OBESITY

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OBESITY

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 body fat. Skin-fold calipers are already gaining acceptance because of their convenience and because much of the excess 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. The data we have suggest that prevalence of obesity reaches a peak at age forty when 35 percent of men and 40 percent of women can be so designated. Prevalence has been increasing slightly for men,

and decreasing slightly for women, during the past thirty years.

There have been studies of more limited populations utilizing more reliable data and permitting more valid inferences. Unfortunately these studies have differed in their criteria of obesity, making their data difficult or impossible to use for comparisons among 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 be providing far more satisfactory data.

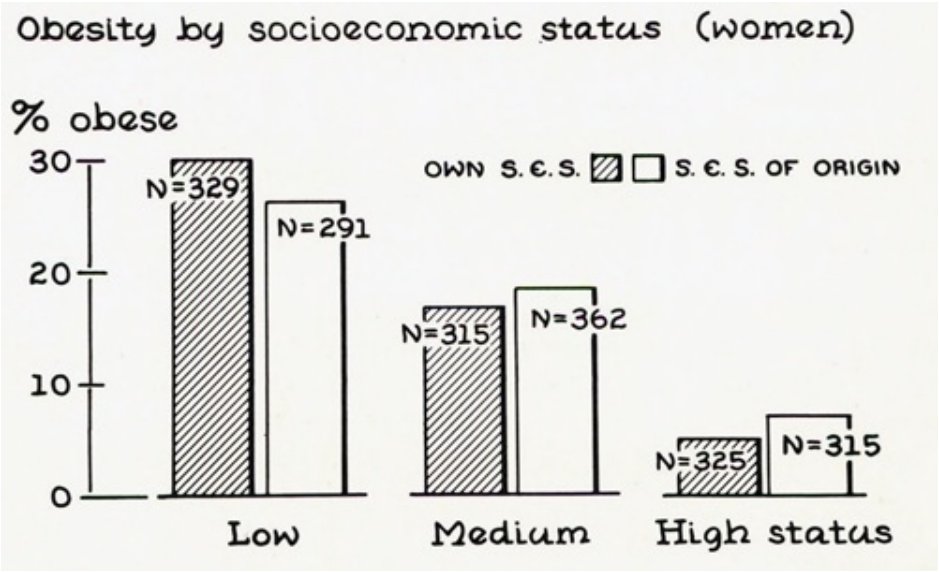
All studies that have compared the sexes report a higher prevalence of obesity among women; this discrepancy is particularly pronounced after age fifty because of the higher mortality rate among obese men in that age group.

Social factors exert a powerful influence on the prevalence of obesity. In many countries undernutrition limits the development of obesity. Freed of this constraint in the affluence of American society, many ethnic groups show

a marked increase in the prevalence of obesity during their first generation in this country. 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 country.

The most striking antiobesity influence is that of socioeconomic status. Figure 31-1 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 31-1 shows, social class of origin 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 31-1.



Decreasing prevalence of obesity with increasing socioeconomic status (S.E.S.) among women in New York City. Socioeconomic status of origin is almost as strongly linked to obesity as is the person's own socioeconomic status. (Reprinted from P. B. Goldblatt, M. E. Moore, and A. J. Stunkard, "Social Factors in Obesity," Journal of the American Medical Association, 192 (1965), 1039-1044. Copyright c 1965 by the American Medical Association.)

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. These factors must also be presumed to be important in human obesity, although clear-cut evidence of genetic transmission has been obtained only in such rare conditions as the Laurence-Moon-Biedl syndrome.

A number of studies have confirmed the layman's impression that obesity "runs in families." In one study obesity was reported in the offspring of approximately 80 percent of obese-x-obese matings, in 40 percent of obese-x-nonobese matings, and in no more than 10 percent of nonobese-x-nonobese matings. In another series, Davenport reported that among fifty-one children of slender parents none was of more than average weight and most were slender; among thirty-seven children of obese parents, on the other hand, none was slender, all were of at least average weight, and a third were obese. But such figures inevitably confuse genetic and environmental influences. Although there have been efforts to separate these influences—by studies of twins and of adopted children—none has elucidated the mechanism of transmission nor provided more than rough estimates of the relative contribution of inheritance. Because so many nongenetic factors can influence body weight, it is generally agreed that overweight per se is an unsatisfactory phenotype for the study of the genetics of human obesity.

Interest is now shifting to the transmission of somatotypes. Their relevance is clear from Seltzer and Mayer's demonstration that obesity occurs with much greater frequency in some physical types than others. Obese adolescent girls, for example, show extremely low ratings for ectomorphy; the presence of even a moderate degree of ectomorphy appeared to protect against obesity. It has been estimated that two-thirds of women in the general population may be sufficiently ectomorphic to receive significant protection

against obesity. Preliminary studies by Withers suggest that somatotypes are heritable, particularly father-daughter transmission of mesomorphy and mother-son transmission of endomorphy. Further investigations of the inheritance of body types, and of their relation to obesity are sorely needed.

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.

A brilliant series of studies of adipose tissue has recently helped to explain the remarkable persistence of juvenile-onset obesity. Many obese persons, particularly those with juvenile-onset obesity, show a marked increase in total number of adipocytes in subcutaneous tissue and in other depots. Whereas the average nonobese person has a total of 25-30 X 10^9 adipocytes, obese persons may have five times this number. The average lipid content of the adipocytes of normal-weight and obese persons varies to a far smaller degree: 0.7 *μg.* for the nonobese and 1.0 *μg* for the obese.

With weight reduction, individual cells shrink greatly, but the total adipocyte number remains constant. A number of animal studies suggest that early in life adipose tissue grows both by increasing cell size and increasing cell number. If feeding patterns are changed during the first three weeks of a rat's life, there will be marked changes in cell number. But when the animal is made obese in adult life, he grows no new adipocytes, the ones he has simply enlarge.

These studies of cellularity in obesity focus attention on the influence early feeding patterns have on the later development of obesity. Adipocyte size and number may be another factor that influences hypothalamic activity and feeding behavior. Obese persons who have lost weight but whose increased number of adipocytes persists tend to overeat and thus refill these extra cells. We have no biochemical data as yet to indicate the nature of the signal from adipose tissue to the hypothalamus.

Etiology

What causes obesity? In one sense the answer is quite simple—eating more calories than are expended as energy. In another sense, the answer still eludes us. For we do not know why some people eat more calories than they expend. But we are making progress. We no longer, for example, expect to find *the* cause of obesity, and we are far more aware than were our predecessors of the many factors involved in the regulation of body weight.

Our increased awareness of the problem's complexity has resulted in the development of an appealing framework for considering the etiology of obesity. Obesity may profitably be viewed as the end product of a disturbance in energy balance, or in the regulation of body weight. This view has helped us organize current information about obesity and has encouraged and informed the search for new information. I will consider at some length what

is now known about the regulation of body weight, to understand better how six disparate factors may influence this regulation. I have already discussed three of these, the genetic, social, and developmental. I will take up the other three later i.e., physical activity, brain damage, and emotional problems.

The Regulation of Body Weight

An average nonobese man stores fat to the extent of about 15 percent of his body weight, enough to provide for all of his caloric needs for nearly a month. This same man consumes approximately one million calories a year. His body fat stores remain unchanged during this time, because he expends an equal number of calories. An error of no more than 10 percent in either intake or output would lead to a thirty-pound change in body weight within this year.

Rats who are force-fed or deprived of food rapidly return to their normal body weights when permitted to return to ad libitum feeding. Similar studies of man are extraordinarily difficult to carry out. Yet in the rare instances when the body weight of human volunteers has been experimentally altered, it, too, rapidly returned to normal values. Sims found that normal-weight volunteers who were fattened by overfeeding and underactivity returned to their normal body weight soon after returning to their usual patterns of eating and activity. Keys' classic study of experimental

semistarvation showed a similar rapid return to normal body weight when the subjects were permitted free access to food. Clearly body weight is regulated with the greatest precision in all nonobese animals, including man. This regulation has been described in detail in two recent scholarly reviews, as has the powerful glucostatic theory of the regulation of food intake.

As befits such a vital function, the neural control of food intake is widely distributed throughout the brain. Within the limbic system alone six thousand different sites have been found to influence eating behavior. Nevertheless, certain structures seem to play a more important part than others.

The discovery that two different hypothalamic areas control hunger and satiety—one in the lateral hypothalamus mediating the former, the other in the ventromedial nucleus mediating the latter—initiated our current understanding of these clearly separable functions. More recent anatomic studies have identified extrahypothalamic structures which play a part as important as that of the hypothalamus in the regulation of food intake. Feeding, for example, is controlled by a diffuse circuit that links the forebrain limbic system (and particularly areas in the amygdala) and the globus pallidus to the midbrain tegumentum via the lateral hypothalamus. The satiety area in the ventromedial hypothalamus similarly links forebrain limbic structures and the head of the caudate nucleus to the midbrain. There are also more direct connections between the feeding and satiety systems, for

example, the inhibitory fibres that run from the ventromedial to the lateral hypothalamic areas.

Noradrenalin serves as a major neurotransmitter in both the feeding and satiety systems. Alpha- and beta-adrenergic subsystems have recently been identified, although their precise functions and locations are still unclear.

Most of our information about the role of the central nervous system in food intake regulation has been obtained experimentally, by destroying or stimulating certain areas electrically or chemically. But what signals normally activate this complex neural apparatus?

A common-sense view holds that we stop eating at the end of a meal because we have replenished some nutrient that had been depleted. And we become hungry again when the nutrient, which had been restored by the meal, is once again depleted. Specifically it has been proposed that some metabolic signal, derived from food that has been absorbed, is carried by the blood to the brain. There this signal activates receptor cells, probably in the hypothalamus, to produce satiety. Hunger is the consequence of the decreasing strength of this same metabolic signal, secondary to the depletion of the critical nutrient.

Four classical theories of hunger and satiety have been based upon this

argument, differing from each other only in the nature of the signal to which they ascribe primary significance. The thermostatic theory, for example, proposes that postprandial increases in hypothalamic temperature mediate satiety; hunger results 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, and carbohydrate.

Although each of these theories explains some of the many phenomena involved in the control of food intake, the glucostatic theory has had by far the greatest predictive power. It starts, as do the others, with the assumption that the signal to the central nervous system comes from one of the three major foodstuffs, i.e., fat, carbohydrate, or protein, or from a metabolic product of one of them. When we consider that the body stores of the key nutrient must be significantly depleted in the hours between meals, we must rule out the role of fat and protein. For such a tiny fraction of the total body stores of both these is used up in those few hours that it is very unlikely that any brain center could detect the change.

With carbohydrates the situation is quite different. The body as a whole can store only very small amounts and the liver, which is the principal storage site of readily available carbohydrate, can actually store no more than half the body's daily requirement of calories. In the few hours between meals, therefore, an enormous percentage of the body's carbohydrate stores is used

up. Any center sensitive to the depletion of carbohydrate stores should have no trouble in detecting a depletion of this size, and in letting the brain know that more carbohydrate is needed.

According to the glucostatic theory, depletion of carbohydrate stores is signaled by the amount of "available glucose" in the circulating blood; a fall in the level of available glucose, signaled to hypothalamic glucoreceptors, becomes the signal for hunger. An increase in available glucose, with carbohydrate repletion, activates the hypothalamic satiety areas, and terminates eating. The heuristic value of this theory was demonstrated when hypothalamic "glucoreceptors" first postulated by Mayer but unknown at the time that he proposed the theory, were discovered.

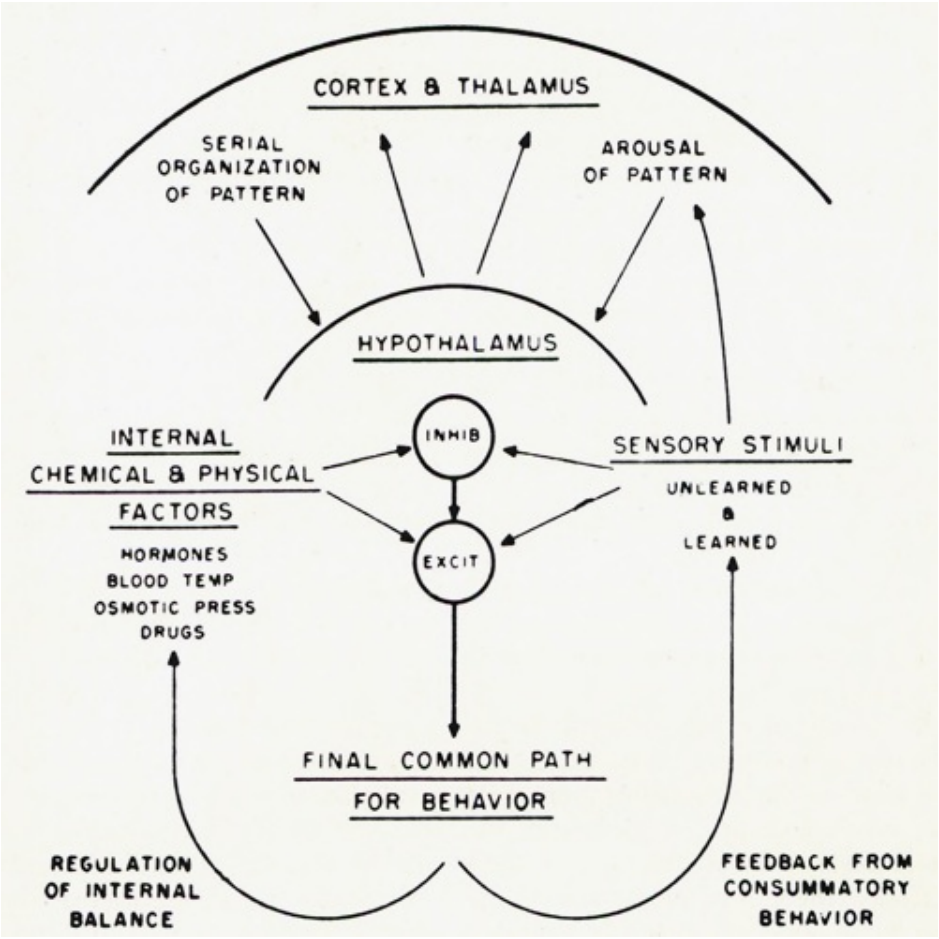
The vigor of the glucostatic theory, twenty years after it was first proposed, is manifested by the many studies it continues to stimulate. Direct measurements of the electrical activity of the hypothalamus have confirmed the findings of earlier, indirect studies of the influence of intravenous glucose. Glucose infusion increases electrical activity in the ventromedial area, and decreases such activity in the lateral area in accordance with the prediction that glucose should increase satiety and decrease hunger. Another line of evidence in support of the glucostatic theory has been derived from studies using the nonmetabolizable glucose analogue 2-deoxy-D-glucose (2-DG). Administration of 2-DG to animals produces decreased intracellular

utilization of glucose, particularly in the brain. Such a decrease in available glucose, a classic example of the signal postulated by the glucostatic theory, produces a prompt and significant increase in food intake. Recently Russek has proposed an imaginative variation of the glucostatic theory. He has amassed considerable evidence in support of the idea that the primary site of glucoreception is not in the hypothalamus, but in the liver.

Despite the attractiveness of the glucostatic theory, it shares with all single-factor theories the difficulty of encompassing the many events involved in the regulation of food intake. Figure 31-2 shows, in schematic outline, the many variables that must be considered in this regulation.

In addition to this general problem, single factor theories of the control of food intake encounter also two specific problems. First, 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 often marked short-term fluctuations? Second, how can a single-factor theory, or indeed any physiological theory, account for the function of satiety? For satiety occurs very soon after the beginning of a meal, when only a small proportion of the total caloric intake of the meal can have been absorbed. If satiety were based solely on the limited information about food intake available at that point, if could contribute little or nothing to the regulation of food intake.

Figure 31-2.



Scheme of the factors contributing to the regulation of body weight. (Copyright © 1954 by the Amer. Psychol. Assoc. Reprinted by permission.)

Recent work by LeMagnen has further documented just how imprecise the mechanism of satiety really is. He has shown that when animals feed ad libitum, meal size bears little relation to the length of the preceding period of

food deprivation. But meal size does determine, quite precisely, the length of time until the next meal. The energy needs of the body determine when a meal is initiated, but not the size of the meal. In other words, what is regulated is meal-interval, not meal-size. Or, in the regulation of food intake, hunger is controlled precisely, satiety only approximately.

Man's ability to alter the interval between meals is sharply limited by the routines of daily life. As a consequence, he is forced to rely primarily on changes in meal size to regulate his food intake. Thus the imprecise mechanism of satiety is burdened even more heavily in man than in animals. If satiety depended solely on humoral factors such as ingested glucose or other nutrients, it would be hopelessly inadequate to the task of regulating food intake.

If humoral factors do not terminate eating, what does? "A full stomach" may be a better answer than we would have thought even a few years ago. Certainly common sense and personal experience suggest that the smell and taste of food, and the feeling of a full stomach, play a part in satiety. Recent systematic clinical investigations support this view. In man as in animals, gastric filling, quite irrespective of the nutritive value of the meal, is the major determinant of satiety in single-meal experiments. The neural mechanism that mediates this response has also recently been demonstrated: gastric distention and direct stimulation of the mechanoreceptors of the stomach

wall increase the firing rate of single units within the ventromedial nucleus.

Although the nutritional value of the meal, as we have noted, plays little or no part in satiety in single-meal experiments, man seems to learn (as do other animals) to change his food intake and even his meal size, in response to changes in energy expenditure and in the character of his food. Is this learning? If so, how does he learn?

Alimentary Learning

An understanding of the mechanism for adjusting food intake, and particularly changes in meal size and frequency, has long eluded us. Our areas of ignorance are still vast. But some recent discoveries have made it possible to entertain a theory which would have been untenable until now. I propose that the adjustment of meal size and meal frequency is a learned process involving Pavlovian, or respondent, conditioning. In this theory oral and gastric factors serve as conditioned stimuli, while humoral factors absorbed from the gastrointestinal tract serve later as the unconditioned stimuli. This sequence can account 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.

Until recently this theory had an apparently fatal flaw. The interval between presentation of the postulated conditioned stimuli and presentation

of the unconditioned stimuli may well be an hour long. Pavlov showed early in this century that classical conditioning cannot occur if the interval exceeds a few seconds.

The idea that the interval between conditioned and unconditioned stimuli (the CS-US interval) could not exceed a few seconds went unchallenged through fifty years of research on conditioning. Then two lines of investigation produced evidence demolishing this constraint. In a little-noticed paper in *Science,* Garcia reported a striking exception to the belief that the CS-US interval cannot exceed a few seconds in duration. Using saccharine as the conditioned stimulus, and x-radiation (and the consequent radiation sickness) as the unconditioned stimulus, he was able to produce in rats a conditioned aversion to saccharine with CS-US intervals of *hours* in length. Further studies showed that, in contrast to the frequent CS-US pairings necessary to produce most conditioned responses, such aversion can occur with only one pairing of CS and US. Finally, and again in contrast to the usual conditioned response, the aversion is remarkably resistant to extinction.

More recently, Rozin has shown that positive reinforcers can produce food *preferences,* "specific hungers," under conditions similar to those in which aversive reinforcement produces aversions. It has been known for years that animals deficient in thiamine select diets that contain thiamine (even if only a trace) out of a wide variety of possible foods. Rozin showed

that this preference results from two factors: (1) a learned aversion, of the type demonstrated by Garcia, to diets that do not contain thiamine, and (2) the positively reinforcing effect of the vitamin on the vitamin-deficient animal. Learning about beneficial consequences over long CS-US intervals is clearly weaker than learning about the aversive consequences. We do not know the upper limit of the interval between the conditioned stimulus of thiamine ingestion and the unconditioned stimulus of well-being, but it can hardly be shorter than many minutes, and it may be as long as hours.

The food preferences and aversions demonstrated by Rozin and Garcia seem to represent a special form of Pavlovian conditioning which, for convenience, we may call "alimentary learning." The distinctive features of "alimentary learning" are: (1) the conditioned stimulus must be either taste or smell; (2) the unconditioned stimulus must be a general body state, either a dysphoric one such as radiation sickness, fever, nausea or, on the other hand, a euphoric one such as is presumably produced by thiamine repletion; (3) the learning can occur with unusual rapidity, after as few as one CS-US pairing, (4) the CS-US intervals can be as long as ten hours; and (5) these conditioned responses are unusually resistant to extinction.

The food preferences and aversions which have taught us about "alimentary learning" are of great importance in their own right. Furthermore, they have freed us from those constraints of Pavlovian

conditioning which have limited our understanding of control of food intake. But I believe that they are best understood as special cases of a more general phenomenon. The primary purpose of "alimentary learning" may be the mediation of satiety. I see "alimentary learning" as a bridge between the long-term, physiological regulation of food intake based upon humoral factors, and the short-term cessation of eating based on gastric filling. If this view is even approximately correct, then impaired "alimentary learning" may underlie the eating disorders found in obesity. An impairment of satiety surely plays a major role in these disorders.

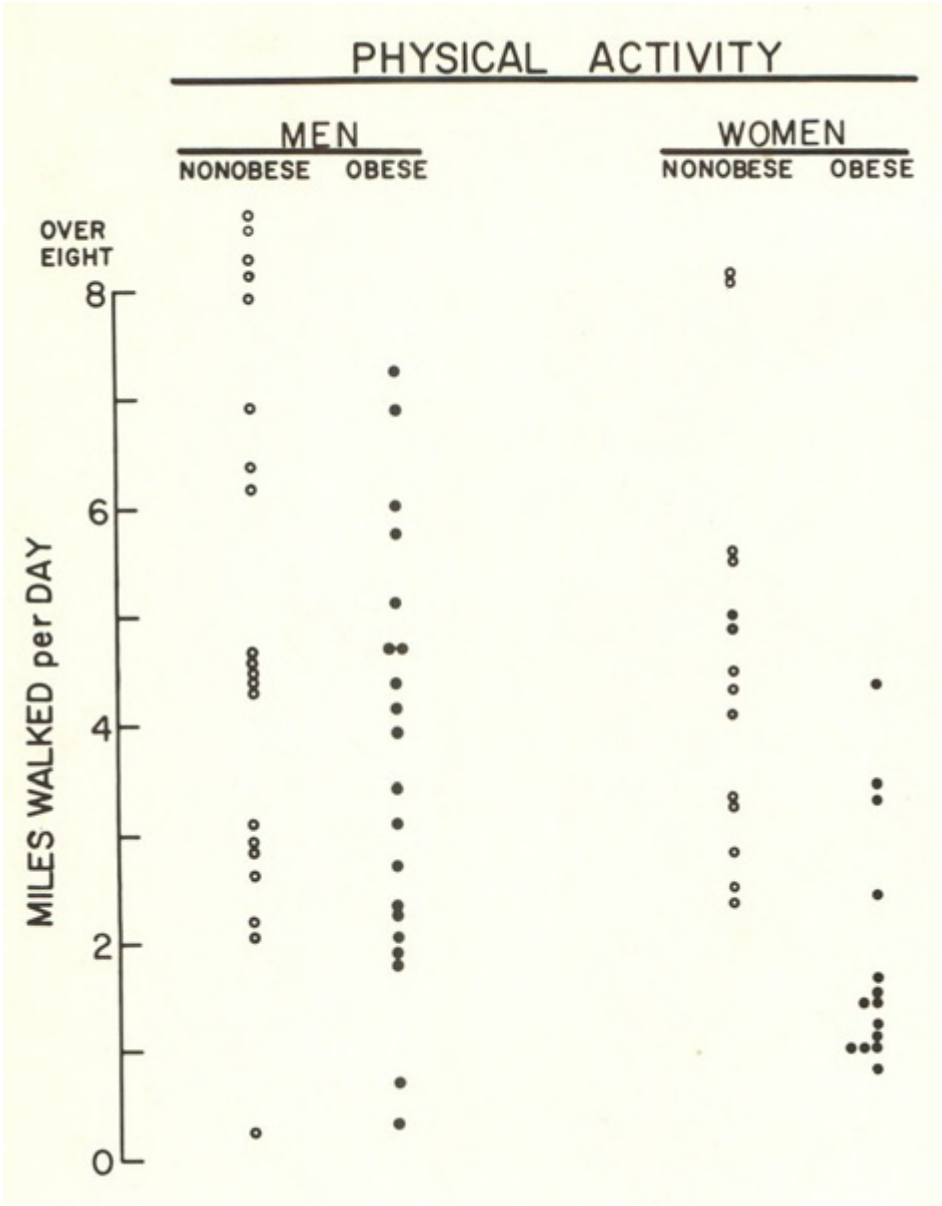
Physical Activity

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 31-3 shows marked reduction of physical activity of 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 31-3 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.

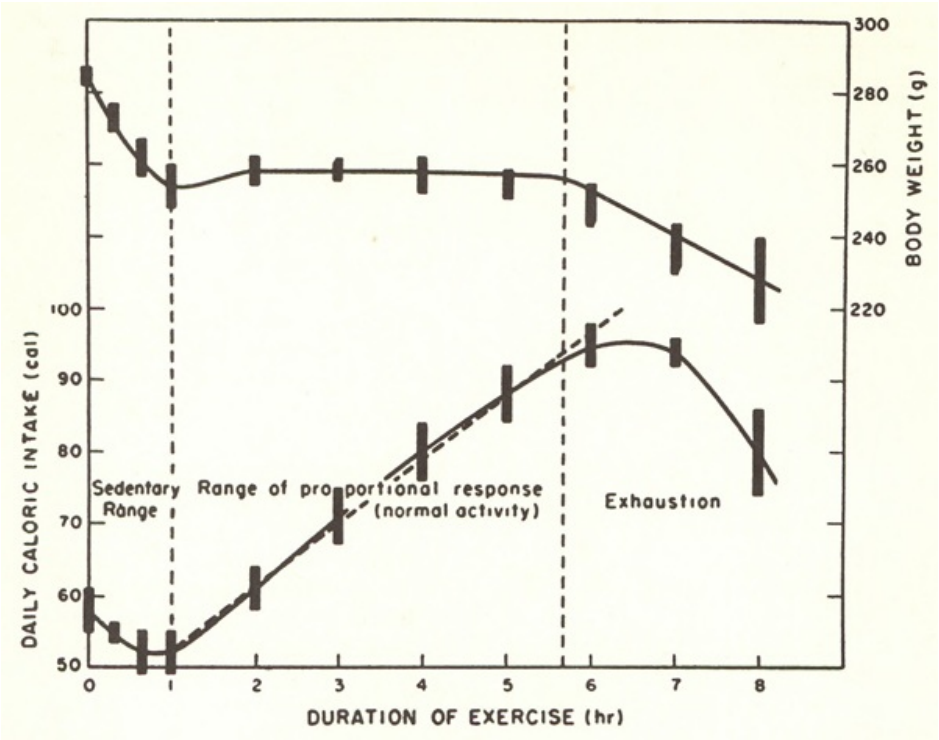
Figure 31-3.


PHYSICAL ACTIVITY
MEN
NONOBESE
OBESE
WOMEN
NONOBESE
OBESE
OVER EIGHT
8
6
4
2
0
MILES WALKED per DAY

Comparison of the physical activity of obese and nonobese men and women. Each point represents the average distance walked each day by each subject, 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. (Reprinted by permission from N. Engl. J. Med., 263 (1960), 935-946.)

Until quite recently, physical inactivity was considered to cause obesity primarily via 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 31-4. In fact, restricting physical activity may actually *increase* food intake! Conversely, when sedentary persons increase physical activity, their food intake may decrease. The mechanism involved in this intriguing control are still unclear, but its great therapeutic potential makes it worthy of careful study.

Figure 31-4.



Caloric 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. (Copyright © 1967 by the Amer. Assoc. Advance. Science. Reprinted by permission.)

Disorders in the Regulation of Body Weight

Our general understanding of the regulation of body weight was greatly advanced during the late 1940s when it was discovered that destruction of

the satiety areas of the hypothalamus could produce obesity. Many of the features of the disordered food intake produced by these lesions were described by Neal Miller in 1950 in a paper with the trenchant title, "Decreased Hunger and Increased Food Intake in Hypothalamic Obese Rats." This remarkably prescient report described for the first time the peculiar feeding behavior of rats made obese by hypothalamic lesions.

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 about their nutritional state, and they responded imperfectly to signals both of satiety and of deprivation.

On the other hand, the obese rats seemed hyperresponsive 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, such as in the genetically determined

yellow obese mouse, in the rat when it becomes obese with aging, and even in the dormouse during the hyperphagia which precedes its hibernation.

The impaired satiety found in different forms of experimental obesity also characterizes the eating patterns of many obese persons. In the exceptional instance this disorder results from unequivocal hypothalamic damage as a result of a strategically placed tumor or vascular lesion; usually we do not know the cause of this impaired satiety. Such persons characteristically complain of being unable to stop eating; it is the unusual obese person who reports being driven by hunger or who eats in ravenous manner. Instead, obese persons seem inordinately susceptible to food cues in their environment, to the palatability of foods, and to the inability to stop eating if food is available.

Bruch has documented these problems of the obese without brain damage in her vivid descriptions of 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. Strong support for Bruch's position has come from a recent study

which shows 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.

Schachter has reported a long series of experiments documenting that obese persons are unusually susceptible to all kinds of "external" stimuli to eating, while they remain relatively unresponsive to the usual "internal," i.e., physiological, signals of hunger. In one comparison with normal-weight controls, obese subjects overate a palatable ice cream and underate an unpalatable one. Similarly, they overate in an experimental setting when food was in front of them, while they underate when getting additional food required no more than opening a refrigerator door. Their eating was even influenced inordinately by what time of day they thought it was.

These findings suggest the ways in which social factors may influence the prevalence of obesity. For the ways in which a culture makes food available (and relatively less available) to its obesity-prone, "externally controlled" members may well determine the degree to which such individuals overeat.

Disorders in the Regulation of Body Weight, 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.

The "binge-eating syndrome," 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 serious health hazards associated with it that have warranted its description as the nation's greatest preventable cause of death. This effect is largely the result of obesity's impact on the cardiovascular diseases which now cause half the deaths in this country.

Effects on Mortality

The evidence is strong that obesity adversely affects morbidity and mortality rates. The death rate from a variety of diseases is significantly higher among obese persons, and the rate increases in proportion to the severity of the obesity and to its duration during adult life. Sudden death is particularly closely linked to obesity. Furthermore, obese persons who lose weight and maintain the loss show radically reduced mortality rates. For women, the rate after weight reduction was as low as if they had never been obese.

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, i.e., adult-onset diabetes and hypertension, are also highly correlated with obesity. Here again, 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 on the basis of data from prospective population studies. These studies report that, if such associated conditions as hypertension and elevated cholesterol and triglyceride levels are factored out, obesity is a poor, and often a very poor, predictor of coronary disease. But since obesity contributes to both hypertension and to disorders of lipid metabolism, such an analysis may not be justified. Furthermore, many laboratory studies have demonstrated how obesity may contribute to coronary disease. Adult-onset obesity predisposes to a response to a carbohydrate load characterized by hyperglycemia, hyperinsulinism, and hypertriglyceridemia, all implicated in the pathogenesis of atherosclerosis.

Keys' seven-country study is worthy of note. Although coronary heart disease was only weakly correlated with obesity, it was closely correlated with the saturated fatty acid content of the diet. The very high percentage of calories from this source in the American diet suggests that treatment of obesity should include restriction of saturated fatty acids as well as of calories.

Physical and Laboratory Abnormalities

The most serious physical manifestation of obesity, and the only one which is (very rarely) life-threatening, is the encircling of the thorax with fatty tissue together with pressure on the diaphragm from below due to intra-abdominal accumulations of fat. The result is reduced respiratory excursion, with dyspnea on 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, i.e., the presence of masses of subcutaneous tissue between the blood pressure cuff and the brachial artery. This problem can sometimes be overcome by using a wider blood-pressure cuff. But any serious doubt as to the existence of hypertension should be resolved by direct intraarterial measurement, particularly before starting specific antihypertensive therapy.

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 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; again both cholesterol and triglyceride levels decline with weight reduction.

Emotional Disturbances

Numerous reports on psychological disturbances among the obese have flooded the literature, making it difficult even for the expert to reconcile the varied and conflicting observations. Many of the problems are the result of difficulties in establishing suitable control groups. It has generally turned out that the better the control group the less the evidence for distinctive psychological features and disabilities. The view that obese persons have a specific personality pattern is no longer held.

Carefully controlled studies do show that the obese have a higher degree of psychopathology than do the nonobese. The differences, however, are relatively small for the obese population as a whole. For certain subgroups, on the other hand, the differences may be quite significant. Prominent among these are young women of upper- and middle-socioeconomic status. The reasons for the special vulnerability of these groups are of interest.

Since both obesity and emotional disturbance are common among persons of lower socioeconomic status, any association between the two in this stratum is quite likely to be coincidental. Higher up on the socioeconomic

ladder, however, obesity is far less prevalent and the sanctions against it 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 higher. Among young, upper-class women any obesity is very often closely 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. The first is overeating, the second, disturbance in body image.

The obese person whose body image is disturbed characteristically feels that his body is grotesque and loathsome, and that others view it with hostility and contempt. This feeling is closely associated with self-consciousness and impaired social functioning. It would 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; less than half, even among these juvenile-onset obese, suffer from it. But it is in the group with body image disturbances that 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 patients routinely treated for obesity by family physicians may develop mild anxiety and depression. In addition, a high incidence of emotional disturbance has been reported among obese persons undergoing long-term, in-hospital treatment by fasting or severe caloric restriction. 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 intestinal by-pass surgery appear to carry less risk of emotional disturbance.

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.

Treatment

General Considerations

Weight reduction confers such great benefits upon obese persons, and it is apparently so simple, that we might expect to find large numbers of

formerly fat people. How can obese people fail to reduce?

The best evidence is that they not only can, but do, fail. Perhaps the large number of women who try to reduce without medical assistance, on diets and advice from the women's magazines, have success. But most obese persons will not enter outpatient treatment for 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 poor results are due not to failure to implement any simple therapy of known effectiveness but to the fact that 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, i.e., establish a caloric deficit by bringing intake below output. All of the many treatment regimens have as their goal this simple task. Perhaps its very simplicity helps to account for an unfortunate aspect of treatment. Unable to understand why his patients cannot carry out this task, the physician often reacts punitively towards them. We have recently become aware that intense discrimination is practiced against obese persons, that it begins in childhood, and that it is continued throughout life to a degree that approaches that practiced against other minority groups. Before undertaking treatment of an obese person, the physician should assure himself that he will not add to this burden. For given the low probability that sustained weight reduction can be achieved, it may

be wisest to try to dissuade the patient from a treatment that may come to nothing more than still another experience of failure for him and a source of frustration for his physician.

The simplest way to reduce caloric intake is by means of a low-calorie diet. The best longterm 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 with the aid of 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 have been a profusion in recent years. Whatever effectiveness these diets may have is due in large part to their monotony—almost anyone will get tired of almost any food if that is all that he gets to eat. As a consequence, when he stops the diet and returns to his usual fare, the incentives to overeat are multiplied.

Fasting, which results in rapid weight loss, has had a considerable vogue as a treatment for obesity in recent years. Many obese persons find it relatively easy to tolerate. After two or three days without food, hunger decreases radically and the patient is able to get along well, as long as he

remains in an undemanding environment. For some massively obese persons, or for the occasional patient in whom rapid weight loss is indicated, it has some small rationale. However follow-up studies of persons who have undergone long-term fasts, show that almost all regain at least all of the weight they lost. Short-term (ten-day) intermittent fasts, by moderately obese persons, appear to have had somewhat better results, but adequate follow-up studies are not available. Because of the occasionally serious complications of fasting, this treatment should be carried out in a hospital.

Effective pharmacological treatment of obesity is largely confined to the amphetamines. These agents suppress appetite and, when used in conjunction with diet in a carefully planned treatment program, they may have limited usefulness. This usefulness is seriously limited, however, by the fact that the initial dose loses its effectiveness within a few weeks. Effectiveness can be restored by an increase in dose—a course that has been so frequently pursued by unscrupulous "diet doctors" and unsupervised dieters as to cast serious doubt on whether these drugs have any place in the treatment of obesity. In the face of today's widespread drug abuse, the mild and transient value of amphetamines in the treatment of obesity is probably outweighed by the danger posed by their abuse. This seems to be the view of the Bureau of Narcotics and Dangerous Drugs which is now taking away from physicians the option of prescribing amphetamines for obesity.

Thyroid or thyroid analogues are indicated for the occasional obese person with hypothyroidism, and probably not otherwise. Bulk producers may have limited value in eliminating the constipation that follows a marked decrease in food intake, but their effectiveness in weight reduction is doubtful. Four controlled studies of chorionic gonadotropin have found it to be ineffective in the recommended dosage.

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 those of normal weight. Furthermore, as mentioned earlier, increased physical activity may actually cause a *decrease* in the food intake of sedentary persons. This combination of increased caloric expenditure and (probably) decreased food intake makes an increase in physical activity a highly desirable feature of any weight-reduction program.

Treatment of obesity has generally followed a traditional medical model in which an authoritarian physician prescribes a diet and appetite-suppressing medication. The patient loses weight, if at all, largely to please the doctor and to meet his expectations. When the relationship is terminated or attenuated, the patient discontinues the diet and regains weight.

Until recently a surgical treatment for obesity would have seemed highly improbable. Within the past decade, however, a number of surgeons have attempted to treat obesity by decreasing food absorption via an intestinal bypass that short-circuits most of the absorptive surface of the intestine. Early results were discouraging. Although the first jejunocolic shunts produced large amounts of weight loss, there was a high incidence of sometimes lethal complications; the reestablishment of intestinal continuity was followed by rapid weight gain. More recently, experience with a jejunoileal shunt has been more promising. Complications have been fewer and less severe. Furthermore, the discovery of a critical length of absorptive surface that will maintain body weight at approximately normal levels has made a second operation, to restore intestinal continuity, unnecessary. Despite these advances, a jejunoileal shunt is fraught with many dangers and should still be considered an investigative procedure. Until further information is available, it seems wisest to limit it to carefully selected patients with massive obesity that has proved uncontrollable by other methods.

Group therapy extends the number of obese patients the physician can treat and probably also increases the effectiveness of treatment. One convincing study showed that patients treated in groups lost more weight than those treated individually on each of three regimens: anorexigenic agents, placebo capsules, and no medication. Evidence for the greater

effectiveness of group over individual therapy for obesity is sufficient to encourage the family physician to attempt this modality, and the psychiatrist can provide valuable consultative help in encouraging such an undertaking.

Group methods are being applied by two different kinds of nonmedical groups with promising results. Each may provide useful adjuncts to medical treatment. TOPS (Take Off Pounds Sensibly), a self-help group with a membership of over 350,000, has over 15,000 chapters in all parts of the country and welcomes collaboration with physicians. The TOPS program suffers from a high rate of drop-outs but those who remain may lose encouraging amounts of weight. Membership is almost exclusively female, and predominantly middle-aged and middle-class; such persons would seem to be good candidates for the program. Weight Watchers, a commercial organization, has been less carefully studied than TOPS. But the size of its membership—over two million—must provide a measure of satisfaction with its results. Both organizations provide powerful vehicles for introducing safe new measures for the control of obesity.

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 the medical-treatment failures reach the psychiatrist. This

process of selection makes it easy to understand why there is no evidence that psychodynamic psychotherapy is any more effective than other, less expensive, aids to weight reduction. Less understandable is the widespread belief in the efficacy of such psychotherapy.

Another unsupported belief is that there is value in uncovering the unconscious causes of overeating so that the patient no longer resorts to this form of response to conflict (according to the psychodynamic model proposed for the resolution of neurotic symptoms). Obese patients may produce interesting fantasies and memories in response to the therapist's interests, but, with one exception discussed below, such production is rarely useful in producing favorable changes in behavior. Many obese persons seem particularly vulnerable to the overdependency upon the therapist and inordinate regression which can occur during the uncovering psychotherapies. Psychodynamic psychotherapy probably cannot modify the symptom choice of persons who overeat in response to stress. Years after successful psychotherapy *and* successful weight reduction, persons who overeat under stress continue to do so.

Despite these limitations, psychodynamic psychotherapy has a place in the management of obesity, and an important place in the treatment of some carefully selected obese persons. Furthermore, obese persons may seek psychotherapy for other reasons than their obesity; helping them to cope

with their obesity may help them resolve their other problems. We have noted that many obese persons overeat under stress. When psychotherapy can help them to live less stressful and more gratifying lives, they are less apt to overeat. They may reduce and sometimes stay reduced. These benefits are not less significant for being nonspecific results of treatment.

Two conditions may constitute specific indications for psychodynamic psychotherapy: disturbances in body image and the binge-eating syndrome. Both have been successfully treated, with enduring weight losses. Neither condition has been influenced by other forms of treatment including, significantly, weight reduction. Psychotherapy of patients with these conditions frequently requires years to ensure lasting results. The process may be facilitated by modifications in traditional technique designed to minimize intellectualization and regression, to cope with the "conceptual confusion" described earlier, and to increase the patient's often seriously inadequate sense of personal effectiveness. Bruch has provided excellent descriptions of such measures in her extensive writings.

Behavior modification has recently been applied to the control of obesity, and it has already proven a major advance in treatment. Within four years of its introduction in 1967, every one of seven controlled studies demonstrated that behavior modification was more effective than a wide variety of alternative treatments. It is particularly important for psychiatrists

to know of these developments, for they have lagged in their appreciation of the potential of behavior modification. The discipline has been developed largely by psychologists, educators, and social workers. The extent to which the medical profession has defaulted is nowhere better illustrated than in behavioral therapy of the "medical" problem of obesity. Physicians participated in only one of the first nine applications of behavior modification to obesity.

Stuart has recently published an extensive description of the application of behavior modification to the control of obesity. A brief outline of a typical program consists of the following four elements:

1. Description of the behavior to be controlled. Patients are asked to keep daily records of the amount, time, and circumstances of their eating. The immediate results of this time-consuming and inconvenient procedure frequently are grumbling and complaints. But most patients acknowledge, often reluctantly, that keeping these records proves very helpful, particularly in increasing their awareness of how much they eat, the speed with which they eat, and the large variety of environmental and psychological situations associated with eating.

2. Modification and control of the discriminative stimuli governing eating. Most patients report that their eating takes place in a wide variety of

places and at many different times during the day. It has been postulated that these times and places become so-called discriminative stimuli for eating. In an effort to decrease the potency of the discriminative stimuli, patients are encouraged to confine eating, including snacking, to one place. In order not to disrupt domestic routines of the housewives, who form such a large part of the patients so far treated in these programs, the kitchen is usually selected as the site for eating. Further efforts to control discriminative stimuli include using distinctive table settings, perhaps an unusually colored place mat and napkin. In addition, patients are encouraged to make eating a pure experience, unaccompanied by other activities such as reading, watching television, or arguing with their families.

3. Development of techniques to control the act of eating. Specific techniques are utilized to help patients decrease the speed of their eating, to become aware of all the components of the eating process, and to gain control over these components. Exercises include counting each mouthful of food eaten during a meal, placing utensils on the plate after every third mouthful until that mouthful is chewed and swallowed, and introducing a two-minute interruption of the meal.

4. Prompt reinforcement of behaviors that delay or control eating. A reinforcement schedule, using a point system, is particularly helpful in the control of eating behavior. Exercise of the suggested control procedures

during a meal earns the patient a certain number of points. By this means, rapid reinforcement of the behavior is achieved. The points can then be converted into various other reinforcers, such as money or gifts from the spouse.

Conclusion

Obesity, a condition characterized by excessive accumulations of fat, is profitably viewed as a-result of a disturbance in the regulation of body weight. This disturbed regulation can result from several different kinds of causes: genetic and developmental, social and emotional, physical (inactivity) and neural (impaired brain function). The relative contributions of these different influences probably varies among different obese persons.

We have strong evidence that obesity adversely affects morbidity and mortality, particularly in this country and probably because of the high saturated fatty acid content of the diet by means of which most Americans become obese. Obesity is also closely associated with many physical disabilities. The increased morbidity, mortality, and physical disability are all reversed by successful weight reduction. Because of these evident health benefits, and for cosmetic reasons, and because weight reduction should be easy, large numbers of obese persons are always trying to diet. For the most part they are unsuccessful. The poor results of weight-reduction efforts are

due not to failure to implement any therapy of known effectiveness but to the fact that no simple or generally effective treatment exists. Obesity is a chronic condition, resistant to treatment, and prone to relapse.

New therapies, developed within the past decade, have achieved somewhat better results than the older ones. Most promising among these is behavior modification, which has proved more effective than a number of alternative measures. Intestinal by-pass surgery may, for the first time, offer some hope to massively obese persons who are willing to accept its risks. Lay groups may provide a useful vehicle for the introduction of new treatments as they are developed. But the main hope for 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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