The polarity in the level and type of concern generated by obesity is consistent with the controversies that surround almost every aspect of this condition. The purpose of this review is to examine some of the data that bear on this problem and thereby to reduce the dogmatism and increase the skepticism of the reader. For purposes of discussion the controversies are grouped under five headings: (1) definition and measurement, (2) natural history, (3) etiology, (4) significance, and (5) treatment. This paper does not include any discussion of the extreme states of obesity characterized by body weights in excess of 100% above expected values.

**Definition and Measurement**

Obesity is the presence of excessive body fat. Such a definition is a simple one but contains two controversial concepts: how to measure body fat quantitatively and how to determine what is excessive.

The amount of body fat in a dead subject has been measured directly on an experimental basis for many years. Such measurements form the fundamental standard against which less direct methods are evaluated. The two methods currently used on a research basis and reasonably well validated against direct measurements are densitometry and whole body $^{40}$K content. Each of these techniques has restrictions in application and requires acceptance of certain assumptions about the constancy of the lean body mass—the measurement of which is also subject to controversy. Nevertheless, weight submerged in water compared to weight in air (densitometry) and whole body scintillation counting of the naturally occurring $^{40}$K are the best methods currently available for living subjects. Measurement of total body water is similar to the $^{40}$K technique, relying upon an assumption of constancy of lean body composition.

### Abbreviations used

- TSNS: Ten State Nutrition Survey
- SES: socioeconomic status

Body weight is the most commonly used measurement for assessment of obesity. In spite of the relative ease of determination, there are four major difficulties with the use of body weight to reflect fatness. The first relates particularly to children, in whom weight is a function of age and must be evaluated on an age-specific basis. The second difficulty is that weight is highly correlated with stature independent of age; thus, weight needs to be related to both age and height. Sex differences are well
substantiated and must be included in the standards. Finally, there is the less well-defined problem of body build. At any age or height, an individual can have a different lean body mass and, therefore, a different weight independent of the amount of body fat. Thus, for weight to be a reliable measure of fatness, age, height, sex, and body build are all variables to be considered.

At least two approaches have been developed to evaluate body build: radiographic analysis of bone and muscle mass in an extremity and somatographic classification of nude body photographs. Although both techniques have their proponents and have been useful investigative tools, each requires more equipment and/or technical skill than is available for widespread clinical utilization.

Another approach involving weight has been to examine exponential functions of weight and height that would provide an approximation of a linear relationship between the two. Three equations have been proposed and examined: (1) \( \frac{\text{weight}}{\text{height}^2} = \text{body mass index} \); (2) \( \frac{\text{weight}}{\text{height}^{1.75}} = K \), a constant; (3) \( \frac{\text{height}}{\text{weight}^{1/3}} = \text{ponderal index} \).

The ponderal index has been utilized to re-examine insurance company life expectancy data. When weight for height was used nonexponentially there appeared to be a continually decreasing life expectancy as weight increased above the mean value. However, when the ponderal index was utilized there was a curvilinear relationship so that life expectancy did not decrease markedly until the index fell below 12 to 12.3,11 which tended to correspond to a weight that was more than 20% above the median.

Since body weight is the most commonly used measure to reflect obesity, it is important to indicate the limitations and problems inherent in this approach. Below age 6 to 7 years, use of weight tends to result in an underestimate of fatness, so that some children who are no more than 10% overweight may actually be obese. At adolescence, however, use of weight frequently results in an overestimate of the amount of fat; adolescents who are 20% overweight may not actually be obese. Without independent measures of the skeletal and muscle components of the body, therefore, weight is not a precise indicator of obesity in the individual person.

There are two other problems inherent in the use of weight: what to use as a standard of reference and what level of weight to consider excessive.

None of the six commonly used standards is ideal because all but the National Health Survey are based on specific groups of children studied years ago. Although the National Health Survey used a national probability sample and is contemporary, separate standards for black and for white children are necessary. Nevertheless, for following the progress of the individual child, any one of these can be used because one is looking for major deviations from a standard pattern; any error in the standards is minor in this context.

For centuries “a pinch of the flesh” was an unscientific, but generally useful, way to assess adiposity. With the advent of calipers that yielded reproducible measurements, the lay approach became a scientific method. Although there has been no officially recognized site for measuring the skin fold thickness, the triceps and the subscapular areas are commonly used. Standards for these measurements have been published by Tanner and Whitehouse and by the Committee on Nutritional Anthropology. Many of the results discussed in this paper were derived from the skin fold study of the Ten State Nutrition Survey. The method can be done practically as part of a clinical evaluation, is reproducible and reliable, and is the method of choice for assessing fatness in children of all ages.

The level of fatness which should be considered indicative of obesity will determine the prevalence of obesity in a population. The question of prevalence is, therefore, a definitional one, since we have no well-defined biologic basis on which to determine any particular definition of the cutoff point between “normal” and “obese.”

**NATURAL HISTORY**

The paramount question in the natural history of obesity is, “Does the fat infant become the fat child and in turn the fat adult?” One can go back further and insert the fat fetus into the progression, or begin with a fat mother leading to a fat fetus. The concept of a fat fetus developing into a fat adult has become increasingly attractive with the advent of reports on the growth of the adipose cells, first in number and then in size. Presumably there are two major periods of increase in the number of adipocytes—the perinatal period and adolescence; during the remainder of life they change only in size. Inherent in the thesis is that the number of fat cells never decreases from whatever level has been reached; reduction in body fat is accomplished solely through reduction in cell size. One must then postulate a mechanism which allows the increased number of fat cells of the obese infant to exert some constant stimulus which would tend to maintain their usual stores of lipid. No such mechanism is known.

The validity of the cell number–cell size hypothesis as the basis for a positive reinforcement of the fetus-infant-child-adult progression of obesity is not firmly established. Application of the experimental findings to the human life cycle is basically inferential and founded on both cross-sectional and retrospective research in man.
and extensive investigations in the rat and other animal species. Other criticisms include the technical problem that very small or "empty" fat cells will not be counted in most methods; the extrapolation from the few milligrams of fat tissue examined to the kilograms of fat in the entire body poses major sampling problems and, finally, there is the comment from Mann: "This notion that an excess of a special cell leads inexorably to an excess of its special function is bizarre in biologic affairs."

A more direct approach to the question of the chronologic progress of obesity is to examine the same individuals at successive intervals in their life span. Few studies cover the entire period from infancy to adulthood in a prospective approach but some cover shorter periods of life. Eid examined the physical growth of children who had excessive weight gain in the first six months of life. He divided the infants into three groups, rapid, medium, and slow weight gainers, in terms of the rate of weight gain in the first half year of life. A significantly greater proportion of the rapid group than of the slow and medium groups were more than 10% above expected weight at 6 to 8 years of age (20% vs. 7%, p < 0.025). However, the rapid group was also taller and had had a lower birth weight; males in this group outnumbered females 2:1, though the overweight children in all groups were equally divided between males and females. In addition, the skin fold data, when analyzed by the distribution of values, suggest that the slow group had the greater degree of fatness, a conclusion opposite to that of the authors.

Mellbin and Vuille also examined physical development at age 7 years in relationship to infantile weight gain, using height increase from 0 to 7 years and weight for height at 7 years as the dependent variables, and a variety of weight gain periods in infancy as the independent variables. They concluded that weight gain in the first year of life, or in any fraction of the first year, was not a strong predictor of obesity at 7 years of age. In examining the relationships between events at one period of life and subsequent abnormal states at a later period, two terms were introduced, relative risk and population attributable risk. Relative risk refers to the likelihood that a group with some characteristic early in life will develop the later sequelae; this is then a prospective view. Population attributable risk represents a retrospective view, examining the percent of the group with the later sequelae who, in fact, had the earlier factors in their history. These concepts are illustrated in Table I, in which data from one of the three models are presented, representing the model with the strongest relationships. Boys who gained weight rapidly (the upper 15%) had a significant risk for being overweight at age 7 and, of the obese boys (> 20% overweight), 70% were in the initial criteria group. The girls did not follow this pattern in this model nor were they similar to the boys in the other models.

Huenemann has presented data on the factors related to the development of obesity at 6 months of age and then followed the children for 3 years using the Wetzel grid. None of the children with high weight for height at age 3 years had been in that group at 6 months, and there had been no consistent direction of change for the other children.

Asher studied children from two obesity clinics in terms of the onset of their obesity. Forty-four percent were stated to have been obese since infancy; about half became obese after 5 years of age. In addition, the school health records of 2,000 children were reviewed at age 15. Of the 101 who were above the ninety-seventh percentile in weight for height at age 15, 16 were obese at age 5; and of the 28 known to be obese at age 5, 16 were still obese at age 15. The attributable risk was low but the relative risk was higher.

A retrospective study of 158 obese adolescent girls by Heald and Hollander noted that the mean weight of these girls at one year of age had been 1.5 pounds greater than that of a control group of nonobese girls. The birth weight of the obese girls was less than that of the control group, but not at a significant level.

### Table I. The concepts of relative risk and attributable risk

<table>
<thead>
<tr>
<th>Initial criteria</th>
<th>Weight gain 0-4 mo (kg)</th>
<th>Weight gain 9-12 mo (kg)</th>
<th>Weight groups 6-8 yr</th>
<th>Relative risk for excessive final weight</th>
<th>% of final excess weight group who met initial criteria (attributable risk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boys</td>
<td>&gt; 4.15</td>
<td>&gt; 2.27</td>
<td>3.5</td>
<td>16.9</td>
<td>27%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>under relative risks, the numbers indicate the increased likelihood of being overweight when infancy weight gains were high as compared to the &quot;average&quot; infant; thus, a boy who gained more than 4.15 kg in the first four months of life and more than 2.27 kg in months 9 to 12, would have 16.9 times the likelihood of being overweight by more than 20% at age 7 years than would an &quot;average&quot; male infant. The attributable risk figures represent the percent of the overweight group that were in the high weight gain group as infants. Thus, of the boys who were more than 20% overweight at age 7 years, 70% had been in the large weight gain group in infancy.</td>
<td></td>
</tr>
<tr>
<td>Girls</td>
<td>&gt; 3.75</td>
<td>&gt; 2.08</td>
<td>1.6</td>
<td>1.3</td>
<td>8%</td>
</tr>
</tbody>
</table>

Data from Mellbin and Vuille.

*Under relative risks, the numbers indicate the increased likelihood of being overweight when infancy weight gains were high as compared to the "average" infant; thus, a boy who gained more than 4.15 kg in the first four months of life and more than 2.27 kg in months 9 to 12, would have 16.9 times the likelihood of being overweight by more than 20% at age 7 years than would an "average" male infant. The attributable risk figures represent the percent of the overweight group that were in the high weight gain group as infants. Thus, of the boys who were more than 20% overweight at age 7 years, 70% had been in the large weight gain group in infancy.
Table II. Relative risk for women to be overweight at 30 to 35 years of age; the risk for overweight adolescents (>120%) to be overweight as adults is 18 times greater (72% vs. 4%) than for girls who were 95 to 105% of expected weight at age 10 to 13 years

<table>
<thead>
<tr>
<th>Childhood weight status, age 10-13 yr</th>
<th>No. examined</th>
<th>&lt; 95% expected weight</th>
<th>95-105% expected weight</th>
<th>105-120% expected weight</th>
<th>&gt; 120% expected weight</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>95-105% expected weight</td>
<td>50</td>
<td>82%</td>
<td>82%</td>
<td>14%</td>
<td>4%</td>
<td>100%</td>
</tr>
<tr>
<td>105-120% expected weight</td>
<td>14</td>
<td>50%</td>
<td>50%</td>
<td>14%</td>
<td>36%</td>
<td>100%</td>
</tr>
<tr>
<td>&gt; 120% expected weight</td>
<td>36</td>
<td>8%</td>
<td>8%</td>
<td>20%</td>
<td>72%</td>
<td>100%</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>51%</td>
<td>51%</td>
<td>18%</td>
<td>33%</td>
<td></td>
</tr>
</tbody>
</table>

Data from Abraham and Nordsieck.34

Table III. Relative risk data for males; males at 10 to 13 years had a sixfold greater risk of being equally overweight at 45 to 50 years than did 10- to 13-year-old boys who were 95 to 105% of expected weight at that age (63% vs. 10%)

<table>
<thead>
<tr>
<th>Childhood weight status, age 9-13 yr</th>
<th>No. examined</th>
<th>&lt; 95% expected weight</th>
<th>95-105% expected weight</th>
<th>105-120% expected weight</th>
<th>&gt; 120% expected weight</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 95% expected weight</td>
<td>223</td>
<td>50%</td>
<td>29%</td>
<td>17%</td>
<td>4%</td>
<td>100%</td>
</tr>
<tr>
<td>95-105% expected weight</td>
<td>358</td>
<td>26%</td>
<td>33%</td>
<td>30%</td>
<td>10%</td>
<td>100%</td>
</tr>
<tr>
<td>105-120% expected weight</td>
<td>117</td>
<td>10%</td>
<td>29%</td>
<td>41%</td>
<td>20%</td>
<td>100%</td>
</tr>
<tr>
<td>&gt; 120% expected weight</td>
<td>19</td>
<td>0%</td>
<td>16%</td>
<td>21%</td>
<td>63%</td>
<td>100%</td>
</tr>
<tr>
<td>Total</td>
<td>716</td>
<td>30%</td>
<td>31%</td>
<td>28%</td>
<td>11%</td>
<td></td>
</tr>
</tbody>
</table>

Data from Abraham and associates.35

Abraham and associates34, 35 studied adults who had had height and weight measurements in elementary school. Some of the results are shown in Tables II to V. In Tables II and III the outcome of the various groups of children is given to depict relative risk. Of the boys and girls who were more than 120% of the expected weight just prior to adolescence, two-thirds were in the same category as adults, whereas those who had average or below average weight were only one-ninth as likely to become an obese adult.

Tables IV and V reflect attributable risk. Of the obese adult males, only 15% had been obese and most had been of average weight as children. In contrast, about four out of five obese adult females were obese as children. If obesity in childhood could have been abolished, we might have prevented obesity in 15% of the obese men in this study and in a large number of women—if, in fact, there actually is a cause-and-effect relationship between childhood and adult obesity. These data establish a relationship, but it could be other than a cause-and-effect one, as for example a genetic trait or the persistence of strong environmental factors.

In a study by Charney and associates,36 the weights of 366 infants born between 1945 and 1955 were compared with their adult weights. Infants whose weights were above the ninetieth percentile during the first six months of life had about a 2.5 times greater chance of being 10% or more overweight as an adult (36% vs. 14%). On the other hand, examining attributable risk, just over half of the overweight adults were identified in the heavy group of infants. Level of education and social class were shown to be significant variables but the number of males and females was not stated nor were the analyses of social class differentiated by sex. The sample was predominantly middle to upper class, as might be expected of a group derived from the practices of private pediatricians. Other problems inherent in this report are the implied similarity between overweight and obesity, and the use of a sample derived from “regular attenders” in pediatricians’ practices and whose parents remained in the Rochester area for 20 to 30 years.

In considering the data from the Ten State Nutrition Survey, it should be recognized that (1) this was a cross-sectional study, so the adults had been children in an earlier generation; (2) the TSNS was not done on a representative sample of the United States population;
Table IV. A modified attributable risk table; in this situation, 79% of the women who were greater than 120% of expected weight had also been in that category at 10 to 13 years of age

<table>
<thead>
<tr>
<th>Childhood weight status, age 10-13 yr</th>
<th>Adult weight status—females* age 30-35 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. examined</td>
</tr>
<tr>
<td>95-105% expected weight</td>
<td>50</td>
</tr>
<tr>
<td>105-120% expected weight</td>
<td>14</td>
</tr>
<tr>
<td>&gt; 120% expected weight</td>
<td>36</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

Data from Abraham and Nordsieck. Since the below average weight group is not included, the figures in this table are not a correct representation of attributable risk.

Table V. Attributable risk data from males indicates that only 15% of the men who were overweight as adults (> 120% of expected weight) had been in that weight classification at age 9 to 13

<table>
<thead>
<tr>
<th>Childhood weight status, age 9-13 yr</th>
<th>Adult weight status—males age 45-50 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. examined</td>
</tr>
<tr>
<td>&lt; 95% expected weight</td>
<td>223</td>
</tr>
<tr>
<td>95-105% expected weight</td>
<td>357</td>
</tr>
<tr>
<td>105-120% expected weight</td>
<td>117</td>
</tr>
<tr>
<td>&gt; 120% expected weight</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>716</td>
</tr>
</tbody>
</table>

Data from Abraham and associates.

and (3) obesity was defined on the basis of fat fold thickness rather than weight. On that basis, a sex difference was readily apparent. Females were fatter than males at all ages; adolescence was a period of increasing fatness in the female whereas there was a transient decrease of fatness in the male. The higher the socioeconomic status of children, both male and female, the greater was the amount of adipose tissue. For comparable income groups, white children were also fatter than blacks. The direct relationship between socioeconomic status and adiposity continued into adult life for the males but became reversed for females; the lower income adult females were fatter than their wealthier counterparts. Abraham and Nordsieck may have observed the same phenomenon in a nonpoverty group.

Stunkard and others examined the relationship between social class and obesity in adult populations from midtown Manhattan and from London. For all adults, this work demonstrated an inverse relationship between obesity and socioeconomic status, lower status being highly correlated with obesity. In contrast to the TSNS, however, Stunkard's group noted the same, though weaker, relationship between obesity and SES for men as they did for women. The differences may be due to the use of weight by Stunkard's group versus fat fold in the TSNS, different measures of socioeconomic status, and/or the highly urban source of his study populations. There was also great social mobility in the Manhattan population; no comparable data are available from the TSNS.

The contrast between the work of Stunkard and the data of the TSNS is not readily explained, but might indicate that there is nonlinear distribution of adiposity among children, with a lower middle class that is most obese and decreasing obesity in the upper middle and very low socioeconomic classes. In adult populations, a nonlinear relationship between obesity and socioeconomic class could also explain these discrepancies if the distributions were highly skewed in opposite directions for men and women.

In summary, the data on the natural history of obesity substantiates a significant tendency for obese children to be obese as adults when compared to children who are not obese. It would appear, however, that most adult obesity does not originate in childhood. These trends, moreover, are greatly modified both by gender and socioeconomic class.
ETIOLOGY

If basic thermodynamic principles are to be maintained, obesity or storage of calories can occur only if there is increased caloric input, reduced energy output, or more efficient caloric utilization. There are reasonable arguments for each of these mechanisms and each could be operative in given individuals.

The qualitative aspects of eating have been studied, but most authors have cited excessive caloric intake as one of the principal causes of obesity at any age. In recent years, however, there is an increasing tendency to acknowledge that many obese individuals may not eat more than their peers. Johnson and associates found that obese adolescent girls consumed about 2,000 calories and the nonobese, 2,700 calories. In the study of Hampton and associates, obese boys ate 2,600 calories versus 3,200 calories for the nonobese; for girls in this study, the difference was 300 calories. Huenemann found that 6-month-old infants in the upper 5% for weight ingested 790 calories whereas those in the lower 5% for weight ate 713 calories; on a per kilogram basis the direction of this difference was reversed, the leanest consuming 98 calories/kg and the heaviest, 87 calories/kg.

Median caloric intake, by definition, is excessive for half our population. Half of the population, then, has the propensity to become obese if it consumes the “average” intake for age. This becomes of great consequence for infants, since the amount they are fed is frequently based on their age and size, and does not take into account their activity levels and thus their actual requirements for calories. Infants whose caloric needs are above average seem to have no trouble making their wants known, whereas the relatively overfed are peaceful and quiet—further reducing their needs and increasing the net surplus of energy.

The factor now receiving increased attention and support is reduced activity. Although activity remains difficult to quantitate, both observational and controlled studies have documented the reduced activity levels of obese infants, adolescents, and adults. An early study by Edholm and Fletcher of students at a military school illustrated the phenomenon well. The caloric intake of all students was nearly identical but some of the young men were gaining weight and others losing on a daily activity schedule that was similar for all. Careful measurements of caloric consumption and energy expenditure were made. For the same activity, the variation in caloric expenditure was large and could have explained their observations. Other quantitative studies have confirmed such patterns in various age groups. Jean Mayer has stated that activity may account for 17 to 39% of an infant’s caloric intake whereas growth accounts for less than 10%. His film studies of adolescents at play show that obese girls, for example, were in motion 50% of the time while playing tennis and thin girls were in motion 90% of the time.

Although a variety of metabolic alterations have been considered as possible factors affecting the balance between caloric intake and output leading to increased energy storage as fat, most of these hypothetical mechanisms have not been substantiated. Recently, work by Derek Miller and associates has revived interest in what was called specific dynamic action; they postulate that activity following meals markedly increases thermogenesis in people of normal weight, so that many more calories are expended as heat during physical activity following meals than when the same physical activity takes place at other times. One could infer that obese persons have decreased postabsorptive thermogenesis and that calories that should have been lost as heat are stored as fat. The significance of this factor in terms of all obese individuals remains to be clarified.

Of the three factors leading to caloric surplus—increased intake, reduced activity, and altered metabolism—current opinion tends to emphasize reduced activity. Underlying these physiologic factors are biologic, social, and psychic forces.

In the psychosocial area, much of the literature on the psychodynamics of the obese person is “after the fact”; thus it is difficult to know whether psychologic forces led to the obesity or resulted from it. Without negating the importance of such internal psychic factors, this review focuses on the external biologic and social issues that may be etiologically significant.

Much has been stated both pro and con regarding the genetic basis of obesity. What we are from birth on is almost always the result of nature and nurture, albeit in differing proportions for different conditions. Most investigators accept the idea of a genetic messenger playing some role in obesity. Reduced levels of physical activity, alteration in food-intake control mechanisms, differing metabolic responses, or the psychic makeup that increases the propensity to oral gratification instead of other methods of satisfaction are a few traits that could be involved genetically, but none is established. Studies of mono- and dizygotic twins reared in different environments, of natural and adopted children raised in the same family, and of parent-child correlations have been used to substantiate some genetic element in the etiology of obesity. On the environmental side, Shenker and associates found that infants placed with obese foster mothers tended to become more obese than infants placed with nonobese foster mothers. In the Ten State Nutrition Survey, strong family-line trends were also evident; the child of obese parents was three times as...
likely to be obese as the child of lean parents. If only one
parent was obese, obesity in the father increased the
likelihood of obesity in the child of either sex to a greater
extent than did obesity in the mother. Sibling correlations
in fat-fold thickness were about 0.4 and the parent-child
correlation coefficient was about 0.3; perhaps more signif-
icant was the correlation between husband and wife of
approximately 0.25. Garn and associates found that the
similarities in fat-fold thickness between children and
their adoptive parents were only minimally less than those
for children and their biologic parents. Thus, those
studies that have been cited to support a genetic element
in obesity are equally supportive of strong familial envi-
ronmental influences.

Maternal weight and weight gain during pregnancy
have been shown to have a positive effect on the birth
weight of the human infant and studies have shown a
positive correlation between birth weight and weight in
the first year of life. Other work, however, demonstrates
that infants of larger birth weight tend to gain less weight
in the first year of life and may be lighter as children than
smaller newborn infants, who tend to gain more weight in
the first year of life. From the environmental view
point, an obese woman is more likely to have an obese
husband, and the offspring of either one or two obese
parents is more likely to be obese than the offspring of
lean parents. Women of lower SES are more obese, but
in the TSNS men and children of higher SES tended to be
fatter; this may explain why obesity in the father appears
to correlate better with obesity in the child than does
obesity in the mother.

The type of food and the frequency of eating have been
implicated as biologic factors in obesity. Hypothetically,
bottle-fed infants should be more likely to eat excessively
than breast-fed infants. The ease of seeing the volume of
milk consumed could be one element in such an hypothe-
sis. A prospective study by Thomson on 40 pairs of first-
born, singleton female infants, divided into breast- and
bottle-fed groupings, and matched for birth weight,
showed identical weight gains by both groups at 16 weeks,
prior to the introduction of other foods. studied 21 breast-fed and 240 bottle-fed infants and noted that a
significantly higher percentage of the bottle-fed infants
were above the ninetieth percentile in weight gain velocity
between birth and six weeks of age. However, Huenemann
found an equal percentage of 6-month-old breast-
fed infants in the upper 10% and in the lower 10% of her
obesity index. Bernal and Richards point out that the
infant who is breast fed (and the mother who chooses
breast feeding) may be different in many ways beside the
type of feeding from the bottle-fed infant (and its moth-
er). Eid’s study of 138 infants showed no significant
difference in the incidence of obesity at age 6 to 8 years
between breast- and bottle-fed babies. Mellbin and
Vuille also found no difference in the weight for height
relationships between 7-year-old children who had been
predominantly bottle fed and the 7-year-old children of
30 years previously, when the majority of Swedish infants
were breast fed. Thus, the available evidence does not
support bottle feeding as a specific factor leading to
obesity.

Closely allied to the breast and bottle controversy is the
one involving the early introduction of solid foods. Dwyer
and Mayer state: “The current trend toward early
feeding of solid foods has perhaps generated a new type of
overfeeding: increased caloric concentration per unit
volume.” Those who espouse this view contend that the
infant responds to volume of intake and that the use of
those infant foods with caloric densities greater than milk
will lead to excessive energy intake and thereby to obesity.
The problem is further complicated if these foods are high
in protein, which increases renal solute load, thus raising
urine volume and creating thirst which is often responded
to with more milk, further increasing the caloric over-
load. Data supporting the predicted result of the earlier
introduction of solid foods are difficult to obtain,
however. Shukla and associates believe that if obesity
resulting from the early introduction of solid foods is a
serious problem, it is only so for the first 6 months of life. At 6 months of age, Huenemann found that the age at
which solid foods had been introduced was the same in
the most obese and least obese groups in infants.

Thus, there is no incontrovertible evidence that bottle
feeding and early introduction of solid foods are potential
factors leading to infantile obesity. There is also little to
indicate that the type of diet fed to children beyond
infancy in the United States is of itself resulting in
significant adiposity. In the TSNS, dietary analysis indi-
cates that the quality of the diet, in terms of essential
nutrients per 100 calories, is similar for individuals of all
socioeconomic levels. The poor appear to have fewer
calories to eat and their growth records reflect this
fact.

Frequency of feeding has been found to affect the
development of obesity in experimental animals. The
less often the animal is fed an isocaloric diet, the more
likely and the greater the extent of obesity, but there have
been no reports substantiating this in man.

The forces derivative from social status, attitudes, and
customs are probably the dominant ones in the estab-
ishment of adiposity. Social factors, to the extent that
they are effective, must operate through individuals; two
of the most significant are the mother and the physician.
Since the child of the middle class is fatter than the lower-
class child, one can examine the socially derived attitudes and practices of the middle-class mother. One of the most well-established attitudes is the equating of "fat baby" with "healthy baby." Probably related historically to the fat-health concept is the "clean plate club" and slogans such as "waste not, want not." The use of such phrases must encourage some children to eat beyond the appropriate levels of satiety. The conversion of eating from a means of satisfying hunger to a means of relieving tension, showing affection, or getting attention probably makes it difficult for older children, adolescents, and adults who have been obese since early life to restrict their intake over long periods of time. Successful parenting, especially mothering, is hard to recognize in the abstract, so it seems quite natural to try to demonstrate one's aptitude to be a parent in more concrete ways, such as providing a surfeit of food and getting the infant or child to exemplify this by being "robust" or, more blatantly, fat. One English study found that children without siblings and without fathers tended to be more obese.8

Interestingly, among the more nutritionally sophisticated mothers, an anxiety about undernutrition and the possibility of resultant subtle, but serious, brain damage is beginning to appear as a rationalization for overfeeding an infant. The use of high carbohydrate snack foods as a reward system, and its promotion on television, both indicate the social pressure to give food and the eating process a significance far beyond a nutritional one. Similar social pressures have been directed toward weight reduction. Because it is difficult to quantitate the social and psychologic significance of food and eating, substantiating data are difficult to identify.19

The physician is also significantly affected by social attitudes, customs, and practices.89 The typical physician of today is likely to be a male of the upper middle class and moderately obese himself, which may decrease his concern about the problem. (Women physicians would be likely to be thin and, accordingly, might be less tolerant of obesity in others.) Physicians have not been inclined to work enthusiastically, and thereby effectively, in areas of medicine in which emotional and social factors tend to predominate. If obesity in infancy is a serious long-range problem, some technique will have to be found that will alert physicians to recognize and to consider the seriousness of early excessive fat and to prescribe measures that can be used to control it. Furthermore, unless the use of skin-fold calipers becomes routine, the lack of correlation of fatness with weight in the first 6 years of life could lead to inappropriate caloric restriction in the large but not obese child, or to equally inappropriate reassurance to the mother of the child of normal weight who is, in fact, obese.

**SIGNIFICANCE**

From the early experiments of McCay93 and the later ones of Ross,84 which demonstrated a longer life expectancy for the underfed rat than for his better nourished litter mates, a growing body of literature has shown the adverse effects of excessive nutrient intake in experimental animals. In recent years the public and the medical profession have become concerned with the idea that "fat is fatal"—and the more fat, the more fatal. The result has been a proliferation of weight reduction specialists and clinics, diet books, and "lose weight without dieting" gimmicks. If all this activity actually resulted in sustained weight loss, would the health of the country be improved? There are beginning to be those who doubt it.

Hutchinson,90 commenting on an extensive study of height, weight, and mortality by the Society of Actuaries, stated that one had to be more than 25% overweight before the risk of increased mortality was apparent. This position was in sharp disagreement with that of most insurance companies, which tended to ascribe increasing mortality to the degree of overweight, beginning at the lowest level of excess weight. Seltzer95 re-examined the data and calculated the ponderal index (height/cube root of weight)* for males; he found that the mortality did not rise appreciably until the ponderal index was below 12.3, values found in those 20 to 30% overweight.

In 1974, Mann96 reviewed the influence of obesity on health. His work represents a balanced view of the entire problem and his conclusions are summarized below.

1. Hypertension and obesity do have a positive correlation but the evidence for a causal relationship is scant: "In particular, there is little to support the widespread dogma of health-education programs that regard obesity as a cause of high blood pressure and treatment of obesity as a useful way of managing high blood pressure." Mann emphasized that unless the rubber bladder of a blood pressure cuff completely encircles the arm, a spuriously high reading will be obtained. There has been great emphasis on cuff width but it appears that cuff length is also critical for obtaining accurate values for blood pressure, especially in obese children.

2. On the basis of several large studies, Mann concluded that obesity had little effect on coronary heart disease.

3. In spite of the questionable effect of obesity on hypertension, there appears to be a significant increase in cerebrovascular accidents in obese individuals. This observation is confirmed and extended in a study which

*The original ponderal index was \(\sqrt{\text{Wi/\text{Ht}}}\) but this was reversed by Sheldon and associates.11 Using the original version, the numbers would be smaller and opposite in direction, but the results would be the same.
relates the incidence of stroke to weight at age 20 and weight gain thereafter.36

Maturity onset diabetes mellitus is highly associated with obesity and improvement is noted with weight reduction. Relative insulin resistance is noted generally in obese individuals and the obese non-diabetic person has higher levels of serum insulin than does the nonobese.37

Abraham and Nordsieck31 concluded: "Childhood relative weight at ages 9 to 13 had no significant relationship to adult levels of fasting blood sugar, serum cholesterol, beta-lipoprotein, or blood pressure, or to cardiovascular renal disease. The markedly overweight children (> 20%) had a tendency to higher blood sugars, blood pressures and cardiovascular renal disease as adults than did the other children, but the differences were not statistically significant. Childhood relative weight at age 9-13 was significantly related to hypertensive vascular disease. The below average weight group experienced a higher prevalence than observed in either average or moderately overweight childhood groups." The markedly obese children had a higher incidence than all other groups but the difference was not significant. It appears that whatever morbidity or mortality risks are associated with childhood and/or adult obesity, the problem is predominantly one for those more than 20% overweight.37

For all persons, but particularly children, the greatest hazards from excessive adiposity are psychologic and social. Many studies have been made of the personality characteristics of obese children but it is often difficult to distinguish what preceded the obesity and what followed from it. The obese child often has a poor body-image, a sense of failure, and a passive external approach to life situations.37-39 Other than among those of the lowest SES, obesity has low-social value and there exists discrimination against the obese in hiring and promotion practices as well as in admission to organizations such as colleges and clubs.37-38 Thus, combining low self-esteem with social rejection poses extremely serious handicaps for attainment of goals by the obese child.

TREATMENT

The American Academy of Pediatrics’ Committee on Nutrition’s view that no treatment program has achieved more than “minor success” is probably still applicable today.36 About all that most individuals can agree upon are that the prognosis for a permanent response to treatment is poor and that the three basic requirements to achieve weight reduction are motivation, increased activity, and decreased caloric intake. Disagreements abound in terms of the relative importance of each of these requirements but the more serious question is whom to treat. Unfortunately, the vast amounts of time, effort, and money that are spent on weight reduction are almost entirely spent on those who are least at risk, i.e., those less than 20% overweight who will benefit least from weight reduction.36 Furthermore, since whatever weight is lost is usually regained soon thereafter and the cycle tends to be repetitive, we may be inadvertently increasing the risk for cardiovascular disease. Serum lipid levels are elevated when one is in positive caloric balance and gaining weight,36 and elevated serum lipid values may be related to increased risk for cardiovascular disease. Thus, losing and then regaining weight may create a greater risk than if once fat, remaining that way.

With occasional exceptions,17 most authors have stated that adults who have been fat from childhood have more difficulty losing weight than do those who become obese as adults. Knittle and Hirsch have suggested that this may lend credence to the cell hypertrophy/cell hyperplasia concept.74, 90 It is equally reasonable, however, to relate this difference to the fact that the behavioral and social factors leading to obesity may be much more intense and have operated for a longer period of time when the adult has been obese since childhood. Nevertheless, weight reduction of the very obese child probably has value because that child has a high likelihood of being a very obese adult with some health hazards and even greater social pressures.

The modalities available for treatment have focused on the three basic requirements of motivation, activity, and caloric restriction.164-166 Summer camps for obese adolescents have utilized all three elements; other group programs that stress all three usually leave the diet and activity aspects to family or self-enforcement.

Caloric restriction has varied from 20 to 100%. Total fasting has been done with children but the accompanying protein loss makes this approach, though effective, too dangerous except for brief periods of time under maximal medical supervision.

Under one year of age, dietary restriction, per se, does not seem indicated. Rather, intensive work with parents is warranted to assist them in better understanding how to meet the needs of their infant other than with food. Between the ages of one and five years, the dietary approaches that can be safely undertaken include the use of skimmed or half-skimmed milk, the elimination of carbohydrate snack foods, the removal of fat from meats by careful trimming, removing the skin from fowl, and the use of broiling, roasting, or boiling for cooking. Current consensus is that significant (20 to 25%) caloric restriction should generally be limited to children over five years of age and that the goal should be either stable weight while linear growth continues or weight loss of less than a half pound per week.37 Iron is usually the only nutrient that
could fall below accepted standards with this approach, assuming that a reasonably balanced diet is maintained. Fad diets or those with specific restrictions ultimately are contrived to restrict total calories."\(^{25}\)

There is no evidence that anorexic agents or chorionic gonadotropins are of any value and the potential danger is too high to consider them as ever useful in the growing child.

Physical activity, both specific and general, is valuable.\(^{25}\) If the increased thermogenesis resulting from exercise that follows meals is a generalizable phenomenon, recommendations about not exercising immediately after eating may have to be reversed. Increasing physical activity may be effective partly because children who are not physically active often eat instead.

The most successful approach to the treatment of obesity to date has been the psychosocial one. Group programs (e.g., Weight Watchers, TOPS) probably fall into this category and may be effective if tied to a financial incentive.\(^{19}\) Behavior modification is currently popular and apparently most effective,\(^{19,101-103}\) Stunkard\(^{104}\) and Mann\(^{26}\) have reviewed these modalities and endorsed them. The long-range value of the behavior modification approach remains to be established.

Closely allied with the behavioral methods for treatment is the general approach to prophylaxis. Although no one has ventured to predict what percentage of obesity could be obviated by a more rational approach to food and to eating, the assumption is that the impact would be large. The magnitude of the endeavor may make it impossible in the short run to implement a full prophylactic program for everyone, and efforts should be focused on individuals and families at higher risk. This would include those with a history of cerebrovascular accidents, diabetes mellitus, hyperlipidemia, and with one or both parents obese. Parents need to appreciate the social forces involved, their personal needs to appear as loving parents, and alternative methods for meeting their infants’ and children’s needs for love and attention, and information about nutrition—that which is established, that which is debatable, and that which is purely mythology. Of equal importance is the orientation of medical personnel, including physicians, in these same concepts in addition to careful observations of growth characteristics and the patterns of food utilization and consumption by the family and the child.

**SUMMARY**

The conclusions that one can state with assurance are remarkably few. Adiposity in children is best monitored clinically by the measurement of skin-fold thickness. The likelihood that obesity will progress from infancy to childhood to adult life is still questionable and may be more important in the individual child than as a significant cause of adult obesity. Behavioral and social factors predominate in etiology, yet biologic factors, including genetic, are also involved. Behavioral and social factors are critical in the significance of obesity to the individual. Although there are definite disease hazards, they appear to be predominantly confined to the very obese. The best treatment appears to be prophylaxis, again heavily involving behavioral and social factors as well as the widespread distribution of sound nutritional information. When prophylaxis fails, the behavioral modification approach to therapy currently holds the greatest promise.

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