

CONFERENCE SPEAKERS PREVIEW

► **TISSUE ISSUES**

Pediatric Orthopedics Part II

SOFT TISSUE MODELING

By Beverly Cusick, PT, MS, COF/BOC

In Part 1 of this series, we reviewed the influences of normal, strain-inducing mechanical loads on developing bone geometry and joints. Here, we'll briefly examine the connective and muscle tissues in similar contexts, with a discussion of the relationship between the history of muscle recruitment and clinical evidence of muscle imbalances and soft tissue transformation. In Part 3 in a future issue of *Network*, I'll discuss the normal neonatal soft tissue constraints described here as they influence the skeletal and joint modeling process.

MUSCLE TISSUE MODELING AND GROWTH

Nutrition, hormones, electrical activity, and the application of mechanical forces—particularly tension—combine to regulate the configuration and size of skeletal muscle. Active and passive mechanical forces significantly influence muscle tissue morphology, though the operative physiologic mechanisms are poorly understood. Muscle growth rate is tightly coupled to bone growth rate.¹ The rapidly growing embryonic skeleton applies tension to the attached muscles. The tensile strain contributes to muscle fiber formation— *(continued on page 9)*

Acquiring Speech Motor Control

COORDINATING RESPIRATORY, LARYNGEAL, VELOPHARYNGEAL, AND ORAL-MOTOR SUBSYSTEMS IN CHILDREN WITH CP

By Carol A. Boliek, Ph.D., speaker at the upcoming NDTA Annual Conference, October 5-8, 2006.

Cerebral palsy (CP) is an “umbrella term” for a group of non-progressive, but often changing, motor impairment syndromes secondary to abnormalities of the brain arising in the early stages of its development.^{1 p.547} It is the most common movement disorder in children. Its prevalence is 2 to 2.5 cases per 1000 live births²⁻³ and is increasing due to better survival rates of very low birth weight infants. Spastic CP is the most common type. Spastic CP generally correlates with a lesion(s) in the motor cortex and its descending white matter tracts. Affected individuals exhibit a constellation of motor signs such as: (a) increased tone, (b) hyperactive reflexes, (c) discordant mass activation of muscles, (d) weakness, (e) decreased speed of movement, and (f) decreased endurance.⁴ Although motor signs are often discussed in relation to limb and body movements, these signs can negatively affect the speech of children with spastic CP.

Associated speech disorders include hypernasality, breathy voice quality, monotonous speech, reduced loudness, uncontrolled rate and rhythm of voice, disordered respiration, weak respiratory muscles, and disordered articulation.⁵⁻¹⁴ Children with spastic CP are at risk for deteriorating speech

as they mature.^{5,8} Speech deficits associated with CP have significant functional consequences including difficulties in academic advancement, social and emotional development, eventual independent living, and work force participation. There are limited reports on the efficacy of speech treatment for children with CP, so there is a great need

for research in this area.^{11,13} The neuromotor bases of speech and voice disorders, which may guide treatment delivery models in children with spastic CP, are not well understood.^{5,14,15} Physiological studies of children with spastic CP to test the plasticity of their sensorimotor systems are virtually non-existent.

Speech deficits associated with CP have significant functional consequences including difficulties in...social and emotional development...and eventual independent living.

Pharmacological (e.g., Botulinum toxin, Baclofen) and surgical (e.g., selective dorsal rhizotomy) interventions can alleviate some of the positive motor signs associated with spastic CP.^{4,16} Behavioural approaches have been used to treat motor and sensory function in children with spastic CP. Of these, neurodevelopmental treatment (NDT)¹⁷⁻²⁰ has been the predominant form of intervention. There is however, a growing body

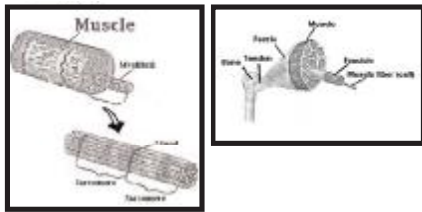
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I N S I D E T H E N E T W O R K :

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with increases in sarcomere (muscle cell) number, length, and cross-sectional width—and to muscle fascicle (fiber bundle) organization. Only Type I (tonic) muscle fibers are present in early gestation. By the end of gestation, Type I and II (phasic) muscle fibers are about equal in number.²



The postnatal, innervated and activated skeletal muscle endures a variety of patterns of normally-applied tensile (elongation) strains that regulate longitudinal and cross-sectional myofiber growth.^{3,4} Neonatal skeletal muscle contains less than 20% of the normal adult number of sarcomeres that align in series to form a muscle fiber. Prostaglandins might be the most significant of all physiological factors in the relationship of mechanical forces and growth as they influence both muscle and bone. Sarcomere length, size, and number are continually regulated by means of total protein (prostaglandin) synthesis and degradation.

The muscle growth center is at the musculotendinous junction. Muscle fiber differentiation is followed by muscle growth in cross-sectional area, as existing fibers hypertrophy, and by growth in length with the addition of sarcomeres.⁵ Muscle fiber properties become tailored to their history of activation, resulting in a relatively homogeneous population of fiber types within a normal motor unit.⁶

FIBROUS TISSUE MODELING

The main component of the fibrous tissue matrix is Type I collagen, which consists of tropocollagen molecules with fixed lengths, assembled in variable quantity and configurations that alter tissue length, weight, and tensile strength. As occurs in bone and muscle, predetermined growth in fibrous tissue length occurs at the ends of the tendons, ligaments, and fascia, rather than in the middle. Growth in length responds

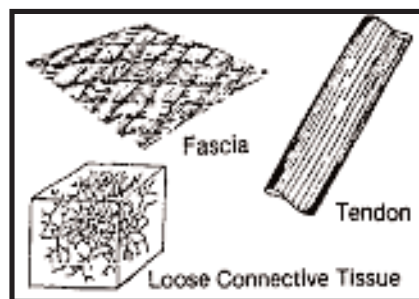
primarily to circulatory and systemic, rather than mechanical, factors. As fibrous tissue is made, existing tension loads and strains direct the organization of the collagen fibers to align parallel to the major loading vectors. They therefore model in response to mechanical usage.⁷

Fibrous tissues model into three kinds of structures⁷:

1. Fascial sheets, like cloth, provide tension, rigidity, and strength in various directions within the plane of the structure, and can transfer tensile loads (example: the intermuscular septae).

2. Ligaments or tendons confine 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 (examples: the iliotibial band and the plantar aponeurosis).

3. Loose, three-dimensional networks or mesh bind together the various cells and noncollagenous intercellular components of organs such as subcutaneous tissue (example: the perimysium surrounding muscle fibers)



The tropocollagen fibers are bound together by cross-links that 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.⁸

Infants show ligament laxity as intrauterine soft-tissue constraints resolve. In a British study of more than 3000 nondis-

abled children age 1 week to 18 years, 50% showed evidence of generalized joint laxity at age 3 years; 5% at age 6 years; and less than 1% at age 12 years⁹. Females showed a higher incidence of joint laxity than males at all ages. However, 2 other studies found that 10% to 12% of participating school-aged children showed evidence of generalized ligament laxity.^{10,11}

Clinical implications of fibrous tissue modeling deficits

Jaffe et al¹² identified joint hypermobility, with no evidence of other problems such as generalized developmental delay, in 126 of 715 infants, ages 8 to 14 months. Of the infants with hypermobility—particularly into hip abduction, elbow hyperextension, and ankle dorsiflexion (DF)—30.2% showed early motor delay. Of the 569 remaining subjects with normal joints, 10.9% showed motor delay. Six months later, 65% of the delayed hypermobility group, and 79.2% of the delayed infants in the other group had resolved their motor delays.

The reported prevalence of persistent or excessive ligament laxity in children of all ages warrants the clinician's attention. Joint hypermobility has been associated with pain, an increased incidence of ligament injuries in sporting activities, and premature osteoarthritis in adulthood.¹³ The attending physical therapist might suggest specific strengthening postures and activities, perhaps with the judicious use of taping, or elastic or orthotic joint support systems, to promote muscular stability at joints that exhibit ligament laxity.

Example: An infant with ligament laxity might routinely distribute her weight antero-medially on her feet, or exhibit predominantly static, rather than dynamic and variable, foot postures in standing positions. In such cases, the arch-supporting ligaments can be protected from enduring accelerated creep deformation with the use of appropriately designed and fitted heel stabilizing cups or shoe inserts^{14,15,16}, such as those from SureStep™ Dynamic Stabilizing System (www.surestep.net). (continued on page 10)

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THE LENGTH-TENSION RELATIONSHIP

Muscle can generate contractile force of varying magnitudes, depending upon:

- The joint angle
- The length of the moment arm connecting the joint axis and a line of muscle tension, intersecting perpendicular to the line of pull
- The cross-sectional area of the muscle fibers
- The overlap of contractile filaments within the sarcomeres
- The angular velocity occurring during the motion
- The existing coefficient of friction.
- The speed of a precontractile elongation

The active and passive length-tension relationships are functionally interrelated in the lower extremities. Resting muscle length (RML) occurs on the arc of passive muscle extensibility (the passive length-tension curve) at the length at which the muscle first exhibits resistance to stretch.¹⁷ This first encounter with resistance to rapid, passive elongation is described by various authors as “ L_1 ” and “ L_0 ”^{17,18,29} “ R_1 ”²⁰ “initial end range,”^{21,22} “ A_0 ”²³ “functional” or “spastic” end range,²³ “dynamic length,”²⁴ and, commonly, “first-catch” end range.

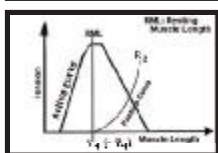
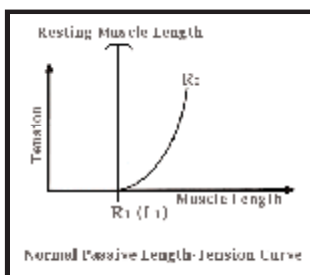


Figure 1. Normal passive length-tension curve.

Figure 2. Normal active and passive length tension curves.

For recording convenience and consistency, I use Maitland’s terms: R_1 end range describes this clinically evident indicator of RML, and R_2 end range indicates the maximum length both available and tolerable.²⁰

The passive length-tension relationship normally exhibits a relatively easy excursion

through the first half of applied elongation, revealing more gain in length than in tensile resistance, and appears as a low slope on the length-tension curve (Figure 1). The last half of the range of available extensibility demonstrates increasing resistance to passive elongation compared with a diminishing gain in muscle length, and appears as a steeper slope on the curve.

The relationship between joint angle, existing muscle length, and the capacity to generate isometric contractile force, is described as the active length-tension relationship (Figure 2). The active length-tension curve is bell shaped. Resting muscle length (RML) describes the joint angle and muscle length at which a muscle or muscle group can generate maximum isometric contractile force, and is evident as the peak of the active length-tension curve. At RML, actin and myosin filaments are overlapped for optimum tensile force generation.^{17,21}

Neonates exhibit “physiologic flexion”—a resistance to passive extension—revealing the presence of adaptive shortening and elevated muscle tone (resistance to passive stretch) in the shortened musculature and associated soft tissues located on the concave side of all flexed joints.²⁵

As the neonate lacks volitional movement, the force generation potential of physiologic flexion is unknown, though one might consider that there is some association with survival by clinging (in flexion) to a tree-climbing, branch-leaping mother.

Physiologic flexion resolves most notably—though not entirely—in the first two postnatal months. Nondisabled children between ages 1 and 3 years usually exhibit no discernable R_1 end range during tests of muscle extensibility. Physiologic adaptation to the typical history of lower-extremity muscle use appears to set the R_1 end range on the slope of passive extensibility, and is a developmental characteristic that generally appears in hamstring and calf musculature at about age four years. The length of muscle at which R_1 occurs gradually diminishes, and the magnitude of resistance encountered at R_1 end range increases with growth in the context of appropriate physical activity. Children who are highly active show higher

magnitudes of resistance at R_1 end range than their less active peers.

A developmental reduction in extensibility between R_1 and R_2 end ranges also occurs normally in response to the history of muscle use. The normal ankle dorsiflexes to about 30 degrees in children less than age four years who are positioned comfortably in prone with the knee extended to 0 degrees and the foot joints aligned in congruity. This R_2 end range diminishes to a more common 10 degrees in adults.

Clinical implications of use-related development of soft-tissue stiffness

The RML is the strongest point in the range of available muscle length for generating isotonic force, and corresponds with a discernable clinical finding of resistance to lengthening applied quickly, lightly, and passively (R_1 end range). Therefore, R_1 end

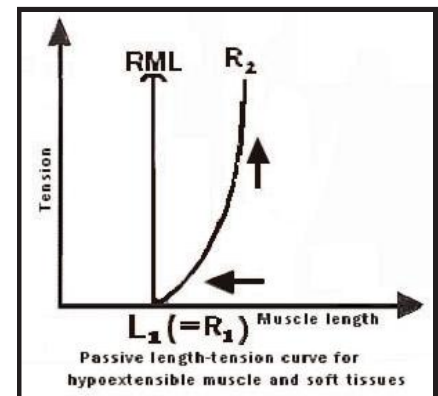


Figure 3. Passive Length-Tension Curve for hypoe extensible muscle and soft tissues.

range is a meaningful and measurable clinical observation.²⁴

Tardieu et al (1987) suggest that R_1 end range, in children with CP and shortened soft tissues, indicates the presence of connective tissue elaboration and strengthening, and that the growth-potential, length, and health of the muscle fibers are evident in the extensibility to maximum end range. Based on reports of animal studies of in vivo cast immobilization effects, these authors hypothesized that an observed gain in R_1 extensibility represents increased connective tissue and/or tendon length, and that an increase in the excursion between R_1 and R_2 end range implies that the (continued on page 11)

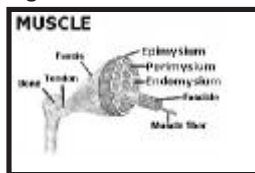
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sarcomeres were added in series to the muscle fiber. Furthermore, a clinical observation of a lack of excursion past R₁ end range indicated a dysfunctional trophic capacity and a poor prognosis for a lasting effect of conservative soft-tissue lengthening measures, such as casting, stretch splinting, or positioning.

EFFECTS OF EXCESSIVE RECRUITMENT AND IMMOBILIZATION IN SHORTENED STATE

The adaptive changes in structural configuration at the cellular level of muscle and soft tissues with extraordinary functional and length demands are a relatively recent observation. These transformations that increase soft tissue stiffness are often misconstrued as “spasticity”. The presence of spasticity can increase the magnitude of stiffness, but stiffness is not evidence of—and can develop without—spasticity.^{26,27,28}

Figure 4.



extremity muscle fascicles^{29,30} and sophisticated histological examinations of biopsied tissues of children with and without cerebral palsy.^{31,32,33} Researchers using these technologies have led the medical community to reconsider accepted theories—drawn from small mammal studies—regarding the length-changing behavior of human muscle and connective tissues. For example, we now know that serial sarcomere number adaptation to imposed changes in muscle length and activation appears to occur in small mammal muscle, and not in human muscle unless the tendon has been surgically lengthened.³⁰ Sarcomeres lose extensibility at shorter lengths.^{30,34} Collagen proliferation,

Since 2001, we’ve seen the emergence of in vivo ultrasound examination of lower

primarily in the perimysium, remains a major human transformation trait (Figure 4).³⁵

Peripheral nerves can become entrapped, and blood vessels, nerves, and skin adapt to the shortened state of the transformed muscle.^{36,37} Gracies (2005a and b) reviews the literature describing the patho-physiology of the cycle of spastic paresis leading to disuse leading to paresis following UMN lesion and chronic, excessive muscle activity vs. immobilization. The scope and detail of this two-part review article exceed the scope of this work. Readers of this essential resource might keep in mind the current evidence suggesting that the small mammalian striated sarcomeres do not appear to behave like comparable human muscle. I interpret the common, age-related increase in stiffness between R₁ and R₂ end ranges in individuals with CP that features either excessive, tonic-type muscle recruitment, with or without spasticity, as evidence (continued on page 12)

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of progression of the process of pathologic soft tissue and muscle transformation.

Clinical implication: Herbert long ago differentiated between the capacity of muscle and soft tissues to exhibit increased extensibility and ROM by way of transitory viscous deformation—a common event in the context of a therapeutic exercise session that reveals the transient, elastic properties of the tissues—and the conditions that are required for true physiologic adaptation to occur in the same tissues.¹⁹ The effects of prolonged, low-load (gentle) elongation on animal striated muscle are well-documented.³⁸ Clinical observations of improved extensibility following routine positioning and serial casting interventions support the premise that long, gentle stretch over time fosters lengthening physiologic adaptation. Though we have learned that sarcomere number loss occurs in calf muscles in children with CP who have undergone surgical tendon lengthening, we have yet to learn the characteristics of lengthening changes at the cellular level in human muscle after casting and positioning.³⁰

MUSCLE BALANCE THEORY AND THE DEVELOPING ACTIVE LENGTH-TENSION RELATIONSHIP

Sahrmann (2002) describes in detail the physiologic and kinesiologic effects of muscle recruitment strategies on muscle and soft-tissue extensibility, though she does not address the pediatric developmental aspects of this process.³⁹ However, Bly (1994) has elegantly and (I expect inadvertently) illustrated Sahrmann’s principles as they apply to early neuromotor development. For this reason, I refer the reader to both sources as essential reading, and present a brief overview of Sahrmann’s principles here. The relationship between Sahrmann’s and Bly’s principles will be made evident in the next article in this series.

Sahrmann’s fundamental observations and hypotheses pertaining to muscle balance and muscle imbalance are these:

- Resting postures influence muscle recruitment strategies—for better or worse.

- All muscles operate in force couples.
- Force couple balance maintains kinesiologically correct joint alignment and longevity.
- Force couple imbalances feature dominant (over-recruited) and dominated (under-recruited) muscles.



Figure 5

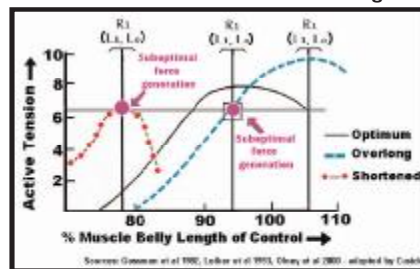


Figure 6

Sahrmann observed that dominant muscles in a force couple:

- Lose extensibility over time, and appear shortened or hyperactive in a passive ROM assessment
- Are recruited first in the activation sequence—which is also true of Type-1 (tonic) muscle fibers
- *Reveal the significance of the role of that muscle or group in the existing functional recruitment strategy. Shortened or “irritable” muscles (pre-shortened) tell you that they are used a great deal.*
- Lead the clinician to identify the under-recruited, dominated muscles by postural observation (underused muscles permit postural convexities), and by deduction (i.e. if the rectus femoris is dominant, the quadriceps and iliopsoas are evidently dominated; if the triceps surae are dominant at the ankle, the anterior tibialis and long toe extensors are dominated).
- Lead the clinician to identify relative flexibility site(s), where the person draws compensatory motion. Examples: The dominant rectus femoris imposes mobility demands on the lumbosacral joint and the patella (Figure 5). Or the dominant triceps

surae restricts ankle dorsiflexion, imposing mobility demands on various neighboring joints, including the midtarsal joint, the metatarsophalangeal joints, the knee joint, and the first ray.

- Force couple imbalances induce joint malalignment at the site at which they operate, and eventually, microtrauma and pain at the primary site and/or at relatively flexible site(s).
- Force couple imbalances produce length-tension alterations revealing *weakness in both the dominant and the dominated muscle* when compared with the normal-length muscle (Figure 6).⁴⁰ Shortened (dominant) muscle – though it activates first in the recruitment sequence, generates less maximum force than normal-length muscle. Overlong (dominated) muscle generates more force at its RML, but less than normal maximum force at the kinesiologically correct length.

Implication: Strengthening both short (weak) and long (weak) musculature is an essential component of therapeutic management and of lifelong body care.

Muscle imbalances can develop in the presence of:

- Disturbances in muscle recruitment due to CNS dysfunction, e.g. synergistic action, mass action, coactivation, and altered activation timing.
- Pathomechanical resting postures, e.g. reclined in an infant seat, or sitting with a rounded spine.
- Inappropriate or excessive distances between load-bearing joints and the vertical body weight load lines in sagittal (Figure 7) and/or frontal (Figure 8) planes.
- Inappropriate distribution of the body center of mass (COM) over the feet, e.g. anterior displacement of the torso, results in chronic and excessive recruitment of the dorsal musculature in order to remain upright. This is a *key concern* for children for muscle and soft-tissue status and function in children with diplegic CP.

Sahrmann’s Principles for Managing Muscle Imbalance

- Optimize resting postural alignment;

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Figure 7

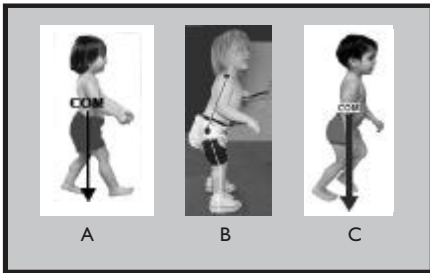


Figure 8

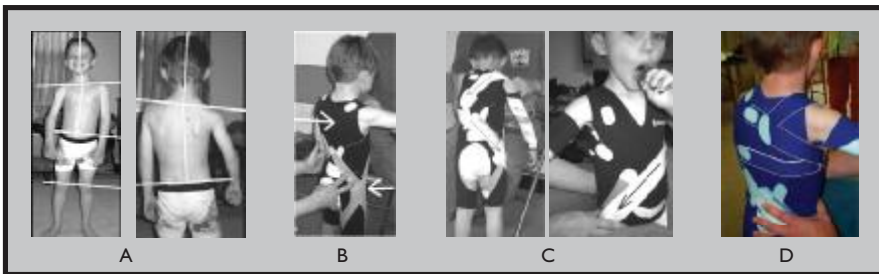


Fig 7. A: 2 year-old nondisabled girl

Fig 7. B: 2 year-old girl with diplegia: excessive distance between joints and vertical load line

Fig 7. C: 4 year-old boy with diplegia: anterior CoM placement.

Fig 8. 7 year-old boy with diplegia; frontal plane CoM placement shows lack of lateral shift, with compensatory lateral lurching.



A. 4-year-old with right Obstetric Brachial Plexus Injury, post 3 surgeries shows many postural compensations that are seen in children with hemiplegic CP. **B.** Pelvis strapped to reduce deviation to right (shortening the long abductors). **C.** Thorax strapped to reduce left upper trunk deviation, restoring alignment over pelvis. Right arm strapped for shoulder retraction, lateral rotation, and forearm supination. **D.** Scapular stabilization straps assist with the plan to gain scapulo-humeral disassociation.

improve postural alignment and use of the base of support.

- Address relative flexibility sites.
- Work toward optimum muscle length.
- Give a more effective strategy before you remove an existing one.
- Shorten long muscles *before* you lengthen short muscles.
- Work toward optimum gravitational force acting on limb segments.
- Integrate *optimum performance during functional activities* into the therapy program.
- Identify functional strategies, and *change them gradually*.

Most of these principles are inherent in the current Neuro-Developmental Treatment approach. Sahrman's principles suggest that the patient is able to selectively operate and control his/her musculature. I advocate adapting Sahrman's principles to the population of children and adults with CNS dysfunction to promote live-in practice in functional circumstances by using appropriate posting on orthotic devices, adapted furniture, positioning programs, Kinesio-Taping or orthopedic taping, and TheraTogs™

strapping systems as training and assistive aids where selective motor control is deficient. TheraTogs strapping applications collectively address all of Sahrman's principles when used for improving standing and sitting posture, for increasing body awareness, for facilitating targeted movements, and for enhancing postural and functional performance during and between treatment sessions. The primary operative principle of TheraTogs strapping systems is that they shorten the long muscle—providing sensory awareness of the underlying muscles, and of the improved position—to provide more effective stability and movement strategies before attempting to reduce existing, deformity-producing strategies.

The following images of a TheraTogs strapping system for a child with right Obstetric Brachial Plexus Injury demonstrates these principles. The same approach applies to children with hemiplegic CP. The Tank Top and Pelvic Anchor provide Velcro® “real estate” for attaching elasticized strapping that mimics the underlying musculature.

NEXT TIME IN THIS SERIES

The normal length-tension relationship

evolves with the developmental progression of voluntary, antigavity muscle recruitment over time. The neonatal flexion constraints and kyphotic spinal configuration present an original resting posture that directs the course of development, resulting in optimum and long-lasting upright function against gravity. In Part 3 of this series, we'll begin to examine the relationship between neonatal postural and soft tissue constraints and the skeletal modeling process. ■

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REFERENCES

- ¹ Vandenburgh HH, Hatfaludy S, Karlisch P, Shansky J. Mechanically induced alterations in cultured skeletal muscle growth. *J Biomech.* 1991; 24:91-99. Supplement.
- ² Mastalgia FL. Growth and development of skeletal muscle. In: Davis JA, Dobbing J, eds. *Scientific Foundations of Paediatrics.* Baltimore, Md: University Park Press; 1981: 590-620.
- ³ Williams PE, Goldspink G. Longitudinal growth of striated muscle fibres. *J Cellular Sci.* 1971;9: 751-767.
- ⁴ Goldberg AL, Etlinger JD, Goldspink DF, Jablecki C. Mechanism of work-induced hypertrophy of skeletal muscle. *Med Sci Sports Exerc.* 1975;7:248-261.
- ⁵ Ziv I, Blackburn N, Rang M, Koreska J. Muscle growth in normal and spastic mice. *Dev Med Child Neurol.* 1984 Feb;26(1):94-9.
- ⁶ Lieber R. Skeletal muscle adaptability. I. Review of basic properties. *Develop Med Child Neurol.* 1986;28:390-397.
- 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.*

(Pediatric Orthopedics Part II continued from page 13)

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: 704-716.
- ¹⁰ Gedalia A, Press J. Articular symptoms in hypermobile schoolchildren: a prospective study. *J Pediatr.* 1991; 94:494-6.
- ¹¹ Carter C, Wilkinson J. Persistent joint laxity and congenital dislocation of the hip. *J Bone Joint Surg.[Br]* 1964; 46: 40-45.
- ¹² Jaffe M, Tirosh E, Cohen A, Taub Y. Joint mobility and motor development. *Arch Dis Child.* 1998;63:159-161.
- ¹³ Gedalia A, Brewer EJ. Joint hypermobility in pediatric practice - a review. *J Rheumatol.* 1993;20: 371-374.
- 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. Foot-ground pressure pattern of flexible flatfoot in children, with and without correction of calcaneovalgus. *Clin. Orthop.* 1992; 278: 177-182.
- ¹⁶ Bordelon RL. Correction of hypermobile flatfoot in children by molded insert. *Foot & Ankle Int'l.* 1980;1:143-150.
- ¹⁷ Lieber R, Bodine-Fowler SC. Skeletal muscle mechanics: Implications for rehabilitation. *Phys Ther.* 1993; 73: 844-856.
- ¹⁸ Gajdosik CG, Gajdosik RL. Musculoskeletal development and adaptation. In: Campbell SK, ed. *Physical Therapy for Children*. Philadelphia, Pa: WB Saunders Company; 1994: 105-126.
- ¹⁹ Herbert R. The passive mechanical properties of muscle and their adaptations to altered patterns of use. *Austral Physiother J.* 1988; 34: 141-149.
- ²⁰ Maitland GD. *Peripheral Manipulation*. Second Edition. Boston, Mass: Butterworths; 1977; 348-353.
- ²¹ Gajdosik RL. Passive compliance and length of clinically short hamstring muscles of healthy men. *Clin Biomech.* 1991 ;6: 239-244.
- ²² Tardieu G, Tardieu C. Cerebral palsy: Mechanical evaluation and conservative correction of limb joint contractures. *Clin Orthop.* 1987; 219: 63-70.
- ²³ Reimers J. Contracture of the hamstrings in spastic cerebral palsy: A study of three methods of operative correction. *J Bone Joint Surg.[Br]* 1974; 56: 102-109.
- ²⁴ Reimers J. Clinically based decision making for surgery. In: Sussman MD, ed. *The Diplegic Child - Evaluation and Management*. Rosemont, IL: American Academy of Orthopedic Surgeons; 1992; 151-161.
- ²⁵ Bly L. *Motor Skills Acquisition in the First Year: An Illustrated Guide to Normal Development*. San Antonio, Texas: Therapy Skill Builders/Psychological Corporation; 1994.
- ²⁶ Ada L, Vattanaslip W, O'Dwyer NJ, et al. 1998. Does spasticity contribute to walking dysfunction after stroke? *J Neurol Neurosurg Psychiatr.* 64: 628-635.
- ²⁷ Crenna P. 1998. Spasticity and 'spastic' gait in children with cerebral palsy. *Neurosci Biobehav Rev.* 22(4): 571-578.
- ²⁸ Booth CM, Cortina-Borja MJ, Theologis TN. 2001. Collagen accumulation in muscles of children with cerebral palsy and correlation with severity of spasticity. *Dev Med Child Neurol.* 43(5): 314-320.
- ²⁹ Shortland AP, Harris CA, Gough M, Robinson RO. 2002. Architecture of the medial gastrocnemius in children with spastic diplegia. *Dev Med Child Neurol.* 44(3): 158-63.
- ³⁰ Shortland AP, Fry NR, Eve LC, Gough M. 2004. Changes to medial gastrocnemius architecture after surgical intervention in spastic diplegia. *Dev Med Child Neurol.* 46(10): 667-673.
- ³¹ Fry NR, Childs CR, Eve LC, Gough M, Robinson RO, Shortland AP. 2003. Accurate measurement of muscle belly length in the motion analysis laboratory: potential for the assessment of contracture. *Gait Posture.* 17(2): 119-124.
- ³² Lieber RL, Steinman S, Barash IA, Chambers H. 2004. Structural and functional changes in spastic skeletal muscle. *Muscle Nerve.* 29(5): 615-627.
- ³³ Foran JR, Steinman S, Barash I, Chambers HG, Lieber RL. 2005. Structural and mechanical alterations in spastic skeletal muscle. *Dev Med Child Neurol.* 47(10): 713-717.
- ³⁴ Friden J, Lieber RL. 2003. Spastic muscle cells are shorter and stiffer than normal cells. *Muscle Nerve.* 27(2): 157-164. Comment in: *Muscle Nerve.* 2003 27(2): 131-132.
- ³⁵ Gracies J-M. 2005a and b. Pathophysiology of spastic paresis. I and II. *Muscle Nerve* 31: 535-571.
- ³⁶ Castle ME, Reyman TA, Schneider M. 1979. Pathology of spastic muscle in cerebral palsy. *Clin Orthop Relat Res.* 142: 223-232
- ³⁷ Frascarelli M, Frascarelli F, Gentile MG, et al. 2005. Entrapment neuropathy in patients with spastic cerebral palsy. *Acta Neurol Scand.* 112(3): 178-182.
- ³⁸ O'Dwyer NJ, Neilson PD, Nash J. 1989. Mechanisms of muscle growth related to muscle contracture in cerebral palsy. *Dev Med Child Neurol.* 31:543-552.
- ³⁹ Sahrman SA. 2002. *Diagnosis and Treatment of Movement Impairment Syndromes*. St. Louis, MO: Mosby.
- ⁴⁰ Wiley ME, Damiano DL. 1998. Lower-extremity strength profiles in spastic cerebral palsy. *Dev Med Child Neurol.* 40(2): 100-107.