The Treatment of Obesity

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The prevailing concept of obesity is based on the assumption that a gain in weight represents always an excessive intake of food. It is true that investigation of the dietary habits of the obese patient often reveals excessive food intake though no adequate statistics are available to show how large a percentage of the obese population actually overeats and how many eat no more or even less than the average. Considering the prevailing dietary pattern of our population with its emphasis on carbohydrate and fat, we might wonder why most of the people are not obese. On the other hand, physicians who see a large number of obese patients, time and again are confronted with the claim that the patients subsist practically on a starvation diet and even that is hardly adequate to prevent a gain in weight.

These observations, however, are discounted and explained as the result of unadmitted eating, for according to the caloric school, it would constitute a violation of the laws of thermodynamics to consider even the possibility that a person could gain weight on food intake less than the calculated caloric requirements. This view assumes an unchanging efficiency of body metabolism, independent from the internal environment and from the effects of the manifold catalytic agents, hormones, etc. which are supposed to regulate the chemical reactions of the body. The caloric school is not much concerned with the facilities engaged in the limitation of the waste of energy, as the actual heat or in any other form; nor do they like to enter the discussion of the variable energy requirements for tissue synthesis which can be satisfied with fewer calories than we are accustomed to think as necessary (Cannon 2), so that the remaining surplus may become available for storage in the form of fat. Actually the chemical processes which go on in the body are far more complex than for instance the assumed burning of carbohydrate to carbon dioxide; the intricacies of these processes are not suitable for the simplifications of the caloric school which had to give up already some of their former mainstays such as the D/N ratio or ketogenic/antiketogenic ratio as untenable figments of a mechanistic imagination.

According to the caloric theory of obesity, it is maintained that appropriate restriction of food intake reduces weight in accordance with prediction in every case. This is undeniably true for complete starvation which causes weight loss irrespective of the patient's constitution or metabolic status. Even 450-600 calories as recommended by Evans and Strang (3) and other orthodox nutritionists, though not constituting actual starvation levels, do accomplish weight reduction in practically all cases, yet it is doubtful whether such a diet can be enforced for any length of time on ambulatory patients. Thus, a diet calculated for a person supposed to weigh 60 Kg. permits daily 60 Gm. of protein and altogether six to eight calories per Kg. body weight. Such a diet would actually consist of one egg and one ounce of bread for breakfast; one egg and four ounces of the low caloric vegetables for luncheon; a cup of broth and three ounces of lean meat and four ounces of vegetable for dinner. There is no doubt that enforcement of these limitations over a period of ten weeks can produce a weight loss of 37 to 42 pounds, but the claim that patients maintain their weight after termination of this dietary experiment is completely at variance with our own experience. Actually, the weight loss achieved by such near starvation procedures is not necessarily commensurate with the hardship involved and is moreover likely to yield to a rapid gain in weight as soon as the diet is interrupted. It is understandable that the majority of obese patients refuse to follow such a diet for an extended period of time (Rony 21).

Based on such observations, it has been suggested to substitute for starvation, a diet high in proteins but low in carbohydrate and fat content which is supposed to be tolerated well without emotional strain and yet produces satisfactory reduction of weight. The high protein diet is meant to allow quantitatively practically unlimited food intake and does not expose the patient to nitrogen deficiency or inadequacy of vitamin intake. It is regrettable that there are patients who do not lose weight according to prediction on the high protein diet even if the total caloric intake is restricted to 1000 calories per day. On the unlimited protein diet, the number of such refractory patients must be correspondingly greater. Denial of failures does not eliminate them from consideration nor are the facts explained away by the facile generalization that all these patients violate their diet.

It is true that there are obese people who, anxious though they are to lose weight, are unable to observe dietary restrictions; this lack of will power, according to prevailing opinion, is due to psychological causes. For those who assume that overindulgence in food is the sole cause of obesity, it seems clear that the essential pathogenetic factors are of an emotional nature. This belief seems to be borne out by the observation that people who are unhappy either lose their appetite or try to find solace in the consumption of more food. Hence, their gain in weight has been spoken...
of as “anhedonic obesity” (Meyerson 18). It is also true and a matter of common experience that frustration or other complex and often obscure emotional factors can be translated by the patient into overindulgence in food irrespective of the coexistent desire to lose weight. Nevertheless, the claim of the psychological school that weight cannot be reduced unless attention is paid to the patient’s emotional situation (Richardson 20) does not hold true; for over the past 15 years we have successfully reduced the weight of several thousand patients in our clinics; the mass attendance precluded any individual psychological attention, yet the therapeutic results obtained were satisfactory enough to cause a continued increase in the attendance of these clinics. It is equally impossible to agree to another generalization, namely that the frequent coexistence of obesity and neurosis should be interpreted in the sense that the obesity is a manifestation of the neurosis, for the latter causes excessive food intake and hence gain in weight.

It is far more common in our observation that the neurotic state develops as a consequence of the obesity because the latter, especially if combined with undesirable fat distribution and other disfiguring changes of appearance (hypertrichosis), is quite likely to induce emotional instability. The obese, neurotic woman is unsociable, quarrelsome and hard to live with, yet she changes into a nicer, more livable and socially acceptable person after reduction of her excessive weight. Thus it is putting the cart before the horse to claim that the coexistence of these two conditions establishes the neurosis as the cause and obesity as the consequence.

Contrary to earlier assertions in the literature, based on thorough studies on a limited number of patients, that the obese do not deviate essentially from the normal in respect to their metabolism (Newburgh 18), a detailed study of a large obese population has revealed a metabolic pattern which deviates to a statistically significant degree from the normal mean (4). These deviations are most conspicuous in patients whose fat distribution is localized to typical areas of the body. Various stigmata indicative of endocrine disorders are usually associated with abnormal fat distribution.

The association of obesity with an endocrinopathy obviously is no proof of a causal relationship, for the abnormal nutritional state is likely to affect the function of the endocrine glands and account for symptoms of a secondary endocrinopathy. This interpretation of the sequence of events, however, is at variance with the facts for the onset of endocrine symptoms usually precedes the gain in weight. A further bone of contention is the frequency of endocrinopathies in the obese. According to the caloric school this coincidence is unusual and unrelated to the problem of obesity. Apparently, only those extreme instances of endocrine disorders are recognized by the energetic school which do not permit any other interpretation. This may include cases of acromegaly, myxedema, dwarfism and perhaps the adrenogenital syndrome. The manifold manifestations of pituitary deficiency, however, with their unquestionable relationship to genital function, carbohydrate and fat metabolism, water retention, etc., are consistently ignored (5).

On the basis of the preceding discussion, the treatment of obesity should be planned separately for those whose caloric intake is above the average and for those who do not overeat. The overeating group should be subdivided again according to the motivating forces, for the cause of overeating may be simply a habit or may be psychogenic or due to pathological influences.

The habitually overeating person should be systematically educated to control himself and change to reasonable dietary habits. If overeating is a consequence of emotional difficulties, the situation should be dealt with by appropriate psychotherapy. In both cases, patients must be induced to acquire sensible ways of eating. Such voluntary restriction of food intake need not approach the starvation level, especially if the protein content of the diet is sufficiently high. The glutton who consumes 4,000 to 5,000 calories a day loses weight readily if the food intake is restricted 30 or 50 percent, in other words, to a level of average food consumption. Educational measures as well as psychotherapy, however, often need support by procedures which help to curb the patient’s appetite. This purpose is well served by the use of amphetamine sulphate in doses of five to ten mg. t. i. d. Adverse reactions such as insomnia, nervousness, headaches or cutaneous manifestations rarely call for termination of this medication. Another useful procedure is sedation with a mixture of atropine sulphate 1/300 to 1/200 of a grain with 1/4 to 1/2 grain phenobarbital t. i. d. alone, or in combination with amphetamine.

An abnormally increased appetite, however, is not always a mere habit or the manifestation of emotional difficulties, for it may express some organic disease. Thus the appetite of the diabetic is often enormous and his overconsumption of food helps to maintain or even increase his weight. Similarly, hyperthyroidism is often associated with increased appetite and the large consumption of food successfully copes with the patient’s excessive catabolism. Of even greater practical importance is the abnormally increased appetite observed in case of hypoglycemia; the postprandial fall of the blood sugar elicits the sensation of hunger and almost insatiable craving for carbohydrates.

The abnormal appetite referable to diabetes or hyperthyroidism does not need elaboration in the discussion of the therapy of obesity. Management of hypoglycemia, on the other hand, is of particular importance, for no cooperation can be expected from a patient whose blood sugar is permitted to fall to a low level; his craving for carbohydrates cannot be conquered by will power. Such patients must be given frequent meals preferably every three hours, with complete elimination of sugar and all high caloric carbohydrates. Thus the diet consists essentially of proteins, the five and ten per cent vegetables and fruit.