

# Orthopedic Complications of Childhood Obesity

Mary Wills, PT, DHS, OCS

Shelby Memorial Hospital, Shelbyville, Illinois

**Purpose:** The purpose of this article is to describe the orthopedic problems known to be associated with being overweight or obese during childhood to assist the clinician in the evaluation and management of these patients. **Summary of Key Points:** Children who are overweight or obese are becoming an increasing concern in our society; the number of children and teens described as overweight or obese tripled from 1980 to 2000. Many problems have been associated with obesity and are well described in the literature, including cardiovascular problems, diabetes mellitus, liver complications, cholelithiasis, sleep apnea, and specific types of cancer. Orthopedic complications are also related to being overweight or obese during childhood. Specifically, the incidence of spinal complications, slipped capital femoral epiphysis, Blount disease, and acute fractures has been related to being overweight or obese. **Conclusions:** Clinicians should be aware of the orthopedic problems related to obesity to better educate individuals as well as to better treat children with this condition. (*Pediatr Phys Ther* 2004;16:230–235) **Key words:** obesity/complication, child, adolescent, musculoskeletal abnormalities, epiphyses/slipped, joint deformities/acquired, physical therapy, review literature

## INTRODUCTION

The number of people classified as overweight or obese continues to rise throughout the world.<sup>1–3</sup> This is especially true in countries with established market economies, including the United States.<sup>1</sup> In a survey conducted in 1999 and 2000 as part of the National Health and Nutrition Examination Survey (NHANES), 30.5% of the 4115 adults in the United States who were surveyed were described as obese [body mass index (BMI)  $\geq 30$  kg/m<sup>2</sup>].<sup>4</sup> This was an increase from the 22.9% found in NHANES III conducted between 1988 and 1994.<sup>4</sup> The prevalence of overweight persons (BMI  $\geq 25$  kg/m<sup>2</sup>) also increased from 55.9% to 64.5% in this same time period.<sup>4</sup>

Although the use of a BMI of 30 kg/m<sup>2</sup> is recognized internationally as the cutoff point for adult obesity, there have been varying definitions of childhood obesity, making international comparisons difficult.<sup>5,6</sup> In 2000, the International Obesity Task Force (IOTF) published the international standards of BMI defining the conditions of over-

weight and obesity by gender between two and 18 years of age, defined to reach a BMI of 25 and 30 kg/m<sup>2</sup> at 18 years of age.<sup>5</sup> These standards were based on data obtained from six large nationally representative cross-sectional surveys on growth from Brazil, Great Britain, Hong Kong, the Netherlands, Singapore, and the United States with more than 10,000 subjects in each survey.<sup>5</sup> Reilly et al<sup>7</sup> and Fu et al<sup>8</sup> used these standards to compare BMI of 4175 seven year olds and 623 Chinese children aged six to 11 years, respectively, and concluded that the IOTF standards had low sensitivity for detection of childhood obesity. Using population-specific BMI-for-age charts, they found greater sensitivity in determination of obesity when a BMI in the 95th percentile was compared with body fat percentage.<sup>7,8</sup> Also, a committee of pediatric experts convened by the Department of Health and Human Services<sup>9</sup> and the American Obesity Association (AOA)<sup>10</sup> has recommended that children with a BMI  $\geq 85$ th percentile of a nationally representative survey be classified as overweight and those with a BMI  $\geq 95$ th percentile be described as obese. A comparison of the standards established by each of these classification systems is shown in Table 1. Although these standards have similar figures, the IOTF generally has higher cutoff points. For American youths, use of the growth charts developed by the Centers for Disease Control and Prevention (CDC)<sup>11</sup> and the AOA guidelines<sup>10</sup> might be a more sensitive way to detect overweight and obesity.

Regardless of the definition used, overweight and obesity are problems in children and adolescents today. In the

0898-5669/04/1604-0230  
Pediatric Physical Therapy  
Copyright © 2004 Lippincott Williams & Wilkins, Inc.

Address correspondence to: Mary Wills, PT, DHS, OCS, Physical Therapy, Shelby Memorial Hospital, 200 South Cedar Street, Shelbyville, IL 62565 Email: mwills@one-eleven.net

DOI: 10.1097/01.PEP.0000145911.83738.C6

**TABLE 1**  
Comparison of Overweight and Obesity Standards as Set by IOTF<sup>5</sup> and AOA<sup>10</sup>

Age (y)	A		B		C		D	
	Males BMI (kg/m <sup>2</sup> )	Females BMI (kg/m <sup>2</sup> )	Males BMI (kg/m <sup>2</sup> )	Females BMI (kg/m <sup>2</sup> )	Males BMI (kg/m <sup>2</sup> )	Females BMI (kg/m <sup>2</sup> )	Males BMI (kg/m <sup>2</sup> )	Females BMI (kg/m <sup>2</sup> )
2	18.4	18.0	18.2	18.0	20.1	20.1	19.3	19.1
3	17.9	17.6	17.4	17.2	19.6	19.4	18.3	18.3
4	17.6	17.3	16.9	16.8	19.3	19.1	17.8	18.0
5	17.4	17.1	16.8	16.8	19.3	19.2	17.9	18.2
6	17.6	17.3	17.0	17.1	19.8	19.7	18.4	18.8
7	17.9	17.8	17.4	17.6	20.6	20.5	19.1	19.6
8	18.4	18.3	17.9	18.3	21.6	21.6	20.0	20.6
9	19.1	19.1	18.6	19.0	22.8	22.8	21.0	21.7
10	19.8	19.9	19.3	19.9	24.0	24.1	22.1	22.9
11	20.6	20.7	20.1	20.8	25.1	25.4	23.1	24.0
12	21.2	21.7	20.9	21.7	26.0	26.7	24.1	25.2
13	21.9	22.6	21.8	22.5	26.8	27.8	25.1	26.2
14	22.6	23.3	22.6	23.3	27.6	28.6	26.0	27.2
15	23.3	23.9	23.4	24.0	28.3	29.1	26.8	28.1
16	23.9	24.4	24.1	24.6	28.9	29.4	27.5	28.8
17	24.5	24.7	24.9	25.2	29.4	29.7	28.2	29.6
18	25.0	25.0	25.6	25.6	30.0	30.0	28.9	30.3

A = IOTF<sup>5</sup> BMI cutoff points for overweight at BMI for 2 to 18 year olds defined to pass through BMI of 25 kg/m<sup>2</sup> at age 18; B = AOA<sup>10</sup> cutoff points for overweight at BMI at 85th percentile according to CDC growth charts<sup>11</sup>; C = IOTF<sup>5</sup> BMI cutoff points for obesity at BMI for 2 to 18 year olds defined to pass through BMI of 30 kg/m<sup>2</sup> at age 18; ; D = AOA<sup>10</sup> cutoff points for obesity at BMI at 95th percentile according to CDC growth charts.<sup>11</sup>

United Kingdom, childhood obesity has been described as an epidemic with the frequency of overweight (BMI >85th percentile) ranging from 22% at age six years and 31% at age 15 years when data were reviewed from a nationally representative sample of 2630 English children.<sup>12</sup> Obesity (BMI >95th percentile) ranged from 10% at age six years to 17% at age 15 years in this same sample.<sup>12</sup> In the United States, a survey of 4722 children from birth to 19 years of age was performed as part of NHANES in 1999 and 2000.<sup>13</sup> The results showed the prevalence of overweight (defined as ≥95th percentile) as 15.5% among 12 through 19 year olds, 15.3% among six through 11 year olds, and 10.4% among two through 5 year olds, which compared with 10.5%, 11.3%, and 7.2%, respectively, in 1988–1994 (NHANES III).<sup>13</sup> This number tripled for children and teens aged six to 19 years between 1980 and 2000.<sup>13</sup> Among this same survey in 1999 and 2000, the prevalence of children at risk of being overweight (defined as ≥85th percentile and <95th percentile) was 14.9% for 12 through 19 year olds, 15.1% for six through 11 year olds, and 10.4% for two through five year olds.<sup>13</sup>

The multiple consequences related to overweight and obesity in childhood have been well documented.<sup>2,3,14–17</sup> Childhood obesity has been documented as a predictor of many health problems in adulthood.<sup>3,14,15</sup> The primary reason for this association is that being overweight or obese in adolescence has been associated with an increased risk of being overweight or obese in adulthood.<sup>3,16,17</sup> Obesity in adulthood has been linked to an increased risk of cancer,<sup>2,14</sup> cardiovascular diseases,<sup>2,3,14</sup> type 2 diabetes mellitus,<sup>2,14</sup> musculoskeletal disorders including osteoarthritis and chronic back pain,<sup>2</sup> and respiratory disorders such as shortness of breath and sleep apnea.<sup>2</sup>

Being overweight or obese during childhood also has a direct effect on health status during childhood.<sup>3,14</sup> Some of the most immediate consequences are psychosocial.<sup>14</sup> Children who are overweight or obese are often targets of discrimination at all ages and a negative self-image often occurs in the adolescent years.<sup>14</sup> Also, medical problems including cardiovascular conditions,<sup>3,14</sup> glucose intolerance and non-insulin-dependent diabetes mellitus,<sup>14</sup> liver complications related to high concentrations of liver enzymes,<sup>14</sup> cholelithiasis,<sup>14</sup> sleep apnea,<sup>14</sup> polycystic ovary disease,<sup>14</sup> and orthopedic complications<sup>14</sup> have been documented in children who are obese.

The physical therapy professional who works with children and adolescents must thoroughly understand the orthopedic consequences of obesity and potential obesity. Clinicians may have the opportunity to intervene to educate and treat children who present with orthopedic problems. Also, knowledge of problems related to obesity provides valuable information in the screening and evaluation process. The purpose of this article is to describe the orthopedic problems known to be associated with the conditions of being overweight or obese during childhood to assist the clinician in the evaluation and treatment of these patients. These problems include spinal complications, slipped capital femoral epiphysis, Blount disease, and fracture risk.

### SPINAL COMPLICATIONS

During growth, children who are overweight and obese do not increase their spinal bone mineral content to fully compensate for their excessive weight.<sup>18</sup> Goulding et al<sup>18</sup> evaluated bone mineral content in 362 fracture-free children between three and 19 years of age. They reported that girls who were overweight had 8% less bone mineral in

their lumbar spine for their bone area, height, weight, and pubertal development than gender-matched children of healthy body weight. Girls and boys who were obese had 12% less and 13% less bone mineral, respectively.<sup>18</sup>

Since the bone mineral content in the vertebrae has been shown to adapt to increased stress,<sup>19,20</sup> these results require further explanation. One explanation is the mechanical properties of the developing spine as compared with the mature adult spine.<sup>21</sup> Growth of the vertebral bodies occurs from the osseous surface of the cartilaginous epiphyseal plates that cover both the upper and lower end plates of the vertebra.<sup>22</sup> These growth plates are not protected by epiphyseal bone plates as are the growth plates of long bones.<sup>21</sup> Instead, the vertebral ring apophyses lie outside the epiphyseal plates and calcify at about six years of age, begin to ossify at about 13 years of age, and begin to fuse with the vertebral body mass at about 17 years of age.<sup>23</sup> The children in the study performed by Goulding et al<sup>18</sup> had a mean age of 11 years, an age at which ossification might not have begun,<sup>23</sup> explaining the lack of an increase in bone mineral content in the vertebrae of children who were heavier and possible the resulting spinal complications.

Karlsson et al<sup>21</sup> concluded that the compressive strength of the spine and the vertebral bone mineral content have a close linear relationship. They also noted that the weakest part of the spinal segment in adolescents is the growth plate. Although this study was performed by compression of functional spinal units of cadavers (mean age of 16.3 years), these results are important as adolescents have experienced avulsion injuries of the vertebral end plate, while adults have more frequently experienced a rupture of a disc or a vertebral fracture in spinal injuries related to accidents.<sup>24</sup>

The developing spine might be more flexible than the mature spine due to the lack of ossification, but overload and resultant pain would still seem to be a plausible concern. One consideration in the etiology of low back pain may be the geometry of bone. The geometry of bone affects bone strength; wide bones are stronger than narrow bones.<sup>18</sup> In the study of 362 children by Goulding et al,<sup>18</sup> both genders showed some adaptive increases in bone mineral accrual, while only boys accommodated for their excessive weight by enlarging projected vertebral bone area.<sup>18</sup> Overall, males have wider and larger vertebrae than females,<sup>25</sup> and low back pain is reported less frequently by adolescent boys than girls.<sup>26</sup>

Kujala et al<sup>27</sup> documented more frequent reports of low back pain and spinal abnormalities shown on magnetic resonance imaging during the adolescent growth spurt when the dissociations between body weight and bone mineral content are the greatest. In a group of 1171 children and adolescents, Taimela et al<sup>28</sup> reported 10.1% of the boys and 9.4% of the girls reported low back pain that interfered with school or leisure activities in the preceding 12 months. Body weight was not considered in this study.<sup>28</sup> But a review of 65 epidemiologic studies of low back pain did report that 32% of the 65 reviewed

studies showed a statistically positive link between weight and low back pain.<sup>29</sup> Since obesity is an external factor that may be controlled, the clinicians should be active in primary prevention strategies. Limiting excessive adiposity in childhood and adolescence could avoid excessive loads and stresses on the lumbar spine and therefore the related complications.

In addition to spinal complications, risks of disease or injury in the extremities of children with obesity are of interest to the practicing physical therapist. These include slipped capital femoral epiphysis, Blount disease, and an increased risk of fracture.

### SLIPPED CAPITAL FEMORAL EPIPHYSIS (SCFE)

SCFE is a change in the anatomic relationship of the femoral head with its neck and shaft due to an epiphyseal plate disruption.<sup>30</sup> This can occur one of two ways: 1) the shear force of the capital femoral growth plate increases to a critical value quickly so the femoral head suddenly separates with an epiphyseal cartilage crack or 2) increased shear force, exerted chronically, causes a gradual slip. Generally, the direction of the slippage of the femoral head is inferior and posterior in relationship to the femoral neck; the femoral head remains in the acetabulum and the entire limb externally rotates.<sup>30</sup> By definition, this disruption occurs before growth plate closure.<sup>30</sup>

Loder et al<sup>31</sup> reviewed the cases of 1630 children with 1993 SCFE. This was a worldwide collection of data consisting of 47.5% white, 24.8% black, 16.9% Amerindian, 7.4% Indonesian-Malay, 2.1% Native American/Pacific Islands, and 1.3% Indo-Mediterranean children.<sup>31</sup> The relative racial frequency of SCFE compared with the white population (reference of 1.0) was 4.5 for the Polynesian, 2.2 for the black, 1.05 for the Amerindian, 0.5 for the Indonesian-Malay, and 0.1 for the Indo-Mediterranean children.<sup>31</sup> Of the 1630 participants, 671 were girls (41.2%) and 959 were boys (58.8%).<sup>31</sup> The average age for the girls and boys was 12 and 13.5 years, respectively, with the age at diagnosis decreasing with increasing obesity.<sup>31</sup> The child's weight status (at the time of the first slip) was known for 1337 children.<sup>31</sup> Of these, 51.5% were obese (body weight >95th percentile), 11.7% were overweight (body weight in 90th to 95th percentile), 8.6% were below average weight (body weight in 10th to 48th percentile), and 4% were underweight (body weight <10th percentile).<sup>31</sup>

As these numbers indicate, body weight may be strongly associated with the occurrence of SCFE.<sup>30-33</sup> Other studies have shown half of the participants with SCFE were in or above the 95th percentile,<sup>32</sup> while Chung<sup>30</sup> described a 72% rate of obesity in children with SCFE.

The mechanism of the relationship of obesity and SCFE is not defined simply by the increased force on the femoral head secondary to the increased load from an increased body mass.<sup>30</sup> Hip abduction increases in those who are obese; hence, the shear component applied to the capital femoral growth plate is also increased, resulting in a higher risk of SCFE.<sup>30</sup> Increased body mass exerted on the epiphysis increases both compressive and shear forces.<sup>30</sup>

Another contributing factor is the decreased femoral anteversion in children who are obese<sup>34</sup> as femoral retroversion is associated with SCFE.<sup>35</sup>

Obesity may further exacerbate problems in those with slow skeletal maturation because increased stress is exerted on a wide growth plate for a longer than the normal period.<sup>30</sup> Loder et al<sup>33</sup> also reported that obese children experience SCFE on average one year earlier than nonobese children due to the threshold stress on the proximal femur physis.

Of those who experience SCFE, bilateral involvement has been reported as 22.3% by Loder<sup>31</sup> in the review of 1630 children worldwide. Of the bilateral cases of sequential presentation, an average of one year lapsed between the initial occurrence and the second SCFE.<sup>31</sup> During this time, 79 of the 89 children remained at the same weight, five increased in weight, and four decreased in weight.<sup>31</sup>

Knowledge of the demographics associated with SCFE may be helpful to the clinician working with the pediatric population. The severity of the problems related to SCFE might be more manageable with weight control as Chung<sup>30</sup> described that 34% of children with slight slippage are obese while 72% of children with bilateral slips are obese. Clinicians should use this information to further educate patients on the importance of weight management to help prevent the occurrence of as well as limit the severity of SCFE.

## BLOUNT DISEASE

Blount disease is a skeletal disorder affecting the medial side of the proximal tibial epiphysis and causing a varus deformity of the tibia.<sup>36–38</sup> This developmental condition is characterized by bowed legs and tibial torsion.<sup>38</sup>

Blount disease is a mechanical response to unequal or early weight-bearing when physiologic genu varum is at its peak, causing undue stress on the medial tibial condyle.<sup>37,38</sup> In accordance with the Heuter-Volkman principle that increased pressure on a physis inhibits growth, the abnormal medial weight-bearing force is transmitted across the tibiofemoral component and slows the growth of the medial tibial physis.<sup>39</sup> Both infantile and adolescent versions of Blount disease occur.

Infantile Blount disease affects children between the ages of one and three years.<sup>37</sup> It is bilateral in 60% to 80% of cases and is characterized by progressive tibial bowing.<sup>37</sup> The incidence of infantile Blount disease has not been widely reported, although a review of 5000 cases of bowleg by Smith<sup>40</sup> found only 37 of these to have Blount disease for an incidence of <1%. Infantile Blount disease can be easily differentiated from physiologic bowleg in which the varus deformity resolves with growth.<sup>37</sup> Additionally, the tibia and femur are both angulated in physiologic bowleg, while only the tibia is involved in Blount disease.<sup>37</sup> The infantile form affects boys and girls equally, but blacks are more commonly affected than whites.<sup>39,41</sup> Some anecdotal reports of hereditary involvement have been reported<sup>42–44</sup> and Meade et al<sup>45</sup> noted a positive family history for 14 of 33

patients. This small sample would suggest a genetic cause, although no hereditary factors have been determined.<sup>39</sup>

Obesity has been linked with the prevalence and degree of angulation of the deformity in infantile Blount disease.<sup>37,38</sup> Dietz et al<sup>38</sup> reviewed 18 cases of children with Blount disease. Of these, 16 had infantile Blount disease, and all the patients described as obese (>120% of ideal body weight) had infantile Blount disease.<sup>38</sup> This was a retrospective study, and therefore the relationship of the onset of obesity and the onset of the Blount disease could not be determined, although activity levels of all the children were reported to be comparable.<sup>38</sup> Dietz et al<sup>38</sup> also noted a correlation between the percentage of ideal body weight and the angle of the femoral condyles to the tibial shafts ( $r = 0.75$  among 15 patients who were a mixture of obese and nonobese and  $r = 0.90$  among nine patients who were obese). Due to this severity, 12 patients defined as obese had a total of 26 osteotomies, while the six patients who were not obese had a total of eight osteotomies.<sup>38</sup> The researchers also noted that no further surgery was needed in one patient who lost weight after the initial osteotomy, while others who remained obese had a recurrence of the deformity leading to further surgeries.<sup>38</sup> Aside from this risk, clinical recovery from surgery would predictably be simpler in a patient without the added complication of obesity.

Adolescent Blount disease is less common than the infantile form.<sup>37</sup> It has been linked to occult trauma, infection, formation of a bony bar, and slowly progressive infantile disease that does not become apparent until adolescence.<sup>37</sup> More than 90% of the reported cases have been in black males who are morbidly obese.<sup>36</sup> To determine the prevalence of adolescent Blount disease, Henderson et al<sup>46</sup> selected all the boys on area high-school football teams who weighed >210 lb. The weight of 210 lb was selected by researchers because it was the 95th percentile for 18-year-old males according to National Center for Health Statistics.<sup>46</sup> Of the 1117 boys, 140 boys (80 black and 60 white) met the weight criteria.<sup>46</sup> Radiographs of the seven boys who clinically screened positive showed that two boys had adolescent Blount disease.<sup>46</sup> The resulting prevalence was 2.5% (two of 80 boys) in the adolescent black male population described as obese.<sup>46</sup>

This epidemiology would support the theory that massive weight gain in adolescents with underlying varus alignment at the knee could lead to excessive medial compartment loading and altered physeal growth.<sup>36</sup> However, Henderson and Greene<sup>47</sup> did not find a correlation between varus static alignment and the development of adolescent tibia vara. Davids et al<sup>36</sup> pursued the hypothesis that an adolescent who is obese with a normal, neutral static standing mechanical axis (center of hip joint to center of knee joint to center of ankle joint) would have dynamic gait deviations to compensate for increased thigh girth that resulted in increased loading of the medial compartment of the knee.<sup>36</sup> Gait deviations found by three-dimensional motion analysis included dynamic stance-limb knee varus,

increased stance-limb knee rotation, and swing-limb circumduction.<sup>36</sup> Pathologic compressive forces were then reproduced in an anthropometric model by using these gait deviations and clinically appropriate excessive body weight.<sup>36</sup> The compressive forces generated at the medial compartment of the knee were considered of sufficient magnitude to alter physeal growth.<sup>36</sup> Although a study using anthropometric models may have some limitation in clinical application, the biomechanical theory that the increased dynamic-loading conditions related to the gait changes, in conjunction with excessive body weight, can result in forces of sufficient magnitude to alter physeal growth and lead to tibia vara is plausible. Clinical notice should also be given to gait patterns in the adolescent patient, especially those of excessive weight. Attention to BMI in children and adolescents can serve in primary, secondary, and tertiary prevention for those at risk of and those with Blount disease.

## FRACTURE RISK

In a study of girls, aged three to 15 years, a group of 100 who had each recently traumatically fractured a forearm was compared with a group of 100 who were fracture free.<sup>48</sup> The purpose was to determine factors that influence fracture risk in young girls.<sup>48</sup> Results showed that previous fractures, low total body areal bone mineral density ( $\text{g}/\text{cm}^2$ ), and high body weight each independently increased the risk of new fractures in growing children.<sup>48</sup> Also, spinal volumetric bone mineral apparent density ( $\text{g}/\text{cm}^3$ ) was a predictor of new fractures.<sup>48</sup> The fracture group had also previously been shown to be heavier than the control group with the eight to 19 year olds being 4.7 kg heavier.<sup>49</sup>

Peak fracture incidence in children coincides with the growth spurt.<sup>50</sup> During this growth period, the metaphyseal/diaphyseal density ratio is lowest, and dissociation between longitudinal growth and mineral accrual increases bone fragility and alters bone quality and microarchitecture.<sup>50</sup> Obesity during this time period may increase the likelihood of fractures during falls as bone development does not adequately cope with excess weight.<sup>51</sup> This weight/bone mass imbalance will also place high levels of stress on growing bones and joints that may result in joint damage and may contribute to osteoarthritis in later years.<sup>51</sup>

Although bone mass is also related to activity, activity level was no different in the study comparing girls who were obese with forearm fractures to a control group.<sup>49</sup> Lack of exercise may be a factor in the pathogenesis of obesity, further contributing to a risk of fracture. Also, children who are overweight or obese may fall with more force and in a more awkward manner than children who are of normal weight.<sup>48</sup> This has been supported by a study by Petti et al<sup>52</sup> involving 938 six- to 11-year-old children. Of this group, 21.3% had experienced traumatic dental injuries from falling; 30.8% of the children who were obese had experienced a dental injury compared with 20.0% of the children of normal weight.<sup>52</sup> They also found that the

children described as obese were much less physically active and almost 40% of the injuries occurred indoors.<sup>52</sup>

Since reduced physical activity is a large contributing factor in obesity and adversely affects bone mineral accrual,<sup>53</sup> promoting exercise is clinically important in the adolescent. By augmenting bone mineral content and reducing body fat through regular activity, the risk of fracture will decline.

In addition to these factors, postural stability and balance have been documented as possible problems in prepubertal boys<sup>54</sup> and adolescents.<sup>55</sup> McGraw et al<sup>54</sup> found that boys aged eight to 10 years who were obese spent a greater percentage of the gait cycle in dual stance and had diminished dynamic stability. Furthermore, a study of 93 boys aged 10 to 21 years supported that adolescents who are overweight have poorer balance than those of healthy weight when results of the Bruninks-Osteretsky balance test and computerized posturography tests were compared.<sup>55</sup> These results would again suggest the need to reduce obesity to prevent falls and injuries.

## SUMMARY

Orthopedic complications related to obesity in adolescents are of interest to the practicing clinician. Both prevention and treatment of these problems need to be addressed among the pediatric population in the clinic and in the community.

Although observation in the clinic would seem to indicate that other orthopedic injuries or problems are related to obesity, no current studies indicate an increased incidence of lower extremity injuries. Further study in this area would provide clinicians with a more comprehensive view of the scope of the problem.

As childhood obesity continues to be a growing problem, active efforts should be made by physical therapists to educate children and those providing for them. Clinicians should participate in controlling obesity and its many complications. The musculoskeletal problems associated with obesity should be addressed along with the many systemic problems.

## REFERENCES

1. Seidell JC. Obesity: a growing problem. *Acta Paediatr Suppl.* 1999; 428:46–50.
2. Visscher TLS, Seidell JC. The public health impact of obesity. *Annu Rev Public Health.* 2001;22:355–375.
3. Reilly JJ, Methven E, McDowell ZC, et al. Health consequences of obesity. *Arch Dis Child.* 2003;88:748–752.
4. Flegal KM, Carroll MD, Ogden CL, et al. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA.* 2002;288:1723–1727.
5. Cole TJ, Bellizzi MC, Flegal KM, et al. Establishing a standard definition for childhood overweight and obesity worldwide: international survey. *BMJ.* 2000;320:1240–1243.
6. Redefining obesity and its treatment. Manila, World Health Organization, Regional Office for the Western Pacific, 2002.
7. Reilly JJ, Dorosty AR, Emmett PM. Identification of the obese child: adequacy of the body mass index for clinical practice and epidemiology. *Int J Obes Relat Metab Disord.* 2000;24:1623–1627.
8. Fu WP, Lee HC, Ng CJ, et al. Screening for childhood obesity: international vs population-specific definitions. Which is more appropriate? *Int J Obes Relat Metab Disord.* 2003;27:1121–1126.

9. Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration, and the Department of Health and Human Services. *Pediatrics*. 1998;102:E29.
10. American Obesity Association. Childhood obesity: prevalence and identification. Available at: <http://www.obesity.org/subs/childhood/prevalence.shtml>. Accessed March 20, 2004.
11. Ogden CL, Kuczmarski RJ, Flegal KM, et al. Centers for Disease Control and Prevention 2000 growth charts for the United States: improvements to the 1977 national center for health statistics version. *Pediatrics*. 2002;109:45–60.
12. Reilly JJ, Dorosty AR. Epidemic of obesity in UK children. *Lancet*. 1999;354:1874–1875.
13. Ogden CL, Flegal KM, Carroll MD, et al. Prevalence and trends in overweight among US children and adolescents, 1999–2000. *JAMA*. 2002;288:1772–1773.
14. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics*. 1998;101:518–525.
15. Nicklas TA, Webber LS, Srinivasan SR, et al. Secular trends in dietary intakes and cardiovascular risk factors of 10-y-old children: the Bogalusa Heart Study (1973–1988). *Am J Clin Nutr*. 1993;57:930–937.
16. Guo S, Roche A, Chumlea, et al. The predictive value of childhood body mass index values for overweight at age 35. *Am J Clin Nutr*. 1994;59:810–819.
17. Dietz WH. Childhood weight affects adult morbidity and mortality. *J Nutr*. 1998;128:4115–4145.
18. Goulding A, Taylor RW, Jones IE, et al. Spinal overload: a concern for obese children and adolescents? *Osteoporos Int*. 2002;13:835–840.
19. Dyson K, Blimkie CJ, Davison KS, et al. Gymnastic training and bone density in pre-adolescent females. *Med Sci Sports Exerc*. 1997;29:443–450.
20. Porter RW, Adams MA, Hutton WC. Physical activity and the strength of the lumbar spine. *Spine*. 1989;14:201–208.
21. Karlsson C, Lundin O, Ekstrom L, et al. Injuries in adolescent spine exposed to compressive loads: an experimental cadaveric study. *J Spinal Dis*. 1998;11:501–507.
22. Bick EM, Copel JW. Longitudinal growth of the human vertebra. *J Bone Joint Surg Am*. 1950;32:803–814.
23. Bick EM, Copel JW. The ring apophysis of the human vertebra. *J Bone Joint Surg Am*. 1951;33:783–787.
24. Jonsson H Jr, Bring C, Rauschnig W, et al. Hidden cervical spine injuries in traffic accident victims with skull fractures. *J Spinal Disord*. 1991;4:251–263.
25. Gilsanz V, Boechat M, Roe T, et al. Gender differences in vertebral body sizes in children and adolescents. *Radiology*. 1994;190:673–674.
26. Harreby M, Nygaard B, Jessen JT, et al. Risk factors for low back pain in a cohort of 1389 Danish school children: an epidemiologic study. *Eur Spine J*. 1999;8:444–450.
27. Kujala UM, Erkintalo MO, Taimeal S, et al. Role of acute injury during adolescent growth spurt in development of lumbar spine abnormalities. *Lancet*. 1994;344:1020.
28. Taimela S, Kujala UM, Salminen JJ, et al. The prevalence of low back pain among children and adolescents. *Spine*. 1997;22:1132–1136.
29. Leboeuf-Yde C. Body weight and low back pain. *Spine*. 2000;25:226–237.
30. Chung SMK. Slipped capital femoral epiphysis (SCFE). In: Chung SMK, ed. *Hip Disorders in Infants and Children*. Philadelphia: Lea & Febiger; 1981;173–191.
31. Loder RT. The demographics of slipped capital femoral epiphysis. *Clin Orthop*. 1996;322:8–27.
32. Kelsey JL, Acheson RM, Keggi KJ. The body build of patients with slipped capital femoral epiphysis. *Am J Dis Child*. 1972;124:276–281.
33. Loder RT, Aronson DD, Greenfield ML. The epidemiology of bilateral slipped capital femoral epiphysis. *J Bone Joint Surg*. 1993;75:1141–1147.
34. Galbraith RT, Gelberman RH, Hajek PC, et al. Obesity and decreased femoral anteversion in adolescence. *J Orthop Res*. 1987;5:523–528.
35. Gelberman RH, Cohen MS, Shaw BA, et al. The association of femoral retroversion with slipped capital femoral epiphysis. *J Bone Joint Surg Am*. 1986;68:1000–1007.
36. Davids JR, Huskamp M, Bagley AM. A dynamic biomechanical analysis of the etiology of adolescent tibia vara. *J Pediatr Orthop*. 1996;16:461–468.
37. Warner WC, Canale ST, Beaty JH. Congenital deformities of the knee. In: Scott WN, ed. *The Knee*. St. Louis: Mosby; 1994:209–227.
38. Dietz WH, Gross WL, Kirkpatrick JA. Blount disease (tibia vara): another skeletal disorder associated with childhood obesity. *J Pediatr*. 1982;101:735–737.
39. Bradway JK, Klassen RA, Peterson HA. Blount disease: a review of the English literature. *J Pediatr Orthop*. 1987;7:472–480.
40. Smith CF. Current concepts review: tibia vara (Blount's disease). *J Bone Joint Surg Am*. 1982;64:630–632.
41. Bathfield CA, Beighton PH. Blount disease: a review of etiological factors in 110 patients. *Clin Orthop*. 1978;135:29–33.
42. Langenskiold A, Riska EB. Tibia vara (osteochondrosis deformans tibiae): a survey of seventy-one cases. *J Bone Joint Surg Am*. 1964;46:1405–1420.
43. Sibert JR, Bray PT. Probable dominant inheritance in Blount's disease. *Clin Genet*. 1977;11:394–396.
44. Tobin WJ. Familial osteochondritis dissecans with associated tibia vara. *J Bone Joint Surg Am*. 1957;39:1091–1096.
45. Meade WC, Schoenecker PL, Pierron RL, et al. Blount's disease—a retrospective review and recommendations for treatment [Abstract]. *Orthop Trans* 1983;7:372–373.
46. Henderson RC, Kemp GJ, Hayes PRL. Prevalence of late-onset tibia vara. *J Pediatr Orthop*. 1993;13:255–258.
47. Henderson RC, Greene WB. Etiology of late-onset tibia vara: is varus alignment a prerequisite? *J Pediatr Orthop*. 1994;14:143–146.
48. Goulding A, Jones IE, Taylor RW, et al. More broken bones: a 4-year double cohort study of young girls with and without distal forearm fractures. *J Bone Miner Res*. 2000;15:2011–2018.
49. Goulding A, Cannan R, Williams SM, et al. Bone mineral density in girls with forearm fractures. *J Bone Miner Res*. 1998;13:143–148.
50. Bailey DA, Wedge JH, McCulloch RG, et al. Epidemiology of fractures of the distal end of the radius in children as associated with growth. *J Bone Joint Surg Am*. 1989;71:1225–1231.
51. Goulding A, Taylor RW, Jones IE, et al. Overweight and obese children have low bone mass and area for their weight. *Int J Obesity*. 2000;24:627–632.
52. Petti S, Cairella G, Tarsitani G. Childhood obesity: a risk factor for traumatic injuries to anterior teeth. *Endod Dent Traumatol*. 1997;13:285–288.
53. Morris F, Naughton G, Gibbs J, et al. Prospective ten-month exercise intervention in premenarchal girls: positive effect on bone and lean mass. *J Bone Miner Res*. 1997;12:1453–1462.
54. McGraw B, McClenaghan BA, Williams HG, et al. Gait and postural stability in obese and nonobese prepubertal boys. *Arch Phys Med Rehabil*. 2000;81:484–489.
55. Goulding A, Jones IE, Taylor RW, et al. Dynamic and static tests of balance and postural way in boys: effects of previous wrist bone fractures and high adiposity. *Gait Posture*. 2003;17:136–141.