Considerations Related to Weight-Bearing Programs in Children with Developmental Disabilities

Wayne A Stuberg

PHYS THER. 1992; 72:35-40.

Considerations Related to Weight-Bearing Programs in Children with Developmental Disabilities

Wayne A Stuberg

Standing is a common modality used in the management of children with developmental disabilities. The purpose of this article is to examine the scientific basis for standing programs, with specific emphasis on the known effects of weight bearing on bone development. Guidelines for the use of standing programs are presented, and the supporting rationale is discussed. [Stuberg WA. Considerations related to weight-bearing programs in children with developmental disabilities. Phys Ther. 1992;72:35–40.]

Key Words: *Bone development; Child development disorders; Kinesiology/biomechanics, general; Orthopedics, general; Pediatrics, development.*

The use of standing is common to physical therapy management of children with developmental disabilities who are chronologically older than 14 to 16 months of age. Although therapists strive for standing without the use of orthoses or adaptive equipment, external support devices are prescribed when active control is inadequate or absent. A *standing program* refers to the use of orthoses or adaptive equipment to position a child in standing when motor control is inadequate to allow standing without such devices.

Standing programs have been recommended for children who have limited mobility in upright posture, including children with cerebral palsy (CP),[1–3] meningomyelocele,[4] muscular dystrophy,[5] and osteogenesis imperfecta.[6–8] The use of adaptive equipment or orthoses has been an accepted method of providing weight bearing in standing for these children. The efficacy of these standing programs has not been thoroughly examined.

The literature has few data-based studies that outline guidelines for standing programs. Clinicians must judge frequency, duration, and device type when recommending standing programs, and, because no standards exist, decisions are left to the clinician's intuition or experience. The purpose of this article is to examine the basis for standing programs for children with developmental disabilities. Specific emphasis is placed on the effects of weight bearing on bone development. Methodologies for assessing bone development will first be discussed, followed by a review of the factors known to affect bone development. Guidelines for standing programs will then be recommended.

Measurement of Bone Mineral Content/Density

Little is known about the effects of weight bearing on the development of bone in children.[2,3] Measurement of linear growth in bone is possible through the use of standard roentgenograms. Techniques to assess bone mass are single-photon absorptiometry (SPA), dual-photon absorptiometry (DPA), and quantitative computed tomography (QCT).[9] Single-photon absorptiometry detects differential photon absorption between bone and soft tissue to allow calculation of bone mineral content (BMC) and is limited to use at peripheral sites such as the radius. By contrast, DPA, which emits two different gamma energies and permits direct measurement of BMC and bone mineral density (BMD) (ie, the BMC per unit of area scanned), can be used to measure the hip, spine, or total body. Neither SPA nor DPA can discriminate between cortical and trabecular bone. Quantitative computed tomography is used specifically to evaluate trabecular versus cortical BMC.

WA Stuberg, PhD, PT, is Director of Physical Therapy, Meyer Rehabilitation Institute, Associate Professor, Division of Physical Therapy Education, and Assistant Professor, Department of Anatomy, University of Nebraska Medical Center, 600 S 42nd St, Omaha, NE 68198-5450 (USA).

The assessment of modeling changes of bone secondary to standing programs or other loading stimuli is possible through the use of DPA and QCT. Research to assess fracture risk and to determine optimal guidelines for standing programs to maintain joint alignment or facilitate bony development is needed.

Factors Affecting Bony Development

Normal bone growth and development is affected by factors including genetic coding,[10–12] nutrition,[13] appropriate levels of some nutrients and hormones (eg, vitamin D, calcium, estrogen, parathyroid hormone),[14,15] and mechanical loading through weight bearing and muscle tension.[16] In weight-bearing bones, where locomotion efficiency depends in part on bone mass, dynamic strains are essential to maintain bone mass.[14] Dynamic strains are repetitive forces that cause minute deformation of the bone. Activity level has been found to be a major determinant in the development of BMC. Disuse, decreased activity, and non–weight bearing have been shown to precipitate a loss of 0.4% to 0.6% per month in adults without developmental disabilities.[17–19] The early bone loss during disuse has been reported to be primarily in trabecular versus cortical bone because of the rapid metabolic turnover of trabecular bone.[20]

Donaldson and associates[19] studied the effects of a bed-rest program on nondisabled men aged 21 to 27 years. The duration of bed rest was 30 weeks for one subject and 36 weeks for two subjects. Serum calcium levels and BMC were assessed. A 25% to 44% loss of BMC was recorded in the calcaneus from week 12 until the end of the trial. During a 36-week exercise program following termination of bed rest, the subjects recovered BMC at approximately the same rate at which they lost BMC during bed rest. Issekutz and colleagues[17] used a 7-week bed-rest program to study the effects of bed rest on urinary calcium levels in 14 nondisabled male subjects (18-21 years of age). One half of the subjects exercised while in bed; the other subjects were sedentary. The authors reported that a 1-hour-per-day exercise program, not including weight bearing, was not effective in retarding urinary calcium loss. The researchers, however, did report that other preliminary work demonstrated that 2 to 3 hours of passive standing on a daily basis, used in conjunction with bed rest, was effective in retarding urinary calcium loss.

The effects of mechanical forces on the development and remodeling of the skeleton have been studied extensively for over a century. Wolff's law states that the remodeling of bone occurs in the presence or absence of physical forces, that is, that bone is deposited in sites subjected to adequate force and is resorbed when forces are reduced.[21] Recently, Frost[22] has made significant contributions to the understanding of bone dynamics by introducing the principle of "flexure drift." The principle pertains to the macroarchitectural responses of bone to dynamic bending strain.[22,23] As Frost's principle applies to this article, the important points are

1. The stimulus for remodeling is mechanical strain (deformation), not stress (pressure), and specifically repetitive, dynamic flexure caused by repetitive mechanical loading on the bone.

2. The response will occur to time-averaged, repetitive strains versus single or occasional strains, with the relative rate, frequency, and magnitude of the strain being unknown.

3. Strains must be provided within physiological limits that achieve the desired response (eg, greater strain to induce greater change).

Electrical potentials resulting from repetitive, dynamic strain have been directly measured within bone.[24–26] Wolff's law and Frost's principle of flexural drift, therefore, may be mediated by electrical potentials.[24,25] The electric potentials created during strain of the bone are thought to signal osteoclastic and osteoblastic cells directly, thus mediating the modeling response. Although the presence of electrokinetic potentials have been recorded in vivo and in vitro, their role in the modeling process has not been fully elucidated.[26]

Specificity of Weight-Bearing Stimulus to Model Bone

Results in Animal Studies

Lanyon and colleagues[15] hypothesized that the first response to loading is a decrease in osteoclastic activity and that only with continued stimuli does osteoblastic activity lead to bone formation. Weight-bearing activities have resulted in increased bone mass and resistance to bending or fracture in animals, including mice,[27–29] roosters,[30] and dogs.[31] Hert and colleagues[32] pioneered a technique of applying known loads to bones in vivo using the rabbit. Rubin and Lanyon[30,33] applied the technique to isolated rooster and turkey ulna preparations using implanted strain gauges. They explored the effect of load duration with static versus intermittent loading and load magnitude on bone mass and architecture. Immobilization with static loading led to rapid and significant bone loss when the load was applied over an 8-week period. This loss was represented by a 15% to 20% reduction in cross-sectional area. These results confirmed the findings of earlier studies.[27,32,34]

Intermittent loading, in contrast to static loading, has been found to retard bone loss. Lanyon and coworkers[15,30,35] studied the effect of intermittent loading at levels measured by in vivo strain gauges during wing flapping on bone loss in rooster and turkey ulnas. They applied intermittent loading for 0, 4, 36, 360, or 1,800 consecutive loading cycles of 0.5 Hz per day for 6 weeks. The four-cycle regimen proved adequate for retarding bone loss, and the 36-cycle regimen demonstrated a 40% increase in BMC, a value that was not significantly improved by the addition of a greater number of loading cycles.

Rubin and Lanyon[36] also examined the effect of load magnitude by varying the strain load from 15% to 100% of physiologic levels at a constant load frequency of 100 consecutive daily reversals over an 8-week period. Maintenance of original bone area was achieved with a strain load corresponding to 30% of strain levels ascertained during wing flapping. Strains greater than 30% showed an incremental increase in the amount of bone deposited, with the greatest amount recorded following the highest strain.

Results in Human Studies

Weight bearing has been described as a key component in decreasing the likelihood of osteoporosis in nondisabled adults.[37–39] The effects of weight bearing and exercise on BMD have been documented in studies of osteoporosis in postmenopausal women.[40–43] The results of these studies consistently showed increased BMD as a benefit of weight bearing and exercise.

In a study of 64 male athletes who participated in full-scale physical exercise programs versus 39 nondisabled, age-matched, sedentary male subjects, Nilsson and Westlin[44] reported athletes to have greater BMD. Bone mineral density is task related, with greater densities recorded in weight lifters and football players than in runners or swimmers.[45,46] Activity-related differences in BMD within an individual demonstrate the importance of mechanical loading in the development of BMD. An example is the significantly higher BMC in the dominant wrist than in the nondominant wrist of professional tennis players.[47]

No studies describing the effects of standing programs on bone modeling for children with developmental disabilities have been published. Research is currently underway, however, in a group of 20 children with CP who are nonambulatory and using standing programs in their educational settings.[48,49] Preliminary results indicate that BMD is significantly less in nonambulatory children with severe to profound CP than in children who are nondisabled. Bone mineral density measurements of the patella, tibial plateau, and supracondylar femur of children with CP demonstrate values of one third to one half of those of age-matched peers without disabilities.[48] Additionally, use of a standing program of 60 minutes' duration four or five times per week appears to result in increased BMD measurements.[49] Reduction of BMD was observed upon removal of the standing program for even a short period of time (ie, summer break) or when the standing program had an average duration of 30 minutes and a frequency of three times per week.[49]

Acetabular development appears to be dependent on articulation of the femoral head in the acetabulum and is promoted through weight bearing.[50–54] The findings of Phelps[50] have been substantiated by Howard et al[53] and Samilson et al[54] regarding the significant role of weight bearing on the development of the acetabulum in children with CP. The use of standing programs to enhance acetabular development appears valid. The justification for the use of standing programs to facilitate acetabular development is particularly strong for children with CP, as hip dysplasia is typically not present at birth in these children.[50,53,54]

Clinical Implications

Children who are known or suspected to have decreased bone mass or bone density should be considered candidates for standing programs. If the results of animal studies of the effect of mechanical loading on bone are applicable to humans (and the similarities across species suggest the assumption may be valid), then important implications can be drawn from these studies about standing programs in children with developmental disabilities. Although specific guidelines for selected disabilities are included in the "Additional Considerations" section later in the article, I believe the following guidelines can be used as a general framework in prescribing a standing program.

Guidelines for Standing Programs

Amount of weight bearing in standing. Results of research using the turkey ulna indicate that strain loads as small as 15% to 30% may have a sparing effect if the loading frequency is adequate.[33] Maximal strain levels were established by direct strain-gauge measurements of the turkey ulna during vigorous wing flapping.[35] The strain level to stimulate bone modeling in children has not been ascertained. If we assume, however, that the force exerted through the lower extremities during standing is within the range to stimulate bone homeostasis and possibly deposition, then standing programs may be an effective stimulus to bone development in children.

The amount of weight bearing that a child is receiving in standing should be ascertained if the goal of the program is to stimulate bone development. The type of orthosis or adaptive equipment used by a child can become important if the equipment redistributes the vertical load by supporting the torso or lower extremities. For example, a child tilted 50 degrees from vertical on a prone stander with the child's arms supported may be placing only one half of the body weight through the legs.[55] Miedaner[55] and Curtis[56] have both reported that widely used standing devices such as prone or supine standers allow loading of up to 70% to 75% of body weight if the devices are adjusted near vertical. I suggest that therapists check for the amount of vertical loading by placing a scale or pressure gauge under the child's feet. In using orthoses, such as knee-ankle-foot orthoses or any orthotic device that supports the legs or torso, the pressure on the bottom of the foot in the brace should be measured.

Standing duration. Duration of the standing program is variable, dependent on whether the goal is bone development, acetabular development,

or contracture management. A standing program of 2 to 3 hours per day for adults has been reported to retard bone resorption.[17,57,58] Preliminary work I have conducted indicates that a duration of at least 60 minutes, in conjunction with a frequency of four or five times per week, is needed to retard bone loss in children with CP who are nonambulatory.

Phelps[50] recommends beginning weight-bearing programs as early as 12 to 16 months of age in children with CP who are at risk for hip dislocation. Phelps reports using a protocol of 3 hours daily with no more than 1 hour at a time. The report by Phelps, however, is anecdotal, without objective outcome measures.

Standing programs of approximately 45 minutes' duration, three times daily, are also reported to control contractures of the lower extremity and to facilitate bone development in children with CP,[1] muscular dystrophy,[59] and meningomyelocele.[60] Specific guidelines to control contractures in children with spastic CP have been advocated by Tardieu and colleagues[61] and include elongation of the muscle for at least 4 hours daily.

Standing frequency. According to animal studies, if loading is near physiologic levels, then a frequency of only four loading cycles per day over a period of 2 weeks would be needed to maintain and possibly stimulate additional bone formation.[30] The duration of the loading cycle was 0.5 seconds for the animal model experiment.[35] Perhaps these four cycles could be carried out in a single session; however, the practice advocated by researchers thus far is daily standing or standing for a minimum of four times per week.[49,50,59,60]

Smith[62] has recommended a three-times-per-week frequency of weight bearing for elderly adults to retard osteoporosis. Based on a review of current practice and animal studies, I believe that children should participate in a standing program at least four or five times per week for a duration of about 60 minutes to facilitate bone development. Standing at a frequency of two or three times daily for a duration of 45 minutes should be considered as an adjunct to a positioning program to control lower-extremity flexion contractures.

Additional Considerations

Chronological age, as opposed to developmental age, is the most common criterion for the use of standing programs chosen by many orthopedists, with the standing program beginning when the child is approximately 12 to 16 months of age.[50,51] Developmental age may be a more appropriate criterion for the use of a standing program for some children, particularly when orthopedic management goals do not preclude postponing the onset of standing. Additionally, standing without appropriate postural support may be detrimental to the child with spasticity, regardless of the age criterion used. Standing equipment should provide correct anatomical alignment of the torso and lower extremities. As most standing devices (eg, a prone or supine stander) do not typically provide distal control, splints or orthoses should be considered.

Monitoring of children's nutritional programs by a dietitian or nutritionist is recommended, particularly for children who are significantly below the normal range on the growth curve or who have osteoporosis. Inadequate dietary intake of calcium or other nutrients required for development of bone mass and bone density will have a detrimental effect, regardless of the appropriateness of the standing or activity program.[38,41]

The use of standing programs for children who have high-lumbar or thoracic meningomyelocele is encouraged by several researchers.[63–65] Rosenstein et al[66] have reported a direct relationship among ambulatory status, lesion level, and the development of BMD in children with meningomyelocele. In comparison with nonambulators, a 38% increase of BMD at the tibia and a 44% increase at the first metatarsal were reported. The use of standing and walking programs for adolescents with high-level defects (eg, thoracic lesions) is controversial, however, because, by adolescence, 70% to 90% of these individuals use wheelchairs for mobility.[63,67] Mazur and colleagues[65] compared 36 children with high-level defects who participated in a standing and walking program with 36 children for whom wheelchair use had been prescribed. The standing program guidelines were not described. The authors reported that 33% of the children in the standing and walking group were able to walk around the community, 20% walked around the home only, and 47% were nonwalkers at the completion of the study. The children who walked early had fewer fractures and were more independent in transfer skills; however, this group had also spent more days in the hospital and were not significantly different from the children who used wheelchairs with regard to skills of daily living.

Standing programs and the prolongation of walking through the use of orthoses are common for children with Duchenne's muscular dystrophy. Spencer and Vignos[68] have reported a dramatic improvement in functional capacity and increased longevity of 2 to 4 years when standing and walking is prolonged through the use of orthoses and adaptive equipment. Vignos et al[59] recommended that standing programs be incorporated into the classroom routine for the nonambulatory school-aged child for at least 3 hours daily. Contracture progression and excessive physical size are primary factors to be considered in discontinuing the standing program. Progression of contractures results in inability to wear orthoses because of skin breakdown and in inability to allow correct alignment in standing. Excessive physical size increases the risk of injury to the child or caregiver by making transfers difficult.

The use of standing programs for children with osteogenesis imperfecta is recommended by most experts; however, the recommended duration of the program has not been specified.[6–8] The use of specialized or-

thoses, including contoured orthoses[7] or vacuum pants,[8] is reported to provide support and reduce the risk of fracture during weight bearing.

Conclusions

Standing programs have been shown to have an effect on bone development in humans and animals. Bone mineral density has been demonstrated to increase with exercise programs that provide a physiologic stimulus for bone modeling. Intermittent loading appears to be a key stimulus during standing, as opposed to increasing the time of a static program. Therefore, active participation from the child is recommended to increase strain on the bone through muscle activity.

Reports in the literature indicate there is a decreased incidence of contractures and fractures in children with developmental disabilities who participate in standing programs.[4,6–8,65] Although suggestions related to standing have been introduced in this article, programs for contracture management and fracture prevention need to be elucidated further. No guidelines have been developed to ascertain fracture risk for children with developmental disabilities. Further study could have a significant effect on the use of standing programs as a management modality for contractures and fractures.

As loading with a constant pressure has not been found to be an effective stimulus for bone modeling in animals, an apparent controversy exists regarding the current method of using static programs in humans.[34,35] Perhaps static standing programs using orthoses or adaptive equipment are not truly static, because some motion is allowed. Anecdotal evidence for the use of standing programs for children with developmental disabilities has been demonstrated, and, until a more efficacious method of providing mechanical stimulation to the bone is identified, the use of standing programs with loading administered for at least 60 minutes, four or five times per week, is recommended as a general guideline for bone development.[48–50,59,60,62]

References

1 Salter RB. *Textbook of Disorders and Injuries of the Musculoskeletal System.* 2nd ed. Baltimore, Md: Williams & Wilkins; 1983:5–14, 257–265.

2 Bleck EE. *Orthopaedic Management in Cerebral Palsy.* Philadelphia, Pa: MacKeith Press; 1987:142–212.

3 Tachdjian MO. *Pediatric Orthopedics.* Philadelphia, Pa: WB Saunders Co; 1990;3:1620–1622.

4 Anschuetz RH, Freehafer AA, Shaffer JW, Dixon MS. Severe fracture complications in myelodysplasia. *J Pediatr Orthop.* 1984;4:22–24.

5 Seigel IM. *Muscle and Its Diseases: An Outline Primer of Basic Science and Clinical Method.* Chicago, Ill: Year Book Medical Publishers Inc; 1986:218–245.

6 Bleck EE. Nonoperative treatment of osteogenesis imperfecta: orthotic and mobility management. *Clin Orthop.* 1981;159:111–122.

7 Binder H, Hawks L, Graybill G. Osteogenesis imperfecta: rehabilitation approach with infants and young children. *Arch Phys Med Rehabil.* 1984;65:537–541.

8 Letts M, Monson R, Weber K. The prevention of recurrent fractures of the lower extremities in severe osteogenesis imperfecta using vacuum pants: a preliminary report in four patients. *J Pediatr Orthop.* 1988;8:454–457.

9 Hassager C, Christiansen C. Usefulness of bone mass measurements by photon absorptiometry. *Public Health Rep.* 1989;104(suppl): 23–33.

10 Matkovic V, Chesnut C. Genetic factors and acquisition of bone mass. *J Bone Miner Res.* 1987;1(suppl):329. Abstract.

11 Smith DM, Nance WE, Kang DW, et al. Genetic factors in determining bone mass. *J Clin Invest.* 1973;52:2800–2808.

12 Lutz J. Bone mineral, serum calcium, and dietary intakes of mother/daughter pairs. *Am J Clin Nutr.* 1986;44:99–106.

13 Santora AC. Role of nutrition and exercise in osteoporosis. *Am J Med.* 1987;82(suppl 18): 73–79.

14 Martin AD, McCulloch RG. Bone dynamics: stress, strain, and fracture. *J Sports Sci.* 1987; 5:155–163.

15 Lanyon LE, Rubin CT, Baust G. Modulation of bone loss during calcium insufficiency by controlled dynamic loading. *Calcif Tissue Int.* 1986;38:209–216.

16 LeVeau BF, Bernhardt DB. Developmental biomechanics: effect of forces on the growth, development, and maintenance of the human body. *Phys Ther.* 1984;64:1874–1882.

17 Issekutz B, Blizzard JJ, Birkhead NC, Rodahl K. Effect of prolonged bedrest in urinary calcium output. *J Appl Physiol.* 1966; 21:1013–1020.

18 Goldsmith RS, Killian P, Inghar SH, Bass DE. Effect of phosphate supplementation during immobilization of normal men. *Metabolism.* 1969;18:349–368.

19 Donaldson CL, Hulley SB, Vogel JM, et al. Effect of prolonged bedrest. *Metabolism.* 1970;19:1071–1084.

20 Courpron P. Bone tissue mechanisms underlying osteoporosis. *Orthop Clin North Am.* 1981;12:513–545.

21 Wolff J. Die Lehre von den funktionellen Knochengestalt. *Virchows Arch [A].* 1899;155: 256–262.

22 Frost HM. Mechanical determinants of bone modeling. *Metab Bone Dis Rel Res.* 1982;4:217–229.

23 Frost HM. *The Laws of Bone Structure.* Springfield, Mo: Charles C Thomas, Publisher; 1964.

24 Fukada E, Yasuda I. Piezoelectric properties of bone. *J Phys Soc Jpn.* 1957;12:1158–1163.

25 Bassett CAL, Becker RO. Generation of electric potentials by bone in response to mechanical stress. *Science.* 1962;137:1063–1064.

26 Chakkakal DA. Mechanoelectric transduction in bone. *J Mater Res.* 1989;4:1034–1046.

27 Woo S-L, Kuei S, Amiel D, et al. The effect of prolonged physical training on the properties of long bone: a study of Wolff's law. *J Bone Joint Surg [Am].* 1981;63:780–786.

28 Kisskinen A, Heikkinen E. Physical training and connective tissues in young mice: biochemistry of long bones. *J Appl Physiol.* 1978;44:50–54.

29 Bell RR, Tzeng DY, Draper HH. Long-term effects of calcium, phosphorus and forced exercise on the bones of mature mice. *J Nutr.* 1980;110:1161–1167.

30 Rubin CT, Lanyon MR. Regulation of bone formation by applied dynamic loads. *J Bone Joint Surg [Am].* 1984;66:397–402.

31 Martin RK, Albright JP, Clarke WR, et al. Load-carrying effects on the adult beagle tibia. *Med Sci Sports Exerc.* 1981;13:343–349.

32 Hert J, Liskova M, Landgrot B. Influence of the long-term continuous bending on the bone: an experimental study on the tibia of the rabbit. *Folia Morphol (Praha).* 1969;17: 389–399.

33 Rubin CT, Lanyon LE. Osteoregulatory nature of mechanical stimuli: function as a determinant for adaptive remodeling in bone. *J Orthop Res.* 1987;5:300–310.

34 Carter DR, Vasu R, Spengler DM, Dueland RT. Stress fields in the unplated and plated canine femur calculated from in vivo strain measurements. *J Biomech.* 1981;14:63–70.

35 Lanyon LE, Rubin CT. Static vs dynamic loads as an influence on bone remodeling. *J Biomech.* 1984;17:897–906.

36 Rubin CT, Lanyon LE. Regulation of bone mass by mechanical loading: the effect of peak strain magnitude. *Calcif Tissue Int.* 1985;37: 411–417.

37 Aisenbrey JA. Exercise in the prevention and management of osteoporosis. *Phys Ther.* 1987;67:1100–1104.

38 Goodman CE. Osteoporosis: protective measures of nutrition and exercise. *Geriatrics.* 1985;40:59–70.

39 Notelovitz M. How exercise affects bone density. *Contemp Ob/Gyn.* 1986;27:108–116.

40 Krolner B, Toft B, Porsnielsen S, Tondevold E. Physical exercise as prophylaxis against involutional vertebral bone loss: a controlled trial. *Clin Sci.* 1983;64:541–546.

41 Smith EL, Reddan W, Smith PE. Physical activity and calcium modalities for bone min-

eral increase in aged women. *Med Sci Sports Exerc.* 1981;13:60–64.

42 Ayalon F, Simkin A, Leichter I, Raifmann S. Dynamic bone loading exercises for postmenopausal women: effect on the density of the distal radius. *Arch Phys Med Rehabil.* 1987;68:280–283.

43 Dalsky GP, Stock KS, Ehsani AI, et al. Weight-bearing exercise training and lumbar bone mineral content in postmenopausal women. *Ann Intern Med.* 1988;108:824–828.

44 Nilsson BE, Westlin NE. Bone density in athletes. *Clin Orthop.* 1971;77:179–182.

45 Nilsson BE, Anderson SM, Hardrup TV, Westlin NE. Bone mineral content in ballet dancers and weight lifters. In: *Proceedings of the Fourth International Conference on Bone Measurement; University of Toronto, Toronto, Ontario, Canada.* 1978:81–86.

46 Dalen N, Olssen KE. Bone mineral content and physical activity. *Acta Orthop Scand.* 1974;45:170–174.

47 Jones H, Priest J, Hayes W, et al. Humeral hypertrophy in response to exercise. *J Bone Joint Surg [Am].* 1977;59:204–208.

48 Stuberg WA. Bone density changes in nonambulatory children following discontinuation of passive standing programs. In: *Proceedings of the American Academy of Cerebral Palsy and Developmental Medicine Conference; Louisville, Ky; October 10, 1991.*

49 Stuberg WA. Comparison of bone density in cerebral palsy and nondisabled children. In: *Proceedings of the American Society for Bone and Mineral Research Annual Meeting; San Diego, Calif; August 29, 1991.*

50 Phelps WM. Prevention of acquired dislocation of the hip in cerebral palsy. *J Bone Joint Surg [Am].* 1959;41:440–448.

51 Beals RK. Developmental changes in the femur and acetabulum in spastic paraplegia and diplegia. *Dev Med Child Neurol.* 1969;11:303–313.

52 Harrison TJ. The influence of the femoral head on pelvic growth and acetabular form in the rat. *J Anat.* 1961;95:12–24.

53 Howard CB, McKibbon B, Williams LA. Factors affecting the incidence of hip dislocation in cerebral palsy. *J Bone Joint Surg [Br].* 1985; 67:530–532.

54 Samilson RL, Tsou P, Aamoth G, et al. Dislocation and subluxation of the hip in cerebral palsy. *J Bone Joint Surg [Am].* 1972;54:863–873.

55 Miedaner J. An evaluation of weight bearing forces at various angles for children with cerebral palsy. *Pediatric Physical Therapy.* 1990;2:215.

56 Curtis L. *The Evaluation of Weight Bearing of Children on Prone, Supine, and Upright Standers.* Chapel Hill, NC: The University of North Carolina at Chapel Hill; 1989. Thesis.

57 Birge SJ, Wheldon GD. Bone. In: McNally M, ed. *Hypodynamics and Hypogravics.* New York, NY: Academic Press Inc; 1968:213–235.

58 Overton TR, Hangartzer TN, Heath R, et al. The effect of physical activity on bone: gamma ray computed tomography. In: DeLuca HF, ed. *Osteoporosis: Recent Advances in Pathogenesis and Treatment.* Baltimore, Md: University Park Press; 1981:147–158.

59 Vignos PJ, Spencer GE, Archibald KC. Management of progressive muscular dystrophy of childhood. *JAMA.* 1963;184:89–96.

60 Tappit-Emas E. Physical therapy intervention. In: Schafer MF, Dias LS, eds. *Myelomeningocele Orthopaedic Treatment.* Baltimore, Md: Williams & Wilkins; 1983.

61 Tardieu C, Huet de la Tour E, Bret MD, et al. Muscle hypoextensibility in children with CP: parts I and II. *Arch Phys Med Rehabil.* 1982;63:97–107.

62 Smith EL. How exercise helps prevent osteoporosis. *Contemp Ob/Gyn.* 1985;25:51–60.

63 Shurtleff DB. Myelodysplasia: management and treatment. *Curr Probl Pediatr.* 1980;10: 64–71.

64 Kupka J, Geddes N, Carroll NC. Comprehensive management in the child with spina bifida. *Orthop Clin North Am.* 1978;9:97–113.

65 Mazur JM, Shurtleff DB, Menelaus MB, et al. Orthopaedic management of high-level spina bifida. *J Bone Joint Surg [Am].* 1989; 71:56–61.

66 Rosenstein BD, Greene WB, Herrington RT, et al. Bone density in myelomeningocele: the effects of ambulatory status and other factors. *Dev Med Child Neurol.* 1987;29:486–494.

67 Stillwell A, Menelaus MB. Walking ability in mature patients with spina bifida. *J Pediatr Orthop.* 1983;3:184–190.

68 Spencer GE, Vignos PJ. Bracing for ambulation in childhood progressive muscular dystrophy. *J Bone Joint Surg [Am].* 1962;44:234–242.

Considerations Related to Weight-Bearing Programs in Children with Developmental Disabilities

Wayne A Stuberg

PHYS THER. 1992; 72:35-40.