Nutritional Aspects of Obesity in Infancy and Childhood

Obesity is characterized by an excess of adipose tissue relative to lean body mass. With rare exceptions, it simply reflects a long-term imbalance in energy intake vs expenditure. The excess energy is stored as fat. The known metabolic correlates of this state are, for the most part, secondary events. The day-to-day "error" in intake or expenditure necessary to derange long-term energy balance is smaller than the accuracy with which either factor currently can be measured over long periods; therefore, the question of etiologies remains unanswered.

The systems regulating mammalian fuel homeostasis and food intake are complex, and many potential "lesions" could alter long-term energy balance. There are a number of experimental and genetic animal models in which regulatory or apparent metabolic disturbances result in obesity, but no similar abnormalities have been consistently demonstrated in individuals with simple obesity. However, the traditionally accepted causes of obesity, relative overeating and/or physical underactivity, may not be operating in all instances of simple obesity.

ANTHROPOMETRY

Criteria for the diagnosis of obesity are difficult to establish because "optimal fatness is a conditional state. A man preparing for an emergency trek, a population entering a period of famine, a child entering a febrile illness or a growth spurt or a woman becoming pregnant will have physiological advantages from abundant stored fat." Medical considerations suggest that excessive adiposity (or leanness) is unhealthful; but cosmetic and other social considerations are generally preeminent in determining the acceptable range of body composition within a culture.

A variety of definitions of obesity have been devised for adults (weight-height indices that produce relative independence of weight from height). However, these measures do not provide an accurate assessment of obesity in children. There is considerable variance in body composition (fat-free weight) among growing children at different ages, particularly at the extremes of stature. In addition, most obese children (unlike obese adults) have an increased lean body mass for height, evidence of the auxotrophic effect of hypernutrition. An average of 10%, and in some instances more than 50%, of the obese child's excess weight is lean tissue.

Relative adiposity in childhood is most readily determined by comparing the triceps and subscapular skinfold thicknesses with age/sex appropriate standards. If skinfold calipers are not available, it seems reasonable to use weight-for-height grids as a practical screening device. However, these grids assess weight relative to height, do not control for age, and do not identify obesity per se. "False-positives" will occur with heavy muscular children, and "false-negatives" will occur with lighter children who have a relative excess of body fat. In addition, weight-for-height grids tend to underestimate adiposity in the youngest age groups (particularly those less than 3 years old). For these reasons, when a child's weight-for-height exceeds the 90th percentile, or there is a discrepancy between the child's appearance and his weight-height percentile, skinfold measurements should be used to assess body composition more accurately. The adolescent's rapidly changing body composition renders weight-height indices particularly inaccurate; therefore, the use of skinfold measurements for obesity assessment is strongly recommended for adolescents.

PREVALENCE AND MORBIDITY

The Ten State Nutrition Survey showed that the prevalence of obesity in adults (defined as triceps skinfold thickness greater than the 85th percentile) ranged from a low of 8.3% in 21-year-old black men to a high of 52.7% in 50-year-old black women. When a criterion of triceps skinfold thickness greater than the 95th percentile was used, there was a prevalence rate for obesity in children of 6% to 13%, depending on age, sex, and a variety of demographic factors. In general, obesity is more
common in females than males, and its prevalence tends to be directly related to socioeconomic status in childhood and inversely related in adolescence and adulthood.

Obesity in adults is associated with increased risks of cardiovascular and gallbladder disease, diabetes mellitus, hyperlipidemia, and endometrial carcinoma. Increasing excess mortality begins at 25% overweight. In children, diminished self-esteem, ostracism by peers, reduced physical activity and exercise tolerance, and increased stress on weight-bearing joints appear to be the major morbidity.

**ETIOLOGY**

The issues confronting the pediatrician are: (1) Is there a causal relationship, mediated either by learned behaviors or acquired adipose organ characteristics, between obesity in early life and the same condition in adulthood? (2) Are genetic factors largely responsible for body size and composition, with environment playing only a permissive role? (3) If either or both of these factors are true, are there safe means for moderating the processes?

Because "simple obesity" probably is a group of disorders lumped under one designation by our ignorance, the answer to all three questions may be "yes" in specific instances.

1. Recent studies regarding correlations of infantile obesity with that occurring in childhood and adulthood seem to indicate that: (a) Intrauterine nutrition, as reflected by size at birth, apparently influences the risk of obesity in childhood to a small but detectable extent. (b) The heaviest infants (upper decile for weight or size during the first six to 12 months of life) have a two- to threefold increased risk of being obese in childhood and adulthood. The reverse is true for the lightest, skinniest infants. However, most obese infants do not become obese adults, and most adult obesity is "unexplained" by obesity in infancy. (c) The correlations between obesity in late childhood, adolescence, and adulthood are considerably stronger than those in infancy. As many as 75% of obese adolescents are obese as adults. Thus, a "channeling" tendency for obesity is apparent from infancy and becomes stronger with increasing age. (d) Both genetic and environmental factors interact to produce strong correlations of obesity among family members.

2. New data from animal studies suggest that precursor cells for adipocytes may be constantly and rapidly turning over, even in the adult animal, and can be induced to mature into fat-filled cells by overfeeding. Once present, these adipocytes can be decreased in volume by weight reduction but probably will not disappear. The studies of Knittle et al on the ontogeny of the fat organ in humans point to the absence of a temporarily limited critical period in adipocyte differentiation and suggest that certain life intervals (before age 2 years and the adolescent growth spurt) may be the most important in childhood with regard to the development of fat organ cellularity.

3. In animals there is an apparent interaction of genotype and early nutrition in the establishment of adipose organ geometry (cell size and number). The adipocyte number can be altered by calorie intake manipulation in preweanling rats, but certain obese strains show persistent cell replication even when there is severe caloric restriction. When overfed, these animals develop even more severe adipocyte hyperplasia. If these models have any validity relative to human obesity, early caloric restriction might not fully correct a genetic effect in some instances of obesity. The few data available in humans imply that adipose organ development possibly could be modified by diet in both infancy and childhood.

Relative obesity in humans may be regarded as a complex force, the magnitudes of which vary from individual to individual. The description, quantitation, and therapeutic manipulation of these forces is largely uncharted territory.

**TREATMENT**

There is no known safe, effective, long-term treatment for obesity. The more radical treatments used in adults—jaw wiring, gastric stapling, small-bowel bypass—and central appetite suppressants and hormones should not be used in children. Caloric restriction to the point of weight loss should not be used for children, whose statural growth and CNS development could be impaired by a prolonged catabolic state. Growth of the child's lean mass should be supported, and the child's adipose mass should be held constant.

The diet should be designed to provide enough protein, minerals, and vitamins to meet lean tissue growth requirements. There is no reason to believe that one type of foodstuff calorie is not essentially equivalent to another; therefore, diets should be composed of usual foods in proportions of a normal, "balanced" diet. Specially concocted formulas and diets are to be eschewed. Well run, sensible weight-reduction programs (eg, Weight Watchers or Taking Off Pounds Sensibly [TOPS]) may be beneficial for older children and teenagers. The entire family (other members of which are also likely to need reduced-calorie diets) should become involved in long-term weight loss programs. Any child subjected to a weight-losing regimen must be observed.
assiduously for evidence that lean tissue growth is not being impaired; head circumference (for children less than 2 years old) and statural growth velocity are the best, readily available indicators. Adolescent girls desiring weight reduction should be carefully evaluated by a medical and social history to ensure that the regimen will not exacerbate incipient or actual anorexia nervosa.28

Long-term success is unlikely. Undue expectations should not be raised. A temporary downward deflection in adipose organ growth velocity may be all that can be reasonably expected from any regimen.

Current knowledge of the natural history of obesity suggests that a high percentage of obese older children and adolescents will be obese adults. These individuals should be taught that their “treatment” probably will be lifelong and necessitate continuing attention to caloric intake.

Treatment regimens for all age groups were discussed in a recent symposium on pediatric nutrition.29

PREVENTION

Because the prognosis for the nonsurgical treatment of obesity—only 10% to 30% of patients achieve and maintain a weight loss regardless of the type of therapy—is somewhat dismal, emphasis has been placed on the prevention of obesity. However, there are a number of complex issues involved in the treatment of already obese children.

1. Obesity is probably the somatic expression of a group of behavioral, metabolic, and regulatory syndromes. No single, preventive measure or treatment would be expected to be both safe and effective in all instances.

2. Obese or otherwise normal infants subjected to such prophylactic measures as feeding of low-fat milk may be exposed to excess renal solute loads and may learn behaviors (eg, increased volume ingestion) not conducive to subsequent caloric regulation.30

3. There is no proof that breast-feeding prevents or that early introduction of solid foods causes obesity.31,32 However, as long as there is a reasonable doubt regarding a causal relationship and there are good reasons other than obesity prophylaxis to encourage breast-feeding and a delay in introducing solids, it is prudent to continue to support both practices to promote optimal nutrition in infancy.

4. The role of moderate exercise in the prevention and treatment of obesity is not entirely clear. Regular exercise improves certain aspects of systemic carbohydrate and lipid metabolism and cardiovascular status; but the extent of its contribution to energy balance, via either caloric expenditure or appetite regulation, has been hotly and cogently contested.3 Therefore, regular exercise should be encouraged in children, but the justification for it perhaps should not be placed entirely, or even largely, on weight regulation.

5. In humans there is no proof that relatively short-term nutritional or exercise intervention of any kind can prevent obesity in an individual destined by elements of nature or nurture to become obese.

All efforts at prevention or treatment of obesity should be tempered by the foregoing considerations. Our knowledge of the pathogenesis of simple obesity in childhood is meager, and careful consideration should be given before any preventive or therapeutic regimen is prescribed. Nowhere is this more true than in regard to the urge of some health professionals to make firm dietary and life-style recommendations for large, heterogeneous populations. Thus, although one might like to believe that universal breast-feeding, delay in the introduction of solid foods, general caloric restriction in infancy, the abolition of all “junk foods,” and nationwide programs of physical exercise for children and adolescents would prevent childhood and adult obesity, there is no substantial evidence to support any of these proposals for the purposes alleged.

We do not yet have the theoretical framework and scientific data base from which a rational, effective, medical, or public health approach to obesity can be derived. Our most pressing need is for studies of the pathogenesis of obesity in early life, with an emphasis on the ontogeny of the fat organ and possible differences in intermediary metabolism/energetics in those destined to become obese. In addition, carefully controlled tests of prevention should be conducted in children who are at high risk for obesity by virtue of family history.

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John D. Benson, PhD
Technical Advisory Group
Rudolph M. Tomarelli, PhD
George A. Purvis, PhD
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