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The Epidemic of Pediatric Obesity: Clinical Implications for the ED Physician

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Obesity is becoming an increasingly prevalent problem in the United States and other countries; even children and adolescents are not excluded from this epidemic. The proportion of children and adolescents who are overweight—defined as a body mass index (BMI) exceeding the 95th percentile for age- and sex-based norms from the 1960s—has tripled in the past three decades.[1,2] Obesity is associated with many potentially serious health problems that the emergency department (ED) physician must recognize and include in the differential diagnosis when a pediatric patient with this premorbid condition seeks health care. Diabetes, cardiovascular and pulmonary disease all have an increased incidence in this population and may require acute therapy in the ED. The author provides a comprehensive review of obesity and its associated complications.

— The Editor

Introduction

BMI is the simplest and most common assessment tool for categorizing childhood obesity and is calculated by dividing weight (kg) by height (m^2). BMI charts for boys and girls can be found at *www.cdc.gov/growthcharts*. BMI accurately reflects the proportion of excess body fat and correlates with markers of secondary complications of obesity and long-term mortality.[3] An expert panel on obesity evaluation and treatment recommends that a BMI greater than the 95th percentile for age and sex should be used as a cutoff for medical evaluation of obesity.[3] In older adolescents, a BMI in the 95th percentile is associated with elevated blood pressure and lipid profiles, increasing risks of obesity-related disease and mortality.[3] A patient with a BMI above the 85th percentile or a large increase in BMI should be observed closely for secondary complications. To maintain consistency with the existing literature, this review will use the term "obesity" to describe a BMI greater than or equal to the 95th percentile and "overweight" for BMI greater than or equal to the 85th percentile.

Being overweight is now the most common medical condition of childhood.[3,4] Nearly one of every three children is at risk of being overweight (defined as BMI between the 85th and 95th percentiles for age and sex), and one of every six is obese (defined as BMI at or above the 95th percentile).[5]

Obesity is associated with significant health problems in the pediatric age group and is an important early risk factor for much of adult morbidity and mortality.[5,6] Medical problems are common in obese children and adolescents and can affect cardiovascular health (e.g., hypercholesterolemia, dyslipidemia, hypertension),[5, 7-11] the endocrine system (e.g., hyperinsulinism, insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus, menstrual irregularity),[12-15] and mental health (e.g., depression, low self-esteem).[16-20] The psychologic stress of social stigmatization imposed on obese children may be just as damaging as the medical morbidities. The negative images of obesity are so strong that growth failure and pubertal delay have been reported in children practicing self-imposed caloric restriction because of fears of becoming obese.[5]

Other important complications and associations include pulmonary (e.g., asthma, obstructive sleep apnea syndrome, pickwickian syndrome),[21-25] orthopedic (e.g., genu varum, slipped capital femoral epiphysis),[5] and gastrointestinal/hepatic (e.g., nonalcoholic steatohepatitis)[26] complications. All these disturbances are seen at an increased rate in obese individuals and have become more common in the pediatric population.

The probability of childhood obesity persisting into adulthood is estimated to increase from approximately 20% at 4 years of age to approximately 80% by adolescence.[27] In addition, it is probable that co-morbidities will persist into adulthood.[5] Thus, the potential future health care costs associated with pediatric obesity and its co-morbidities are staggering, prompting the surgeon general to predict that preventable morbidity and mortality associated with obesity may exceed those associated with cigarette smoking.[5] Current data suggest that life expectancy for obese young adults may be reduced by as much as 20 years.[28]

Annually, obesity-related diseases in adults and children account for more than 400,000 deaths and more than $100 billion per year in treatment costs.[3, 29] Pediatric obesity-related hospital costs have increased three-fold during the last 20 years, reaching $127 million per year.[30] The median reimbursement rate for pediatric obesity treatment is estimated to be 11%. This rate varies greatly among different types of insurance policies, although there are no significant variations based upon a child's sex, race, or degree of obesity.[30]

Obesity occurs when susceptible (i.e., genetically pre-disposed) individuals are placed in adverse environments.[31,32] Dietz has suggested that there are three critical periods for the development of obesity. These include: 1) the intrauterine environment or early infancy; 2) 5 to 7 years of age (adiposity rebound); and 3) adolescence.[33] Weight and adiposity are entrained during early life. Furthermore, often the obesity observed in adulthood originates during the early childhood years.[32] Gestational events and the adiposity rebound period may influence childhood-onset obesity leading to adult obesity. Studies have observed that famine during gestation can either increase or decrease later obesity prevalence, and infants with higher neonatal amniotic fluid concentrations of insulin show significantly increased obesity at age 6 years.[30] Infants whose mothers had gestational diabetes have a 50% greater risk of obesity at age 10 years.[30] Although some studies indicate that breast-feeding protects against obesity, others indicate either no influence or an increased risk of becoming obese. A strong correlation between low socioeconomic status during early life and adult obesity exists.[30]

Many studies on childhood obesity etiologies have focused upon adiposity rebound, a normal childhood developmental event. Rebound is the increase of BMI that occurs after the normal decrease in adiposity between infancy and approximately age 5 to 7 years. Some studies identify early adiposity rebound in childhood as an obesity predictor.[30] However, no clear consensus exists concerning how early adiposity rebound influences obesity.[30]

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Table 1. Health Implications of Childhood Obesity

• Cardiovascular	Hypertension, obesity cardiomyopathy, cardiovascular disease
• Metabolic	Insulin resistance, dyslipidemia
• Endocrine	Type 2 diabetes, polycystic ovary syndrome, advanced physical maturation
• Renal	Microalbuminuria, glomerulosclerosis
• Pulmonary	Sleep apnea, obesity hypoventilation syndrome, asthma
• Neurological	Pseudotumor cerebri
• Gastrointestinal	Cholelithiasis, hepatic disease
• Orthopedic	Slipped capital femoral epiphysis, genu varum and tibia vara
• Psychosocial	Depression, low self-esteem

Researchers cannot predict reliably which obese children will become obese adults. Intervention studies are needed in high-risk early childhood populations, and pinpointing responsible factors should be a national priority.

There are several environmental factors that contribute to obesity: Low physical activity; high-calorie, high-fat foods; and a lower exercise tolerance are some of the many factors involved in the development of obesity.[32] However, in children younger than 6 years, the most important of these factors is parental obesity.[31] It is well accepted that the environment of the family plays a key role in the development of obesity in young children placing them at risk for adult obesity and related diseases such as diabetes mellitus.[34-37] Research shows that parental inactivity strongly predicts inactivity in the child.[32] Moreover, the exercise patterns of parents have a strong influence on the frequency of exercise in their children. Research also shows that parental influences are early determinants of food attitudes and practices in young children.[32]

Food preferences greatly influence the consumption patterns of young children. Therefore, strategies that positively alter the behaviors and environment of the family may reduce the risk of adult obesity and diabetes mellitus by improving physical activity and nutrition. Efforts to halt and reverse obesity and related metabolic disease, therefore, should begin with young children. More importantly, educational interventions that target the parents of children at risk for obesity should be an integral part of standard pediatric and family medical care.

Familial factors strongly affect the development of childhood obesity. Children with two obese parents have an 80% chance of developing obesity during their lifetime.[32, 38] If only one parent is obese, this risk factor declines to 40%. Remarkably, obesity will develop in only 7% of children born to lean parents.[38] However, it is unclear if these outcomes are based upon environmental issues or genetic predisposition.[39] Bouchard suggests that in most individuals, the human variation in body composition is not associated with genetic predisposition.[40]

These individuals may be affected positively by appropriate clinical and educational interventions. Dietary intake and physical activity are behavioral and, thus, modifiable aspects of the prevention and treatment of childhood and adolescent obesity.[31] It is clear, that human obesity and metabolic disease are determined by a complex matrix of familial factors including genetics, culture, diet, and activity patterns. Research during the past decade has focused upon determining environmental conditions that promote obesity and metabolic disease in genetically vulnerable individuals. Current studies seek to identify genetic markers for obesity and altered metabolism. There are strong arguments for the impact of the genetic profile, as well as the early nutritional environment on the development of obesity from birth to adulthood.[32]

Health Implications of Childhood Obesity

Pediatric obesity is a serious chronic disease that contributes to various conditions. (*See Table 1.*) Obesity and being overweight in childhood can no longer be considered a benign condition or one related only to appearance.

It is important in the obese patient to screen for co-morbidities that necessitate a more aggressive management. (*See Table 2.)* Obesity causes or exacerbates many health problems, both independently and in association with other diseases.

Type 2 Diabetes. Type 2 diabetes increasingly is being diagnosed within the pediatric population and parallels the increased prevalence of pediatric obesity.[41] Type 2 diabetes now accounts for as much as half of newly diagnosed diabetes in children age 10-21 years, depending upon the socioeconomic and ethnic composition of the population.[41] Early signs of insulin resistance such as acanthosis nigricans, a thickened hyperpigmented skin condition found on the neck, axillae or groin, warrant further screening including fasting insulin and glucose levels. (*See Tables 3 and 4.*) The constellation of insulin resistance, compensatory hyperinsulinemia to maintain glucose homeostasis, hypertension, dyslipidemia, and central obesity (abdominal or visceral obesity) has been termed syndrome X or the metabolic syndrome.

This central fat distribution results in an increased delivery of insulin, leading to a marked peripheral insulin concentration and insulin resistance. In the initial phase of the process, the pancreas can maintain a hyperinsulinemic state, but this compensatory mechanism is overwhelmed by the continued abundance of free fatty acids and results in hyperglycemia.[41] The islet cell mass in new-onset type 2 diabetes is normal. Late in the disease, beta cells may be reduced in association with amyloid deposition in islets, but beta cell dysfunction occurs well before the development of hyperglycemia, as suggested by data from the United Kingdom Prospective Diabetes Study (UKPDS).[4] Insulin sensi-

Table 2. Evaluation of the Obese Pediatric Patient

- Depression
- Snoring
- Daytime somnolence
- Exercise intolerance
- Polydipsia
- Polyuria
- Abdominal pain
- Joint pains
- Headaches
- Hirsutism
- Cold intolerance
- Constipation
- Reflux
- Menstrual irregularities
- Skin rashes/lesions
- Developmental delay

IMPORTANT FACTORS IN THE PHYSICAL EXAMINATION OF THE OBESE CHILD

- Degree of obesity and its distribution (truncal, peripheral, generalized)
- Developmental status of the patient
- Size of the tonsils in relation to the nasopharynx
- Thyroid size and consistency
- Presence of acanthosis nigricans (hyperpigmented velvety skin in area of neck or axilla)
- Presence of any right upper quadrant tenderness
- Tanner stage of genitalia and appropriateness for age
- Waist/hip ratios*
- Skin examination for hirsutism, striae, xanthomas, erythematous papules/pustules in groin area (hidradenitis suppurativa)
- General mood and affect of patient

** Complications of obesity correlate best with abdominal fat and less well with lower body fat.*

Table 3. Type 2 Diabetes Laboratory Tests that Define and Diagnose

GLUCOSE LEVELS
- Random level > 200 mg/dL
- Fasting level > 126 mg/dL

FASTING INSULIN AND C-PEPTIDE LEVELS
- Elevated—reflects insulin resistance

AUTOANTIBODY LEVELS (ABSENCE SUGGESTS TYPE 2 DIABETES)
- Islet cell
- Insulin
- Glutamic acid decarboxylase (GAD)

HEMOGLOBIN A_{1c}
- Elevated

tivity and beta cell function are inversely and proportionally related, and based upon this concept, it has become evident that subjects who are at high risk for developing type 2 diabetes have diminished beta cell function at the time when many of them still have normal glucose tolerance test results.[4] The presence of a first-degree relative with type 2 diabetes mellitus increases the relative risk of the patient three- to 10-fold. The Centers for Disease Control and Prevention warns that one in three U.S. children born in 2000 (and one in two Latino children) will become diabetic unless people start eating less and exercising more.[42]

Acute decompensation with diabetic ketoacidosis (DKA) has been recognized to occur at the time of diagnosis in as many as 25% of children with type 2 diabetes mellitus.[43] Hyperglycemic hyperosmolar state (HHS) with serious electrolyte disturbances also may occur in children with type 2 diabetes.[41] Recent reports indicate that approximately 4% of newly diagnosed pediatric, type 2 diabetes mellitus patients will have HHS, with a case fatality of 12%.[41] The criteria for HHS include plasma glucose concentration greater than 33.3 mmol/L (600mg/dL), serum carbon dioxide concentration greater than 15 mmol/L, small ketonuria, absent to low ketonemia, effective serum osmolality greater than 320 mOsm/kg, and stupor or coma.[44] Long-standing hyperglycemia and osmotic diuresis in children with type 2 diabetes can result in severe total body water, potassium, and phosphorus deficits, worsened by the advent of vomiting. Physicians need to have a high index of suspicion for type 2 diabetes and should consider this diagnosis as much an emergency as the recognition of type 1 diabetes. Timely diagnosis and prompt, appropriate treatment of their diabetes is essential.

The onset of type 2 diabetes at an early age is devastating. Long-term research based upon 51 patients diagnosed with type 2 diabetes before age 17 years , and now 18 to 33 years, shows that seven had died, three others were on renal dialysis, one became blind at the age of 26 years, and one had a toe amputation. Out of 56 pregnancies, only 35 had resulted in live births.[45]

Advanced Maturation. Childhood obesity promotes advanced physical maturation in especially severe conditions.[29] Obese children have advanced bone age, higher bone density and area, and increased sex hormone levels.[46] Increased estradiol levels have been associated with obesity in adult females.[32] Precocious puberty or premature puberty recently were associated with insulin resistance, long-term changes in body composition, exaggerated adrenal response, and polycystic ovary syndrome.[32, 47-50] Metabolic defects resulting from parental (environmental and genetic) influences and early onset obesity may promote hormonal alterations that predispose obese children to advanced physical maturation. Thus, biologic mechanisms that promote obesity during childhood also may play a role in premature physical maturation.

Polycystic Ovary Syndrome. This is a complex disorder characterized by infertility, hirsutism, obesity, and menstrual disturbances. Characteristically, the ovaries have atretic follicles and cysts, plasma luteinizing hormone (LH) levels are elevated, and follicle-stimulating hormone (FSH) levels are normal, resulting in an elevated LH/FSH ratio usually greater than 2 to 3. In more than half of women with polycystic ovary syndrome, plasma androstenedione and testosterone levels are increased. One

Table 4. Features and Presentation in Type 1 and Type 2 Diabetes

FEATURE	TYPE 1 DIABETES	TYPE 2 DIABETES
Polydipsia, polyuria, polyphagia	Yes	Yes
Exacerbation by acute illness	Yes	Yes
Diabetic ketoacidosis	Yes	Possible
Accidental diagnosis	Uncommon	Common
Age of onset	All ages, but mostly young children	Puberty
Weight	All ranges	Obese
Acanthosis nigricans	Rare	Common
Vaginal infection	Rare	Common
Hypertension	No	Common
Dyslipidemia	Rare	Common
Autoantibodies	Positive	Negative

theory suggests that this disorder originates as an exaggerated adrenarche in obese girls with insulin resistance. The combination of elevated androgens, insulin resistance, and obesity would result in an acyclic production of extraglandular estrogen. This leads to a positive feedback on LH secretion and negative feedback of FSH secretion.[4]

Weight loss may normalize ovulation in some women. If the woman is hirsute and does not wish to become pregnant, excess androgen production can be suppressed with oral contraceptives, glucocorticoids, luteinizing hormone-releasing hormone analogues, or antiandrogens (spironolactone). If the woman is not hirsute, monthly withdrawal bleeding should be induced to reduce the risk for endometrial neoplasia.[35] Metformin for the treatment of insulin resistance also has been shown to restore normal menses in the majority of patients.[51]

Cardiovascular Consequences of Obesity

Leaders in the emerging field of preventive cardiology increasingly have recognized obesity's role in adult cardiovascular disease. Correspondingly, the guidelines for adult cholesterol and the primary prevention of cardiovascular disease reflect this increased recognition.[52] The guidelines for cholesterol levels (See Table 5.) also target the metabolic syndrome—a constellation of metabolic derangements that predict both type 2 diabetes and premature coronary artery disease—as a newly recognized entity that warrants clinical intervention. According to the National Cholesterol Education Program (NCEP), persons meeting at least three of the following five criteria qualify as having the metabolic syndrome: elevated blood pressure, a low high-density lipoprotein (HDL) cholesterol level, a high triglyceride level, a high fasting glucose level, and abdominal obesity. Because of the increasing rates of adult obesity and obesity's association with insulin resistance and type 2 diabetes, the NCEP panel stated that the metabolic syndrome soon will have a greater impact on premature coronary artery disease than does tobacco.[53] According to recent estimates, the metabolic syndrome affects 22% of the U.S. adult population overall, including 7% of men and 6% of women in the 20- to 29-year-old age group.[52] The metabolic syndrome has been called several other names, including syndrome X, insulin resistance syndrome, dysmetabolic syndrome X, Reaven syndrome, and the metabolic cardiovascular syndrome.[52] Obesity, insulin resistance, dyslipidemia, and hypertension are common to all.

As the number of overweight children increases, its medical complications are becoming more recognized.[54-56] Studies suggest that a substantial percentage of overweight children and adolescents may be afflicted with the metabolic syndrome because many have one or more of the following: an elevated triglyceride level, a low HDL cholesterol level, and high blood pressure.[6,52] Many overweight children also have elevated insulin levels, indicating an increase in insulin resistence.[6]

The overweight condition has important implications for the future health of our young people, especially in terms of coronary heart disease and diabetes. The Pathobiological Determinants of Atherosclerosis in Youth research group, for example, found that overweight (measured by BMI) in young men was associated with fatty streaks, raised lesions, and low-grade stenosis of the coronary arteries.[57] Pathological studies have shown that both the presence and extent of atherosclerotic lesions at autopsy after unexpected death of children and young adults correlate positively and significantly with established risk factors, namely low-density lipoprotein cholesterol, triglycerides, systolic and diastolic blood pressure, body mass index, and presence of cigarette smoking.[58] Findings from the Bogalusa Heart Study indicate that as the number of cardiovascular risk factors increases, so does the pathological evidence for atherosclerosis in the aorta and coronary arteries beginning in early childhood.[59] Electron beam computed tomography of coronary artery calcium and increased carotid artery intima-media thickness, an ultrasound measure of carotid artery atherosclerosis, have been evaluated in 29- to 39-year-olds monitored from 4 years of age.[60] Significant risk predictors for coronary artery calcium were obesity and elevated blood pressure in childhood and increased BMI and dyslipidemia as young adults.

In addition, studies have established that child and adolescent obesity also predicts the metabolic syndrome in adults.[61-63] Results of one of the many reports from the Bogalusa Heart Study[64] show that when insulin concentrations are increased in childhood they tend to remain elevated in adulthood, and those adults with consistently elevated insulin levels also tend to have increased rates of obesity, hypertension, and dyslipidemia. In a recent study, adolescents with the metabolic syndrome had a

mean BMI just above the 95th percentile; thus, they represent a fairly common clinical problem, one likely to be encountered routinely by general pediatricians.[52] Perhaps 4% of adolescents overall and nearly 30% of overweight adolescents meet the criteria for this syndrome, suggesting that almost 1 million adolescents in the United States are affected.

Abdominal or centrally distributed fat is associated with type 2 diabetes and a poor cardiovascular profile in adults.[52, 65-67] In children, an increased waist circumference has been shown to correlate with abnormal systolic and diastolic blood pressures and elevated serum levels of total cholesterol, low-density lipoprotein, triglyceride, and insulin, as well as lower concentrations of HDL.[52, 68] The association between the clustering of cardiovascular risk factors and waist circumference is not only a reflection of the degree of obesity, but also is dependent upon the regional distribution of the excess body fat.[69] Thus, because a more central distribution of fat correlates with worse cardiovascular risk, and waist circumference has been shown to be the strongest correlate of central fat distribution in children, it seems appropriate to use waist circumference in a pediatric definition of metabolic syndrome.[52, 69] In fact, BMI is a less sensitive indicator of fatness in children and fails to account for fat distribution.[52] Perhaps for these reasons, an American Heart Association statement has recommended the inclusion of waist circumference measurements in evaluating children for insulin resistance or those who manifest features resulting from insulin resistance that constitute much of the metabolic syndrome.[52]

Visceral fat is not apparent on physical examination, but is the most metabolically active fat in the body and can undergo lipolysis quickly. It is located around the viscera, so that metabolic products drain immediately into the portal system and into the liver. The intra-abdominal adipose tissue (IAAT) comprises visceral adipose tissue and retroperitoneal adipose tissue, whereas subcutaneous fat, the visible fat on physical examination, is spread throughout the body. It is the visceral fat that has been associated with metabolic derangements in adults, such as dyslipidemia, hyperinsulinemia, and cardiac risk factors. Recent studies support the role of increased IAAT in children as a cause of insulin resistance and dyslipidemia.[70] Although waist-to-hip ratios are used as a substitute for the direct determination of visceral adipose tissue in adults, waist-to-hip ratios do not reflect IAAT in children and adolescents.[70] Furthermore, increased IAAT may cause these metabolic derangements without increased total body fat.

Adipocyte cytokines such as adipsin, leptin, interleukin-6, tumor necrosis factor-α, and resistin may be involved in the link between adiposity, insulin resistance, and increased disease risk. For example, resistin, a novel signaling molecule, is secreted by adipocytes.[71] Resistin levels are increased in both diet-induced and genetic models of mouse obesity.[72] The administration of antiresistin antibodies improves blood glucose and insulin action.[73] Resistin mRNA is detected mostly in abdominal adipose tissue depots of obese humans, perhaps explaining the relation between central obesity and insulin resistance.[71]

Table 5. Classification of Serum Cholesterol

MEASUREMENT (MG/DL)	HIGH	BORDERLINE	DESIRABLE
Total cholesterol	≥ 200	170-199	< 170
LDL* cholesterol	≥ 130	110-129	< 110
HDL† cholesterol	< 35 (low)	35-45 (borderline low)	> 45
Triglycerides (children < 10 years)	≥ 100	75-99	< 75
Triglycerides (children 10-19 years)	≥ 130	90-129	< 90

* LDL - Low density lipoprotein
†HDL - High density lipoprotein

From: National Cholesterol Education Panel. Report of the expert panel on blood cholesterol levels in children and adolescents. Bethesda, MD; National Heart, Lung and Blood Institute, National Institutes of Health, 1991.

Irrespective of the mechanism, reduction of body weight and visceral obesity, in particular, is well known to improve glucose tolerance. Weight loss also has been shown to decrease mortality among patients with diabetes and those without the disease. The extent of loss does not need to be great for health benefits to accrue. As little as 5–10% loss of initial weight appears sufficient.[56]

There are no easy answers to weight loss, of course. In the most general terms it is achievable only when energy intake is less than energy expenditure. In this equation, diet and exercise each play a role.

Moderate dietary restriction has positive effects on glucose tolerance almost immediately, even before there is a significant decrease in body weight. With continuing restriction, fat mass begins to decrease and this increases insulin sensitivity. Weight loss also improves dyslipidemia, especially hypertriglyceridemia, and lowers blood pressure. The greatest problem with weight loss is that in the long term up to 90–95% of patients will regain the lost weight.[74] Behavioral modification, physical exercise, and close follow up appear to increase the long-term success rate.

Children and adults who are obese may have elevated blood pressure, and all obese children should have blood pressure determinations obtained longitudinally; however, there are pitfalls to the measurement of blood pressure in overweight children.[70]

A common source of false measurement of hypertension is the use of an incorrectly sized cuff. Correctly sized cuffs have a bladder width of 40% of the child's arm circumference when measured at a point midway between the olecranon and the acromion.[75] The bladder should cover 80–100% of the child's

arm circumference. Blood pressure is measured 3-5 minutes after the subject is seated comfortably at rest. An adult-size cuff is necessary for heavier adolescents. If a smaller cuff is used inappropriately, the systolic and diastolic blood pressures will be elevated, whereas if a larger than necessary cuff is used, the blood pressure will not decrease sufficiently to mask hypertension. It is better to err on the side of a larger cuff. Blood pressure is compared best with charts that are adjusted for height or percentile of height for age. Recent data suggest no significant difference in blood pressure among ethnic groups, so different guidelines do not seem necessary.[76]

Blood pressure is increased in obese patients with the greatest elevation in obese children who have a predominantly abdominal adipose distribution.[77] Approximately 20–30% of obese children have elevated blood pressure; obese children have a 2.4-fold risk for elevated blood pressure compared with controls.[6] Furthermore, obese adolescents have a 8.5- to 10-fold risk for adulthood hypertension.[70] An excessive rate of weight gain during childhood is a risk factor for systolic hypertension, dyslipidemia, and insulin resistance 15 years later, which suggests that modification of the rate of weight gain may exert beneficial effects.[70]

Obesity-related hypertension is a state of high cardiac output, increased intravascular volume, increased sympathetic nervous system activity, sodium retention, and hyperinsulinemia.[70,78,79] There is an increase in left ventricular mass in obese children and adults, with the major effect of adipose tissue related to central obesity rather than total fat mass.[80] However, although an individual might have elevated blood pressure and excess adipose tissue, it seems that the elevation of serum insulin is related most closely to the change in left ventricular mass rather than simply the elevated BMI.[81]

Left ventricular hypertrophy (LVH) occurs commonly in children with hypertension and is associated with an increased BMI.[82] Individuals with LVH are at greater risk than those without LVH for complications such as myocardial infarction, cerebrovascular events, and congestive heart failure.[82]

Severe childhood obesity is associated with early endothelial dysfunction and increased stiffness of elastic arteries.[83-86] Thus, permanent, obesity-related abnormalities that increase the risk of coronary heart disease may occur in childhood. This vascular dysfunction in children may be an early step in the development of atherosclerosis.

The composition of the diet also may be related to blood pressure; in a study of urban minority youth with blood pressures of more than the 90th percentile, blood pressure was lower in those with higher intakes of a combination of nutrients, including potassium, calcium, magnesium, and vitamins.[87] These are the factors found in a diet more likely to lead to a healthy weight rather than an overweight condition, and the investigators believed that diets rich in a combination of nutrients derived from fruits, vegetables, and low-fat dairy products could contribute to primary prevention of hypertension if instituted at an early age.

Weight loss decreases blood pressure in most cases, with the most effective program combining exercise and caloric reduction and family encounters or counseling.[70] Weight reduction by individual counseling or group treatment led to an improved cardiac risk profile in 6- to 15-year-old children. There were increased HDL cholesterol levels, increased ratios of HDL cholesterol to total cholesterol, and decreased triglyceride concentrations in both groups. Fasting plasma insulin concentrations decreased significantly in some subjects.[70] A program aimed at increasing exercise tolerance and aerobic capacity rather than weight loss was successful in improving the cardiac risk profile in a group of 10 obese adolescents.[70] Thus, weight control need not be the goal as long as healthy habits are instilled.

Sleep disordered breathing (SDB) also may be a cause of hypertension in overweight children.[88] This will be discussed in the respiratory complications section.

The current, large increase in the prevalence of childhood obesity will translate into a rise in cardiovascular morbidity and mortality unless we improve our management of obesity. Weight reduction improves obesity-related metabolic risk factors in children but is very difficult to achieve.[82] Consequently, direct intervention on obesity-associated vascular abnormalities deserves consideration. Improvement in endothelial dysfunction through a direct mechanism rather than through improvements in metabolic abnormalities has been shown during dietary supplementation with antioxidant vitamins and L-arginine (the substrate for endothelium-derived nitric oxide) in children and young adults with hypercholesterolemia.[82] There is now substantial scientific evidence documenting the acquisition of behaviors associated with risk factors in childhood; these include dietary habits, physical activity behaviors, and the use of tobacco.[53,89] An increasing body of research now documents the safety and success of intervention to reduce risk factors in childhood. These studies include the Dietary Intervention Study in Children Trial, which demonstrated the safety and efficacy of a low-fat diet in children with hypercholesterolemia; skill-training programs in smoking prevention in adolescents; the Child and Adolescent Trial for Cardiovascular Health Study, which increased physical activity levels in children by using elementary school-based programs; and other successful long-term, family-based, treatment programs for childhood obesity.[53]

Given the growing concern about metabolic syndrome, coupled with the alarming increase in the prevalence of overweight children and adults, it is not surprising that the American Heart Association set forth a series of guidelines for promoting cardiovascular health as part of comprehensive pediatric care.[53] Evidence shows that obesity and insulin resistance has already started the clock of coronary heart disease in some patients even before the onset of diabetes.

Respiratory Consequences

The association between obesity and disturbed breathing during sleep in children has been recognized since the 1800s when

Charles Dickens described in *The Pickwick Papers* the messenger boy Joe, who was overweight, snored loudly during sleep, had excessive daytime sleepiness, and heart failure.[25] In 1956, Burwell et al coined the term "pickwickian syndrome" for patients with extreme obesity and alveolar hypoventilation.[90] The spectrum of SDB was expanded to include the syndrome of obstructive sleep apnea characterized by periods of repeated partial or complete airway collapse and increased airway resistance during sleep. In a study of the prevalence of SDB among middle-aged adults, Young et al demonstrated that obesity was a significant risk factor for the syndrome of obstructive sleep apnea. An increase of one standard deviation in any measure of body composition was related to a three-fold increase in the risk of obstructive sleep apnea.[91] Similarly, Redline et al demonstrated that obesity increases the risk for SDB in obese children by four- to five-fold compared with nonobese children.[92]

In an attempt to answer the question of whether obesity leads to SDB, several studies described the structural changes of the upper airway in obese and nonobese subjects with the disorder. These studies have demonstrated that obese subjects with SDB have an increase in the size of the soft-tissue structures surrounding the upper airway. One study showed that obese subjects with SDB have an increase in the size of the tongue, the soft palate, and lateral pharyngeal walls.[93] Another study showed a positive correlation between the degree of severity of SDB expressed as the apnea hypopnea index and the volume of adipose tissue adjacent to the pharyngeal airway in humans.[94] In a third study, results showed that obese subjects with SDB had large deposits of fat in the posterior and lateral aspects of the oropharyngeal airspace at the level of the soft palate.[95] In concurrence with the anatomical abnormalities identified in the above studies, Watanabe et al showed that obese subjects were more likely to have obstruction of their airway at the level of the velopharynx rather than at the level of velopharynx and oropharynx, as in subjects with craniofacial abnormalities.[96]

Although similar assessment of airway structures and dynamics in obese children with SDB remains to be investigated, there is strong evidence to suggest that the effect of obesity on upper airway structures and the propensity of the airway to collapse results in a relationship between obesity and SDB, whereby increased body fat increases the risk for obstructive breathing during sleep.

Parallel to the increase in childhood obesity, an increase in the prevalence of cardiovascular risk factors among children has been demonstrated. Nearly 60% of overweight children in the Bogalusa study were found to have at least one cardiovascular disease risk factor.[6] Although SDB has been shown to be an independent risk factor for cardiovascular morbidity and mortality in adults, the relationships between obesity in childhood, SDB, and risk factors for cardiovascular disease have not been thoroughly investigated.[25]

In an attempt to answer the question of whether SDB contributes to obesity and its cardiovascular morbidity, several studies examined the relationship between SDB and cardiovascular risk factors. Metabolic abnormalities that might affect the degree of adiposity and body fat distribution in subjects with SDB have been described. Resistance to the weight loss effect of the protein leptin has been demonstrated among subjects with SDB who were found to have higher levels of serum leptin and a larger accumulation of visceral fat compared with weight-matched controls.[97,98]

Recently, de la Eva et al investigated the link that might exist between SDB in obese children and the presence of insulin resistance.[24] In a study of 62 obese children, they demonstrated a positive correlation between the degree of hyperinsulinemia and the degree of severity of SDB measured as respiratory disturbance index (RDI). In multiple linear regression analysis, the RDI, glucose level, and measures of the degree of hypoxemia during sleep were associated with fasting insulin level. Similar to the findings observed in obese children in this study, studies in adult obese subjects with SDB found a higher degree of insulin resistance compared with weight-matched control subjects without SDB.[99] The importance of these observations stems from the knowledge that insulin resistance is thought to be the primary antecedent abnormality of the metabolic syndrome, a constellation of obesity, dyslipidemia, hypertension, insulin resistance, and hyperinsulinemia.[100] Furthermore, the possible association of SDB and type 2 diabetes mellitus was examined in the prospective Nurses Health Study, which demonstrated that the risk for diabetes increased by two-fold among regular snorers compared with nonsnorers after adjustments for all other diabetes risk factors.[101]

The current knowledge available about SDB in adults suggests a closed loop between SDB and obesity, whereby the initial development of obesity might contribute to SDB by physical factors promoting airway obstruction during sleep.[25] In turn, SDB might maintain obesity and further increase the risk for cardiovascular disease via hormonal or other humoral factors. The study of de la Eva et al is a first step toward the investigation of the relationship of SDB and obesity in childhood.[24] Results from this study point out the need for further investigations that provide deeper understanding of the web association among SDB, obesity, insulin resistance, and other risk factors for cardiovascular disease in children and adolescents.

Obese children carry a risk for: 1) restrictive airway disease caused by the difficulty in respiration from the mass of adipose tissue; and 2) obstructive airway disease caused by fatty deposition along the airway when compounded by the tonsillar and adenoidal hypertrophy that is common at a young age.[102] Obstructive sleep apnea (OSA), with carbon dioxide retention, hypoxia, and right ventricular hypertrophy and failure is a potential cause of severe morbidity or even mortality. Symptoms may vary from snoring to enuresis (this also could suggest type 2 diabetes mellitus and osmotic diuresis), daytime somnolence or irritability, hyperactivity, poor school performance, and neurocognitive deficits.[103] The most severe form is the obesity hypoventilation syndrome, or pickwickian syndrome, consisting of hypoven-

tilation, somnolence associated with carbon dioxide retention, hypoxia, polycythemia, right ventricular hypertrophy and failure, and possibly pulmonary embolism.[70]

It must be mentioned that while enlargement of tonsils and adenoids may further compromise the airway of the obese patient, treatment of OSA by tonsillectomy and/or adenoidectomy in obese children will improve the OSA, but may not help with weight reduction and may even exacerbate the obesity.[104] When OSA complicates obesity, attention must be paid to reducing weight by such measures as exercise, diet and behavior therapy, in addition to treatment of the OSA.

Finally, as previously discussed, OSA is associated with systemic hypertension.[105,106] Surges in blood pressure associated with sleep apnea occurring during sleep (especially rapid eye movement sleep) might be closely linked in the production of sustained elevation of blood pressure during wakefulness.

The increasing prevalence of asthma coincides with an increase in the number of people who are overweight, both children and adults.[107] An increased BMI has been reported to be associated with an increased prevalence of asthma.[22,23,108-110] Furthermore, high BMI appears to be a risk factor for the development of asthma rather than a consequence, as indicated by several prospective studies.[102,110] A gender-specific relationship has been shown consistently in female but not in male adults, whereas the findings are inconsistent in children.[102,110]

The association between high BMI and asthmatic symptoms could be due to a variety of factors, such as low physical activity, diet, hormonal influence, immune modification, and/or mechanical factors. Low physical activity is clearly associated with being overweight and the reduction in deep breathing associated with a sedentary lifestyle may lead to a latching state of airway smooth muscle, and in turn to airway obstruction and hyperactivity.[111, 112] Whether being overweight causes asthma symptoms through low physical activity, or whether asthma symptoms result in avoidance of exercise, which then leads to weight gain, cannot be answered in a cross-sectionally based study. However, the results of prospective studies suggest that high BMI is a risk factor for asthma.[102, 110] In addition, airway obstruction and peak expiratory flow variability in obese asthmatics were improved after moderate weight loss, indicating a causal relationship.[113]

There are several other possible explanations for the association between high BMI and asthma, for example, dietary factors. A low intake of antioxidants may be associated with asthmatic symptoms in children and a high intake of salt with bronchial hyperresponsiveness.[102] It is conceivable that there are differences in the intake of nutrients between children with high BMI and those with normal BMI. Wheezing symptoms in children with a high BMI also may be induced by a low level of systemic inflammation, which is present in overweight and obese subjects (*discussed further in the next section*).[114] Finally, asthma symptoms in children with high BMI simply may be explained by mechanical factors, (i.e., the deposition of adipose tissue in the chest wall and the airways, leading to narrow airways, and/or the presence of gastro-esophageal reflux, which is associated with both overweight and asthma).[102, 112]

Emergency medicine physicians frequently will be involved in the acute resuscitation of obese critically ill or injured patients. There are many clinical and practical problems that emergency medicine physicians encounter in the resuscitation of the obese patient—adult or child.

Airway Management. Management of the airway in an obese patient can be extremely difficult. Morbidly obese patients desaturate more quickly than nonobese patients.[106-115] Bag-valve-mask ventilation is more difficult because of reduced pulmonary compliance, increased chest wall resistance, increased airway resistance, abnormal diaphragmatic position, and increased upper airway resistance.[115] The risk of aspiration is greater in obese patients because of a larger volume of gastric fluid and increased intra-abdominal pressure with a higher incidence of gastroesophageal reflux.[115]

Like in the nonobese patient, endotracheal intubation remains the method of choice for controlling the airway. Obesity increases the risk of a difficult intubation by inhibiting the physician's view of the laryngeal structures during orotracheal intubation.

Effective preoxygenation is extremely important. Obese patients should be kept sitting upright or semirecumbent for as long as possible. If bag-valve-mask ventilation is a requisite, higher-than-normal ventilatory pressures could be needed.

In the spontaneously breathing obese patient, an assessment of the likelihood of a difficult oral intubation should be made. The Mallampati scale, an objective measure of the pharyngeal structures visible with the patient's mouth wide open, can be helpful in predicting the difficult airway. Other potential predictors include limited neck mobility, limited mouth opening, and micrognathia.

Visualization of the glottis by established techniques is difficult in obese children. The upper aerodigestive tract often is narrowed by fatty infiltration of muscles and subcutaneous fat deposits that exert an anteroposterior and lateral (i.e., externo-internal) force on regional structures.[116] These factors may combine with other anatomic variations, such as enlarged, upper airway lymphoid tissue, posterior displacement of the tongue with retrognathism, and elongated soft palate, to make airway obstruction in the obese child a significant concern. Acute respiratory tract infection or decreased muscle tone while asleep can turn a marginal airway into a critical one.

Obesity can produce several physiologic derangements that complicate anatomic obstruction and lead to poor gas exchange. First, there can be a decrease in chest wall and diaphragmatic excursion secondary to fat deposits over the chest and abdomen that leads to decreased pulmonary compliance and increased work of breathing and oxygen requirement. Decreased lung volume, increased airway resistance, and reduced total respiratory volume frequently result in decreased tidal volume and resultant alveolar hypoventilation. Hypoxia and hypercapnia may occur. There is a tendency with obesity to perfuse lower pulmonary

segments while preferentially ventilating upper segments, producing ventilation-perfusion mismatch.[116] Pulmonary hypertension and cor pulmonale are life-threatening sequelae from chronic airway obstruction that result from pulmonary vasoconstriction in response to decreased arterial oxygen saturations and pH and increased arterial carbon dioxide.

These marked pulmonary function changes seen in awake, obese patients are amplified in the sedated, paralyzed, and supine obese patient.[117] Data suggest that obese patients during sedation and paralysis are likely to be hypoxemic with wide, alveolar-arterial oxygen gradients secondary to ventilation-perfusion mismatching, and have marked alterations of the mechanical properties of the respiratory system, including reduction in lung and chest wall compliance, increased lung resistance, and increased intraabdominal pressure.

Pulse oximetry can be unreliable in morbidly obese patients secondary to increased finger tissue thickness and poorly transmitted light waves. If difficulty is encountered in using the finger for pulse oximetry, the ear lobe should be used because it is generally thinner, better perfused, and holds the oximetry probe more readily.[115]

If oral endotracheal intubation is attempted, positioning the patient is vitally important. It can be more difficult to place the morbidly obese patient in the appropriate sniffing position, which facilitates oral intubation. Because of the multitude of problems that may complicate the intubation of the obese pediatric patient, any physician faced with urgent intubation decisions in the obese patient must understand and anticipate the challenges caused by morbid obesity. Alternatives to oral intubation should be considered and available. Such alternatives include blind nasal intubation, fiberoptically aided, nasotracheal intubation, and laryngeal mask airway (LMA). A surgical approach to failed airway management in the obese patient is the final option.

Obesity often is associated with Down syndrome, and these children are at increased risk for obstruction secondary to associated problems of micrognathia, macroglossia, midfacial hypoplasia, and hypotonia. Atlantoaxial instability must always be considered when intubating or performing surgery on a child with Down syndrome. Ten percent of patients with Down syndrome have this problem, and preoperative radiologic assessment is indicated with flexion-extension neck radiography.[118]

Once intubated and on positive pressure ventilation, tidal volume should be calculated based upon ideal body weight, because use of the patient's actual weight is likely to result in excessive peak airway pressure and alveolar overdistention. Further manipulations of tidal volume, peak airway pressures and ventilator rate should be based upon arterial blood gas measurement. The addition of positive end-expiratory pressure (PEEP) may improve arterial oxygenation and respiratory compliance in obese patients who are sedated and supine.

Volume Resusitation. Fluid resuscitation must be planned carefully, especially in the hypertensive obese patient. Such patients may have abnormal, left ventricular systolic and diastolic function as discussed earlier.

Medication Administration. Medications administered to morbidly obese patients are liable to have significantly altered pharmacokinetics. These alterations are the result of changes in volume of distribution, renal clearance, hepatic metabolism, and protein binding.[115] Underlying disease greatly can affect a drug's pharmacokinetic characteristics. The actual pharmacokinetic manner in which a medication behaves in an individual obese patient is dependent upon the interaction of these variables, making the overall response unpredictable. Because of the variable nature of drug pharmacokinetics in morbidly obese patients, careful monitoring of clinical end points, signs of toxicity, clinical response, and serum drug levels are essential.[119]

Chronic Inflammatory State

The American Heart Association has declared that obesity is a major, independent risk factor for the development of cardiovascular disease. It is well known that obesity has an impact on the traditional risk factors—-hypertension, lipid abnormalities, and diabetes; however, it appears to have an impact on cardiovascular disease independent of these effects. The mechanism of this impact is not known. Ford et al have shown that increased BMI is associated with elevated levels of C-reactive protein (CRP), which is a marker of inflammation.[120] Recent research in adults has focused upon inflammation as an important mechanism in the development of atherosclerosis. The work of Ford et al suggests that obesity in childhood and adolescence may be associated with chronic inflammation. This may signal the beginning of a process leading to myocardial infarction and stroke later in life.

The mechanisms by which obesity promotes increased CRP concentration may be due to the production of cytokines by adipocytes. Tumor necrosis factor-α, which is produced by adipocytes, promotes the release of interleukin-6, which in turn stimulates acute phase reactant production by the liver.[120] This effect may be reversible, because weight loss results in decreases of tumor necrosis factor-α and CRP concentration in adults.[120] These observations suggest that body fat is central to the pathophysiology of elevated CRP concentration. The role of various fat compartments and the factors that contribute to elevated CRP concentration in some individuals but not others remains to be elucidated.

Yudkin et al have proposed that obesity may increase the risk for cardiovascular disease by inducing a state of low-grade inflammation, which subsequently may result in insulin resistance and endothelial dysfunction.[121] In this context, CRP may be a marker or mediator of this inflammatory process and may contribute to increased obesity-associated morbidity and mortality later in life.

Although the role of obesity in causing increased morbidity and mortality is well recognized, the underlying pathophysiologic mechanisms often are not well defined for many complications. The recognition that adipocytes are more metabolically

active, indicated by the production of a host of cytokines, has opened new avenues of research into the pathophysiology of obesity-associated complications. The possibility that excess weight initiates and maintains a state of chronic low-level inflammation in many people may offer new insights into the prevention and management of morbidity and mortality associated with excess weight. Furthermore, future studies of excess weight, cytokines, and acute phase reactants may yield new approaches to identifying overweight children at increased risk for obesity-associated complications.

Orthopedic Complications

Because the tensile strength of bone and cartilage did not evolve to carry substantial quantities of excess weight, a variety of orthopedic complications accompany childhood and adolescent obesity. Among young children, excess weight can lead to bowing of the tibia and femurs analogous to the bowing that occurs when downward pressure is exerted on a flexible stick. The resultant overgrowth of the medial aspect of the proximal tibial metaphysis is known as Blount disease. Although the prevalence of Blount disease is low, approximately two-thirds of these patients may be obese.[30, 55]

Slipped capital femoral epiphysis results from the effect of increased weight on the cartilaginous growth plate of the hip. Between 30% and 50% of patients with slipped capital femoral epiphysis are overweight.[55]

Excess body weight and hormonal imbalance have been associated with growth (epiphysis) plate injuries in obese pubertal youth.[122] During weight-bearing exercise, obese children carry an additional load that creates additional stress to the growth plates. There is also a strong endocrine basis for slipped capital femoral epiphysis.[32, 122] Growth hormone reduces plate strength while sex hormones promote plate closure and, therefore, improved strength. Estrogen and growth hormone must be in balance for normal growth plate development to occur. When imbalances occur, the resulting bone abnormalities reduce physical activity, increase obesity, and further enhance the risks for adult obesity and diabetes mellitus. The relationship of hormonal maturation parameters to obesity and insulin resistance in children is an area in need of further research.

Hepatic Steatosis and Cholelithiasis

High concentrations of liver enzymes represent a frequent obesity-associated finding in children and adolescents. In a large series, more than 10% of all obese children seen in a general obesity clinic setting had modest increases of liver enzymes, frequently associated with fatty liver, fatty hepatitis, fatty fibrosis, or cirrhosis.[55] Hyperinsulinemia also may play a role in the pathophysiology of steatohepatitis. Weight reduction induces a normalization of hepatic enzymes.

Cholelithiasis occurs with increased frequency among obese adults and may occur more frequently with weight reduction.[4] Increased cholesterol synthesis and cholesterol saturation of bile occurs in obesity. Although gallstones are a less frequent occurrence among obese children and adolescents, almost 50% of cases of cholecystitis in adolescents may be associated with obesity.[55] Furthermore, as in adults, cholecystitis in adolescents may be associated with weight reduction.

Psychosocial Complications

Current research indicates that the most serious and prevalent long-term consequence of childhood obesity is psychosocial.[17] Obese children may experience adverse psychological consequences of childhood obesity including lowered self-esteem and increased depression ratings.[16] Obese children are targets of early and systematic discrimination by peers, family members, and teachers. Moreover, the early physical maturation associated with childhood obesity is linked to low self-esteem.

The prevalences of depression and obesity have been shown to increase from adolescence to young adulthood. Thus, understanding how depression and obesity interact during this period may provide useful information about the mechanisms involved and potential targets of obesity.[123]

Studies also have demonstrated an association between obesity and decreased quality of life, specifically dimensions related to psychosocial health, self-esteem, physical functioning, and the impact on parental well-being, in school-age children.[20]

It is possible that programs aimed at reducing obesity may help improve health-related quality of life for some children. Additionally, overweight children may benefit from interventions that address issues related to psychosocial functioning and self-esteem. Depressed adolescents are at increased risk for the development and persistence of obesity. Understanding the shared biological and social determinants linking depressed mood and obesity may inform the prevention and treatment of both disorders.[16]

Summary

A new chronic disease has emerged over the past two decades, one that overshadows all others in frequency in the pediatric population—obesity. Changes in the Western lifestyle have led to significant reductions in the energy expenditure of children and have encouraged super-sizing of calorie-dense, high-fat foods and snacks. Several factors have contributed to the reduction of physical activity by our youth, including the downsized requirements for physical education in schools; the increasing recreational use of video and computer games, the Internet, and television; and reliance on the family automobile as the common means for parents to deliver their children to their various activities.[124] The fast-food industry and television advertisements that target children emphasize large portions of tasty, high-fat foods, which are sold with free toys and games.

A barrier to effective obesity management is the toxic environment in which advertising and the availability of unhealthy foods are pervasive and increasingly tempting for children and families.[125] A related barrier involves political and economic

forces that result in several industries benefiting from food consumption and lobbying to discourage governments from doing anything to inhibit overeating.[126] The resulting imbalance between financial resources available to combat obesity and the resources that encourage food intake is staggering.[125]

As a result of this epidemic of childhood obesity, a multitude of chronic illnesses and risk factors for adult disease now are starting in childhood rather than in adulthood. The long-term consequences are projected to be significant.[124]

Seen in its entirety, pediatric obesity becomes a disease; a disease that involves a genetic predisposition, toxic environment, and behavior.

It is now time to bring together pediatric health care providers in mounting an all-out battle against childhood obesity and to begin to approach it as the most common chronic illness in childhood. In addition to advocating healthy diets and appropriate levels of physical activity, pediatricians also should be concerned with policy and programmatic changes that give families and persons the tools and environment they need to be healthy.

References

1. Ogden CL, Flegal KM, Carroll MD, et al. Prevalence and trends in overweight among US children and adolescents, 1999-2000. *JAMA* 2002; 288:1728-1732.
2. Yanovski JA, Yanovski SZ. Treatment of pediatric and adolescent obesity. *JAMA* 2003;289:1851-1853.
3. Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert committee recommendations. *Pediatrics* 1998;102:e29.
4. Duran P, Kramer RE. Pediatric obesity: Concerns and controversies. *Pediatric Case Reviews* 2002;2:168-179.
5. American Academy of Pediatrics, Committee on Nutrition. Prevention of pediatric overweight and obesity. *Pediatrics* 2003;112: 424-430.
6. Freedman DS, Dietz WH, Srinivasan SR, et al. The relation of overweight to cardiovascular risk factors among children and adolescents: The Bogalusa Heart Study. *Pediatrics* 1999; 103:1175-1182.
7. Sorof JM, Poffenbarger T, Franco K, et al. Isolated systolic hypertension, obesity, and hyperkinetic hemodynamic states in children. *J Pediatr* 2002;140:660-666.
8. Reich A, Muller G, Gelbrick G, et al. Obesity and blood pressure–results from the examination of 2365 school children in Germany. *Int J Obes* 2003;27:1459-1464.
9. Deedwania PC. Metabolic syndrome and vascular disease: Is nature or nuture leading the new epidemic of cardiovascular disease? *Circulation* 2004;109:2-4.
10. Csabi G, Torok K, Jeges S, et al. Presence of metabolic cardiovascular syndrome in obese children. *Eur J Pediatr* 2000;159:91-94.
11. Freedman DS, Khan LK, Dietz WH, et al. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: The Bogalusa Heart Study. *Pediatrics* 2001;108:712-718.
12. Shinha R, Fisch G, Teague B, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *N Engl J Med* 2002;346:802-810.
13. American Diabetes Association. Type 2 diabetes in children and adolescents. *Pediatrics* 2000;105:671-680.
14. Nesmith JD. Type 2 diabetes mellitus in children and adolescents. *Pediatrics in Review* 2001;22:147-152.
15. Valente AM, Strong W, Sinaiko AR. Obesity and insulin resistance in young people. *Am Heart J 2001*;142:440-444.
16. Goodman E, Whitaker RL. A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics* 2002;109:497-504.
17. Schwimmer JB, Burwinkle TM, Varni JW. Health-related quality of life of severely obese children and adolescents. *JAMA* 2003; 289: 1813-1819.
18. Eisenberg ME, Neumark-Sztainer D, Story M. Associations of weight-based teasing and emotional well-being among adolescents. *Arch Pediatr Adolesc Med* 2003;157:733-738.
19. Dixon JB, Dixon ME, O'Brien PE. Depression in association with severe obesity: Changes with weight loss. *Arch Intern Med* 2003; 163:2058-2065.
20. Friedlander SL, Larkin EK, Rosen CL, et al. Decreased quality of life associated with obesity in school-aged children. *Arch Pediatr Adolesc Med* 2003;157:1206-1211.
21. American Academy of Pediatrics, Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome. Clinical practice guideline: Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2002;109:704-712.
22. Chen Y. Obesity and asthma in children. *J Pediatr* 2004;144:146-147.
23. de la Eva RC, Baur LA, Donaghue KM, et al. Metabolic correlates with obstructive sleep apnea in obese subjects. *J Pediatr* 2002; 140: 654-659.
24. Amin R. Relationship between obesity and sleep-disordered breathing in children: Is it a closed loop? *J Pediatr* 2002;140:641-643.
25. Berger KI, Ayappa I, Chatr-amontri B, et al. Obesity hypoventilation syndrome as a spectrum of respiratory disturbances during sleep. *Chest* 2001;120:1231-1238.
26. Rashid M, Roberts EA. Nonalcoholic steatohepatitis in children. *J Pediatr Gastroenterol Nutr* 2000;30:48-53.
27. Guo SS, Chumlea WC. Tracking of body mass index in children in relation to overweight in adulthood. *Am J Clin Nutr* 1999;70: 145S-148S.
28. Fontaine KR, Redden DT, Wang C, et al. Years of life lost due to obesity. *JAMA* 2003;289:187-193.
29. Mokdad AH, Marks JS, Stroup DF, et al. Actual causes of death in the United States, 2000. *JAMA* 2004;291:1238-1245.
30. Zametkin AJ, Zoon CK, Klein HW, et al. Psychiatric aspects of child and adolescent obesity: A review of the past 10 years. *J Am Acad Child Adolesc Psychiatry* 2004;43:134-150.
31. Hill J, Throwbridge F. The causes and health consequences of obesity in children and adolescents. *Pediatrics* 1998;101:497-575.
32. Sothern MS, Gordon SJ. Prevention of obesity in young children: A critical challenge for medical professionals. *Clin Pediatr* 2003; 42:101-111.
33. Dietz WH. Critical periods in childhood for the development of obe-

sity. *Am J Clin Nutr* 1994;59:955-959.
34. Strauss RS, Knight J. Influence of the home environment on the development of obesity in children. *Pediatrics* 1999;103:e85.
35. Dietz WH, Gortmaker SL. Preventing obesity in children and adolescents. *Annu Rev Public Health* 2001;22:337-353.
36. Fogelholm M, Nuutinen O, Pasanen M, et al. Parent-child relationship of physical activity patterns and obesity. *Int J Obes Relat Metab Disord* 1999; 23:1262-1268.
37. Dowda M, Ainsworth BE, Addy CL, et al. Environmental influences, physical activity, and weight status in 8- to 16-year-olds. *Arch Pediatr Adolesc Med* 2001;155:711-717.
38. Whitaker RC, Wright JA, Pepe MS, et al. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med* 1997; 337:869-873.
39. Bouchard C. Obesity in adulthood – the importance of childhood and parental obesity. *N Engl J Med* 1997;337:926-927.
40. Bouchard C. Genetic variation in exercise and obesity. In: Hills A, Wahlqvist M, eds. *Exercise and Obesity*. London: Smith-Gordon; 1994:1199-1205.
41. Morales AE, Rosenbloom AL. Death caused by hyperglycemic hyperosmolar state at the onset of type 2 diabetes. *J Pediatr* 2004; 144:270-273.
42. Narayan KMV, Boyle JP, Thompson TJ, et al. Lifetime risk for diabetes mellitus in the United States. *JAMA* 2003;290:1884-1890.
43. American Diabetes Association. Consensus statement: Type 2 diabetes in children. *Diabetes Care* 2000; 23:381-389.
44. American Diabetes Association. Position statement: Hyperglycemic crises in patients with diabetes mellitus. *Diabetes Care* 2001;24: S83-90.
45. Ehtisham S, Barrett TG. The emergence of type 2 diabetes in childhood. *Ann Clin Biochem* 2004;41:10-16.
46. De Simone M, Farello G, Palumbo M, et al. Growth charts, growth velocity and bone development in childhood obesity. *Int J Obes Relat Metab Disord* 1995;19:851-857.
47. Bideci A, Cinaz P, Hasanoglu A, et al. Serum levels of insulin-like growth factor-I and insulin-like growth factor binding protein-3 in obese children. *J Pediatr Endocrinol Metab* 1997;10:295-299.
48. Dimartino-Nardi J. Premature adrenarche: Findings in prepubertal African-American and Caribbean-Hispanic girls. *Acta Paediatr* 1999;88:67-72.
49. Hasanoglu A, Bideci A, Cinaz P, et al. Bone mineral density in childhood obesity. *J Pediatr Endocrinol Metab* 2000;13:307-311.
50. Lazar L, Kauli R, Bruchis C, et al. Early polycystic ovary-like syndrome in girls with central precocious puberty and exaggerated adrenal response. *Eur J Endocrinol* 1995;133:403-406.
51. Ibanez L, Valls C, Ferrer A, et al. Sensitization in insulin induces ovulation in nonobese adolescents with anovulatory hyperandrogenism. *J Clin Endocrinol Metab* 2001;86:3595-3598.
52. Cook S, Weitzman M, Auinger P, et al. Prevalence of a metabolic syndrome phenotype in adolescents. *Arch Pediatr Adolesc Med* 2003; 157:821-827.
53. National Cholesterol Education Program. *Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): Full Report.* Bethesda, MD: National Institutes of Health; 2002. NIH publication No. 01-3670.
54. Gahagan S. Child and adolescent obesity. *Curr Probl Pediatr Adolesc Health Care* 2004;34:6-43.
55. Dietz WH. Health consequences of obesity in youth: Childhood predictors of adult disease. *Pediatrics* 1998;101:518-525.
56. Strauss RS. Childhood obesity. *Pediatr Clin N Am* 2002;49:175-201.
57. McGill HC Jr, McMahan CA, Herderick EE, et al. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation* 2002;105:2712-2718.
58. Kavey RW, Daniels SR, Lauer RM, et al. American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood. *J Pediatr* 2003;142:368-372.
59. Li S, Chen W, Scrinivasan SR, et al. Childhood cardiovascular risks factors and carotid vascular changes in adulthood: The Bogalusa Heart Study. *JAMA* 2003;290:2271-2276.
60. Raitakari OT, Juonala M, Kahonen M, et al. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: The cardiovascular risk in young Finns study. *JAMA* 2003; 290:2277-2283.
61. Guo SS, Roche AF, Chumlea WC, et al. The predictive value of childhood body mass index values for overweight at age 35y. *Am J Clin Nutr* 1994; 59:810-819.
62. Whitaker RC, Wright JA, Pepe MS, et al. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med* 1997; 337:869-873.
63. Vanhala M, Vanhala P, Kumpusalo E, et al. Relation between obesity from childhood to adulthood and the metabolic syndrome: Population based study. *BMJ.* 1998;317:319-320.
64. Bao W, Srinivasan SR, Berenson GS. Persistent elevation of plasma insulin levels is associated with increased cardiovascular risk in children and young adults: The Bogalusa Heart Study. *Circulation* 1996; 93:54-59.
65. Dobbelsteyn CJ, Joffres MH, MacLean DR, et al. A comparative evaluation of waist circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk factors: The Canadian Heart Health Surveys. *Int J Obes Relat Metab Disord* 2001;25:652-661.
66. Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference and health risk: Evidence in support of current National Institutes of Health guidelines. *Arch Intern Med* 2002;162:2074-2079.
67. Janssen I, Heymsfield SB, Allison DB, et al. Body mass index and waist circumference independently contribute to the prediction of nonabdominal, abdominal subcutaneous, and visceral fat. *Am J Clin Nutr* 2002;75:683-688.
68. Moreno LA, Pineda I, Rodriguez G, et al. Waist circumference for the screening of the metabolic syndrome in children. *Acta Paediatr* 2002; 91:1307-1312.
69. Steinberger J, Daniels SR. Obesity, insulin resistance, diabetes, and cardiovascular risk in children: An American Heart Association scientific statement. *Circulation* 2003;107:1448-1453.
70. Styne DM. Childhood and adolescent obesity: Prevalence and significance. *Pediatr Clin N Amer* 2001;48:823-854.
71. Aye T, Levitsky LL. Type 2 diabetes: An epidemic disease in child-

hood. *Curr Opin Pediatr* 2003;15:411-415.
72. Shuldiner AR, Yang R, Gong D-W. Resistin, obesity, and insulin resistance–the emerging role of the adipocyte as an endocrine organ. *N Engl J Med* 2001;345:1345-1346.
73. McTernan PG, McTernan CL, Chetty R, et al. Increased resistin gene and protein expression in human abdominal adipose tissue. *J Clin Endocrinol Metab* 2002; 87:2407-2410.
74. Boyne MS. Prevention of type 2 diabetes mellitus in high-risk patients. *Preventive Medicine in Managed Care* 2000;1:S83-S94.
75. Bartosh SM, Aronson AJ. Childhood hypertension: An update on etiology, diagnosis, and treatment. *Pediatr Clin North Am* 1999;46: 235-252.
76. Rosner B, Prineas R, Daniels SR, et al. Blood pressure differences between blacks and whites in relation to body size among US children and adolescents. *Am J Epidemiol* 2000;151:1007-1019.
77. Lurbe E, Alvarez V, Liao Y, et al. The impact of obesity and body fat distribution on ambulatory blood pressure in children and adolescents. *Am J Hypertens* 1998;11:418-424.
78. Feld LG, Springate JE, Waz WR. Special topics in pediatric hypertension. *Semin Nephrol* 1998;18:295-303.
79. Kahleova R, Palyzova D, Zvara K. Essential hypertension in adolescents: Association with insulin resistance and with metabolism of homocysteine and vitamins. *Am J Hypertens* 2002;15:857-864.
80. Daniels SR, Morrison JA, Sprecher DL, et al. Association of body fat distribution and cardiovascular risk factors in children and adolescents. *Circulation* 1999;99:541-545.
81. Urbina EM, Gidding SS, Bao W, et al. Association of fasting blood sugar level, insulin level, and obesity with left ventricular mass in healthy children and adolescents: The Bogalusa Heart Study. *Am Heart J* 1999;138:122-127.
82. Hanerold C, Waller J, Daniels S, et al. The effects of obesity, gender, and ethnic group on left ventricular hypertrophy and geometry in hypertensive children: A collaborative study of the International Pediatric Hypertension Association. *Pediatrics* 2004;113:328-333.
83. Tounian P, Aggoun Y, Dubern B, et al. Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: A prospective study. *Lancet* 2001;358:1400-1404.
84. Sorot JM, Alexandrov AV, Cardwell G, et al. Carotid artery intimal-medical thickness and left ventricular hypertrophy in children with elevated blood pressure. *Pediatrics* 2003;111:61-66.
85. Gutin B, Treiber F, Owens S, et al. Relations of body composition to left ventricular geometry and function in children. *J Pediatr* 1998; 132:1023-1027.
86. Rocchini AP. Obesity hypertension. *Am J Hypertens* 2002;15: 50S-52S.
87. Falkner B, Sherif K, Michel S, et al. Dietary nutrients and blood pressure in urban minority adolescents at risk for hypertension. *Arch Pediatr Adolesc Med* 2000;154:918-922.
88. Perkin RM, Lin JJ. Obstructive sleep apnea and hypopnea in hypertensive obese pediatric patients. *Chest* 2003;124:73S.
89. Carnethon MR, Gidding SS, Nehgme R, et al. Cardiorespiratory fitness in young adulthood and the development of cardiovascular disease risk factors. *JAMA* 2003;290:3092-3100.
90. Burwell CS, Robin ED, Whaley RD. Extreme obesity with alveolar hypoventilation: A pickwickian syndrome. *Am J Med* 1956;21: 811-818.
91. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328:1230-1235.
92. Redline S, Tishler PV, Schluchter M, et al. Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. *Am J Respir Crit Care Med* 1999;159: 1527-1532.
93. Schwab RJ, Gupta KB, Gefter WB, et al. Upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing: Significance of the lateral pharyngeal walls. *Am J Respir Crit Care Med* 1995;152:1673-1689.
94. Shelton KE, Woodson H, Gay S, et al. Pharyngeal fat in obstructive sleep apnea. *Am Rev Respir Dis* 1993;148:462-466.
95. Horner RL, Mohiaddin RH, Lowell DG, et al. Sites and sizes for fat deposits around the pharynx in obese patients with obstructive sleep apnea and weight-matched controls. *Eur Respir J* 1989; 2:613-622.
96. Watanabe T, Isono S, Tanaka A, et al. Contribution of body habitus and craniofacial characteristics to segmental closing pressures of the passive pharynx in patients with sleep disordered breathing. *Am J Respir Crit Care Med* 2002;165:260-265.
97. Phillips GB, Kato M, Narkiewicz K, et al. Increases in leptin levels, sympathetic drive, and weight gain in obstructive apnea. *Am J Physiol Heart Circ Physiol* 2000; 279:H234-237.
98. Chin K, Shimizu K, Nakamura T, et al. Changes in intra-abdominal visceral fat and serum leptin levels in patients with obstructive sleep apnea syndrome following nasal continuous positive airway pressure therapy. *Circulation* 1999;100:706-712.
99. Vgontzas AN, Papanicolaou DA, Bixler EO, et al. Sleep apnea and daytime sleepiness and fatigue: relation to visceral obesity, insulin resistance, and hypercytokinemia. *J Clin Endocrinol Metab* 2000; 85:1151-1158.
100. Liese AD, Mayer-Davis EJ, Haffner SM. Development of the multiple metabolic syndrome: An epidemiologic perspective. *Epidemiol Rev* 1998;20:157-172.
101. Al-Delaimy WK, Manson JE, Willett WC, et al. Snoring as a risk factor for type 2 diabetes mellitus: A prospective study. *Am J Epidemiol* 2002;155:387-393.
102. Wing Vk, Hui SH, Pak WM, et al. A controlled study of sleep related disordered breathing in obese children. *Arch Dis Child* 2003; 88:1043-1047.
103. Rhodes SK, Shimoda KC, Wald LR, et al. Neurocognitive deficits in morbidly obese children with obstructive apnea. *J Pediatr* 1995; 127:741-744.
104. Soulton Z, Wadowski S, Rao M, et al. Effect of treating obstructive sleep apnea by tonsillectomy and/or adenoidectomy on obesity in children. *Arch Pediatr Adolesc Med* 1999;153:33-37.
105. Kohyama J, Ohinata JS, Hasegawa T. Blood pressure in sleep-disordered breathing. *Arch Dis Child* 2003;88:139-142.
106. Perkin RM, Downey R, MacQuarrie J. Sleep-disordered breathing in infants and children. *Resp Clin N Amer* 1999;5:395-426.

107. Mai XM, Nilsson L, Axelson O, et al. High body mass index, asthma and allergy in Swedish schoolchildren participating in the International Study of Asthma and Childhood. *Acta Paediatr* 2003; 92: 1144-1148.
108. Schachter LM, Peat JK, Salomes CM. Asthma and atopy in overweight children. *Thorax* 2003; 58:1031-1035.
109. Tantisira KG, Litonjua AA, Weiss ST, et al. Association of body mass with pulmonary function in the Childhood Asthma Management Program. *Thorax* 2003; 58:1036-1041.
110. Castro-Rodriquez JA, Holberg CJ, Morgan WJ, et al. Increased incidence of asthma-like symptoms in girls who become overweight or obese during school years. *Am J Respir Crit Care Med* 2001;163: 1344-1349.
111. Crespo CJ, Smit E, Troiano RP, et al. Television watching, energy intake, and obesity in US children: Results from the third National Health and Nutrition Examination Survey, 1988-1994. *Arch Pediatr Adolesc Med* 2001;155:360-365.
112. Tantisira KG, Weiss St. Complex interactions in complex traits: Obesity and asthma. *Thorax* 2001;56:64-73.
113. Hakala K, Stenius-Aarniala B, Sovijärvi A. Effects of weight loss on peak flow variability, airways obstruction, and lung volumes in obese patients with asthma. *Chest* 2000;118:1315-1321.
114. Visser M, Bouter LM, McQuillan GM, et al. Elevated C-reactive protein levels in overweight and obese adults. *JAMA* 1999;282: 2131-2135.
115. Brunette DO. Resuscitation of the morbidly obese patient. *Am J Emerg Med* 2004;22:40-47.
116. Ray RM, Senders CW. Airway management in the obese child. *Pediatr Clin N Amer* 2004;48:1055-1063.
117. Pelosi P, Croci M, Ravagnan I, et al. The effects of body mass on lung volumes, respiratory mechanics, and gas exchange during general anesthesia. *Anesth Analg* 1998; 87:654-660.
118. Goldstein NA, Armfield DR, Kingsley KA, et al. Postoperative complications after tonsillectomy and adenoidectomy in children with Down syndrome. *Arch Otolaryngol Head Neck Surg* 1998;124: 171-176.
119. Varon J, Marik P. Management of the obese critically ill patient. *Crit Care* 2001;17:187-200.
120. Ford ES, Galusta DA, Gillespie C, et al. C-reactive protein and body mass index in children: Findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *J Pediatr* 2001;138: 486-492.
121. Yudkin JS, Stehouwer CD, Emeis JJ, et al. C-reactive protein in healthy subjects: Associations with obesity, insulin resistance, and endothelial dysfunction: A potential role for cytokines originating from adipose tissue? *Arterioscler Thromb Vasc Biol* 1999;19: 972-978.
122. Wilcox PG, Weiner DS, Leighley B. Maturation factors in slipped capital femoral epiphysis. *J Pediatr Orthop* 1988;8:196-200.
123. Richardson LP, Davis R, Poultan R, et al. A longitudinal evaluation of adolescent depression and adult obesity. *Arch Pediatr Adolesc Med* 2003;157:739-745.
124. Sokol RJ. The chronic disease of childhood obesity: The sleeping giant has awakened. *J Pediatr* 2000;136:711-713.
125. Cuttler L, Whittaker JL. Pediatric obesity policy: The danger of skepticism. *Arch Pediatr Adoles Med* 2003;157:722-724.
126. Nestle M. The ironic politics of obesity. *Science* 2003; 299:781.

Physician CME Questions

51. All of the following conditions are consequences of children being overweight *except:*
    A. hyperlipidemia.
    B. obstructive sleep apnea.
    C. hypoglycemia.
    D. social difficulties.

52. The clinical definition of being overweight for children is based on:
    A. calculating the body mass index (BMI) from accurate height and weight measurements and comparing this value with norms for age and gender.
    B. the weight for age percentiles.
    C. skin fold caliper measurements.
    D. None of the above

53. Food preferences of young children significantly correlate with those of their parents.
    A. True
    B. False

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion. When your evaluation is received, a certificate will be mailed to you.

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed.

e.) Provide patients with any necessary discharge instructions.

54. A child or adolescent is defined as being overweight when the body mass index (BMI) is greater than the 75th percentile for age and gender.
    A. True
    B. False

55. All of the following statements concerning obesity in children are true *except:*
    A. Obesity is now the most common medical condition in children.
    B. Obesity is associated with significant health problems in the pediatric age group.
    C. The metabolic syndrome (hypertension, dyslipidemia, high fasting glucose, abdominal obesity) does not occur in children.
    D. Childhood obesity is associated with endothelial dysfunction and increased stiffness of elastic arteries.

56. The probability of childhood obesity persisting into adulthood is estimated to increase from approximately 20% at 4 years of age to approximately 80% by adolescence.
    A. True
    B. False

57. All the following statements concerning pediatric obesity are true *except:*
    A. Parental influences are early determinants of food attitudes and practices in young children.
    B. The most important environmental factor in the development of obesity in children younger than 6 years is parental obesity.
    C. Efforts to halt and reverse obesity and related metabolic disease should begin with young children.
    D. The exercise patterns of parents do not influence the exercise patterns of their children.

58. Which of the following statements concerning type 2 diabetes is *false*?
    A. Diabetic ketoacidosis does not occur in type 2 diabetes.
    B. Type 2 diabetes shows elevations in insulin level and fasting glucose level.
    C. Hemoglobin A_{1c} (glycosylated hemoglobin) is elevated in newly diagnosed type 2 diabetes patients.
    D. Autoantibody levels to islet cells are absent.

59. Which of the following statements is true regarding the management of the airway in an obese pediatric patient?
    A. Airway management may be difficult.
    B. Bag-valve-mask ventilaton may be difficult.
    C. Obese patients have a higher risk of aspiration.
    D. All of the above

60. Which of the following orthopedic problems is *not* associated with obesity?
    A. Legg-Calvé-Perthes disease
    B. Slipped capital femoral epiphysis
    C. Blount disease
    D. Bowing of the tibia

Answer Key:

51. C	56. A
52. A	57. D
53. A	58. A
54. B	59. D
55. C	60. A

In Future Issues:

Viral exanthems