ABSTRACT. The prevalence of pediatric obesity is increasing in the United States. Sequelae from pediatric obesity are increasingly being seen, and long-term complications can be anticipated. Obesity is the most common cause of abnormal growth acceleration in childhood. Obesity in females is associated with an early onset of puberty and early menarche. Puberty is now occurring earlier in females than in the past, and this is probably related either directly or indirectly to the population increase in body weight. The effect of obesity on male pubertal maturation is more variable, and obesity can lead to both early and delayed puberty. Pubertal gynecomastia is a common problem in the obese male. Many of the complications of obesity seen in adults appear to be related to increased accumulation of visceral fat. It has been proposed that subcutaneous fat may be protective against the adverse effects of visceral fat. Males typically accumulate fat in the upper segment of the body, both subcutaneously and intraabdominally. In females, adiposity is usually subcutaneous and is found particularly over the thighs, although visceral fat deposition also occurs. Gender-related patterns of fat deposition become established during puberty and show significant familial associations. There are no reliable means for assessing childhood and adolescent visceral fat other than radiologically. Noninsulin-dependent diabetes is being seen more commonly in the pediatric population. Diabetes and impaired glucose tolerance are noted particularly in obese children with a family history of diabetes. In this situation, a glucose tolerance test may be indicated, even in the presence of fasting normoglycemia. Hypertriglyceridemia and low high-density lipoprotein–cholesterol levels are the primary lipid abnormalities of obesity and are related primarily to the amount of visceral fat. Low-density lipoprotein–cholesterol levels are not typically elevated in simple obesity. The offspring of parents with early coronary disease tend to be obese. Very low-density lipoprotein and intermediate-density lipoprotein particles, which are small in size, may be important in atherogenesis but they cannot be identified in a fasting lipid panel. The propensity to atherogenesis cannot be interpreted readily from a fasting lipid panel, which therefore should be interpreted in conjunction with a family history for coronary risk factors. Hypertriglyceridemia may be indicative of increased visceral fat, familial combined hyperlipidemia, familial dyslipidemic hypertension, impaired glucose tolerance, or diabetes. Almost half of adult females with polycystic ovary syndrome are obese and many have a central distribution of body fat. This condition frequently has its origins in adolescence. It is associated with increased androgen secretion, hirsutism, menstrual abnormalities, and infertility, although these may not be present in every case. Adults with polycystic ovary syndrome adults are hyperlipidemic, have a high incidence of impaired glucose tolerance and noninsulin-dependent diabetes, and are at increased risk for coronary artery disease. Weight reduction and lipid lowering therefore are an important part of therapy. Obstructive sleep apnea with daytime somnolence is a common problem in obese adults. Pediatric studies suggest that obstructive sleep apnea occurs in ~17% of obese children and adolescents. Sleep disorders in the obese may be a major cause of learning disability and school failure, although this remains to be confirmed. Symptoms suggestive of a sleep disorder include snoring, restlessness at night with difficulty breathing, arousals and sweating, nocturnal enuresis, and daytime somnolence. Questions to exclude obstructive sleep apnea should be part of the history of all obese children, particularly for the morbidly obese. For many children and adolescents with mild obesity, and particularly for females, one can speculate that obesity may not be a great health risk. However, there are many individuals for whom obesity will contribute to morbidity and mortality, and in this instance, the family history often provides valuable clues. These patients in particular should be targeted for weight reduction. Only with a considerably increased research effort will we be able to provide answers as to how to prevent and treat the present-day explosion of obesity. Pediatrics 1998;102(1). URL: http://www.pediatrics.org/cgi/content/full/102/1/e4; pediatric obesity, visceral fat, noninsulin-dependent diabetes, polycystic ovary syndrome, obstructive sleep apnea.

ABBREVIATIONS. NIDDM, noninsulin-dependent diabetes mellitus; VLDL, very low-density lipoprotein; HDL, high-density lipoprotein; IDL, intermediate-density lipoprotein; PCOS, polycystic ovary syndrome.

There has been a significant increase in the body weight of children in the United States, particularly since the early 1980s, with a corresponding increase in the prevalence of obesity. Between 1973 and 1994, the body weight of 5- to 14-year-old children in the Bogalusa Heart Study increased by 3.4 kg, and the weight of 15- to 17-year-olds by 5.7 kg. The prevalence of overweight, based on the 85th percentile for body mass index (body weight in kg/height in square meters) using the 1973 data, doubled during this time. The National Health and Nutrition Examination Surveys also found a doubling of overweight between 1963 and 1991 based on the 95th percentile for body mass index. The problem is not confined to the United States, and an increase in
pediatric obesity also is being noted in other developed countries.3-5

As a result of this upward shift in the weight of the pediatric population, a significant increase in sequelae is being seen. Evidence, albeit limited, suggests that long-term complications also can be anticipated. In a 32-year prospective study of Dutch adolescents, 2% of whom were overweight (defined as a body mass index >25 kg/m²), a significant increase in mortality was seen in the adolescents who were overweight.9 Overweight children in Sweden demonstrated excess mortality in comparison with a nonobese Swedish reference population after 40 years’ follow-up, with cardiovascular disease being the most common cause of death.7 Excess mortality and morbidity from adolescent overweight also was demonstrated in a 55-year study of Caucasian adolescents enrolled in the Harvard Growth Study, after adjusting for body mass index at age 50.8 The risk of morbidity from coronary disease and atherosclerosis was increased among men and women who had been overweight adolescents. In addition, the risk of colon cancer was increased among the males, and the risk of arthritis was increased among the females.

In general, pediatric health care professionals have been slow to face the implications of this explosion of obesity. For pediatricians, in particular, a major paradigm shift is needed. Coronary artery disease, stroke, noninsulin-dependent diabetes mellitus (NIDDM), and degenerative arthritis are not the language of pediatricians. However, if preventive pediatrics is to retain its meaning, then identifying, counseling, and treating children potentially at risk from obesity has to be a pediatric priority.

It should be stated at the outset that pediatric health care providers have not been given an easy task. The etiology of the major complications of obesity, such as atherosclerosis and diabetes, is far from being understood. The interaction between physical maturation and the development of these diseases also is not well described. Therefore, our ability to identify those children and adolescents most at risk from obesity is limited.

In this review article, I provide an outline of the physiologic processes underlying the major complications of obesity, with particular emphasis on the importance of visceral fat. I also attempt to bridge the gap between the adult and pediatric data, so as to provide the pediatric practitioner with a framework for dealing with the sequelae and complications of childhood and adolescent obesity.

GROWTH AND PUBERTY IN OBESE CHILDREN

Obesity, particularly that developing early in infancy, leads to accelerated growth.9 Obese prepubertal children are above average in size and have advanced bone ages.9,10 Despite accelerated growth, however, growth hormone concentrations are low in obese children because of reduced growth hormone pulsatile release and increased growth hormone clearance.11 Levels of IgF-1, the main circulating growth factor, also are not increased.9 Obese prepubertal children, however, do show increased free IgF-1 and reduced IgF binding protein concentrations, and it seems likely that the accelerated growth of obese children results from the increased bioavailability of IgF-1.12 Low growth hormone concentrations can probably be explained by a suppressive effect of free IgF-1 at the level of the pituitary–hypothalamus.

Obesity in females leads to an early onset of puberty. This is associated with a normal tempo of puberty and, hence, early menarche.13 Examining >17 000 girls from across the country, Herman-Giddens and colleagues14 have shown recently that the onset of female puberty in the United States is occurring earlier than anticipated from currently used norms. The mean age for breast development in African-Americans was 8.9 years and in whites was 10.0 years. Currently, the average age of breast development is considered to be 10.9 years.15 This study also confirmed that girls are heavier and taller than in the first and second National Health and Nutrition Examination Surveys.16 It seems likely that these trends are related either directly or indirectly to the increased weight of American children.

In contrast to females, the onset of puberty in obese males shows considerable variation, with obesity leading to both early maturation and pubertal delay.13,17 Severe obesity also may suppress the tempo of pubertal maturation.13,18 Thus, there is considerable variability in pubertal progression in obese males.14,19 Gynecomastia is a common finding in obese pubertal males, and the breasts often are greater in size and more persistent than in the usual pubertal gynecomastia.20

The recently discovered hormone leptin may provide a link between body weight and the onset of puberty and menarche. Leptin is an adipocyte-produced hormone that provides a signal to the brain regarding the amount of body fat stores.21 Blood leptin concentrations are correlated strongly with body weight. An approximate 50% rise in leptin has been noted before the onset of puberty in normal weight males.22 Serum leptin increases in early puberty, although levels subsequent decline in males.23 In females, serum leptin remains constant during midpuberty, but rises in late puberty.22 There also is an inverse relationship between serum leptin and the age of menarche up to a critical level of leptin.24

Those who care for children and adolescents should be aware of the physical changes that accompany obesity so that appropriate reassurance can be given to families and unnecessary endocrine referrals for accelerated growth avoided. There are a number of causes for abnormal growth acceleration in childhood, but obesity is by far the most common.25 Significant changes have occurred in the timing of physical maturation of US girls, particularly for African-Americans. The age of normality for female pubertal development appears to have advanced by just <1 year.26 These data are not yet reflected in textbooks and growth charts.

THE IMPORTANCE OF BODY FAT DISTRIBUTION

The metabolic complications of adult obesity are linked strongly to the distribution of body fat, which in turn is heavily influenced by gender. Adipose tissue accumulates in two main sites, intraabdominal
and subcutaneous. Intraabdominal fat comprises visceral fat surrounding the omentum and mesentery, together with a smaller amount of retroperitoneal fat, whereas subcutaneous fat is distributed over the entire body. In males, fat typically accumulates in the upper segment of the body, both subcutaneously and intraabdominally. This is apparent visually as a bulging abdomen in an apple-shaped distribution. In females, adipose tissue accumulates subcutaneously, particularly over the thighs in a pear-shaped gluteal distribution. However, an upper body pattern with visceral fat deposition can occur in females, particularly after the menopause. Gender-related patterns of body fat deposition become established during puberty and, as with total body fat, show significant familial associations.

In adults, the waist–hip ratio (ratio of the waist circumference to that of the hip) has been the most extensively used indirect measure of visceral fat. In actuality, waist circumference and abdominal sagittal diameter show a better correlation with visceral fat as determined by computed tomography. It may well be, however, that the waist–hip ratio provides a measure of the relative distribution of adipose tissue between visceral and subcutaneous fat. In children, there is no correlation between the waist–hip ratio and visceral fat. Furthermore, conventional anthropometric measures such as skinfold thicknesses and limb circumferences are insufficiently sensitive for assessing intraabdominal fat on a clinical basis. Only radiologic methods, such as magnetic resonance imaging or computed tomography scanning of the abdomen, provide an accurate measure of pediatric visceral fat.

In adults, excess visceral fat, but not subcutaneous fat, predicts lipoprotein abnormalities, diabetes mellitus, hypertension, and cardiovascular disease. The adverse effects of visceral fat are thought to be related to an increased flux of free fatty acids directly from the portal system into the liver. The increased flux of free fatty acids increases the hepatic production of very low-density lipoprotein (VLDL) particles. It also decreases hepatic insulin sensitivity and increase hepatic glucose output. This may lead to impaired glucose tolerance and diabetes. Visceral fat is the most metabolically active of the fat depots. It also responds the most rapidly to changes in diet and physical activity.

A large prospective study of adults from Gothenburg, Sweden, demonstrated that the higher the waist–hip ratio, the greater the mortality and the higher the incidence of stroke and ischemic heart disease, which was not the case for increased body mass index. This was true for both males and females. Also observed was that lean adults with an increased waist–hip ratio were at greater health risk than obese individuals with a similar waist–hip ratio. This could imply that subcutaneous fat is in some way protective against the adverse effects of visceral obesity. Others have concluded that subcutaneous thigh fat has a protective influence on lipoprotein levels, which lends support to this concept.

The deposition of visceral fat is very age-dependent; in one study, visceral fat was shown to increase in men from 12.4% of body surface at age younger than 40 years to 18% after age 65. This increase was independent of obesity. By contrast, the figure was 5.4% for adolescents. Adiposity for male and female children is predominantly subcutaneous. As in adults, there is a range of visceral fat in lean and obese children, with the range being greatest in the obese. In adolescent girls, for example, the range of intraabdominal fat is two to three times greater in the obese than in the nonobese.

A wealth of evidence points to the importance of visceral fat as a determinant of adult health. Excess subcutaneous fat, on the other hand, may not constitute a great health risk, and may even provide some protection against the adverse effects of visceral fat. Hence, a significant proportion of mild adult obesity may well be relatively benign, and this may be particularly true for premenopausal females.

To what extent can the abundance of information on adult visceral fat be used in the assessment of the obese pediatric patient? Unfortunately, there is a paucity of research data on pediatric visceral fat. Research in this field has been hindered considerably by the absence of methods other than radiologic for assessing pediatric visceral fat. In addition, although there is a strong relationship between adolescent and adult obesity (although the relationship between childhood and adult obesity is only moderate), the degree to which visceral fat tracks from childhood to adulthood is unknown.

Given this situation, the family history can be extremely useful. There is a strong hereditary component to visceral fat deposition, as well as to NIDDM, hypertension, and ischemic heart disease. These often can be traced through the family tree in obese family members.

ABNORMALITIES OF INSULIN AND GLUCOSE METABOLISM IN OBESITY

Resistance to the action of insulin and compensatory hyperinsulinemia are the hallmarks of obesity, and individuals with upper body obesity show the greatest degree of insulin resistance and hyperinsulinemia. There also is a well-documented relationship between glucose tolerance, visceral fat, and the ratio of visceral fat to peripheral fat for both lean and obese adults. This relationship is independent of total body fat. In addition, increased visceral fat is a feature of NIDDM. Such data implicate visceral fat as an important contributory factor to NIDDM. Nevertheless, NIDDM is a heterogenous condition, and current body weight also may be of importance. Impaired glucose tolerance can be seen in obese children and adolescents, and is noted particularly in obese children with a family history of diabetes. As in adults, obese children manifest insulin resistance and postprandial hyperinsulinemia. In adolescent females, insulin resistance correlates strongly with the amount of visceral fat, but there is no correlation with subcutaneous fat.

A matter of concern is that NIDDM is becoming a common diagnosis in pediatric diabetes clinics throughout the United States. Between 1982 and 1995, a 10-fold increase in the number of children and adolescents diagnosed with NIDDM has been noted at the Children’s Hospital Medical Center in
Cincinnati, OH.62 NIDDM now accounts for 33% of diabetics between 10 and 19 years of age newly diagnosed at this institution. It is suggested that this represents a true increase in the incidence of NIDDM in the catchment population rather than an increased awareness of the diagnosis. The following details regarding their patients with NIDDM are noted: the female to male ratio is 1.7:1; there are twice as many African-Americans as whites; 38% are morbidly obese (body mass index >40 kg/m²) with only a small percentage being of normal weight; and 65% have a first-degree relative with NIDDM. Arkansas Children’s Hospital also reports that the incidence of NIDDM has been increasing on a yearly basis, from 1 to 3 patients per year in years 1990 and 1991 to 17 patients per year in 1995.63 The mean body mass index of their patients with NIDDM was 35 ± 1.1 kg/m² compared with 20 ± 0.8 kg/m² for their type-1 insulin-dependent diabetics. Thirty-two percent were hypertensive at presentation. An increased incidence of pediatric NIDDM also has been observed in other American cities64 and in Japan.65 Contrary to conventional wisdom, >25% percent of young, obese African-Americans with NIDDM show ketosis.63,65 Ketoacidosis also may occur.66 A similar phenomenon has been described in African-American adults.67

NIDDM in pediatrics is a serious condition. Many families, particularly those from the inner city, lack the education and internal resources to deal adequately with their child’s obesity and diabetes. Not all pediatric diabetes clinics are set up adequately to provide intense nutritional and lifestyle counseling. Yet without adequate treatment of their obesity and diabetes, many of these patients are at risk for early atherosclerosis, renal complications, eye disease, and death.

An argument can be made that even moderate degrees of impairment of glucose tolerance require attention. Haffner and coworkers68 describe an atherogenic profile of increased levels of triglyceride and decreased levels of high-density lipoprotein (HDL)–cholesterol in confirmed pre-diabetic adults, many of whom were normoglycemic at the time of initial testing. Prediabetes, mild glucose intolerance, and diet-controlled diabetes are common findings in adult coronary disease patient,69 and many coronary patients do not develop frank diabetes until years after their initial coronary event. Approximately half of adults diagnosed with NIDDM have complications by the time of diagnosis.70 A glucose tolerance test would not constitute excessive testing for an obese adolescent if there is a close family history of NIDDM, even in the presence of fasting normoglycemia.

LIPID ABNORMALITIES IN THE OBESE CHILD

Hypertriglyceridemia and low levels of HDL–cholesterol are the primary lipid abnormalities accompanying adult obesity. These abnormalities are related primarily to the amount of visceral fat.71,72 The relationship between obesity and HDL–cholesterol is stronger for men than it is for women, and this probably is related to the fact that males have greater amounts of visceral fat.72 Obesity induces an increase in the hepatic production of triglyceride-bearing VLDL particles. Hypertriglyceridemia results when there is an imbalance between the production of VLDL triglyceride and its clearance.

Hypertriglyceridemia with low HDL–cholesterol is as much a predictor of ischemic heart disease as a high LDL–cholesterol level, and this combination should be regarded as potentially atherogenic.73 It is generally agreed that hypertriglyceridemia can be an atherogenic risk factor, although there is considerable controversy as to the relative importance of hypertriglyceridemia versus hypercholesterolemia in coronary disease and the mechanisms whereby hypertriglyceridemia may exert its atherogenic effect.74–76 In the presence of hypertriglyceridemia, a sequence of lipoprotein changes occur. These include a decrease in the amount of lipid relative to protein and a decrease in lipoprotein particle size. Lipoproteins that become lipid-depleted are heavier and smaller in size than lipid-laden particles.

The nature of the obesity determines the type of VLDL particle secreted by the liver. Increased subcutaneous fat is associated with large, lipid-laden VLDL particles, whereas smaller particles accompany central obesity.77 Small VLDL particles are richer in cholesterol. They also differ in their metabolic fate. After delipidation, smaller particles are more likely to be converted to LDL through the intermediary of intermediate-density lipoproteins (IDL), whereas large particles are removed directly from the circulation.78 Despite the increased flux of lipoproteins into LDL, LDL–cholesterol concentrations usually remain within the normal range because of a concomitant increase in LDL clearance.79

There is a significant body of evidence that small VLDL particles and IDL are important in atherogenesis.80,81 In relation to small VLDL and LDL particles, IDL particles are intermediate in size, density, and composition of lipid relative to protein. In case-control studies, IDL and small VLDL frequently emerge as major independent contributors to coronary disease,82–84 whereas LDL–cholesterol is not associated with the progression of coronary disease in most serial coronary angiographic studies.76 It has been suggested that potentially atherogenic IDL remnants may accumulate in excess particularly in the post-prandial state.85,86

As in adults, childhood obesity leads to increased concentrations of fasting triglyceride and decreased HDL–cholesterol, and these abnormalities are most marked in children with a central distribution of body fat.88,90 Nevertheless, it is debatable that these children are in a markedly atherogenic state. The appearance of atherogenic particles may well be age-dependent. Fasting IDLs, for example, are lower in normal children than in normal adults.91 Postprandial VLDL and chylomicron clearance also are more accelerated in the young.92 Factors such as these could influence the propensity to atherogenesis.

The usually obtained fasting lipid panel is a single cut in time which reflects poorly the heterogeneity of lipoprotein particles. It provides no information, for example, as to small VLDL and IDL concentrations, lipoprotein particle size, and lipoprotein flux. Not surprisingly, therefore, the information that can be derived from it is limited. This
is particularly so for the obese in whom multiple abnormalities may be present.

Current thinking on lipids is very much LDL-oriented. However, if attention is focused exclusively on LDL–cholesterol (which, in any case, is an imprecise calculated value), nuances will be missed. Frequently, the significance of hypertriglyceridemia cannot be determined. Nevertheless, hypertriglyceridemia may be indicative of visceral obesity, familial combined hyperlipidemia, familial dyslipidemic hypertension, impaired glucose tolerance, and diabetes. These are all potentially atherogenic conditions, which are inherited in a dominant manner, and which show varying degrees of expression in childhood. Bao and associates have shown recently that offspring of parents with early coronary disease are overweight, and that this begins in childhood. Seeking a family history of early coronary disease and dyslipidemia is essential for obese children, and this should be complemented by the fasting lipid panel.

POLYCYSTIC OVARY SYNDROME (PCOS)

PCOS is a common condition that affects ~6% of women of reproductive age and that frequently has its origin in adolescence. Premature adrenarche in childhood can be an even earlier manifestation of this syndrome. Typical features include hirsutism, amenorrhea or oligomenorrhea, and infertility, although these may not be present in every case. Between 38% and 50% of adult patients with PCOS are obese. Many have an increased waist–hip ratio, suggesting that central obesity may be part of this syndrome.

Multiple ovarian cysts may be noted on ovarian ultrasonography, although this is not an essential finding for the diagnosis. Characteristic hormonal features include increased ovarian androgen secretion, a predominance of luteinizing hormone to follicle-stimulating hormone secretion, and a variable increase in adrenal androgen concentrations. Ovarian testosterone secretion is increased, although total testosterone concentrations may be normal because of a decrease in the binding-protein sex hormone binding globulin. However, free testosterone concentrations are invariably increased.

The etiology of PCOS is unclear, although it is probably related in some manner to hyperinsulinemia. Hyperinsulinemia may increase ovarian androgens by an effect on the ovaries or through increased gonadotropin secretion. Both insulin resistance and free testosterone concentrations are decreased by weight reduction. A genetic influence is suggested by the findings of hyperinsulinemia, polycystic ovaries, and premature male baldness in close family members.

Of note is that in addition to these hormonal abnormalities, women with PCOS are hyperlipidemic and at increased risk for coronary artery disease, NIDDM, and hypertension. Adults with PCOS have increased triglyceride, LDL–cholesterol, and VLDL–cholesterol concentrations, and decreased HDL–cholesterol concentrations. Adult PCOS patients followed prospectively for 6 to 12 years showed a significantly increased risk for myocardial infarction. Premenopausal PCOS adults demonstrate increased intima–media thickness on carotid artery ultrasonography, suggesting the presence of subclinical atherosclerosis. Forty-two percent of women ≤60 years undergoing coronary angiography for chest pain or valvular disease have PCOS.

Obese and nonobese patients with PCOS have increased area under the glucose curve after an oral glucose challenge, and between 20% and 40% of obese adult women with PCOS have impaired glucose tolerance or frank NIDDM. Women with PCOS also have higher ambulatory blood pressures. There are no comparable data for adolescents, but there is no reason to doubt that younger patients express these abnormalities in varying degrees.

Adolescents with PCOS usually present with hirsutism, with or without menstrual dysfunction. These patients also are at risk for developing coronary artery disease and NIDDM as adults. Weight reduction and lipid lowering is as much part of therapy as hirsutism and infertility treatment. This can be an unexpected and hence difficult message to convey.

OBSERVANT SLEEP APNEA

Obstructive sleep apnea with daytime somnolence is a common problem in adults, affecting 2% to 4% of middle-age men and 1% to 2% of middle-age women. During normal inspiration, negative upper airway pressure is induced in the pharynx, and this is counteracted by muscles of the upper airway. During sleep, the muscles relax, and this can lead to airway turbulence and snoring. Excess adipose tissue adjacent to the pharyngeal airway puts the obese at risk for complete airway occlusion. Most patients with obstructive apnea have a central distribution of body fat, even in the absence of obesity. Affected individuals experience disturbed sleep because of nighttime apnic spells, and this can result in impaired daytime functioning, and industrial and traffic accidents.

How common is obstructive sleep apnea in obese children and adolescents? Marcus and colleagues performed sleep studies in 22 moderately obese predominantly inner city, African-American children and adolescents, none of whom had sleep-related symptoms, and found that 36% of the subjects had abnormal polysomnography findings. Seventeen percent of the total group had moderate to severe obstructive sleep apnea, and 9% had mild abnormalities. There was a moderate correlation between the degree of obesity and the number of apnic spells and SaO₂ nadir (r = 0.47 and r = −0.60, respectively). Between 37% and 59% of obese children and adolescents with symptoms suggestive of obstructive sleep apnea have abnormal sleep studies. Sleep studies also were performed in 10 overweight infants, who showed a small but significant increase in brief apneic spells compared with control subjects. The significance of this finding is unclear, but suggests that some childhood obstructive sleep apnea may be very longstanding.

The most common symptom of obstructive sleep apnea is snoring. However, this is not a discriminating symptom for either adults or children. Other
features include nighttime restlessness, difficulty breathing, arousals and sweating, and daytime somnolence.\textsuperscript{123} Nocturnal enuresis can be a valuable diagnostic pointer.\textsuperscript{123,126} Silvestri and coworkers\textsuperscript{122} demonstrated that sleep apnea in obese children can be predicted by the combination of severe obesity, adrenonsillar hypertrophy, and day and nighttime symptomatology. Carroll and colleagues,\textsuperscript{127} on the other hand, discussing predominantly nonobese children, argue that adult-type symptoms are unreliable in children, who may have undisturbed sleep and absence of daytime somnolence despite severe oxygen desaturation. The Pickwickian obesity-hypventilation syndrome is a rare but extremely dangerous complication of morbid obesity associated with day time somnolence and cor pulmonale.\textsuperscript{128} It has a high morbidity.

A study by Rhodes and associates\textsuperscript{129} on 14 morbidly obese children, 5 of whom had obstructive sleep apnea, suggests that obstructive sleep apnea can have a profound effect on learning potential. Affected children showed neurocognitive abnormalities with deficits in learning, memory, and vocabulary. Memory and learning performance were correlated inversely with the number of apneic/hypopneic episodes.

Studies suggest that obstructive sleep apnea occurs in $\sim$17% of obese children and adolescents, and that many of these children are academically compromised as a result. As noted by the authors, however, the subjects in the prevalence study of Marcus et al\textsuperscript{123} and the psychological study of Rhodes and associates\textsuperscript{129} were not truly representative of the obese population in terms of race or socioeconomic status. Nevertheless, if the figures are true by only a fraction, this still would rank obstructive sleep apnea as a major cause of school failure and learning disability in this country. Clearly, a large sleep study in a well-randomized population of obese children and adolescents is needed to confirm these preliminary findings.

Questions to exclude obstructive sleep apnea should be part of the history of all obese children, particularly for the morbidly obese. The devastating impact that a sleep disorder can have on an obese child needs to be emphasized. Nocturnal enuresis is a common symptom in children with obstructive sleep apnea. This, together with morbidity obesity and poor academic performance cannot but lead to a low self-image. If in addition there is teasing, unsatisfactory peer relationships, restricted mobility, and limited drive, there may be few solaces in life other than eating. Placing such a child on an unsuccessful diet may decrease his or her sense of self-worth further.

CONCLUSIONS

The increase in pediatric obesity in the United States has profound implications for the present and future health of our children. For many children and adolescents with mild obesity—and this includes many females—one can speculate that obesity may not be a great health risk. However, there are many individuals for whom obesity will contribute to morbidity and mortality, and the family history often will provide valuable clues. Much of this increased risk may be linked to excess visceral fat. It is these individuals in particular to whom our treatment efforts need be directed. There is, however, a host of questions that need to be answered before we can focus our attention on these individuals with confidence. What degree of tracking does visceral fat show from childhood to adulthood? How prognostic is the accumulation of childhood and adolescent visceral fat with regard to adulthood diabetes, coronary disease, stroke, and death? And what is the role of visceral fat in the etiology of pediatric NIDDM and PCOS?

As a nation, we are failing to give the present explosion of obesity the seriousness it merits. Only with a considerably increased research effort can we hope to provide the answers required to prevent and treat this ubiquitous problem.

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CHILDHOOD OBESITY AND THE PEDIATRIC PRACTITIONER


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Childhood Obesity, Adipose Tissue Distribution, and the Pediatric Practitioner
Arnold H. Slyper
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