

Several theories have been proposed to explain the process whereby obese individuals maintain an abnormal high regulation of body weight. Keesey et al. suggested the concept of a "set point" for body weight regulation [179–181]. Consistent with his reports, the organism regulates a stable body weight by means of a precise system tuned to a fixed "set point". Keesey et al. proposed that obese patients possess an abnormally elevated set point. Thus, their organisms adjust energy metabolism to an increased level of body weight.

Some data contend that obese patients may show an altered activity of "futiless" metabolic cycles [167, 298]. This implies the continual cycling of substrates through a series of synthetic and degradative reactions which have the same initial and final energy status. This type of reaction is energy demanding and results in loss of the energetic efficiency of the system, dissipating a great amount of heat. The following "futiless cycles" have been suggested bear some relevance in obesity (Fig. 8): (a) the fatty acid synthesis–oxidation cycle and (b) the triglyceride hydrolisis–reesterification cycle. Both cycles involve the HSL (hormone-sensitive lipase). This is activated by adrenaline, thus explaining their lipolytic and probably their calorigenic affects on adipose tissue [298].

The issue of whether obese patients show an increased metabolic efficiency, or a thermogenic defect, still remains an open question [23, 88, 117, 142, 176, 277, 283]. More recently, it has been suggested that obese patients may show an impairment of FFA (free fatty acid) mobilization from adipose tissue [210].

Conclusions

Obviously much work remains to be done in this fascinating field of energy metabolism. However, from our perspective the following conclusions may be drawn:

1. Nonobese individuals preserve a stable body weight regardless of wide fluctuations in daily caloric intake [66, 70].
2. Similar regulatory mechanisms seem to operate in obese patients. Unfortunately for them, this regulatory system maintains an increased level of body weight despite periods of forced dieting [206].

With respect to the processes involved in this bioenergetic cycle, Hirsch has elegantly concluded: "The persistence of obesity in the face of well-publicized information on the health hazard of obesity . . . . suggests to me that there is a more subtle problem of obesity that many of us have been lead to believe" [151].

Obesity: A Multifactor Disorder

Evidence suggests that obesity is a multifactor disorder. A huge body of data concludes that genetics [9, 39, 41, 52, 54, 125, 167, 297, 304, 329], the environment [24, 43, 81, 82, 87, 156, 193, 245, 261, 301a], psychological traits [75, 122, 252, 258], socioeconomic level [5, 12, 259, 301a], development [90, 172, 182, 183, 205, 328], and a CNS (central nervous system) disturbance [7, 21, 49, 116, 175, 197, 253] contribute to the genesis or the maintenance of the disorder. Thus "nature" and "nurture", in variable

Table 1A. Site differences in subcutaneous fat metabolism after one week of therapeutic fasting of obese subjects (from [11])

Metabolic event	Abdominal fat	Femoral fat
Basal metabolism	Profound changes	Less profound changes
Catecholamine action	No change	Increased α-adrenergic effect Inhibition at post receptor level
Antilipolytic effect of insulin	No change	Increased sensitivity
Fat cell size	Decrease	No change

Table 1B. Site differences in subcutaneous fat metabolism after one week of therapeutic fasting of obese subjects (from [11])

Fat cell size	F > A[a]
Basal lipolysis rate	F < A
Basal lipoprotein lipase activity	F > A
Insulin action	F > A
Catecholamine action	F < A

[a] F = femoral fat cells; A = abdominal fat cells

proportions, are equally important in the consideration of obesity [17, 37, 51a, 56, 77, 135, 152, 168, 339].

Adipose Tissue

Heterogeneity and Multiple Physiological Aspects of the Adipose Tissue

The adipose tissue is not a uniform mass randomly distributed throughout the human body. Depending on topographic localization, adipocytes possess different sensitivities to hormones, enzymes, drugs, and fasting periods [10, 11, 38, 91, 177, 203, 287, 288, 306, 315]. The processes of lipolysis and lipogenesis in adipocytes are subject to the action of several hormones and drugs (for review see [78]). It has been observed that during fasting catecholamines inhibit femoral fat lipolysis in women [10, 11]. According to Arner [10, 11] these findings may be explained by the recent observation that the β-adrenergic receptor number is decreased in femoral fat pads during therapeutic fasting [10, 11, 240]. Insulin binding to adipocytes is modified in different fat deposits during therapeutic fasting (Tables 1a and 1b) [91]. On the other hand, some evidence concludes that the adipose tissue is quite an active mass as far as the metabolism of FFA [78], steroid hormones [97a,b], and amino acids [310] is concerned.

Lipoprotein Lipase (LPL)

Lipoprotein lipase (LPL) is an enzyme that hydrolyzes plasmatic VLDL (very-low-density lipoproteins) and chylomichrons, thus releasing FFA from the intravascular lumen. The adipocyte takes up these FFA and reesterifies them to triglycerides (TG) in the interior of the fat cell (Fig. 9). Several reports suggest that a high LPL adipose tissue activity, as seen in some obese subjects, predisposes them to increased fat storage [131, 238, 257, 274].

LPL activity was found to be higher in the femoral than in abdominal regions in women, except during lactation. During lactation, however, LPL activity is decreased in women's femoral fat pads. These findings suggest that femoral adipose tissue possesses a particular metabolic specialization during lactation [254].

Steroid Hormones

There exists an extremely large pool of steroid hormones in adipose tissue, several times that observed in plasma [97a,b]. Consequently, the adipose mass may be an important variable of steroid hormone metabolism in humans.

Adipocyte Regeneration in Adult Individuals?

Earlier reports suggested that the adipose cell does not multiply in adults [29, 30, 47, 149, 268, 269]. Based on this preliminary data, it was speculated that SAL could be the appropriate tool for the treatment of obesity [162]. Nevertheless, this enthusiastic surgical approach to a longstanding problem of human metabolism soon was over shadowed by a series of reports that concluded that the adipocyte may, indeed, multiply in adult individuals. Evidence of this came from experiments in animals and the long-term followup of obese patients.

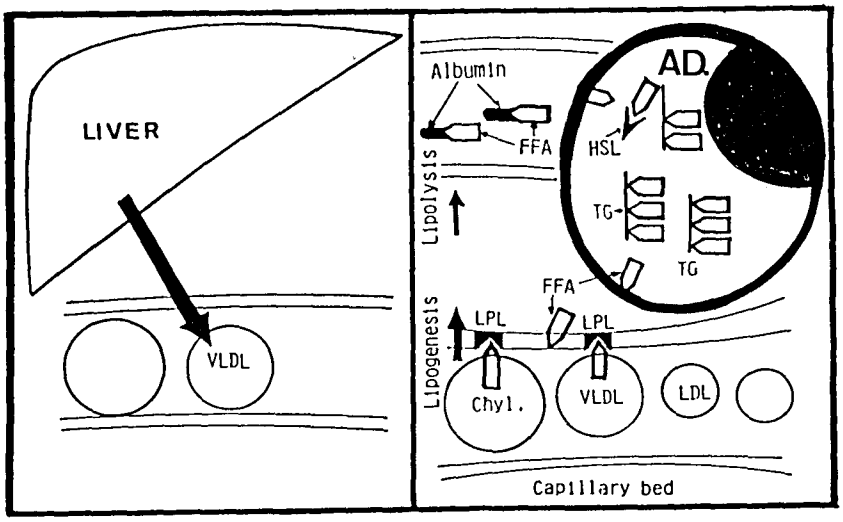


Fig. 9. The mechanism of action of LPL (lipoprotein lipase). The enzyme hydrolyzes VLDL (very low density lipoproteins) and Chyl. (chylomicrons) from plasma, releasing FFA (free fatty acids) in the extracellular space. The adipocyte (Ad.) uptakes these FFA and reesterifies them back to TG (triglycerides). When needed, these TG are hydrolyzed back to FFA by the HSL (hormone-sensitive lipase) and released in the circulation coupled to the albumin (Alb.) fraction.

Adipocyte Regeneration in Rodents

Roth [264] demonstrated that after a lipectomy, the remaining fat cells multiply in rats. Miller [226] showed evidence concerning a *de novo* production of adipocytes in adult rats. Faust showed that under certain conditions, specific rat adipose tissue pads may regenerate [93, 96].

Adipocyte Regeneration in Humans

Foley [105] concluded that overeating leads to recruitment of new adipose cells in moderately obese patients. Sjöstrom et al. suggested that the adipose cells multiply in adult subjects [282a,b]. One of his reports was a long-term followup of obese women [282a]. Kral [199a,b], in a followup of three lipectomized women, observed that one had regained the weight she had before the operation, a second patient had to keep a rigorous diet to maintain her weight, and there is no data concerning the third patient. Kral concludes: "Surgical reduction of fat mass does not seem to prevent a future weight gain." Taken together, these data suggest that the total adipose mass is under the control of some type of regulatory mechanism. This "homeostatic" system might compensate for eventual losses of fatty tissue through hypertrophy and/or hyperplasia from the adipocytary pool.

Regulation of the Adipose Tissue Mass

Maintenance of a fairly stable weight throughout life or the recovery of the adipose mass after surgical interventions strongly suggests that there exists a CNS (central nervous system) mechanism that controls fat deposition and release in adipose tissue (for review see [332]). The proposal of the hypothalamic region as the regulatory organ appears plausible [209, 332]. The hypothalamus has been reported to play a regulatory role in the menstrual cycle [20, 100], cardiac frequency [227], and immunity [76]. Thus, it is reasonable to assume that body energy homeostasis is modulated in the hypotalamic region as well [251, 332] (Fig. 10). However, a major drawback to this hypothesis lies in the difficulty to extrapolate some clinical conditions as observed in humans (e.g., obesity) with the results of experiments in animals.

Part II: The hCG Method for Obesity Treatment

"There are, it may be, so many kinds of voices in the world, and none of them is without significance"

I Corinthians

Introduction

As is well known, the first protocol for the management of obesity with hCG was reported in *Lancet* [278] by the late Dr. A.T.W. Simeons. However, previous publications concluded that hCG was a useful drug for the treatment of certain clinical presentations of adolescent obesity, except Fröhlich's syndrome [65, 116].

Since 1966 this clinic has managed obese patients with the hCG method. As experience was gained by treating 12,000 obese subjects, modifications to the original protocol were introduced. Uniform results, excellent body contour after treatment, and absence of clinical complications are the main characteristics of this outstanding form of obesity therapy. The

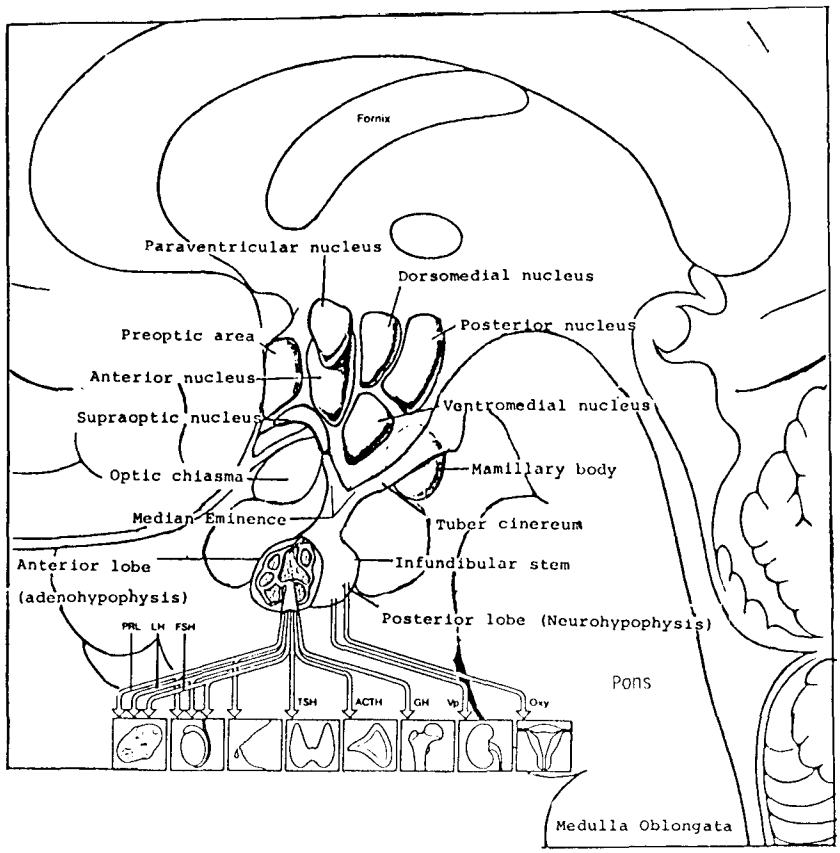


Fig. 10. The hypothalamic region. Different hypothalamic nuclei and their relationships to pituitary gland can be seen. PRL: prolactin; LH: luteinising hormone; FSH: follicle stimulating hormone; ACTH: adrenocorticotrophic hormone; TSH: thyroid stimulating hormone; GH: growth hormone. Hormones from the posterior part of pituitary: Vp: vasopresine; Oxy: oxitocyne

procedure was accepted [80] until the mid-1970s when, for several reasons, it fell from credibility:

1. An excessive proliferation of the so-called "fat clinics." These institutions injected hCG under totally uncontrolled conditions [19]. Unproper management of the hCG protocol resulted in an increased rate of clinical complications [86].
2. An overestimation of the real therapeutic possibilities of hCG. Publicity lead the people to believe that hCG was the "magic wand" that would cure their disease.
3. A series of clinical tests, which nearly all [61, 74, 108, 130, 140, 224, 276, 289, 294, 337] but one well-controlled one [14] concluded that the method was of no use for obesity treatment.

We have postulated elsewhere [320a–325] that hCG is *not* the magic solution to cure obesity. A daily injection of hCG gives optimum results *only when used in a rational weight reduction program. Therefore, strict observation to the complete protocol is mandatory.*

A Hypothetical Framework

This clinic is engaged in a study program on the subject. We have developed a working hypothesis based on the results of our clinical experience, on recent evidence from the field of obesity research, and on some speculative hypotheses. The latter were introduced when experimental data were not available. The model is incomplete and much work remains to be done to test the validity of these hypotheses.

A Working Hypothesis in Obesity Therapy. The basic postulates of our model are (1) obesity is not the same as being overweight, (2) obesity has physical signs, (3) human obesity might be characterized by a hypothalamic disorder, (4) the hCG method comprises pharmacologic, behavior modification, and dietetic aspects. The pharmacologic aspects are (a) hCG is lipolytic *in vivo*, (b) hCG may act at the hypothalamic level, (c) hCG affects mood behavior.

1. Obesity is not the same as being overweight. As we have seen before, recent data indicates that obesity is a different clinical entity than being overweight. Several lines of evidence contribute to this hypothesis:

Gynoid type of obesity is preserved from the clinical complications appearing in android obesity. When compared with similar weights, the latter appears more "malignant" than the former [313, 316].

Recent laboratory tests have reported abnormalities suggesting metabolic complications of obesity in normal or near-normal weight subjects

[267]. Therefore, current classifications of obesity in terms of body weight may not correlate with clinical severity of the disorder.

According to the NHANES survey conclusions, incidence of hypertension is higher in obese but not overweight individuals [166a,b].

The National Institutes of Health consensus panel concluded that appropriate timing to treat obesity may depend on clinical variables other than height and weight [235].

Taken together, these data indicate that obesity and being overweight are not interchangeable terms: the former may be a clinical disease, whereas the latter could be a sign of disease, not a disease itself [50]. Should this be true, then the assessment of obese subjects should include variables other than height and weight alone.

2. Does obesity have physical signs? As far as we know, it was Dr. A.T.W. Simeons who described for the first time physical signs that he considered typical of obesity [279]: (1) one or two folds of skin around both sides of the back or the chest, (2) the presence of a fat pad on the nape of the neck in an otherwise moderately obese patient, (3) a noticeable valgum of the knees, (4) a fat pad inside the knees. These physical signs could be "clinical markers" used to separate obese individuals from those who are simply overweight. Obese patients should be treated with a more energic weight reduction program because they show a higher incidence of clinical complications.

3. Obesity might be characterized by a hypothalamic disorder. Notwithstanding recent data that suggest that the adipocyte might be the main cause of obesity [94, 95, 132, 262], there is much evidence that the total body fat mass is regulated by a central nervous system modulatory system [209, 214, 332]. Therefore, despite genetic influences may predispose adipose cells to accumulate lipids [41, 79, 132, 147], the overall activity of fat deposition and release must depend on an integratory circuit, which should control the metabolic activity of diverse fat cells all over the organism.

A well-studied topic is the relationship between hypothalamic experimental lesions and the development of obesity in rats. In 1939, Hetherington and Ranson [143, 144] reported that small electrolytic lesions in the VMN (ventral hypothalamus) resulted in hyperphagia and obesity. Though the first publications focused on the metabolic and endocrine disorders accompanying hypothalamic lesions, later on several reports insisted that a basic modification in eating behavior (hyperphagia) heralded the onset of metabolic abnormalities (hyperinsulinemia) [8a,b, 185, 286, 295, 296]. However, recent investigations suggest this interpretation may be incorrect. A current formulation for these syndromes proposes that neurally mediated hyperinsulinemia is the primary factor contributing to excessive fat accumulation [164, 165]. This concept is not shared by all investigators [134, 295, 296].

Despite the controversy on the subject, it is generally agreed the hypothalamus somehow plays a regulatory role in the mechanism of energy metabolism regulation [67, 112, 120, 158, 169, 209, 214, 215, 225, 243, 251, 272, 332]. Nevertheless, a major problem lies in the extrapolation of the results obtained in animal experiment to the clinical condition of obesity as observed in humans. Except for a handful of cases [49, 64] no demonstrable hypothalamic lesions have been reported in common obesities. Thus, it appears that experimental animal models of obesity are of limited value when considering human obesity (for a review on experimental and genetic models of animal obesity see [51b, 101]).

A reasonable alternative to this problem may be that human obesity might be characterized by a subtle hypothalamic disorder, still not accessible to current diagnostic methods [112, 272]. Indirect evidence that supports this hypothesis is seen in several experiences in humans. Amatruda et al. [7] demonstrated that a group of obese males showed an abnormal response to 100 μg of GnRH (gonadotropin releasing hormone). Jung et al. [174] concluded that women with familial obesity have a hypothalamic function disorder which was not totally corrected after weight loss. Kopelman et al. [195, 196], after studying the prolactin (PRL) response to insulin-induced hypoglycemia, concluded that hypothalamic function is disturbed in massive obesity. The causes for this regulatory disorder are presently unknown.

A Hypothalamic Opioid Disorder in Human Obesity?

Recent data from the opioid research field opened a new perspective in the consideration of human obesity: Obesity might result from an opioid regulatory derangement in the diencephalic region [220]. Several data suggest that CNS opioids regulate energy metabolism [192, 216–218, 335] and ingestion of nutrients [126, 178, 207, 228–230, 270, 285, 335]. One of the best studied neuropeptides is β-endorphin. It has been suggested that this opioid acts upon the mechanism that elicits eating through a "food-rewarding" system. This cycle might function as follows: Food ingestion may increase CNS opioid levels [216]. This creates a "self-gratifying" sensation [62]. Therefore, obese subjects should be compelled to elevate their food intake to maintain an elevated CNS opioid concentration [62].

From this perspective, gluttony observed in obese patients could be explained on a biochemical basis: Addiction to food would be a recognizable

CNS opioid disorder. Following this line of reasoning, food restriction in obese subjects would decrease the content of CNS endorphins, creating a "withdrawal syndrome" similar to that observed in drug addicts. This hypothesis finds partial support in the Gambert et al. report [115] that concludes that fasting decreases the content of hypothalamic β-endorphin in rats.

We hypothesize that modification of the content of hypothalamic opioids may be related to energy metabolism as follows:

1. Hypothalamic neuropeptide hypersecretion in obese patients may create a dependence on food because food intake would increase CNS opioid concentration [and thus self-gratification]. In this case, obesity would be maintained by an elevated energy input. A persistent high food intake level could lead to metabolic changes which may in turn perpetuate obesity [280, 281].
2. The hypothalamus might be part of the diffuse neuroendocrine system, as proposed by Margules [217, 218]. He suggested that opioids are the neuromodulators of this system. Any stressful situation—a diet, for example—could disrupt the homeostasis of the system. In the case of a diet, the period of decreased energy input would be compensated by physiological adjustments in energy metabolism. This counterregulatory phenomenon could result in the maintenance of the body weight "set-point."

Finally, some evidence seems to suggest that the diencephalic region plays a regulatory function in the metabolism of fat deposition and release. Research on hypothalamic neuropeptides may shed new light on the interpretation of obesity. Subtle modification of the diencephalic opioid concentration may be the cause or an indication of an underlying neuromodulatory disturbance. This would, in turn, initiate the metabolic changes that lead to obesity.

4. *Multiple Aspects of the hCG Protocol*

Pharmacologic Aspect: hCG is lipolytic in vivo. According to Simeons [278], obesity was characterized by the presence of an "abnormal" adipose tissue. These fat pads were localized in specific body areas. Simeons suggested that hCG showed an affinity for these fat masses. As far as we know, there are no reports proposing that hCG mobilizes this "abnormal" fat. However, some data demonstrate that hCG can mobilize lipids from adipose tissue.

Fleigelman [104] concluded that the administration of hCG in rats decreased the activity of α-glycerophosphate dehydrogenase and glucose-6-phosphate dehydrogenase from the liver and adipose tissue. This could mean a diminished lipogenic activity in both tissues under hCG (Fig. 11).

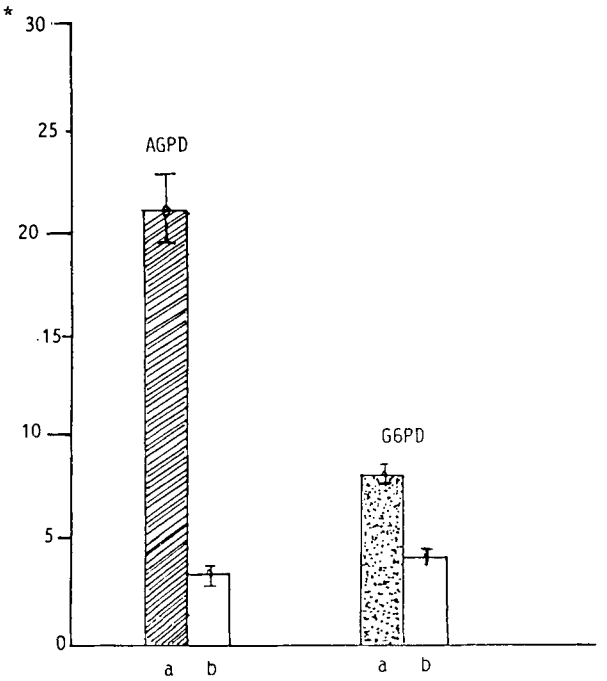


Fig. 11. The activity of two enzymes from rat adipose tissue before **(A)** and after **(B)** hCG administration. AGPD: soluble α-glycerophosphate dehydrogenase; G6PD: glucose-6-phosphate dehydrogenase. (All enzymatic activities are expressed as micrograms formazan/μg nitrogen) (Drawn from [104])

Yanagihara [334] reported that hCG accelerates "not only mobilization of fat from fat deposits, but also its utilization in peripheral tissues. hCG increased the metabolism of injected fat emulsions, suggesting not only the acceleration of not only oxidation of fat, but increased ketone production in the liver and its utilization in peripheral tissues." Romer [260] reported that hCG intensifies the metabolism of rat brown adipose tissue.

Administration of hCG in humans appears to increase the release of free fatty acids that varies with the age of the subjects. Melichar et al. [221] demonstrated that hCG causes a marked FFA release in newborn infants. In adults, a single injection of hCG stimulated the release of FFA by $P > 0.05$ when compared with placebo-treated individuals. This lipolytic action of hCG appears to be mediated. Tell [309] reported that adipocytes do not possess receptors for hCG. Therefore, the lipolytic action of hCG is mediated through an organ or system, which may release a lipid-mobilizing substance in response to hCG stimulation.

Alternatively, chCG (crude, commercial hCG) may exert an *in vitro* lipolytic activity: commercial preparations of hCG contains β-endorphin [139], an opioid peptide with a suggested *in vitro* lipolytic activity [255].

hCG might act at hypothalamic level. At this point, it seems relevant to discuss some data regarding hCG. hCG is a glycoproteic hormone, normally secreted by trophoblastic cells of the placenta during pregnancy [275]. It consists of two dissimilar, separately but coordinatedly translated chains called the alpha and beta subunits [27, 59, 102, 249, 317, 318]. The three pituitary hormones LH (lutenizing hormone), FSH (follicle stimulating hormone), and TSH (thyroid stimulating hormone) are closely related to hCG in that all four are glycosylated and have a dimeric structure comprising Alpha and Beta chains as well. The amino acid sequences of the alpha chain of all four human glycoprotein hormones are nearly identical. the amino acid sequences of the beta subunits differ because of the unique immunological and biological activities of each glycoproteic hormone [263]. β-hCG contains a carboxilic residue of 30 amino acids that are characteristic of hCG [25–27].

Its name, human chorionic gonadotropin originated when it was found that hCG matured the infantile sex glands (gonadotropin) and that it was secreted by the placenta (chorionic) [13, 338]. Recent data suggest, however, that both terms can be quite misleading: normal human tissues [45, 305, 336], plasma from nonpregnant subjects [40, 242], trophoblastic and nontrophoblastic tumors [55, 68, 71, 153, 236, 265, 266, 291, 317], bacteria [3, 16, 213, 219, 284], and plants [85, 109, 110] express hCG or a hCG-like material (for review see [157]). Recently, it has been suggested that this hCG-like material may act as a local growth modulatory factor (D. Belluscio, unpublished).

As far as the scope of this article is concerned, we see that the hypothalamic region is a target organ for the extragonadal actions of hCG. Yaginuma [333] showed that in rats peripherally injected ^{125}I-hCG crosses the blood–brain barrier and accumulates in the hypothalamic region. Hirono [148] reported that hCG has a direct effect on hypothalamic median eminence (ME), inhibiting the synthesis and release of FSH and the release of LH from the anterior pituitary through the hypothalamus. Board [36] demonstrated that hCG administration increases the secretion of the growth hormone in humans. hGH (human growth hormone) may perform lipolytic [98] and calorigenic [46] functions in humans. Thus, hCG could stimulate hGH secretion. This would, in turn, stimulate lipolysis from adipose tissue. Alternatively, and from a purely speculative viewpoint, hCG could stimulate, through the hypothalamus, the secretion of a pituitary lipid-mobilizing factor [57].

hCG and mood behavior. A most intriguing clinical aspect of the hCG program is the sense of well-being observed in treated patients [14, 278, 279, 319–325]. However, these findings were refuted by several publications [130, 224, 294, 337]. Nevertheless, recent data may shed some light on the subject: Hashimoto and Sawai reported that commercial preparations of hCG contain β-endorphin [139], a neuropeptide related to changes in mood behavior [136, 271]. Pure hCG contains β-endorphin as well [2]. Consequently, we hypothesized that the content of β-endorphin in hCG might be responsible for the slight "euphoria" observed in our patients. This opioid might act in the hypothalamic region, an area of major synthesis of β-endorphin [113, 133, 136, 198, 271]. But a major drawback to this supposition lies in the fact that except for a few reports [35, 63, 192], several studies conclude that peripherally injected β-endorphin does not cross the blood–brain barrier, or, if it does, it is either taken up by the brain or broken down with extreme rapidity [137, 154, 200, 208]. Only direct administration to the central nervous system seems to show clinical effects [248].

It occurred to us, from a pure hypothetic viewpoint, that hCG might be the "carrier" for β-endorphin into the brain, delaying its catabolism and facilitating its penetration into the brain: hCG crosses the CNS blood–brain barrier [18], and it accumulates in the hypothalamus [333]. Extremely low concentrations of the complex hCG/β-endorphin at the hypothalamic level should be sufficient to exert a therapeutic effect: β-endorphin is one of the most potent of the tested neuropeptides [136]. Alternatively, β-endorphin could enter the brain at the spinal cord level [118].

The complex hCG/β-endorphin may act in obese patients as follows: (1) The hypothalamic content of β-endorphin decreases during starvation [115]. If the same observation was seen in humans, then exogenous β-endorphin may prevent the withdrawal syndrome that accompanies a dieting period. (2) It could stimulate the secretion of the hypothalamic GHRH (growth hormone releasing hormone) factor and hence lipolysis [98].

Since the above are speculations, they should be read with caution: much work remains to be done to test the validity of these hypotheses.

Behavior Modification Aspect. After Stuart's report [299], data on the utility of a behavior modification program for obesity treatment became available [99, 301b, 302, 303, 327, 330, 331]. The idea behind these programs is that the primary behavior to be changed is eating, and a number of exercises are designed to slow the rate of eating. Former behavioral programs based on only behavior modification had little success [106, 330]. Recently, however, satisfactory long-term results have been reported with a combination of behavior modification and the administration of a very low calorie diet [32, 33, 211].

In our opinion, the protocol hCG contains behav-

Table 2. A comparison between the basic behavior modification techniques comprised in the hCG protocol **(left)** and those from a current behavior modification program **(right)**

(A) Daily visits to the doctor	(A,B) Reinforcement of prescribed behaviors
(B) Daily weighing of the patient	
(C) Extreme sensitivity of the method to daily dietary errors	(C) Self-monitoring of the patients
(D) Modification of daily eating habits	(D) Development of techniques to control the act of eating
(E) A programmed maintenance period	(E) Maintenance period after treatment

ior modification procedures that are similar to the basic guidelines of a standard behavior modification protocol (Table 2).

Dietetic Aspect. Diet plays a specific role in obesity therapy: It decreases energy input thus stimulating energy consumption from fat deposits. No study that we know of has reported complications with the 500-kcal diet. Recently it has been shown conclusively that the use of the very-low-calorie diet for managing of obese patients is safe [114, 155, 190, 237, 290].

Results

First we want to ask: Does a new classification of obesity require a new clinical test? In our opinion, the value of a standard double-blind test to evaluate the hCG program seems questionable. There is no doubt that a 500-kcal diet will render an acceptable weight loss in patients who receive a daily injection of hCG, a placebo, or simply dietary advice [150, 278]. On the other hand, if obesity and being overweight are medical terms that define different clinical conditions, hCG should be tested against a placebo only in obese patients [278, 279]. Such a clinical study has never been done before and would require the close cooperation between a research laboratory and a department of internists. The latter should be fully acquainted with the minimal details of the hCG complete protocol. The research lab should be willing to initiate a research program on this poorly investigated relationship between hCG and obesity.

The following results are from our clinical experience. We prepared a random selection of 450 patients treated with the hCG method between 1971 and 1979. Results can be seen in Tables 3a and 3b. It became clear that weight loss under the hCG protocol is most substantial when compared with standard weight reduction programs. Figures 12–17 show some of our obtained results.

At the total dose indicated for a complete course of treatment with hCG (5000 IU), no complications have been reported. Gonadal hyperstimulation syndrome [15] and acute Meigs syndrome [111] are always related to a higher dose of hCG (20,000 IU or more), generally used in combination with hMG (human menopausal gonadotropin) [233, 307]. Minor complications have been reported: loss of telogen effluvium hair [202, 311] observed in patients subjected to a dieting period and pain at the injection site of a commercially prepared hCG, but not with a different one [141, 234].

In our experience the following minor complications were observed: (1) menstrual cycles disturbances in 0.1% of patients. Several of our obese patients who presented amenorrheic disorders prior to treatment reverted to normal cycles when weight was decreased; (2) hair loss in less than 0.01% of the treated subjects. The hair was of the Telogen effluvium (mature hair) type. Normal regrowth was observed after treatment in all cases. There were no cases of permanent alopecia reported in well over 12,000 treated subject.

Conclusions

The advent of SAL has dramatically increased the number of obese patients coming to our consultation offices. Therefore, a cooperation with a physician who specializes in obesity is of utmost importance. This team work will help in the proper selection of patients and to decide whether a medical weight reduction program should be performed in the first place.

Obesity is a multifaceted disorder. Present classifications include the assessment of height, weight, adipose tissue distribution, and familial antecedents of obesity or obesity-related diseases. Current decisions on the appropriate timing of obesity treatment are based on a careful analysis of the variables listed earlier. Patients who are five or ten pounds overweight should be treated with a weight reduction program when personal or familial antecedents advise it.

The adipose tissue reacts differently to hormones and drugs, depending its topographical localization. Conspicuous body areas seem to be more resistant to fasting because of the particular metabolic char-

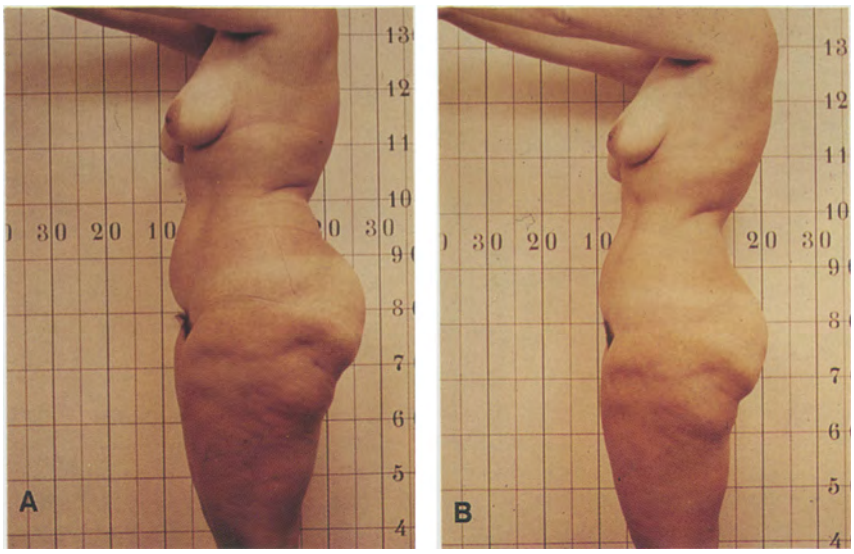


Fig. 12. A 45-year-old patient before **(A)** and after **(B)** a weight loss of 13.5 kg in a 35 day course with the hCG program. Note absence of skin sagging despite the significative weight reduction

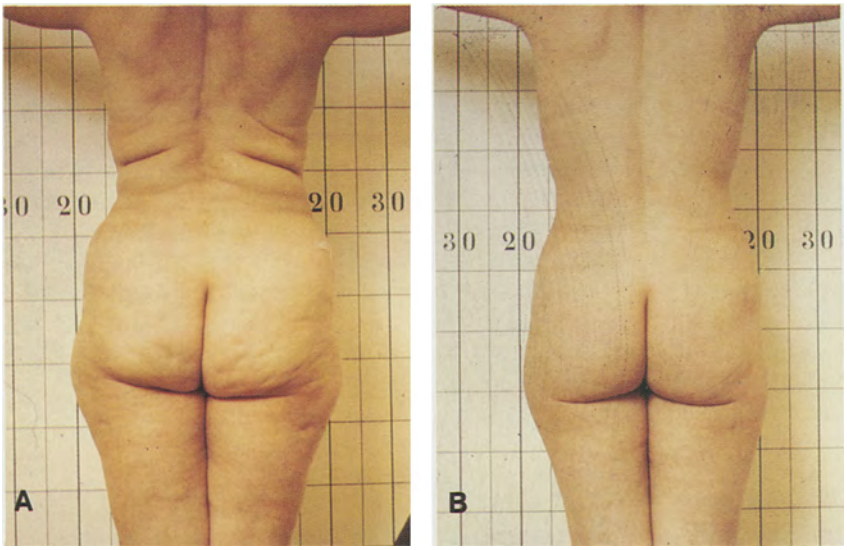


Fig. 13. Harmonious 12 kg weight loss in a 39-year-old patient treated with our protocol (40 days). Physical signs characteristic to obesity have dissappeared **(A)** before; **(B)** after

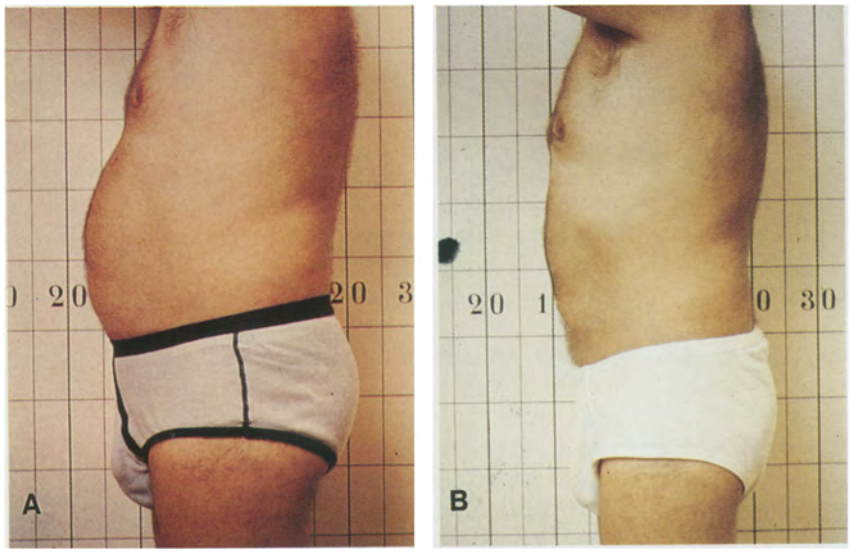


Fig. 14. A 30-year-old male before **(A)** and after **(B)** a weight loss of 17.3 kg, managed with the hCG method. It can be noticed the net improvement of his abdominal type of obesity

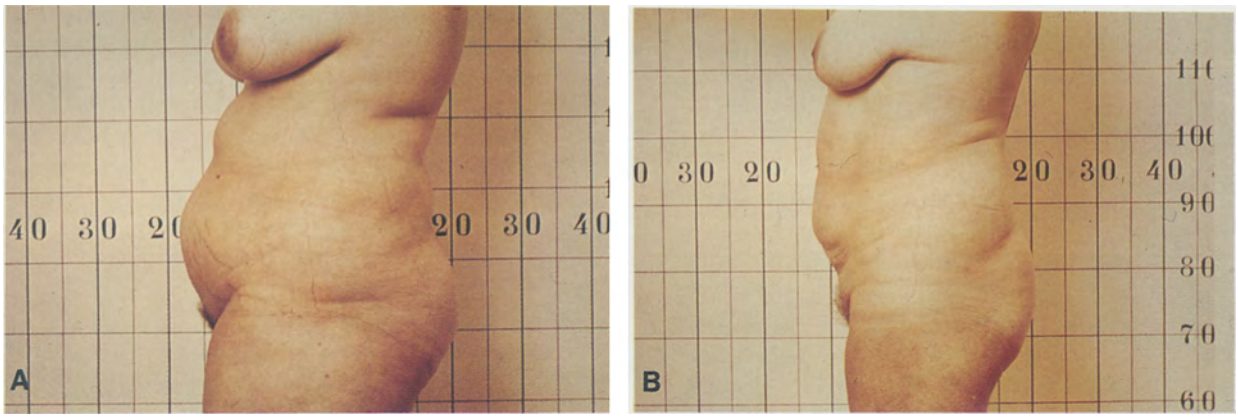


Fig. 15. Obese 51-year-old patient before **(A)** and after **(B)** a weight reduction of 16.2 kg in the course of a hCG treatment (42 days). Observe the dissappearance of striae cutanea from the lower abdomen

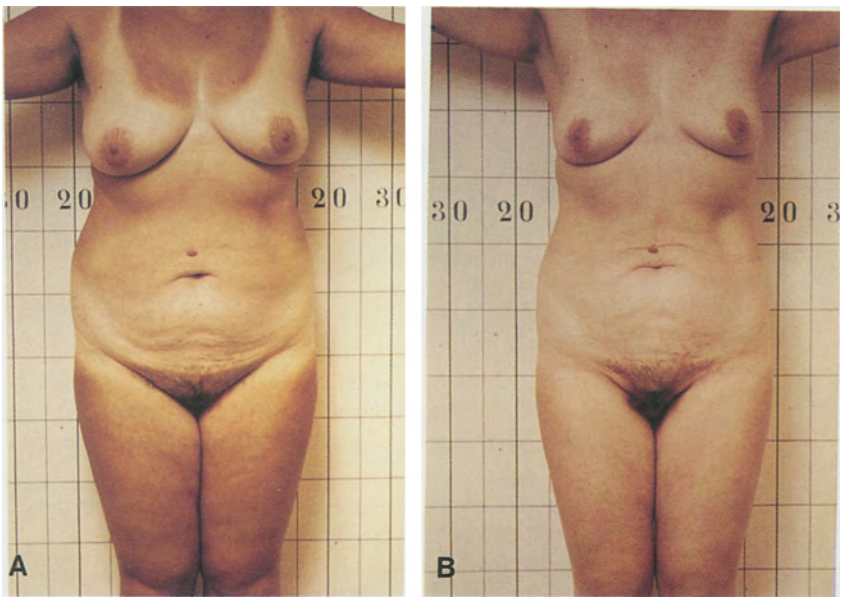


Fig. 16. Obese patient before **(A)** and after **(B)** a weight loss of 12.4 kg after a course of 40 days of treatment with our hCG protocol. Symmetric body fat reduction. Minimal sagginess of skin

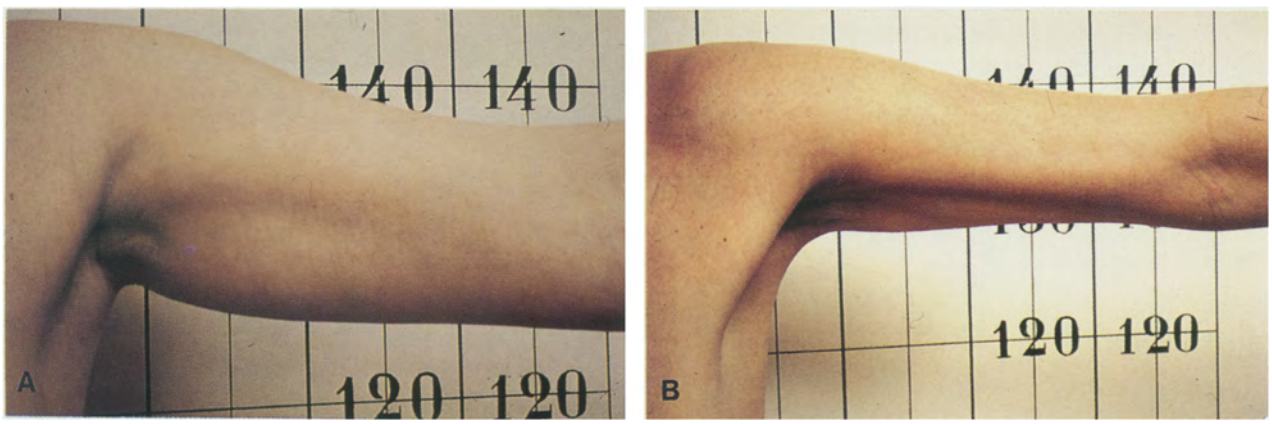


Fig. 17. This photograph shows that proper aesthetic results can be obtained with the hCG method without any surgical procedure: arm from an obese patient before **(A)** and after **(B)** the hCG treatment

acteristics of regional adipocytes. It seems that some cases of "resistant" obesity might be explained by the decrease of the release of free fatty acid from adipose tissue, or a relative decrease in the number of beta-receptors from adipose tissue during therapeutic fasting. This relative decrease in beta-receptor number may favor the alpha action (accumulation of lipids) of hormones.

Individuals tend to maintain a fairly stable weight throughout their life. For the obese, this "set point" of body weight regulation appears abnormally elevated. This hypothesis indirectly proposes that there exist a mechanism that controls fat deposition and release. Some evidence points to the hypothalamic region as the control organ.

Counterregulatory mechanisms tend to compensate for the loss of fat mass. This compensatory growth may occur in body areas where adipocyte hypertrophy and/or hyperplasia could result in increased morbidity. On the other hand, regrowth of adipose tissue in lipectomized areas may result in the recurrence of both obesity and body contour deformity. Thus, surgical intervention on adipose tissue (SAL or lipectomies) is not advised as the method of choice for obesity therapy. Nevertheless, SAL has a definite place in body contour surgery for the management of small, localized fat accumulations.

Table 3A. Randomized study of 450 patients treated between 1977 and 1979 in our clinic with the hCG protocol

Total patients: 450	
Females: 351	
Males: 99	
Age: females: 15–71 years	
males: 16–75 years	
Degree of overweight (%)[a]: females:	54.23% (±25.28)
males:	74.03% (±41.06)
Average treatment (days): females:	41.24
males:	41.68

[a] Degree of overweight is expressed according to the indications of the table published by the Metropolitan Life Insurance Company, 1959

The hCG protocol is a safe, appropriate approach to obesity. It combines pharmacological, behavior modification, and dietetic aspects. When properly managed, it results in a rapid weight loss and excellent body contour. Clinical complications and unfavorable results are related to hazardous modifications of the original protocol.

There is some evidence that suggests that hCG possesses lipolytic activity. Therefore, the basis of use of hCG for obesity treatment might be biochemical. Because hCG does not mobilize lipids *in vitro*, the hypothalamic region might be the intermediate organ in hCG lipolytic action. On the other hand, hCG stimulates hGH secretion. Thus, it was hypothesized that hGH might be the lipolytic hormone secreted to hCG stimulation.

Commercial preparations of hCG contain β-endorphin, and opioid peptide that may affect mood behavior. We speculated that this neuropeptide may be responsible for the sense of well-being observed in our hCG-treated patients.

Since it has been reported that β-endorphin does not cross the blood–brain barrier, it was suggested that hCG might act as a "carrier" for β-endorphin in the brain. Alternatively, β-endorphin may account for an *in vitro* lipolytic activity.

The hCG method comprises a behavior modification program that helps to a better handle obese patients. There is some correlation between the behavioral program included in the hCG protocol and a current behavior modification program for obesity treatment.

The 500-kcal diet as prescribed in the original method proved to be safe and effective.

Obesity is a widespread condition afflicting millions of individuals all over the world. It is a slow killer disease, causing disability, morbidity, and diminution of the quality of life.

Table 3B. Randomized study of 450 patients treated between 1977 and 1979 in our clinic with the hCG protocol

	Before	After	Reduction
Females			
Weight (kg)	81.60(±14.25)	70.61(±10.04)	10.99
Circumference (cm)			
Breast	105.23	98.10	7.13
Waist	89.40	79.10	10.30
Hip	111.64	101.54	10.10
Males			
Weight (kg)	101.60(±16.3)	88.46(±8.04)	13.13
Circumference (cm)			
Waist	107.72	97.05	10.67
Hip	108.97	98.57	10.40

The hCG method of treatment might be a reasonable therapeutic alternative aimed at offering relief to a longstanding unresolved problem of human metabolism.

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