American Handbook of Psychiatry

OBESITY

Revisited

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OBESITY is a condition characterized by excessive accumulations of fat in the body. By convention, obesity is said to be present when body weight exceeds by 20 percent the standard weight listed in the usual height-weight tables. This index of obesity, however, is only an approximate one at lesser degrees of overweight, since bone and muscle can make a substantial contribution to overweight. In the future, diagnosis will probably be based upon newer and more accurate methods of estimating. Skin-fold calipers have already gained acceptance because of their convenience and because half of body fat is localized in subcutaneous tissue. But for most clinical purposes the eyeball test is still the most reasonable: If a person looks fat he is fat.

Epidemiology

Strikingly little information is available about the prevalence of obesity. Since most good diagnostic methods are too cumbersome for use in large-scale studies, much of our information is derived from height and weight data of poor quality, averaged over populations, and subjected to the criterion of 20 percent over standard weight. Present data suggest that prevalence of obesity reaches a peak at age forty when 35 percent of men and 40 percent of women can be designated as obese. There have been studies of more limited

populations utilizing more reliable data that permit more valid inferences. Unfortunately, these studies differ in their criteria of obesity, making their data difficult or impossible to use for comparisons with other studies. These studies show a striking effect of age, with a monotonic increase in the prevalence of obesity between childhood and age fifty, and a twofold increase between ages twenty and fifty. At age fifty, prevalence falls sharply, presumably because of the very high mortality of the obese from cardiovascular disease in the older age groups. Since these studies use the height-weight criterion, and since the fat content of the body increases per unit weight with age, these studies almost certainly underestimate the prevalence of obesity in older persons. The increasing use of skin-fold calipers should soon provide far more satisfactory data.

All studies comparing the sexes report a higher prevalence of obesity among women; this discrepancy is particularly pronounced after age fifty because of the higher mortality rate among obese men in this age group.

Social factors exert a powerful influence on the prevalence of obesity. In many countries undernutrition limits the development of obesity. Where there is no shortage of food, as in an affluent American society, many ethnic groups show a marked increase in the prevalence of obesity in the first generation. Thereafter, a variety of social influences combine to radically reduce the prevalence of obesity. One study reported a fall from 24 to 5

percent between the first and fourth generations in this county.

The most striking anti-obesity influence is that of socioeconomic status. Figure 23-1 shows that obesity is six times as common among women of low status as among those of high status in New York City. A similar, though weaker, relationship was found among men. Two findings suggest that a causal relationship underlies these correlations. First, as figure 23-1 shows, the social class of one's parents is almost as closely linked to obesity as is the subject's own social class. Although obesity could conceivably influence a person's own social class, his obesity can hardly have influenced the social class of his parents. Furthermore, obesity is far more prevalent among lower-class children than it is among upper-class children; highly significant differences are already apparent by age six. Similar analyses have shown that social mobility, ethnic factors, and generational status in the United States also influence the prevalence of obesity.

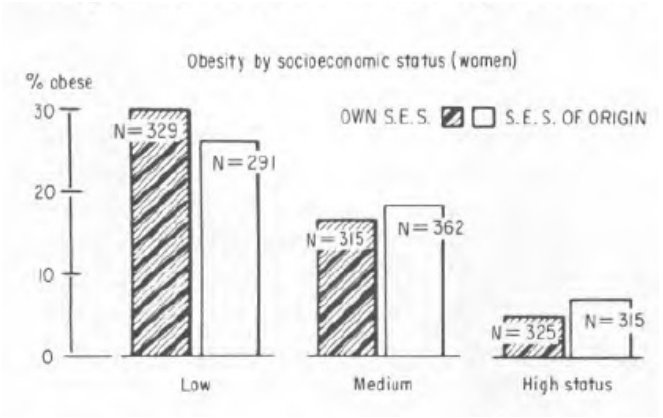

Figure 23-1.
Decreasing prevalence of obesity with increase in socioeconomic status (SES). (From Goldblatt, P. B., et al. By permission of the Journal of the American Medical Association, 192 (1965)11039-1044.)

Genetics

The existence of numerous forms of inherited obesity in animals and the ease with which adiposity can be produced by selective breeding make it clear that genetic factors can play a determining role in obesity. For years, textbooks of biology and medicine have included brief accounts implying that we know a good deal about the topic and that genetic factors play an important part in human obesity. It therefore comes as a surprise to realize the extent of our ignorance about the heritability of human obesity. Not much research has been done and we know very little about it.

Animal models of obesity have helped to put into perspective the either-

or controversy of nature versus nurture; these models have indicated how several possible channels of influence can each have its own particular interaction of genetic and environmental variables, including interactions among and between genes. Genetic influences, for example, may be expressed in determining fat cell number in response to different diets at different periods of development, in regulating the efficiency of metabolic processes, and in establishing the sensitivity of different parts of the nervous system to nutrient depletion and repletion. Already a few such interactions have been studied.

One of the most instructive examples of gene-gene interaction was effected by Mayer's breeding the "waltzing gene" into genetically obese (ob/ob) mice. The increased physical activity of these mice prevented the development of obesity. A similarly interesting example of gene-environmental interaction is the different responses to a high-fat diet observed in two different strains of mice. The C_3H or A strain became fat while the $C_{57}BL$ or I Strain remained thin or lost weight.

With problems of human obesity, there are probably examples of single-gene obesity, comparable in some ways to the genetic obesity of the ob/ob mouse or the Zucker obese rat. But such single-gene obesity in humans, if it exists, seems confined to such rare conditions as the Lawrence-Moon-Biedl and Prader-Willi syndromes. Most human obesity is probably of polygenic

origin and quite possibly encompasses a large number of different conditions. Efforts to study this problem have utilized three methods: the study of familial resemblance, of twins, and of adoptees.

Examining familial resemblance has provided strong support for the familiar belief that obesity runs in families. But little progress has been made in determining whether this phenomenon is the result of genetic or environmental influences, and no progress has been made at all in determining genetic-environmental interactions. Garn's quantitative studies have been the most revealing. He has established that there is a significant correlation between the skin-fold thicknesses of those parents and children who share common genes and environment (r=0.30). However, the correlation coefficient between the skin-fold thickness of marital partners, who share only their environment, is almost as high (r=0.25). These data have led Garn to propose that human obesity is primarily of environmental origin.

The study of twins, the second method of investigating human obesity, accords primary importance to genetic factors. Three studies utilized estimates of heritability based on the difference between the intrapair resemblances of monozygotic and dizygotic twins. All were high: 0.74 for 200 English twin pairs (meaning that 74 percent of the variance was accounted for by heredity), 0.88 for 100 Swedish twin pairs, and 0.78 and 0.77 for 4,000 American twin pairs (studied by the author at the time of induction into the

Army and again twenty-five years later).

The results of the three major adoption studies, the third method of investigating human obesity, are in complete disagreement with each other. Withers reported findings that were originally interpreted as supporting a genetic origin of human obesity. In fact, however, these results are probably more supportive of an environmental origin. More recently, Garn posited a purely environmental origin on the basis of his finding that the correlation of skin-fold thickness between parents and adopted children was the same as that between parents and biological children. Biron, on the other hand, proposed that obesity is of genetic origin. He reported zero correlation between measures of obesity of parents and adopted children and highly significant correlations between parents and their biological children.

Etiology

What causes obesity? In one sense, the answer is simple: Fat is accumulated when more calories are taken in as food than are expended as energy. In another sense, the answer still eludes us. The regulation of body weight in normal-weight organisms is understood only poorly, and in obese organisms it is understood even less well. It does appear, however, that body weight is regulated even by many obese organisms. This idea that body weight may be regulated (even in obesity) rather than being the result of a

number of unconnected influences is relatively new. The evidence supporting it, however, is strong.

The Regulation of Body Weight

It has been known for some time that the body weight of animals of normal weight is regulated. After the body weight of most experimental animals has been altered—lowered by starvation or raised by forced-feeding—it returns promptly to baseline. Only recently, however, has it become clear that animals suffering from a variety of forms of experimental obesity possess the same capacity for regulation. Thus, in animals, at least, obesity need not be due to a disorder in the regulation of body weight, as had been believed in the past. Instead, it can be due to an elevation in the level about which the regulation occurs, a level that has been viewed by some as a regulatory "set point."

We know far less about the regulation of body weight in humans. Their weight tends to be the same year after year, despite the exchange of vast amounts of energy. For example, the average nonobese man consumes approximately one million calories a year; his body fat stores, however, remain unchanged during this time because he expends an equal number of calories. An error of no more than 10 percent either in intake or output would lead to a thirty-pound change in body weight within a year. There are only

two studies of perturbation of this system in humans, and each supports the idea of regulation. Sims found that normal-weight volunteers who were fattened by overfeeding and underactivity returned to their normal body weight without any special effort soon after they resumed their usual patterns of eating and activity. Keys' classic study of experimental semi-starvation showed that when subjects were permitted free access to food, their body weight also rapidly returned to normal. There are no similar studies of obese persons and we do not know whether they regulate body weight. The evidence, however, suggests that they do not.

Lifetime weight histories of obese persons rarely show a level about which body weight appears to be regulated. How can we explain this curious phenomenon? Why should obese people seem to be the only organisms that do not regulate body weight?

Nisbett has proposed an ingenious explanation for this apparent failure of regulation. According to his theory, obese people may well have the capacity to regulate body weight. However, the set point about which their weight would be regulated, if only biological pressures existed, is higher than that which is tolerated by the society in which they live. As a result, such people go on reducing diets. And even if their weight does not fall to normal levels according to the height/weight charts, it still falls below what would be biologically normal for them. The result is the paradox of people who are

statistically overweight and biologically underweight. Nisbett has described seven ways in which the biology and the behavior of obese people resemble that of people whose usually normal body weight has been reduced by starvation or other caloric restriction. In brief, they act as if they are hungry.

Nisbett's theory, attractive as it is, poses a major problem—it cannot be tested, at least not directly. Such a test would require, first, that obese people gain to their putative body-weight set point and, second, raise or lower their weight to see if it would return to this (elevated) baseline. This test is unfeasible on theoretical as well as on ethical grounds, since the putative set point cannot be estimated.

Although Nisbett's theory cannot be tested directly, indirect tests are possible. One such test, which will be described, supports the theory at least as far as *some* obese people are concerned. It was the results reported by Björntorp of the treatment of obese people who possess such an excess number of fat cells that they can reduce to a statistically normal weight only by reducing the lipid content of their individual fat cells below normal values.

The second test of Nisbett's theory has been carried out by Herman, who has shown that some obese people (and some nonobese people) who habitually exercise restraint in the amount they eat share psychological characteristics that distinguish them from persons who do not restrain their

food intake. Such "restrained eaters," for example, may show "counter-regulation" of food intake and eat to excess when their habitual restraint is disinhibited. The range of such disinhibiters is impressive and includes dysphoric emotions such as anxiety and depression, alcohol, and even a high-calorie preload in a taste-testing experiment. Indeed, Herman has proposed that most of the behaviors attributed to the "externality" of obese persons, as is suggested by Schachter's popular theory, is actually due to disinhibition of restrained eaters, who are more common among obese than among nonobese populations. Since this restraint may prevent obesity in persons of normal weight and mitigate its severity in those already obese, it suggests that the body weight of these people is below a biological set point.

A number of theories of the regulation of body weight have been proposed. Most ascribe this regulation to the regulation of a single nutrient. They start with the common-sense view that a person stops eating at the end of a meal because of the repletion of some nutrient that had been depleted. And one becomes hungry again when the nutrient, which had been restored by the meal, is once again depleted. It seems reasonable that some metabolic signal, derived from food that has been absorbed, is carried by the blood to the brain, where it activates receptor cells, probably in the hypothalamus, to produce satiety. Hunger is the consequence of the decreasing strength of this metabolic signal, secondary to the depletion of the critical nutrient.

Four classical theories of the regulation of body weight have been based upon this argument. They differ from each other only in the signal to which they ascribe primary importance. The thermostatic theory, for example, proposes that postprandial increases in hypothalamic temperature mediate satiety, with hunger resulting from a decrease in temperature at this site. Lipostatic, aminostatic, and glucostatic theories each assign the critical regulatory role to blood-borne metabolites of fat, protein, or carbohydrate, respectively.

Although each of these theories explains some of the many phenomena involved in the control of food intake, Mayer's glucostatic theory has had the greatest influence on the field. According to this theory, depletion of carbohydrate stores decreases the amount of "available glucose" in the circulating blood; a fall in available glucose, signaled to hypothalamic glucoreceptors, becomes the stimulus for hunger. An increase in available glucose, with carbohydrate repletion, activates hypothalamic satiety areas and terminates eating. For more than twenty years this theory has exerted great organizational and heuristic power, and evidence for it continues to accumulate.

Despite the attractiveness of the glucostatic theory, it shares with all single-factor theories the general difficulty of encompassing the many events that are involved in the regulation of body weight. In addition to this difficulty,

single-factor theories encounter two specific problems.

1. How can a mechanism of short-term, meal-to-meal control of food intake account for the remarkable stability of body weight over long periods of time and in the face of frequently marked short-term fluctuations? As a result of the problems of single-factor theories in modeling such stability, the newer theories of the regulation of body weight are multifactorial ones. They are achieving increasing success in predicting food intake under a wide variety of conditions.

2. The second specific problem of single factor, primarily physiological theories is how they can explain the function of satiety. For satiety occurs so soon after the beginning of a meal that only a small proportion of the total caloric content of the meal can have been absorbed. If satiety were based solely upon the limited information about food intake available at that time, it could contribute little or nothing to the regulation of food intake.

If humoral factors do not terminate eating, what does? A “full stomach” may be the answer. In addition to common sense and personal experience, Jordan has added experimental evidence indicating that gastric filling, quite irrespective of the nutritive value of the meal, is the major determinant of satiety in single-meal experiments.

Although the nutritional value of meals plays little part in satiety in single-meal experiments, humans learn, as do other animals, to change food

intake and even meal size in response to changes both in energy expenditure and in the nutritive value of the food. Booth and Stunkard have proposed that this learning is a special case of Pavlovian, or classical, conditioning. In this theory, oral, gastric, and perhaps duodenal factors serve as conditioned stimuli; humoral factors absorbed from the gastrointestinal tract serve as the later, unconditioned stimuli. This sequence accounts both for the termination of eating early in the process of food absorption from the intestine and for the long-term adjustment of meal size to changing caloric needs. Booth has extended this theory to encompass "conditioned hunger" as well as conditioned satiety.

Until recently, it was believed that classical conditioning could not occur when the interval between conditioned and unconditioned stimuli was more than a few seconds. CS-US intervals longer than these have been reported only in the special case of taste aversions. But taste aversions may simply be special cases of a more general "alimentary learning." It has been proposed that this alimentary learning serves as the bridge between the long-term physiological control of body weight based on humoral factors and the short-term control of hunger and satiety based upon conditioning. If this view is even approximately correct, impaired alimentary learning may lead to obesity and, more important, therapies based upon classical conditioning may become feasible.

General Determinants of Obesity

There are at least six known determinants of obesity. Social determinants have been discussed under "Epidemiology," and genetic determinants under "Genetics." The other four determinants are: developmental, physical activity, brain damage, and emotional.

Developmental Determinants

A key to the understanding of obesity is provided by our growing knowledge of the anatomy of adipose tissue. It has become clear that the increased adipose tissue mass in obesity can result either from an increase in fat cell size ("hypertrophic obesity"), from an increase in fat cell number ("hyperplastic obesity"), or from an increase in both size and number ("hypertrophic-hyperplastic obesity"). Johnson and Hirsch's study of six forms of experimental obesity in rodents reveals that most are either hypertrophic or hypertrophic-hyperplastic, and it appears that obese humans also usually fall into one of these two categories. These findings have important implications for prevention and treatment.

Most people whose obesity began in adult life suffer from hypertrophic obesity. When they lose weight it is solely by a decrease in the size of their fat cells; fat-cell number does not change. Salans and colleagues have elegantly illustrated the dynamics of hypertrophic obesity in their study of human

experimental obesity. When normal-weight men were induced to gain forty to sixty pounds, they did so solely through an increase in fat-cell size; when they lost the weight it was solely by a decrease in fat-cell size. Fat-cell number remained constant.

Persons whose obesity began in childhood are more likely to suffer from hyperplastic obesity, usually of the combined hypertrophic-hyperplastic type. They may have up to five times as many fat cells as persons of normal weight or those suffering from pure hypertrophic obesity. We still do not know all of the reasons for the elevation in fat-cell number, and at the present time the field is wracked with controversy. Recent research has challenged the old orthodoxy that fat-cell number cannot increase after early childhood and that events during a relatively brief "critical period" early in life is largely responsible for adipose tissue hyperplasia. Instead, it has been proposed that fat-cell hypertrophy is a major stimulus for fat-cell hyperplasia and that this circumstance, and not a critical period, accounts for the common association of hyperplastic adipose tissue and juvenile-onset obesity.

This view is compatible with Stern and Johnson's review that describes at least two periods when cellular proliferation is enhanced in normal-weight children. One is before the age of two years and one is between the ages of ten and fourteen. In obese, presumably overfed children, however, the period of cell proliferation may extend well past two years of age, with consequent

development of pronounced hyper-cellularity even early in life. Whether over-nutrition alone can account for this prolonged period of cellular proliferation is not known; a genetic predisposition may also be required. Johnson and Hirsch's studies of the genetically obese Zucker rat suggest the intriguing possibility that genetic factors may exert their influence by extending the period during which proliferation is particularly susceptible to the influence of over-nutrition. The hyper-cellularity of the adipose tissue of these animals results from an ability to produce new fat cells well beyond the period of regulated proliferation found in their nonobese litter mates. Whatever the final outcome of this research, enough is already known to single out the early years of life as particularly important in the genesis of hyperplastic obesity. The public health implications are clear. To the compelling psychological reasons for the prevention of childhood obesity must now be added these compelling anatomical ones.

Our growing understanding of adipose tissue has clinical implications of equal importance. They are highlighted in a report by Björntorp and colleagues on twenty-six outpatients who lost thirty-three pounds on a dietary regimen. Body weight and body fat content at the end of treatment varied widely and had reached normal limits in only ten of the subjects. Individual fat-cell size, however, was quite similar in all patients at the end of treatment and had fallen to normal in twenty-three of them. Most patients stopped treatment just at the point when further weight loss could be

achieved only by the reduction of their fat cells to subnormal size. It was as if fat-cell size (perhaps particularly certain events at the cell membrane) had set a biological limit to weight reduction. If this is the case, it would explain the difficulty that hyperplastic obese persons experience in reducing to normal body weight and their proclivity to regain the weight that they have lost. More speculatively, it also suggests that reduction to a normal body weight may not be as important a health measure for these patients as we had believed. There is evidence that increased cell size, and not increased body fat alone or increased cell number, is responsible for the malignant metabolic sequence of insulin resistance, hyperinsulinemia, and lipid derangement.

Physical Activity

The only component on the energy-expenditure side of the caloric ledger that both fluctuates and is under voluntary control is physical activity. As such, it is a vital factor in the regulation of body weight. Indeed, the marked decrease in physical activity in affluent societies seems to be the major factor in the recent rise of obesity as a public health problem. Obesity is a rarity in most underdeveloped nations, and not solely because of malnutrition. In some rural areas, a high level of physical activity is at least as important in preventing obesity. Such levels of physical activity are the exception in this country. If the trend exemplified by automatic can openers and mechanized swizzle sticks continues, we may succeed in reducing our energy expenditure

to near basal levels. Among many obese women, the trend is already far advanced.

Figure 23-2 shows marked reduction of physical activity in a group of Philadelphia housewives; this reduction is so great as to account almost entirely for their excess weight. But such low levels of physical activity are not present among all obese persons. Figure 23-2 shows that the differences in physical activity among the men were so small that the additional energy expended by obese subjects in moving their heavier bodies produced a caloric expenditure equal to that of nonobese men.

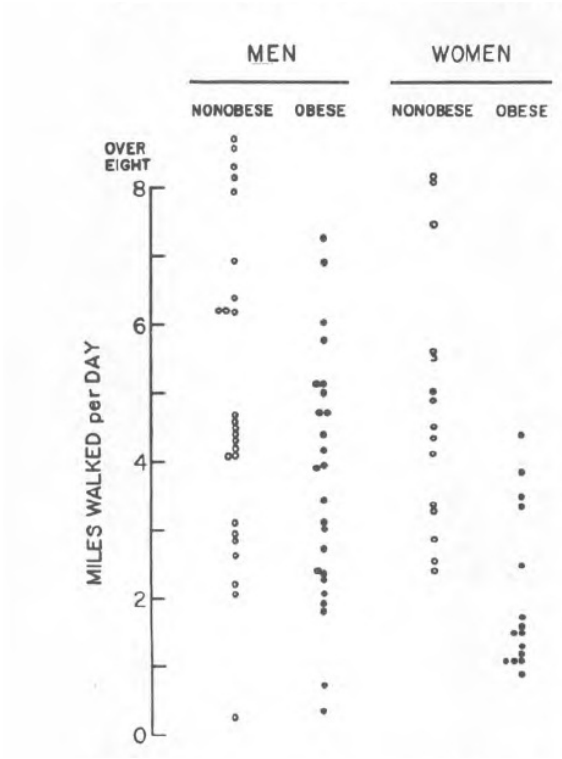

Figure 23-2.
Comparison of the physical activity of obese and nonobese men and women. Each point represents the average distance walked each day by the subjects, as measured by a mechanical pedometer. Most obese women walked shorter distances than nonobese women. Among men, there is less difference in the distances walked. (From Chirico, A. M., and Stunkard, A. J. By permission of the New England Journal of Medicine, 263 (1960): 935-946.)

Until recently, physical inactivity was considered to cause obesity primarily through its restriction of energy expenditure. There is now good evidence that inactivity may contribute also to an increased food intake.

Although food intake increases with increasing energy expenditure over a wide range of energy demands, intake does not decrease proportionately when physical activity falls below a certain minimum level, as shown in figure 23-3. In fact, restricting physical activity may actually increase food intake. Conversely, when sedentary organisms increase physical activity, their food intake may decrease. The importance of this phenomenon is probably even greater than was realized when it was first demonstrated by Mayer in Sprague-Dawley rats of normal weight. Quite recently, studies of three forms of experimental obesity in rodents have shown that activity controls food intake even more powerfully in obese animals than in those of normal weight. The mechanism involved in this intriguing control is still unclear, but it is great therapeutic potential makes it worthy of careful study.

Brain Damage

Brain damage can lead to obesity, although it probably does so only rarely in humans. Nevertheless, brain damage is of great theoretical interest in understanding obesity. This discovery, during the 1940s, that destruction of the ventromedial hypothalamus could produce obesity initiated the modern investigation of the condition. Subsequent work has delineated two broad anatomical systems mediating hunger and satiety—the former with special representation in the lateral hypothalamus, the latter in the ventromedial hypothalamus.

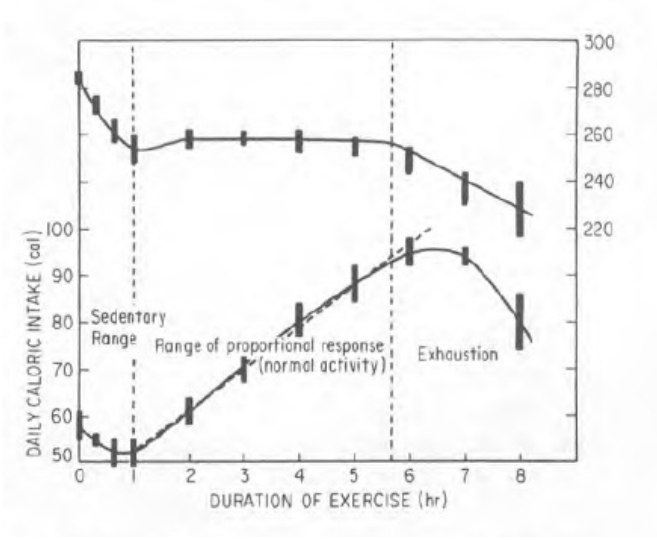

Figure 23-3.
Calorie intake and body weight as functions of duration of exercise in adult rats. Within the range of normal physical activity, food intake increases with increasing physical activity and body weight remains stable. In the sedentary range of activity, however, decreasing physical activity is associated with increased food intake and an increase in body weight. (From Mayer, J., and Thomas, D. W. By permission of the American Association for the Advancement of Science, 156 (19671:328-337.)

The way obesity is produced by ventromedial hypothalamic damage is particularly relevant to the thesis that obesity results from the level of regulation of body weight rather than from a disorder of regulation itself. Animals with such damage regulate body weight, but at a higher level. In the course of maintaining their new fat stores and body weight, such animals demonstrate interesting and potentially highly significant changes in behavior. Many of the features of this behavior in obese rats with

hypothalamic lesions were described years ago by Miller and coworkers in their report on the paradox of "Decreased Hunger and Increased Food Intake in Hypothalamic Obese Rats."

The cardinal feature of the rats' behavior was that they overate when food was freely available; but when an impediment was placed in the way of their eating, they not only decreased their food intake but actually decreased it to a far lower level than that of control rats without hypothalamic lesions. Furthermore, it seemed to make little difference what kind of impediment was used; motivation to work for food was impaired in every manner of task that could be devised. These rats seemed to be relatively unresponsive to all physiological cues concerning their nutritional state, and they responded imperfectly to signals both of satiety and of deprivation.

Nonetheless, the obese rats seemed hyper-responsive to the taste of food and to its availability. They increased their overeating when fat and sweet substances were added to their diet and radically restricted intake when the palatability of their food was decreased by the addition of quinine. Similar eating patterns have been reported in a wide variety of animals when they became obese for natural reasons—for example, in the genetically determined yellow obese mouse, in the rat when it becomes obese with aging, and even in the dormouse during the hyperphagia that precedes its hibernation. Experimental obesity of various types in animals thus seems to

possess some common behavioral correlates. Clinical research has revealed intriguing parallels to the behavior of obese humans.

In the exceptional case, obesity in humans results from hypothalamic damage from a strategically placed tumor or vascular lesion. Usually, however, the cause of the impaired satiety exhibited by many obese persons remains unknown. Such persons characteristically complain that it is difficult to stop eating; it is the unusual obese person who reports being driven by hunger or who eats in a ravenous manner. Instead, obese persons seem particularly susceptible to the palatability of foods and find it difficult to keep from eating if food is available.

Studying the problems of obese persons without brain damage, Bruch has described their misperception of important visceral events. Some obese persons, who are also neurotic, have difficulty in identifying hunger and satiety. They frequently seem unable to distinguish between hunger and other kinds of dysphoria. Bruch has linked this “conceptual confusion” to severe deficits in identity and to feelings of personal ineffectiveness. She has convincingly described the need on the part of these patients for external signals to tell them when to eat and when to stop eating. Support for Bruch’s position has come from studies that show that neurotic obese persons have a strong response bias that impairs their perception of gastric motility. Unfortunately, correction of the bias did not result in weight loss.

On the basis of a long series of experiments, Schachter has proposed a theory of obesity compatible with these ideas, which has achieved wide popularity. According to this theory, obese people are unusually susceptible to all kinds of "external" stimuli to eating, while remaining relatively unresponsive to the usual "internal" or physiological signals of hunger. At the present time this theory is under revision. Although externality as a personality trait seems well established, its relationship to obesity remains inconclusive.

Emotional Determinants

Many obese persons report that they often overeat and gain weight when they are emotionally upset. But it has proved singularly difficult to proceed from this provocative observation to an understanding of the precise relationship between emotional factors and obesity. The most clear-cut evidence of how emotional factors influence obesity has come from two small subgroups of obese persons, each characterized by an abnormal and stereotyped pattern of food intake. About 10 percent of obese persons, most commonly women, manifest a "night-eating syndrome" characterized by morning anorexia and evening hyperphagia with insomnia. This syndrome seems to be precipitated by stressful life circumstances and, once present, tends to recur daily until the stress is alleviated. Attempts at weight reduction when the syndrome is present have an unusually poor outcome and may even

precipitate a more severe psychological disturbance.

Bulimia, found in fewer than 5 percent of obese persons, is one of the rare exceptions to the pattern of impaired satiety. It is characterized by the sudden, compulsive ingestion of very large amounts of food in a very short time, usually with great subsequent agitation and self-condemnation. It, too, appears to represent a reaction to stress. But in contrast to the night-eating syndrome, these bouts of overeating are not periodic and they are far more often linked to specific precipitating circumstances. Binge eaters can sometimes lose large amounts of weight by adhering to rigid and unrealistic diets, but such efforts are almost always interrupted by a resumption of eating binges.

Complications

Troublesome as obesity may be from a cosmetic standpoint, it is the health hazards associated with it that have caused it to be described as the nation's greatest *preventable* cause of death.

Effects on Mortality

Obesity has a strong adverse effect on morbidity and mortality rates. The death rate from several diseases is significantly higher among obese persons, and the rate increases in proportion to the severity of the obesity.

The most serious consequence of obesity is its impact upon the cardiovascular diseases that now cause more than half of all deaths in this country. The Framingham and Chicago Peoples Gas Company studies have shown a very strong relationship between obesity and coronary artery disease.

This evidence of the direct effect of obesity on mortality is matched by evidence of its indirect effect. Two of the most potent risk factors for coronary artery disease—adult-onset diabetes and hypertension—are also highly correlated with obesity. Weight reduction has a powerful effect: 75 percent of adult-onset diabetics may discontinue medication, and the blood pressure of 60 percent of hypertensives returns to normal levels after significant weight loss.

In recent years, arguments against the importance of obesity in coronary disease have been raised, citing evidence that it is at best a weak independent risk factor. Its powerful indirect effects, via diabetes, hyperlipidemias, and hypertension, however, detract from the strength of this argument.

Physical and Laboratory Abnormalities

The most serious physical manifestation of obesity, and the only one that is life-threatening, albeit very rarely, is the encircling of the thorax with fatty tissue, together with pressure on the diaphragm from below due to

intraabdominal accumulations of fat. The result is reduced respiratory excursion, with dyspnea from even minimal exertion. In very obese persons, this condition may progress to the so-called “Pickwickian syndrome,” characterized by hypoventilation with consequent hypercapnia, hypoxia, and finally somnolence.

Severe obesity leads to a variety of orthopedic disturbances, including low-back pain, aggravation of osteoarthritis, particularly of the knees and ankles, and often enormous calluses over the feet and heels. Even mild degrees of obesity are associated with amenorrhea and other menstrual disturbances. Subcutaneous fat is an excellent heat insulator, and the skin of obese persons is often warm and sweaty, particularly after meals. Hyperhidrosis leads to intertrigo in the pendulous folds of tissue, making itching and skin disorders common. Mild to moderate edema of the feet and ankles often occurs, probably due to venous obstruction; diuretics are not indicated. What is most notable about all of these complications is the ease with which they can be controlled and eliminated by weight reduction, often of only a moderate degree.

Blood-pressure elevations are frequently found in obese persons, often due to an artifact, that is, the presence of masses of subcutaneous tissue between the blood-pressure cuff and the brachial artery. This problem can usually be overcome by using a wider blood-pressure cuff.

Hyperuricemia is sometimes found in obesity, and it may reach a significant degree in persons who fast intermittently. When obesity has produced respiratory distress, hypercapnia may develop along with a respiratory acidosis.

A particular problem in the laboratory evaluation of obesity is the impaired glucose tolerance, and even the presence of fasting hyperglycemia, that occurs in many obese persons without a family history of diabetes. The high insulin levels in the fasting state and after a glucose load, usually associated with obesity, are related to the presence of muscle and adipose tissue resistance to carbohydrate metabolism. The precise relationship between tissue resistance and insulin levels is not clear. It may be that tissue resistance signals the pancreas to produce more insulin, or that a high-calorie diet may increase insulin production, with tissue resistance a secondary phenomenon. However these questions are finally resolved, the response to weight reduction is highly gratifying. Most such abnormalities disappear completely unless the patient is truly diabetic.

Plasma-lipid levels are often moderately elevated in the obese. Weight reduction decreases both total cholesterol and triglycerides, and it is one of the few measures that produces elevations (among men, at least) in the protective high-density lipoprotein cholesterol.

Emotional Disturbances

Reports on emotional disturbances among obese people have flooded the literature. The better the study the less the evidence for distinctive psychological features and disabilities. The two most careful studies have shown little differences in psychopathology between obese and nonobese people. Moore and colleagues reported slightly higher levels of psychopathology among obese people; Crisp and McGuiness reported slightly lower levels. Even massively obese people do not seem to suffer undue psychiatric disability. The view that obese persons have a specific personality pattern is no longer held.

Although the differences in psychopathology are relatively small for the obese population as a whole, they may be quite significant for certain subgroups. Prominent among these are young women of upper and middle socioeconomic status. The reasons for the special vulnerability of these groups are of interest. Both obesity and emotional disturbances are common among persons of lower socioeconomic status; any association between the two conditions in persons in this stratum is apt to be coincidental. Higher up on the socioeconomic ladder, however, obesity is far less prevalent, and the sanctions against it are far stronger. There is also far less emotional disturbance at this level. As a result, when obesity and emotional disturbance coexist in this group, the likelihood that they are associated is far greater.

Among young, upper-class women, obesity is usually linked to neurosis. What is the nature of this linkage?

Of the various emotional disturbances to which obese persons are subject, only two are specifically related to their obesity. One is overeating, which has been discussed; the other is disparagement of the body image. Persons suffering from disparagement of the body image characteristically feel that their bodies are grotesque and loathsome and that others view them with hostility and contempt. This feeling is closely associated with self-consciousness and impaired social functioning. While it may seem reasonable to suppose that all obese persons have derogatory feelings about their bodies, such is not the case. Emotionally healthy obese persons have no body-image disturbances and, in fact, only a minority of neurotic obese persons have such disturbances. The disorder is confined to those who have been obese since childhood; and even among these juvenile-onset obese, less than half suffer from it. But in the group with body-image disturbances, neurosis is closely related to obesity, and this group contains a majority of obese persons with specific eating disorders.

The extent and severity of complications following weight-reduction programs have been the subject of controversy in recent years. It now appears that as many as half of the patients routinely treated for obesity by family physicians may develop mild anxiety and depression. An even higher

incidence of emotional disturbance has been reported among morbidly obese persons undergoing long-term treatment by fasting or severe caloric restriction even when carried out in the hospital. These complications should be balanced against the likelihood of a decrease in anxiety and depression among those who diet successfully. Such newer treatments as behavior modification and by-pass surgery carry far less risk of emotional disturbances.

Obese persons with extensive psychopathology, those with a history of emotional disturbance during dieting, or those in the midst of a life crisis should attempt weight reduction, if at all, cautiously and under careful supervision. For others, the possibility of complications need not preclude treatment when it is indicated.

General Considerations

Weight reduction confers such great benefits on obese persons and is apparently so simple that we might expect it to be a common occurrence. Perhaps the large number of women who try to reduce without medical assistance (following diets and advice from the women's magazines) have more success. But "most obese persons will not enter outpatient treatment of obesity; of those who do, most will not lose a significant amount of weight, and of those who do lose weight, most will regain it." Furthermore, these

results are poor not because of failure to implement any simple therapy of known effectiveness, but because no simple or generally effective treatment exists. Obesity is a chronic condition, resistant to treatment and prone to relapse.

The basis of weight reduction is utterly simple—establish a caloric deficit by bringing intake below output. All of the many treatment regimens have this simple task as their goal. The simplest way to reduce caloric intake is by means of a low-calorie diet. The best long-term effects are achieved with a balanced diet that contains readily available foods. For most people, the most satisfactory reducing diet consists of their usual foods in amounts determined by tables of food values available in standard works. Such a diet gives the best chance of long-term maintenance of the weight lost during dieting. But it is precisely the most difficult kind of diet to follow during the period of weight reduction.

Many obese persons find it easier to use a novel or even bizarre diet, of which there has been a profusion in recent years. Whatever effectiveness these diets may have is due, in large part, to monotony—almost everyone gets tired of almost any food if that is all he or she gets to eat. As a consequence, when one ends the diet and returns to the usual fare, the incentives to overeat are multiplied.

Fasting has had considerable vogue as a treatment of obesity in the recent past, but it is now rarely used. Its importance lies primarily in what it has taught us about radical dietary restriction in the treatment of obesity. The great virtue of fasting is the rapid weight loss it engenders, often with relatively little discomfort. After two or three days without food, hunger largely disappears and patients get along well as long as they remain in an undemanding environment. The main problem with fasting as a treatment is the failure to maintain weight loss: Most patients regain most of the weight they have lost. Furthermore, fasting, although surprisingly safe for such a radical procedure, is not without complications and deaths have been reported.

The most important consequence of the experience with therapeutic fasting has been the interest it has aroused in the idea that one can take advantage of the benefits (rapid weight loss) while avoiding the complications. These effects can be achieved by the administration of very small amounts of protein. The so-called “protein-sparing-modified-fast” can effectively maintain nitrogen balance by amounts of protein small enough to have only a negligible effect upon the rapid weight loss. The diet of Genuth and others, for example, contains no more than 320 calories (45 grams of egg albumen and 30 grams of glucose). Vitamin and mineral supplements are necessary. These diets appear to be safe and have the merit of being able to be carried out on an outpatient basis.

The largest series of patients—1,200— treated by protein-sparing-modified-fasts has been reported by Genuth and others. Seventy-five percent lost more than 18 kilograms, and blood pressure was reduced to normal in 67 percent of hypertensives. Few complications were reported: mild hyperuricemia and occasional mild orthostatic hypotension, cold intolerance, and anemia. The major problem of the protein-sparing-modified-fast is that of all conservative treatments for obesity: failure to maintain weight loss. The limited data on long-term follow-up are not encouraging.

An important distinction must be made between the carefully studied protein-sparing-modified-fast and the spate of commercially exploited "liquid protein diets" that have appeared in recent years. The best known of these is Linn's "Last Chance Diet." Generally composed of hydrolysates of cowhide, collagen, and gelatin, these "liquid proteins" are of low biological quality and do not contain an adequate balance of essential amino acids. Not unexpectedly, complications began to be reported soon after these diets were introduced, and already a number of deaths have been reported. "Liquid protein" diets have no place in the treatment of obesity.

Pharmacological treatment of obesity has been greatly altered by recent directives of the Drug Enforcement Administration, which has progressively restricted the use of amphetamines as appetite suppressants. A variety of agents is taking their place: diethylpropion (Tenuate), fenfluramine

(Pondimin), and mazindol (Sanorex) are the common examples. The efficacy and side effects of these agents are similar and their potential for abuse is limited.

Pharmacotherapy of obesity is currently out of favor. Nevertheless, its efficacy in weight reduction has been underestimated. In a recent study, weight loss with fenfluramine was increased from thirteen to thirty-two pounds simply by changing the circumstances of its administration from a traditional doctor's office format to a weekly group meeting. Patients regained weight rapidly after medication was stopped. This finding suggests that tolerance to the effects of fenfluramine did not develop and that this is not a reason for restricting its use. It seems likely that pharmacotherapy of obesity has been prematurely written off; new medications and new circumstances of administration, however, may restore its popularity.

An interesting new treatment for bulimia (with or without obesity) has been reported by Wermuth and coworkers. They administered phenytoin (Dilantin) to nineteen obese and nonobese persons who suffered from eating binges at least three times a week. Six patients reported a marked decrease in eating binges during the double-blind trial, and two of the four who continued treatment with phenytoin reported no binges in the next eighteen months. This report of such a simple treatment of a distressing disorder is most promising, and replication is sorely needed.

Thyroid or thyroid analogues are indicated for the occasional obese person with hypothyroidism, but should be discouraged otherwise. Bulk producers may help control the constipation that follows decreased food intake, but their effectiveness in weight reduction is doubtful. Four controlled studies of chorionic gonadotropin have found it to be ineffective.

Increased physical activity is frequently recommended as a part of weight-reduction regimens, but its usefulness has probably been underestimated, even by many of its proponents. Since caloric expenditure in most forms of physical activity is directly proportional to body weight, obese persons expend more calories with the same amount of activity than do people of normal weight. Furthermore, increased physical activity may actually cause a *decrease* in the food intake of sedentary persons. This combination of increased caloric expenditure with probable decreased food intake makes an increase in physical activity a highly desirable feature of any weight-reduction program.

Group methods, propagated by the burgeoning self-help movement, are being used by increasing numbers of obese people. The two largest organizations are the nonprofit Take Off Pounds Sensibly (TOPS) with over 300,000 members, and the profit-making Weight Watchers, which is even larger. Costs for attending meetings are small, and many people report that the group support and frequent weighings are quite helpful. Objective

assessment of these organizations, however, has lagged. A recent report makes it clear that dropout rates are very high. This problem is partly compensated for by high reentry rates, but we know very little about the weight losses of representative samples of participants. These organizations are unique in the weight-reduction field in providing economical and readily accessible assistance, in making few demands upon the participants, and in permitting them to leave and rejoin without penalties. As such they should have an important place in an overall approach to obesity.

Surgical treatments for obesity are relatively new but are highly significant for that small fraction of people who suffer from "morbid" obesity, that is, 100 percent over ideal weight. Four factors have made surgery the treatment of choice for many such people: (1) recent demonstration of the severity of the various physical complications often with profound psychosocial disability, resulting in a twelvefold increase in mortality among younger persons, (2) the inefficacy of conventional treatments; (3) the continuing development of newer surgical measures; and (4) many health benefits at acceptable levels of risk.

Although the prevalence of morbid obesity is very low—less than 1 percent—over half a million Americans suffer from this condition and many of them seek psychiatric help at some time in their life. It is, therefore, worth describing the newer surgical procedures and some of their surprisingly

favorable behavioral sequelae. Two operations currently dominate the treatment of morbid obesity—jejunoileal bypass and gastric bypass. In the jejunoileal bypass operation, fourteen inches of proximal jejunum is anastomosed to four inches of terminal ileum, bypassing the remaining bowel and radically reducing the absorptive surface. In the gastric bypass operation, a stomach pouch of 50 ml capacity is constructed with a 1.2 cm outlet to the proximal jejunum, radically reducing the amount of food that can be consumed at one time. The original rationale for the jejunoileal bypass was to decrease intestinal absorption of nutrients. Although malabsorption occurs, most of the weight loss following jejunoileal bypass (and all of the weight loss following gastric bypass) is due to voluntary restriction of food intake. Weight loss following both procedures occurs at a decelerating rate for twelve to eighteen months, during which time at least 50 percent of excess weight is lost, although there is, of course, considerable individual variability. This weight loss is accompanied by a number of significant benefits; among them, relief of the Pickwickian syndrome, reduction of elevated blood pressure and blood glucose to normal levels among most hypertensives and diabetics, and correction of a wide variety of the mechanical ill effects of excessive weight. One of the most gratifying results is marked amelioration of the psychosocial disabilities that afflict most morbidly obese persons. Against these benefits must be considered the risks of bypass surgery. Mortality in the operative and postoperative period is below 3 percent for both procedures, if they are

carried out as they should be by skilled interdisciplinary teams able to provide continuing supervision. Postoperative results, however, favor gastric bypass. Jejunoileal bypass is often followed by severe diarrhea, fluid and electrolyte disturbances over the short-term, and liver disease that is fatal in 2 percent of patients over the longer term. Hyperoxaluria leads to nephrolithiasis in 10 percent of patients and to serious focal nephritis induced by oxalate crystals in an undetermined number of other patients. Complications of jejunoileal bypass do not seem to decrease over the years and the long-term adverse effects have been serious enough to cause surgeons to turn increasingly to gastric bypass. The only common complications of this surgery are epigastric distress and vomiting; these are readily controlled as the patient learns new eating habits.

Some unexpected behavioral consequences of both operations are of interest for both theoretical and practical reasons. Five studies have reported that bypass surgery is followed by unusually benign behavioral consequences, but four of these studies have understated the benefits of this surgery. The latter studies used what was probably an inappropriate control period. For comparison with the emotional status after the surgery period, they used as a control the time just before surgery. A more appropriate control period would seem to be the time when the patient was attempting to lose weight (usually with far less success) without surgery. During such times a majority of these patients had experienced depression, anxiety, and a variety of depressive

affects. By contrast, during the weight loss that follows surgery patients rarely experience such affects and, on the contrary, usually report enhanced feelings of well-being. Furthermore, the restriction of food intake, which plays the largest part in the weight loss, is achieved without particular effort and is accompanied by a striking normalization of eating patterns. There is a marked decrease in snacking, night-eating, and binge eating; the ability to stop eating is also enhanced. Even more surprising is the effect upon the large percentage of persons who until then did not eat breakfast: during a period of rapidly falling weight, most of these people began to eat breakfast.

These phenomena are striking enough to suggest that far from merely altering the mechanics of the bowel, bypass surgery brings about a major change in the biology of the organism. It has been proposed that this surgery has the effect of lowering the set point at which the body weight of obese people may be regulated. According to this view, the lack of dysphoric reactions to weight loss and the normalization of eating patterns result from the body adjusting to this new, lower set point, since it no longer has to struggle to reduce against the pressures of a higher set point.

Specialized Psychotherapeutic Techniques

Information about reducing diets is so widely available that only those who have already failed to lose weight on their own come to the doctor's

office. And only those who have failed with medical treatment seek out the psychiatrist. This process of selection makes it understandable why there has not been, until recently, a systematic study of the effects of psychoanalysis upon obesity and why such an approach has fallen from favor in treating obesity. No more than 6 percent of persons entering psychoanalysis do so for treatment of their obesity, and analysts themselves have been skeptical of their ability to deal with this problem.

Psychoanalysis

A recent study by Rand and Stunkard suggests that a more optimistic view of the influence of psychoanalysis upon obesity may be justified. The weight losses of a sample of eighty-four obese men and women treated by seventy-two psychoanalysts compared favorably with those achieved by other conservative methods. Thus, mean weight loss was 9.5 kg during treatment that averaged forty-two months in duration. Furthermore, 47 percent of patients lost more than 9 kg, and 19 percent lost more than 19 kg during this time. Analysts reported a striking decrease in severe body-image disparagement in their patients. At the beginning of treatment 40 percent reported severe disparagement; at its termination this figure had fallen to 14 percent. Obese patients were generally more difficult to treat than nonobese patients. For example, more obese than nonobese patients terminated treatment prematurely, and those who remained in treatment showed less

improvement in psychological functioning than did nonobese patients.

This study may reawaken interest in psychoanalytic psychotherapy of obese persons. Some general observations are in order. First, there is no evidence that uncovering putative unconscious causes of overeating can alter the symptom choice of obese people who overeat in response to stress. Years after successful psychotherapy and successful weight reduction, persons who over ate under stress continue to do so. Second, many obese people seem inordinately vulnerable to the over-dependency on the therapist and to the severe regression that can occur in psychoanalytic therapy. Bruch has provided excellent descriptions of measures designed to minimize such regression, to cope with the "conceptual confusion" described earlier, and to increase the patient's often seriously inadequate sense of personal effectiveness.

Although psychoanalysis and psychoanalytic therapy are very expensive ways to lose weight, they may be indicated for persons suffering from severe disparagement of the body image. This condition has not been influenced by other forms of treatment, even those which effect weight reduction. Psychoanalysis and psychoanalytic therapy may also be indicated for treatment of bulimia, another particularly resistant condition. Furthermore, obese people may seek psychotherapy for reasons other than their obesity; helping them to cope with their obesity may help them to resolve other

problems. We have noted that many obese people overeat under stress. If psychotherapy helps them to live less stressful and more satisfying lives, then they are less likely to overeat. As a result, they may reduce and stay reduced. These benefits are not less significant for being nonspecific results of treatment.

Behavior Therapy

Behavior therapy was introduced into the treatment of obesity a decade ago, and within five years the topic had achieved a popularity bordering on faddism. The next five years, however, saw the appearance of over fifty controlled clinical trials, and it is now possible to ascertain what has and has not been accomplished by this vast expenditure of effort.

It is clear that behavior therapy represents an improvement over traditional outpatient treatments for mild and moderate obesity, and it is the treatment of choice for these conditions. It is also clear that its great early promise has been only imperfectly realized. There is consensus on six issues:

1. Dropouts from outpatient treatment have been greatly reduced, from figures as high as 25 to 75 percent to not more than 10 percent.

2. Emotional complications of behavioral weight-reduction regimens are uncommon, in contrast to rates as high as 50 percent among persons in traditional outpatient regimens.

3. Weight losses, although greater than those achieved by alternate treatments in controlled clinical trials, have been modest and of limited clinical significance. There are many reasons for these limitations: Most of the programs were short-term, many involved patients who were only mildly overweight, and a large number were conducted by inexperienced therapists. Nevertheless, mean weight loss exceeded fifteen pounds in only a minority of clinical trials.

4. There is great variability in weight losses and still no way to predict which patients will do well and which ones will not.

5. Weight lost during behavioral treatment tends to be regained. However, weight losses are probably better maintained than they are with other forms of treatment.

6. The most important aspect of behavior therapy may be the fact that its procedures can be so clearly specified and so readily taught. Detailed instructions for the conduct of behavioral weight-reduction programs for small groups and for individuals have been provided by Ferguson, Jordan and colleagues, the Mahoney's, and Stuart. Brownell's recent Partnership Diet Program describes an original self-help program for couples.

There is a growing trend for the behavior therapy of obesity to be carried out by persons with less and less formal training, and these services are increasingly delivered by nonprofessionals. The most ambitious of these efforts is that of Weight Watchers, which enrolls 400,000 persons a week in

classes taught entirely by lay persons. The program is outlined in a series of "modules" that contain a brief written summary of the behavioral tasks to be accomplished during the next two weeks, along with forms for recording progress in this endeavor. Within the medical profession the lead in behavioral treatment has been taken by the Society of Bariatric Physicians, many of whose members carry out behavioral treatment programs, often with the aid of their office nurses.

Obesity in Childhood

The obesity of persons who were obese in childhood—the so-called "juvenile-onset obese"—differs from that of persons who became obese as adults. Juvenile-onset obesity tends to be more severe, more resistant to treatment, and more likely to be associated with emotional disturbances.

Obesity that begins in childhood shows a very strong tendency to persist. Long-term prospective studies in Hagerstown, Maryland, have revealed the remarkable degree to which obese children become obese adults. In the first such study, 86 percent of a group of overweight boys became overweight men, as compared to only 42 percent of boys of average weight. Even more striking differences in adult weight status were found among girls: 80 percent of overweight girls became overweight women, as compared to only 18 percent of average-weight girls. A later study showed that the few

overweight children who reduced successfully had done so by the end of adolescence. The odds against an overweight child becoming a normal-weight adult, which were 4:1 at age twelve, rose to 28:1 for those who did not reduce during adolescence. An even more recent study, which used a longer interval (thirty-five years) and, unfortunately, different (more rigid) criteria for obesity, found the difference in adult weight status continuing to grow: 63 percent of obese boys became obese men, as compared to only 10 percent of average-weight boys.

It is widely believed that obese children are very inactive and that their inactivity plays a major part in the development and maintenance of their obesity. Recent research suggests that excessive food intake is a far more important factor. At least four studies that used objective measures of physical activity failed to reveal significant differences between obese and nonobese children, while only one study reported that obese children were less active.

A recent intensive study of energy intake and expenditure suggests that excessive food intake and not decreased physical activity maintains childhood obesity and may even produce it. Waxman and Stunkard directly measured food intake and energy expenditure in four families at meals and at play in three different settings. In each family there was one obese boy and a nonobese brother whose ages were within two years of each other. The

subjects' oxygen consumption at four levels of activity was measured in the laboratory to permit calculation of energy expenditure from time-sampled measures of observed activity. The study showed that the obese boys consumed far more calories than did their nonobese brothers. Furthermore, their levels of physical activity did not differ greatly from those of the nonobese boys. When measures of activity were converted into calories, it was found that the obese boys actually had higher levels of energy expenditure than did their nonobese controls. These findings need confirmation with larger samples and with studies of girls. Nevertheless, the evidence that obese boys overeat is so striking that it justifies directing treatment at this problem.

Reduction of food intake has been the major focus of behavioral treatments of obesity in children. Although behavioral treatment of childhood obesity has lagged behind the application of comparable methods to obese adults, it is attracting increasing attention. Nine studies have already been reported and more are currently underway. The results to date warrant a cautious optimism; behavior therapy of children may well prove as effective with children as it has been with adults.

Conclusion

Obesity, a condition characterized by excessive accumulations of body

fat, is widely distributed within the population, affecting one-third of adult Americans. It has recently been viewed, not as the result of a disturbance in the regulation of body weight, but rather as the result of an elevation of the set point at about which body weight is regulated. At least six factors may affect this regulation: genetic and developmental, social and emotional, physical (inactivity) and neural (impaired brain function). The relative importance of these different factors probably varies among different obese persons. Recent studies suggest that the behavior of many obese persons is affected by their efforts to restrain their natural inclinations to eat in order to maintain socially approved levels of body weight.

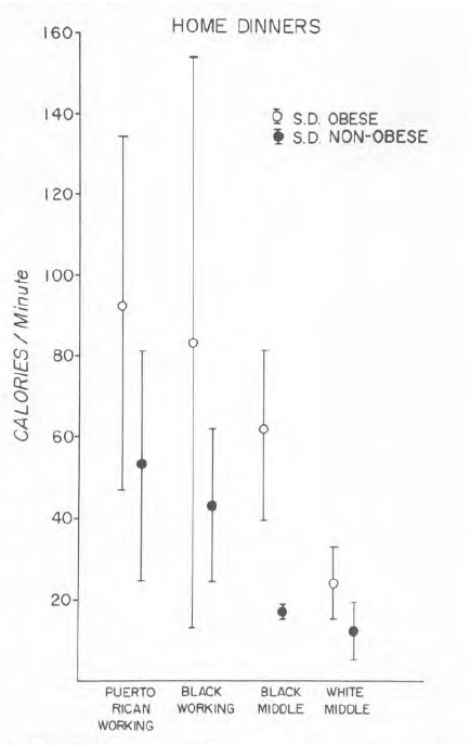

Figure 23-4.
Significantly greater calorie intake by obese boys at home dinners compared with that of their nonobese brothers. (From Waxman, M, and Stunkard, A. J. By permission of the Journal of Pediatrics, 96 (1980: 187-193.)

Obesity adversely affects morbidity and mortality, and the rates increase in direct proportion to the severity of the obesity. Although obesity is not a strong independent risk factor for cardiovascular disease, it is a major determinant of hypertension and insulin-independent diabetes, and both these conditions are markedly improved by weight reduction. Partly for

health benefits and partly for cosmetic reasons, large numbers of obese people try to lose weight. Most are unsuccessful. The poor results of weight-reduction efforts are not due to failure to implement any therapy of known effectiveness but to the fact that no simple or generally effective therapy exists. Obesity is a chronic condition, resistant to treatment and prone to relapse.

The unfavorable therapeutic outlook for obesity has been brightening in recent years with the development of new treatments and renewed interest in old ones. New pharmacological treatments together with new circumstances of administration show promise of improving the results of pharmacotherapy; phenytoin (Dilantin®) has been found to be effective in the management of some cases of bulimia. Jejunoileal bypass surgery and, more recently, gastric bypass surgery have begun to bring hope to some morbidly obese persons. Psychiatric studies have shown favorable emotional concomitants of the large weight losses achieved by surgical means. A recent study indicates that psychoanalysis may have unsuspected merits in reducing body weight as well as in lessening body image disparagement of some obese persons. A very large effort has been expended in the development of behavioral therapies for obesity, making these measures the treatment of choice for mild and perhaps also moderate obesity, as well as providing a useful model for psychotherapy research. Furthermore, the ease with which behavioral measures can be taught has resulted in the increasing delivery of weight-reduction services by

nonprofessionals. Lay groups are providing valuable vehicles for the introduction of behavior therapy to large numbers of people. But the main hope for the control of obesity lies in a better understanding of the factors that regulate body weight. Fortunately research into this problem is proving increasingly fruitful.

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