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MUSCULOSKELETAL IMPAIRMENTS

CLINICIAN'S CORNER

So Is NDT Just Everything, Then?

THE ANSWERS TO THREE QUESTIONS PINPOINT THE NDT APPROACH

By Marcia Stamer, PT

As an NDTA instructor, I learn a lot myself while teaching others. The participants who take the courses I teach have ways of asking questions, describing the children they treat, or discussing their observations of the children we all see during the course that force me to pause and consider the best wording for my response. In having to do this, I am forced to organize and articulate my own observations, opinions, and explanations. Later, I often (obsessively) reflect on my answers to see if I can think of better explanations.

So here are a few of my recent questions:

1) You talk about guided weight shifting with graded handling; everyone already knows about that. What's so different about weight shifting and NDT?

2) You would use a treadmill with a child with CP in NDT? I thought NDT was all about handling patients. I guess NDT just includes everything now, huh?

3) You are teaching us about some of the systems theories of motor control. What does that have to do with NDT? I heard that NDT is an outdated approach and the systems theories now replace it.

Well, those are good questions! Except if you are the one *(continued on page 13)*

Getting Down to the Bare Bones

PEDIATRIC ORTHOPEDICS PART 1: THE MODELING PROCESS

By Beverly Cusick, PT, MS, COF/BOC

This series of articles will address the characteristics of, and developmental changes in, pelvic and lower-extremity bone design, joint configuration and alignment, and soft tissue extensibility. These characteristics change as the modeling process prepares the growing skeleton, muscle, nerve, and fascial tissues to endure the demands of adult daily life. The modeling process is the focus of this article.

Later articles in this series will feature a discussion of the roles of various morphologic constraints in the maturation of postural and lower-extremity joint alignment in infancy.

All segments of this series carry through to clinical implications and proposed management strategies.

SKELETAL MODELING

Skeletal development spans the fifth week of gestation, when the hyaline cartilage skeletal model appears, to the end of skeletal ossification, perhaps as late as age 25 years.[1] To a progressively diminishing degree, postnatal skeletal modeling gradually redesigns the cartilage models of bones and joints as they ossify. Structures which were designed to suit intrauterine confinement are reconfigured to sustain thousands of daily load-bearing demands throughout adulthood.[2] Harold Frost, whose lifetime study of skeletal modeling comprises the primary basis for this discussion, states: "...the postnatal skeleton is architecturally fluid to a significant degree, and …the stereotypism of the mature skeleton reflects the stereotypism of its mechanical *(continued on page 15)*

Terminology: Mechanics of Skeletal Modeling

The Wolff Law (a.k.a. Heuter-Volkmann Law) of bone transformation, (paraphrased): Mechanical stresses can modulate bone shape and internal architecture.

Modeling: The process by which agents external to a growing tissue influence its fiber grain, growth speed, and direction in ways that creates its microscopic and gross architectures. This process is primarily confined to the growing skeleton only.

Remodeling: A process that, in part, maintains the functional competence of already-existing tissues and structures.

Strain: Any deformation or change in the shape or dimensions of a structure caused by any kind of load applied to it. The principle modeling strains are described in Table 1.

Dynamic Strain: Bone modeling evidently responds primarily to changing strains, i.e., to strain rate, rather than to constant strains. If constant strains have physiologic effects on this system they are obscure and poorly understood.

Minimum Effective Signal (MES): This is the threshold—a range of strain magnitudes—above which the feedback signals for modeling evoke a response and effect the system. Strains below MES are considered trivial and ineffective in activating the modeling process.

Strain-Averaging Mechanism: The modeling systems of both bone and fibrous tissue somehow average the peak strains and strain rates over a period of time, perhaps between 1 and 12 months, then produce the changes in the tissue's bulk strength that are needed to fit that history of mechanical usage. The growing system puts bone where it is needed, provided it is needed often enough. Then, the system leaves the bone there, even though the demands of other, more frequent modes of usage do not require it.

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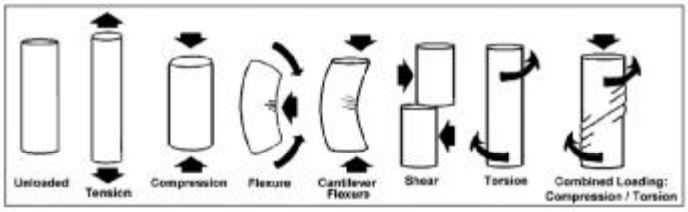
usage during growth, coupled to the initial conditions established by the blind execution of the embryonic and fetal structural blueprint"[2 (p.26)]. "Since the nervous system coordinates muscle contractural strengths and patterns, it follows that the chondral modeling contribution to joint configuration and limb alignment must reflect certain properties of that neurologic control as well" [1 (p. 294)].

Dynamic strain occurs in bone when the cartilage (chondral) model deforms in response to movement-generated, externally applied loads. (Modeling clay strains—changes shape—in response to imposed loads.) In postnatal life, and only while bone is growing, dynamic strain appears to govern the modeling process. Frost states: "Even on weight bearing joints, the major loads come from muscle forces rather than body weight, and they can, during strenuous activities, readily exceed body weight by factors of 2, 5 or even 10x [times]..." [1 (p. 295)]. Those loads and the resulting, deforming strains include compression, tension, cantilever flexure (bending under compression), and shear. Shear occurs in the context of weight-loaded torque (twisting) loads.[3,4] Movement mixes these strains by repeatedly engaging body weight and ground reaction forces in an expanding range of activities.



Adapted from Frankel, et al 1980.

Developing bone evidently requires both a threshold magnitude and a history of strain-related mechanical signals in order to activate the modeling mechanism. Different types of strain result in different modeling effects. The chronic application of gravitational load can also influence bony geometry.[1,5] *(See Table 1, page 14)*

Adolf et al (1998) illuminated the significance of an active usage history and of the role of practice in motor development by obtaining data pertaining to the quantity and variety of loading moments experienced by nondisabled infants engaged in early standing and pre-walking activities. The infants wore shoes with computerized microprocessors embedded in the soles. The microprocessor registered a count when the shoe was off-loaded to less than three pounds. The caretakers plugged the shoes into a counting box each evening. The researchers determined that the infants experienced 500-1500 weight-shifts and loading events per waking hour.[6]

THE ROLE OF CARTILAGE IN SKELETAL MODELING

As Frost tells us, cartilage plays a dominant role in skeletal growth and development, both before and after birth, for it determines the original number, location and shape of the fetal models of most bones, as well as their orientations, alignment and articulations. Hyaline cartilage models comprise the main portion of the fetal skeleton, and begin appearing in the fifth gestational week with cartilage cell formation. The cartilage cells build a matrix material that is then covered by perichondrium, a connective tissue sheath, the inner cells of which produce hyaline cartilage. By seven weeks gestational age, the cartilage models, by then infused with blood vessels, begin undergoing osteogenesis.[2]

Young bone contains less mineral substance and more water than old bone. Hyaline cartilage, which is 78% water, is more compliant than bone, and is capable of much greater unit creep deformation than are bone, mature tendon, or mature ligament.[1] As soon as bone is laid down in the cartilage matrix, the perichondrium becomes a thick and spongy periosteum. Fetal ligaments and muscle tendons attach solely to periosteum and apply tension to the bone model surface. The resulting strains, which begin with movements in utero and continue to occur after birth, eventually produce the strong attachment sites observed on mature bone surfaces, including tuberosities, tubercles, trochanters, and ridges.[1] These superficial enlargements also improve the efficiency of the attached muscles by increasing the distance between the joint axis and the force line of the muscle, and therefore, improving the muscle leverage.

Postnatal cartilage modeling establishes the following skeletal characteristics:

- Bone and limb length
- Tendon, ligament, and fascia attachment locations and sizes
- Fascial and joint size and shape
- Spinal and limb alignment
- Body height and proportions
- Geometric properties of bones and joints.

LAMELLAR BONE MODELING

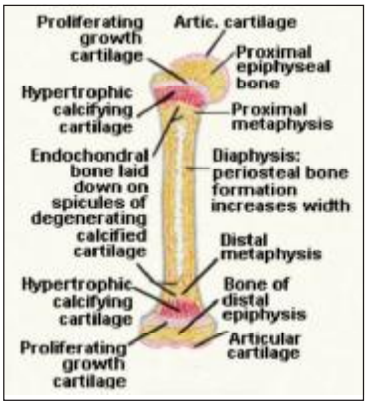
Lamellar bone comprises more than 99% of the adult skeleton. Most bone forms by the process of endochondral ossification—the destruction and replacement of a pre-formed, hyaline cartilage model.

Trabeculae are laid down by osteoblasts in loci and patterns which generally align at right angles to each other, and parallel with typical tension and compression strains.[1]

Lamellar bone deposits only on pre-existing trabeculae. As a result, the

(continued on page 16)

Sites of Growth in Bone Length



Femur: Female—Age 10 years

Adapted from Crelin ES, Netter FH, Estler IA. Clinical Symposia: Development of the Musculoskeletal System.Volume 33(1). Summit, NJ: Medical Education Division, CIBA; 1981.

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TABLE 1

Principle Strains and Modeling Effects

STRAIN	MODELING EFFECT
Compression: a reduction in distance between the ends of an object, with a concurrent increase in diameter, under a load, which is directed through both ends toward the center. Example: weight-bearing. Normal compression loads are applied intermittently—as they are in the context of normal movements—and are of a magnitude that falls within the maximum normal body weight, and therefore do not exceed the chondral growth / force response (CGFR) peak. The CGFR peak is the loading range within which the optimum normal growth response to compression strain occurs.	Compression strains of normal magnitude induce an increase in the growth rate at the compression sites on the epiphyseal plates, and an increase in the cross-sectional diameter of the bone via the periosteum. Epiphyses and compression-loaded apophyseal plates normally take loads that fall close to the CGFR peak, and so compression-related bone growth increases with the increasing magnitude of strains that occurs with normal body growth. Example: The resolution of genu varum and valgum occurs with increased compression strain on the concave side of the knee joint (the tibiofemoral angle as it is viewed from the anterior-posterior perspective). Growth rate increases at the epiphyses on the same side of the joint, while relatively reduced compression strain on the convex side of the joint contributes less stimulus to grow on that side of the joint.
Tension: an increase in distance between two points of application of a load which draws those points away from the center and from each other. Tendon and ligament attachments to bone are normally loaded in tension.	Tension loads on a chondral plate increase its growth. The result is formation of tuberosities, trochanters, tubercles, prominences, and ridges at the attachment sites. This process starts with the first fetal movements.
Flexure: Bending, by applying a lateral load to the center of the [bone] while stabilizing both ends. **Cantilever flexure:** Bending an already bent structure by applying compression loads to the ends of that structure. Cantilever flexure comprises tension, compression, and shear strains.	Flexure drift: Apparent movement of all affected bone surfaces in the concave-tending direction. The concave surface fills (bone grows faster), the convex surface resorbs, and the bone straightens.
Shear: Displacement of successive layers or segments in opposite directions, by applying a pair of equal and opposite parallel loads directed across the part. Shear is a primary component of torque strain (twist), which occurs in the context of normal movement.	
Torque: Torsional deformation: a twisting displacement about the longitudinal axis of a part, such that the ends of the object rotate in opposite directions to each other. Torque strain features a combination of compression, tension, and primarily, shear.	Reduction or increase in torsional declination through the shaft, or about the longitudinal axis, of a bone, in accordance with the direction of the application of the torque load.
Compression and Torque: Example: this combination of strains occurs in the load-bearing lower extremity during single-limb-stance in normal walking. As the swing limb advances in the sagittal plane, the stance limb endures weight-loaded lateral rotation forces—torque toward the loaded side—from the pelvis to the feet.	Torsional alteration occurs, enhanced by compression-induced increase in bone growth rate.

areas of maximum resistance to tension and compression loads usually match the expected, lifetime application of major mechanical loads.

Primary ossification centers appear in the central portion of the long bones. At birth, all long bones reveal primary ossification centers, with secondary ossification centers evident at the distal femur and proximal tibia. Secondary ossification centers continue to develop at variable rates in the chondroepiphyseal regions at the ends of the bones. The mineralization process displaces water from the matrix, and endows bone with characteristic stiffness. Newly formed bone requires many months, often years, to fully mineralize.[1]

CLINICAL IMPLICATIONS OF SKELETAL MODELING

The younger the child, the more responsive the skeletal system is to mechanical usage and related strains. The therapist who works with very young children has the greatest potential to influence the skeletal modeling mechanism. A schedule of weekly or biweekly "therapeutic handling" or "treatment" sessions for a child with movement disorder could not possibly influence the modeling process. Skeletal modeling requires a history of either thousands of movements that, collectively and appropriately, strain the developing tissues, or the chronic application of external loads as in orthotic splinting, (continued on page 17)

(Getting Down to the Bare Bones continued from page 16)

prolonged positioning, and more recently, in the use of such modalities as orthopedic and KinesioTaping, and TheraTogs™ garment and strapping systems. Early intervention—using dynamic devices that improve posture and joint alignment throughout the day—offers the greatest prospect for minimizing the development of bone and joint deformities.

Examples. *Image 1:* Normal movement activities model the bones and joints correctly. *Images 2-4:* Foam-lined, breathable elasticized strapping systems that stay on the skin and stay in place can assist and influence the modeling process.

FIBROUS TISSUE MODELING

The main component of the fibrous tissue matrix is Type I collagen. As occurs at the epiphyses plates in bone, predetermined growth in fibrous tissue length occurs at the ends of the tendons (the musculotendinous junction), ligaments and fascia, rather than in the middle. Growth in length responds primarily to circulatory and systemic, rather than mechanical, factors. As fibrous tissue is made, existing tension loads and strains direct the organization of the collagen fibers to align parallel to the major loading vectors. They therefore model in response to mechanical usage.[7]

Fibrous tissues model into three kinds of structures:

1. Fascial sheets, like cloth, provide tension, rigidity, and strength in various directions within the plane of the structure and can transfer tensile loads.
2. Ligament or tendon confines the same rigidity and strength as fascial sheets to a single axis or line, to transfer tensile loads between bones or from muscle to bone.
3. Loose, three-dimensional networks or mesh binds together the various cells and noncollagenous intercellular components of organs such as subcutaneous tissue and muscle.

Tropocollagen fibers are bound together by chemical cross-links, which prevent them from sliding past each other and which provide the mature tissue with tensile strength and stiffness. Repeated dynamic tension loads of normal magnitude, yet greater than the tissue's threshold for activating the modeling process, bring about an increase in both the diameter of assembled fiber bundles and the number of crosslinks. Thus, strength and stiffness increase throughout the structure.[7,8]

After the first two postnatal months, infants show a status of generalized ligament laxity, with associated soft tissue hyperextensibility and excessive joint range of motion (ROM). In a British study of more than 3000 non-disabled children age one week to 18 years, 50% showed evidence of generalized joint laxity at age three years; 5% at age six years; and less than 1% at age 12 years.[9] Females showed a higher incidence of joint laxity than males at all ages. However, two other studies found that 10%-12% of participating school-aged children showed evidence of generalized ligament laxity.[10,11]

CLINICAL IMPLICATIONS OF FIBROUS TISSUE MODELING

*** Laxity.** Researchers have associated joint hypermobility with such events as joint pain, increased incidence of ligament injuries in sporting activities, and premature osteoarthritis in adulthood.[12]

An infant with excessive ligament laxity might routinely distribute her weight on her medial forefeet or exhibit predominantly static, rather than dynamic and variable, foot postures in standing positions. The arch-supporting ligaments can be protected from enduring accelerated creep deformation with appropriately-designed and fitted heel cups or shoe inserts.[13,14,15,16] (SureStep Dynamic Stabilizing Orthoses at www.surestep.net). Like eye glasses that improve vision only while they are worn, such shoe inserts are likely to be a lifetime intervention for those whose foot alignment improves while wearing them and does not correct without them.

Hypermobility into hip abduction can interfere with rolling, with the timely achievement of transitions into sitting and quadruped positions, and with the execution of efficient weight shifts over the knees and feet. Jaffe et al (1998) identified joint hypermobility in 126 of 715 infants, ages 8-14 months. Of the total group, 30.2% of them showed early motor delay, particularly those with hypermobility into hip abduction, elbow hyperextension, and ankle DF. Of the remaining subjects with normal joints, 10.9% showed motor delay. Six months later, 65% of the hypermobility group, and 79.2% of the larger group had resolved their motor delays.[17]

When an infant exhibits an extremely wide base of support when attempting various activities, the therapist might try applying Hip Helpers—elasticized long-leg shorts (or short pants), made with the inseams sewn together (Hip Helpers Inc. at www.hiphelpers.com.) Hip Helpers are designed to comfortably reduce the functional hip abduction, providing a constraint that helps in training and strengthening the hip musculature, until such loading and movement skills improve.

I used Heel Seats (Heel Seats – AliMed, Inc. at www.alimed.com) and Hip Helpers with my Chinese daughter, Ting, when she was 13 months of age. Immediately upon donning the Hip Helpers, she was able to assume quadruped. I saw that the garment-induced change of functional hip joint alignment placed considerable, even exhausting demands on her musculoskeletal (continued on page 18)

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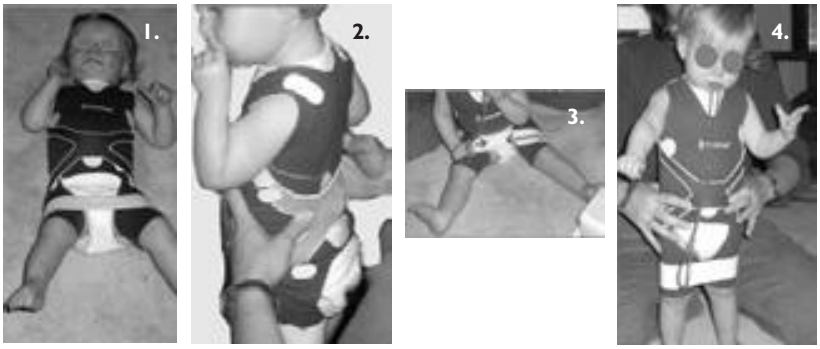
system, and how quickly she acquired the hip joint stability she was lacking after about two weeks of daily wear for about two hours of 'practice.' Similarly, she stood stable and quiet only when wearing the heel seats in her athletic shoes. Without them, she was struggling. Unlike the average Chinese infant who walks independently at around 18 months, Ting walked at age 14 months.

Hip Helpers do not address hip rotation. Orthopedic taping without causing skin irritation is a high-level skill. There is a limit, also set by skin tolerance, to KinesioTaping for mechanical effect. Dissatisfied with the heating effects, the complexity, and the lack of adaptability of other garment-like orthotic systems, I developed the TheraTogs™ Orthotic Undergarment and Strapping System to meet a wider variety of specific needs for supporting the goals of postural and neuromotor training.

The fundamental principle of TheraTogs strapping is to dynamically assist and reduce the length of the underused, over-lengthened musculature in functional context. The strapping system typically replicates some of the sensory and manual effects of manual facilitation, while carrying the session into daily life, all day long. I've come to use them in conjunction with KinesioTaping to influence functional alignment and muscle recruitment. *(continued on page 19)*



Examples. Age 16 months: Partial Agenesis of the Corpus Callosum; Hypotonia, Developmental Delay: *Image 1:* TheraTogs garments increase body awareness. Elasticized split strap across abdominals connects thorax to pelvis. Stretch strap across anterior thighs gently reduces hip abduction and lateral rotation. *Image 2:* Elasticized split strap across lumbar spine connects thorax to pelvis, and adds to lumbar extension and trunk stability. *Images 3 & 4:* Stretch strap across the anterior thighs reduces hip abduction and lateral rotation; increases activation of the postural musculature by reducing the base of support.

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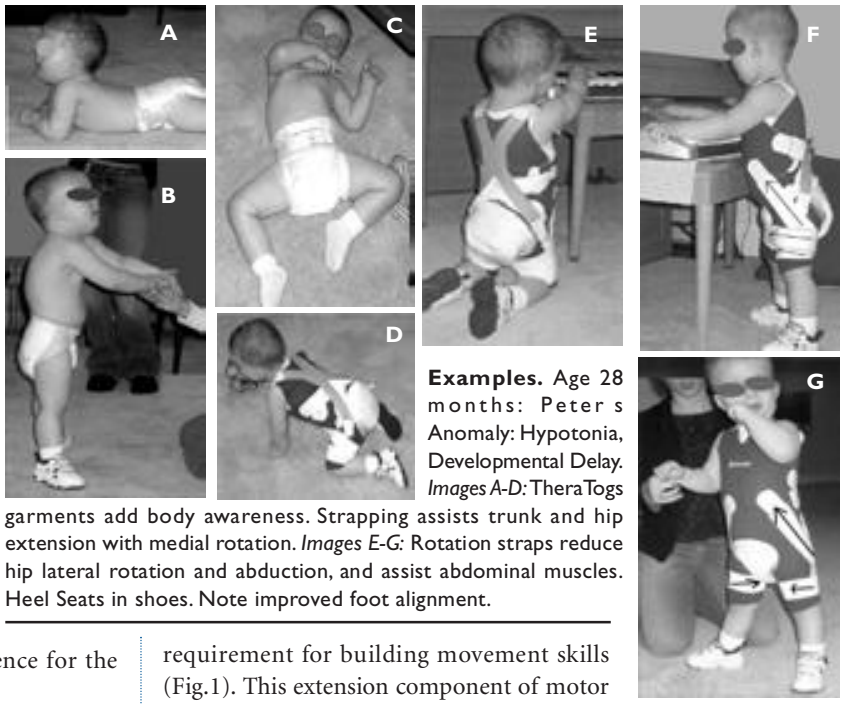

Examples. Age 28 months: Peter s Anomaly: Hypotonia, Developmental Delay. *Images A-D:* TheraTogs garments add body awareness. Strapping assists trunk and hip extension with medial rotation. *Images E-G:* Rotation straps reduce hip lateral rotation and abduction, and assist abdominal muscles. Heel Seats in shoes. Note improved foot alignment.

***Stiffness.** The increasing loss of soft tissue extensibility in children with spastic cerebral palsy reveals the elaboration and strengthening of connective tissue within and between the muscle fibers and cells in the presence of chronic excessive muscle recruitment. Proliferation of connective tissue has also been observed in and around immobilized muscle, particularly the tonic fibers. The proliferating connective tissue eventually binds the nearby nerves and blood vessels. In shortened state, all of these tissues, including the skin, adapt by losing length and extensibility. By physiologic adaptation, the tissues convert to serve the orthotic function of reliance on tonic muscle recruitment.[18,19]

Schleip et al (2006) hypothesize that intramuscular connective tissue, in particular the fascial layer known as the perimysium, is "capable of active contraction and consequently influence passive muscle stiffness, especially in tonic muscles. Evidence for the hypothesis is based on five indications: [20]

1) Tonic muscles contain more perimysium and are therefore stiffer than phasic muscles.
2) The specific collagen arrangement of the perimysium is designed to fit a load-bearing function.
3) Morphological considerations as well as histological observations in their laboratory suggest that the perimysium is characterized by a high density of myofibroblasts, a class of fibroblasts with smooth muscle-like contractile kinetics.
4) In vitro contraction tests with fascia have demonstrated that fascia, due to the presence of myofibroblasts, is able to actively contract, and that the resulting contraction forces may be strong enough to influence musculoskeletal dynamics.
5) The pronounced increase of the perimysium in muscle [after] immobilization…indicates that perimysial stiffness adapts to mechanical stimulation and hence influences passive muscle stiffness. In conclusion, the perimysium seems capable of … adapting passive muscle stiffness to increased tensional demands, especially in tonic musculature."

The early achievement of bilateral symmetrical trunk, neck, and hip extension strength and control against gravity is the primary requirement for building movement skills (Fig.1). This extension component of motor development is soon followed by symmetrical, antigravity, trunk and neck flexion (Fig.2).[21] These 'ingredients' combine to foster appropriate use of the extremities for movement and functional use by providing adequate trunk stability (Fig.3).

Without these fundamental elements, the child who desires to interact with his environment musters positional stability by relying on compensatory and relatively tonic recruitment of the more distal musculature (Fig.4). The result is the development of contractures (shortening) and stiffness (increased resistance to stretch) in the overused musculature.

I recommend the early use of taping and TheraTogs torso garments and strapping to facilitate the acquisition of these two basic ingredients—core extension and flexion—so that the limbs may be used for moving rather than for stabilizing (Figures 5 and 6). This intervention is particularly important during the first year of life, while the neuromotor system is undergoing primary maturation.[22,23]

In the next article, I'll address muscle tissue modeling and growth and the beneficial role of full-term gestation in flexed position in the skeletal modeling processes. ■

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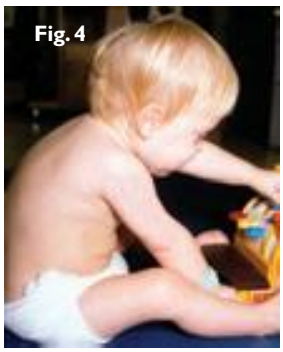

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REFERENCES

1. Frost HM. *Intermediary Organization of the Skeleton*. Volume I. Boca Raton, Fla: CRC Press; 1986.

2. Frost HM. *Intermediary Organization of the Skeleton*. Volume II. Boca Raton, Fla: CRC Press; 1986.

3. Soderberg GL. *Kinesiology: Application to Pathological Motion*. Baltimore Md: Williams & Wilkins. 1986.

4. Frankel VH, Nordin M. *Biomechanics of the Skeletal System*. Philadelphia, Pa: Lea & Febiger; 1980.

5. Brighton CT, Fisher JRS, Levine SE, et al. The biomechanical pathway mediating the proliferative response of bone cells to a mechanical stimulus. *J Bone Joint Surg.*[Am] 1996;78: 1337-1347.

6. Adolf, KE, Avolio AM, Barrett T, Mathur P, Murray A. Step counter: quantifying infant's everyday walking experience. *Infant Behavior & Development*. 1998; 21:43. Special Issue - International Conference on Infant Studies.

7. Frost HM. Skeletal structural adaptations to mechanical usage (SATMU): 4. Mechanical influences on intact fibrous tissues. *Anat Record*. 1990;226: 433-439.

8. Fung YC. Biomechanical aspects of growth and tissue engineering. In: *Biomechanics. Motion, Flow, Stress, and Growth.* New York, NY: Springer-Verlag; 1990; 499-545.

9. Wynne-Davies R. Acetabular dysplasia and familial joint laxity: two etiological factors in congenital dislocation of the hip: A review of 589 patients and their families. *J Bone Joint Surg.*[Br] 1970;52: 704716.

10. Gedalia A, Press J. Articular symptoms in hypermobile schoolchildren: a prospective study. *J Pediatr*. 1991; 944:946.

11. Carter C, Wilkinson J. Persistent joint laxity and congenital dislocation of the hip. *J Bone Joint Surg.*[Br] 1964;46: 40-45.

12. Gedalia A, Brewer EJ. Joint hypermobility in pediatric practice - a review. *J Rheumatol*. 1993;20: 371-374.

13. Jay RM, Schoenhaus HD, Seymour C, Gamble S. The Dynamic Stabilizing Inner Sole System (DSIS): The management of hyperpronation in children. *J Foot Ankle Surg.*1995;34: 124-131.

14. Bleck EE, Berzins UJ. Conservative management of pes valgus with plantarflexed talus, flexible. *Clin Orthop*. 1977; 122:85-94.

15. Aharonson Z, Arcan M, Steinbeck TV. Footground pressure pattern of flexible flatfoot in children, with and without correction of calcaneovalgus. *Clin. Orthop*. 1992; 278: 177182.

16. Bordelon RL. Correction of hypermobile flatfoot in children by molded insert. *Foot & Ankle Int'l.* 1980; 1: 143-150.

17. Jaffe M, Tirosh E, Cohen A, Taub Y. Joint mobility and motor development. *Arch Dis Child.* 1998; 63: 159-161.

18. Williams PE, Goldspink G. Connective tissue changes in surgically overloaded muscle. *Cell Tissue Res*. 1981; 221(2): 465-70.

19. Given JD, Dewald JP, Rymer WZ. Joint dependent passive stiffness in paretic and contralateral limbs of spastic patients with hemiparetic stroke. *J Neurol Neurosurg Psychiatry.* 1995; 59(3): 271-279.

20. Schleip R, Naylor IL, Ursu D, et al. Passive muscle stiffness may be influenced by active contractility of intramuscular connective tissue. *Med Hypotheses*. 2006; 66(1): 66-71.

21. Bly L. *Motor Skills Acquisition in the First Year: An Illustrated Guide to Normal Development.* San Antonio, Texas: Therapy Skill Builders; 1994.

22. Leonard, C.T., H. Hirschfeld, H. Forssberg H. The development of independent walking in children with cerebral palsy. *Develop Med Child Neurol*. 1991; 33:567-577.

23. Leonard, C.T. Motor behavior and neural changes following perinatal and adult-onset brain damage: implications for therapeutic interventions. *Phys Ther.*1994;74:753-767.