

The Neurological Control System for Normal Gait

*Robert S. Lin, C.P.O.
James R. Gage, M.D.*

Introduction

Cerebral palsy is a fairly common neuromuscular disability, and orthotists are frequently called upon to provide orthoses for clients with this condition. If we wish to logically approach the treatment of their ambulation problems, it is necessary to have an understanding of the abnormalities in locomotion which cerebral palsy imposes. However, any such knowledge of gait pathology must be built upon an understanding of the normal control mechanism for human gait. Although the mechanisms of cerebral control of gait are not fully understood, sufficient information is known to at least provide a framework for a treatment approach.

We know that the neurological control of normal gait is extremely complex. Even in lower organisms such as lampreys, it has been demonstrated that "central pattern generators," i.e., neurons which control specific activities via clusters of other neurons, exist in a sort of hierarchical fashion all the way down to the spinal cord level.

The Proprioceptive System

In the human, a sophisticated control system exists which features a complex blend of voluntary and automatic control. In order for this system to function, the brain must have rapid, precise information regarding the position of the body and each limb in space, the length and rate of contraction of each muscle and the tension which each muscle is applying at its point of insertion. The sensory inputs to the control system are the muscle spindles, the golgi tendon organs, and the sensory receptors in joints, subcutaneous tissue, and skin.

The muscle spindle and golgi tendon are the sensory organs responsible for monitoring muscle tone. The muscle spindle is an intrafusal (encapsulated) muscle fiber which occurs in varying numbers in all skeletal muscles. They are particularly numerous in muscles which require fine control such as the intrinsic muscles of the hand. The spindles are attached to the connective tissue septae which also anchor the extrafusal muscle fibers. Hence, they are connected in parallel with the muscle fibers which produce the contraction. As such, the spindle fiber would rapidly lose tension during a muscle contraction if it were not under some sort of central control.

Muscles are innervated by three types of motoneurons: alpha, beta, and gamma. The former innervates only the extrafusal muscle fibers, whereas the latter innervates only the muscle spindles. The gamma afferents set the tension of the spindle cell and, therefore, are felt to control the sensitivity of the spindle receptors. Although most of the descending motor pathways influence the activity of the gamma motoneurons to some degree, the reticular system, cerebellum, and basal ganglia exert particularly strong control. Two types of gamma motoneurons exist:

1. the dynamic gamma motoneurons which affect the afferent response to phasic stretch more than static stretch; and
2. static gamma motoneurons which sensitize the spindle afferents to static stretch only.

Two different types of afferent fibers return to the spinal cord from the spindle:

1. the 1A afferents establish direct monosynaptic connections with alpha motor-neurons innervating the same (homonymous) muscle as well as connections with synergistic (heteronymous) muscles; and
2. group II afferents connect only to the homonymous muscle.

The sensory function of the afferents coming from the spindles is to inform the control system of the length and rate of change in length of the extrafusal fibers.²

The golgi tendon organs are encapsulated organs which are attached in series to the collagenous fibers of tendons at the point of muscle insertions and also exist in the fascial coverings of muscles. IB afferents coming from the golgi tendon organs to the spinal cord are stimulated by muscle tension and selectively inhibit the alpha motoneurons of the agonist muscles while facilitating those of the antagonist muscles.

One of the features of cerebral palsy is abnormal muscle tone. Since the muscle spindles and golgi tendon organs are the ultimate sensory organs which control this tone, there has been a great deal of interest in them. It should be pointed out that it

is the central control of the spindles and golgi tendon organs which is abnormal in cerebral palsy, and not the end organs themselves. However, in the treatment of cerebral palsy, tone alteration has been attempted in various ways including orthotics, physical therapy, drugs, transcutaneous electrical stimulation, cerebellar stimulators, and partial dorsal rhizotomies.

An Overview of the Control System

Looking at the system of control from the bottom up, the simplest of the reflexes is the monosynaptic reflex, which occurs at a single level of the spinal cord, and may involve as few as two neurons, one afferent and one efferent. Hence, tapping the patellar tendon with a reflex hammer stretches the muscle spindles causing a stimulation of the A afferents which, in turn, monosynaptically stimulate the alpha motoneurons supplying the quadriceps muscle. The result is a quick, unsustained contraction of the quadriceps. However, in almost every case, the reflex is more complex. In the example above, the 1A afferent would also polysynaptically inhibit the antagonist muscles (hamstrings) and as such, the reflex would probably involve multiple spinal cord levels.

The next higher level of organization is the "mass limb" reflex which occurs via a multi-segmental interaction of the cord, for example, a withdrawal reflex secondary to a painful stimulus. This consists of flexion of the ipsilateral limb and extension of the contra-lateral limb. If the painful stimulus was to the bottom of the foot, the result would be a withdrawal of the stimulated extremity due to polysynaptic facilitation of alpha motor-neurons innervating the flexors, and concomitant inhibition of the motoneurons innervating the extensor muscles of the same leg. On the contralateral side the intersegmental reflex would produce extension of the opposite limb to support the body.

Continuing upward in the system of motor control, erect postural tone resides at the level of the brain stem and vestibular system. Primitive walking reflexes occur at the midbrain and subthalamic levels.

Finally, selective motor control, which is a prerequisite to normal gait, is controlled at the level of the cerebrum. Selective control allows modification of reflex patterns so that one joint or even one muscle can be moved independently of the normal reflex or habitual pattern. During walking this allows muscles at the hip, knee, and/or ankle to move independently of the subcortical patterns in response to variations of the ground reaction and/or inertial forces.

Perry describes five types of muscle control which allow us to move normally (Table 1) ^{3,4}

CLINICAL SIGN	STIMULUS	NEUROLOGICAL LEVEL
Spasticity (rigidity)	Quick Stretch (slow stretch)	Reflex arc from muscle spindle
Mass limb reflex extension flexion	Hip and knee extension (or flexion)	Spinal cord multisegmental interaction
Erect postural tone	Trunk upright	Vestibular system in brain system
Primitive locomotor patterns	Desire to stand	Midbrain/subthalamus area or step
Selective control	Normal volition	Cerebrum

Table 1. Neurological levels of control.

The more elementary control centers in the spinal cord, brainstem, midbrain and subthalamus areas allow the voluntary control system to respond with speed and efficiency to the functional demands placed upon it during normal locomotion. Thus, these five centers exist at different subcortical and/or spinal cord levels. The first two are forms of hyperreflexia--quick stretch and slow stretch.

Quick stretch is basically the uninhibited reflex arc which appears as clonus in the spastic patient. Slow stretch is a sustained contraction that would be best described as rigidity. This reflex does not appear in the absence of cortical injury which removes the normal inhibition. It is this reflex in the patient with cerebral palsy which makes it impossible to differentiate muscle spasticity from fixed contracture in the non-anesthetized patient.

The third and fourth primitive reflexes relate muscle tone to posture. They are limb position in space and body position in space. Extension of the hip and knee produce gluteus maximus, hamstring, quadriceps, and ankle plantarflexor activity, whereas flexion of the hip and knee produces activity of the hip and knee flexors and the ankle dorsiflexors. The position of the body in space influences postural tone of the extremities and trunk via the vestibular system which will be discussed in more

detail later.

The fifth type of primitive muscle control is the midbrain, extra-pyramidal, locomotor center. This center allows the patient to use primitive reflex patterns voluntarily for ambulation in the absence of selective motor control. Thus a hemiplegic stroke patient who has lost selective cortical control will frequently walk by reciprocating the lower limb mass flexion and mass extension reflex patterns described earlier. Perry points out that all patients with upper motor neuron lesions are subject to varying mixtures of these five primitive control mechanisms.⁵

In the brain, motor activity is controlled by the interactions of three major regions: the cerebral cortex, the cerebellum, and the basal ganglia. These regions influence the lower motoneurons either directly through the pyramidal system or indirectly via the extra-pyramidal system. The pyramidal system consists of the primary motor cortex in the precentral gyrus (Brodmann's area 4); the premotor cortex (Brodmann's area 6) in the frontal lobe, the premotor cortex (Brodmann's areas 1, 2 and 3) in the parietal lobe; and the corticobulbar and corticospinal pathways. The extra-pyramidal system encompasses all other projection pathways that influence motor control. As such, the extrapyramidal system includes the basal ganglia and all projection pathways from the brain system to the spinal cord and thus can be thought of as including the cerebellum as well. Therefore, we can divide the brain's system for motor control into two major areas: the pyramidal or "selective control system," and the extra-pyramidal which might be considered to be the "habitual" or "automatic control system." As observed by Mearns, all cerebral palsy can be classified as either spastic, athetoid, or mixed.⁶ Spasticity and athetosis are very different in character, and each reflects a defect in one of these major control systems. Spasticity results when the pyramidal system is injured and athetosis results with injury to the extra-pyramidal system.

Travis and Woolsey demonstrated that when normal monkeys were taught a particular task, they could still perform that task, although somewhat crudely, after ablation of the pyramidal system. However, if the pyramidal system was injured prior to the teaching of a task, the animals could not learn it. From this work, it appears that these two systems of the brain control motor activity through a complex integration of manual and automatic control. The extra-pyramidal system contains the reflexes and integrated reactions which are inborn. However, additional integrated activities can be programmed into it. Thus, the extra-pyramidal system would appear to be the seat of automatic control.⁷

The pyramidal system would also appear to be the locus of the manual control system. Here all voluntary activities are carried out. If a pattern of voluntary movement is required, it can be learned via the pyramidal system and then transferred as a "programmed activity" to the extra-pyramidal system. Kottke refers to these learned motor activities as "engrams."⁸ Thus, the infant in his playpen who is continually examining objects with his hands learns by repetitive action to transfer control of the elbow and shoulder joints to the extra-pyramidal system. In that way, he can concentrate only on what he is doing with his hand. In a similar manner a child learns to walk, ride a bicycle, and play the piano. Once the activity is under the control of the extra-pyramidal system, the pyramidal system can busy itself in "fine-tuning" the activity. It would appear that the pyramidal system can exert different intensities of control on the extra-pyramidal system. For example, when a boy is walking in quiet surroundings on level terrain, his mind can be elsewhere, perhaps he is thinking about a girl. When he is playing football, and trying to evade opponents, his mind, and particularly his pyramidal system, must give its full attention to the task at hand.

Kottke defines a motor engram as a "pathway of interneuronal linkages involving activation of certain neurons and muscles to perform a pattern of motor activity in a specific sequence of speed, strength, and motion, and at the same time inhibition of other neuron pathways so that muscles which should not be participating in this pattern remain quiet." He states that if the practiced activity has been precise, the engram will be precise, i.e., "practice doesn't make perfect," rather, when it comes to motor engrams, perfect practice makes perfect.⁹

Kottke goes on to describe the central nervous system organization as a reflex system with neuronal activation resulting as a response to external stimuli. The "voluntary system of control" is regulated by maintaining the system at a state of excitation which is just below threshold. Voluntary activation adds just enough impulses to change the excitation from the sub-threshold to the suprathreshold level in order to produce a motoneuron discharge. The only part of this system over which we have direct control is a limited-channel excitatory pathway which is the pyramidal tract from the motor cortex. Therefore, neuromuscular function is based on reflexes, and cerebral modifications of motor function are integrated through the basal ganglia and brain stem ganglia to produce multi-muscular coordination by selective patterns of excitation and inhibition of supraspinal and spinal reflexes. It follows, therefore, that damage to the highest or intermediate centers would cause abnormalities in performance by releasing the activity of the undamaged next lower center from control, rather than by generating a new form of activity originating from the damaged center. Kottke states that "the human infant appears to capture control of reflexes by learning first to facilitate the reflex activity via the pyramidal excitatory mechanism. At first this activity is crude, random, and essentially ineffective. As the association between voluntary excitation and reflex excitation is repeated, the infant becomes aware of the response. The voluntary excitation becomes increasingly effective until eventually the child can produce a desired muscular activity without manifest reflex participation"^{10,11}

If the pyramidal system can facilitate only one activity at a time and can change tasks no more than three times per second, it will obviously not allow the performance of complex coordinated activities. On the other hand, the extra-pyramidal system is a multiple channel system with a much faster response rate. Engrams are learned by repetitions. Kottke states, on the basis of

the work of himself and others, that thousands of repetitions are required to start to form an engram and millions of repetitions are required to perfect it. However, after the coordination engrams have been developed, volition becomes merely the need to excite the engram, to maintain it as long as it is desired, and to discontinue it when the activity is completed. With practice, simple engrams become linked or chained together to produce more complex actions. Optimal training, therefore, would mandate repetitive activities while avoiding incorrect performance.¹²

The modern computer is somewhat analogous to this situation. It consists of a central processing unit (CPU) for processing information plus a disk drive for information storage. The disk, which is placed in the drive, may contain preprogrammed information as well as provide a repository for new information. In the brain's system of movement control, the pyramidal system would be analogous to the CPU and the extra-pyramidal system to the disk drive. The primitive reflexes, which are present from birth, would be analogous to preprogrammed information on the disk (Figure 1) .

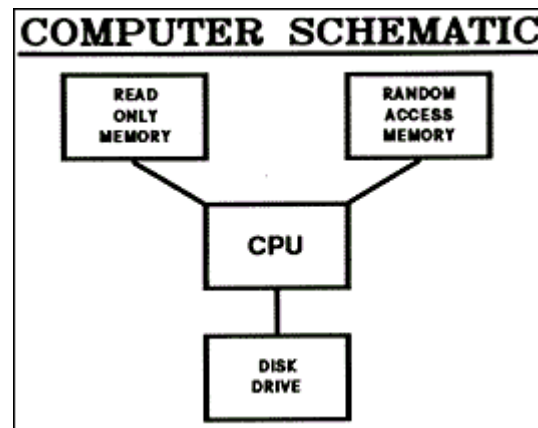


Figure 1. A simplified block diagram of a computer. The CPU is the central processing unit. It processes data which it receives from other areas and then sends it on. This could be likened to the primary motor cortex, which is the center for selective or voluntary control of locomotion. ROM stands for "read only memory." In the computer these are dedicated chips which have been permanently programmed to perform a particular function. For example, when you turn on your calculator and divide 72 by eight, the method by which division is performed is stored in a ROM chip. Our primitive reflexes which provide some of the basic building blocks for walking are somewhat analogous to ROM chips in that they are "preprogrammed" activities. RAM chips are "random access memory." This is where short-term memory is stored and it is instantly lost if the machine is turned off. Each time we perform a new activity it is stored in our short-term memory. However, if the activity is repeated enough times it eventually becomes habitual, i.e., stored in our extrapyramidal system. Thus, the extra-pyramidal system might be likened to the disk drive of the computer since both participate in long-term storage of information.

In a child with a purely athetoid or extra-pyramidal injury, the voluntary control system would be normal, but the automatic control center would be damaged. In this instance, manual activities could theoretically be carried out, but patterns of activity could not be correctly stored. This would cause the child severe motor disability since even a single activity such as picking up a spoon contains a great deal of automatic control. In the lower extremities, walking is carried out almost entirely through patterned movements. In the upper extremity, the fingers may be largely under voluntary (pyramidal) control, but the larger joints, i.e., shoulder, elbow, and wrist, are following the hand activity via patterned movements. Furthermore, we can surmise from complex hand activities, such as playing the piano, that even finger activities are patterned to a large degree. Thus, in the scenario of a severe injury to the extra-pyramidal system, all movement patterns including speech would break down and decomposition of movement would be the rule. In a patient with athetoid cerebral palsy as a result of kernicterus, in which only the extra-pyramidal system is thought to be injured, this model is in agreement with the clinical presentation. In less severe cases of extra-pyramidal injury, gross movements are carried out in an uncoordinated fashion and with a significant element of randomness. Fine motor activities usually can not be performed.

Because of the damage to the pyramidal system, a patient with spastic cerebral palsy will demonstrate some or all of the following features:

1. loss of selective muscle control;
2. dependence on primitive reflex patterns for ambulation;
3. abnormal muscle tone;
4. relative imbalance between muscle agonists and antagonists across joints; and
5. deficient equilibrium reactions.

A simple diagram may give the reader a better understanding of how a lesion in the corti-cospinal tracts could produce such a variety of abnormalities (Figure 2) .

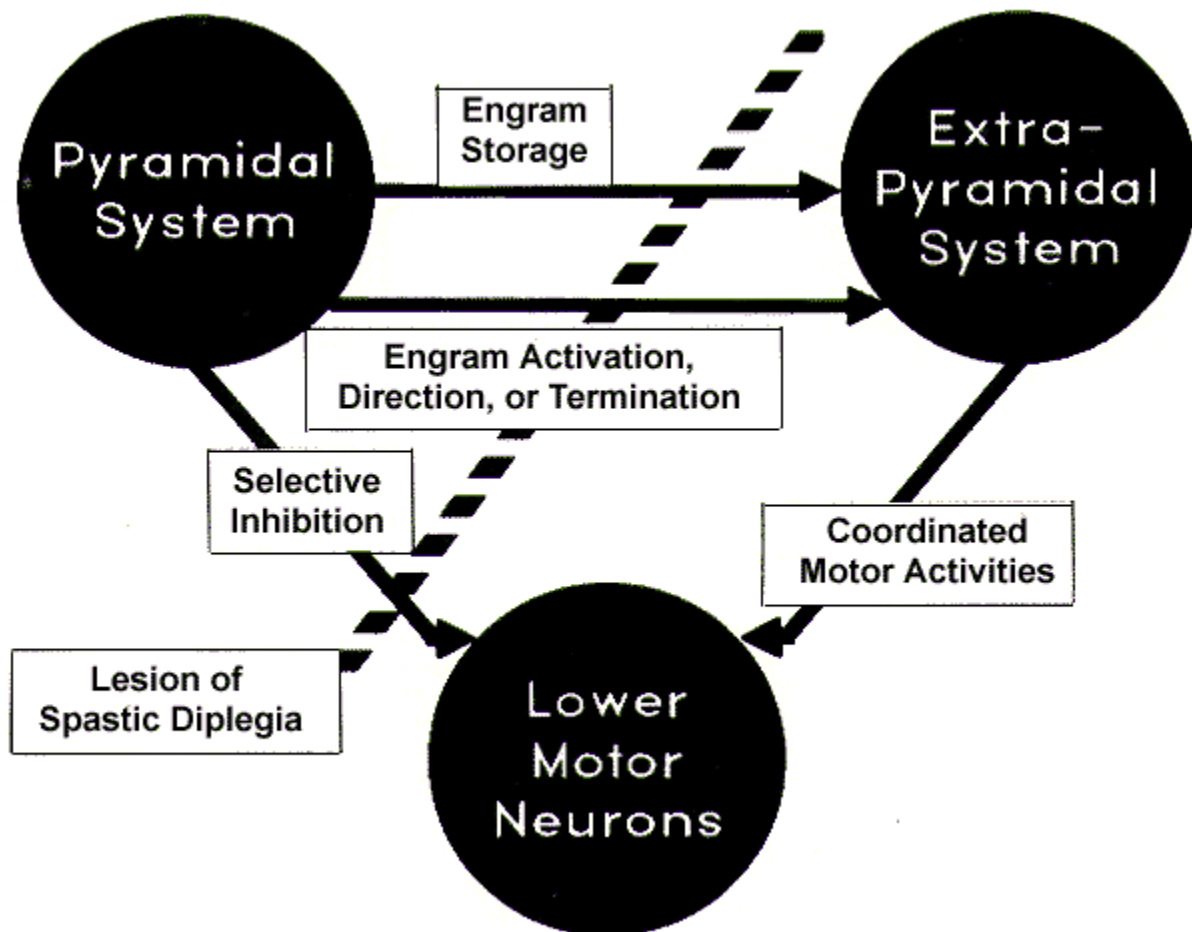


Figure 2. A schematic diagram of the motor control system of the brain. The pyramidal system initiates voluntary motor activities (engrams). It also supervises or oversees the extra-pyramidal system in which the engrams are stored. This is thought to be accomplished by a process of inhibition rather than facilitation. The pyramidal system also prevents excessive muscle tone through its inhibitory action on the spinal cord neurons. A lesion in the region of the internal capsule can disrupt the pyramidal systems connections to both the extrapyramidal system and the spinal cord. The result of such a disruption could produce loss of selective muscle control, dependence on primitive reflex patterns for ambulation, abnormal muscle tone, relative imbalance between muscle agonists and antagonists across joints, and deficient equilibrium reactions.

Loss of Selective Muscle Control and Pattern Dependence

When gait analysis is carried out in a patient with spastic cerebral palsy, the movement patterns in the lower extremity are abnormal, but they do not vary significantly from step to step, run to run, or even from day-to-day. If we go back to the model discussed earlier, in a purely spastic child the system which stores patterned activity would be normal and presumably the inborn patterns or reflexes which that system contained would be normal as well. The problem would lie in the voluntary control system. Unfortunately, this system is required in order to input new information into the extra-pyramidal system; and so the child with a purely pyramidal (spastic) lesion would be unable to input normal movement patterns into his extra-pyramidal system. The abnormal patterns which he attempted to "store" in the extra-pyramidal portion of his brain would, however, be faithfully recorded to be later reproduced by this automatic control system whenever it was called upon to do so. In a sense, the situation could be likened to an orchestra in which some of the musicians knew their parts well and played them properly, whereas others in the orchestra had learned their parts incorrectly, playing them in exactly the same manner each time with the "bad notes" always reappearing at the same point in the score. Furthermore, in our hypothetical example, the conductor might have a speech deficit such that the musicians would have a difficult time in following his instructions. The result might well be that the listening audience would be able to recognize the particular piece of music which was being attempted, but would also readily appreciate the disorganization and disharmony.

Abnormal Muscle Tone

The extent of damage to the pyramidal system determines the amount of selective control remaining. Since the corticospinal tracts act to inhibit the tone setting of the muscle spindles, maintenance of normal muscle tone is also dependent upon the integrity of this system. The amount of spasticity present would depend upon the aggregate damage to the pyramidal system. If there were damage to the extra-pyramidal system, muscle tone will not be spastic, but rather dystonic, which translates literally from Latin as "bad tone." This may vary greatly in degree from decreased tone (hypotonia) to markedly increased tone (rigidity or tension athetosis). In mixed cerebral palsy both patterns of abnormal tone are present and the patient will exhibit a mixture of spasticity and dystonia.

Relative Agonist/Antagonist Imbalance Across Joints

In spastic diplegia, spasticity is most prominent in the hip adductors, flexors, and internal rotators, knee flexors (hamstrings), ankle plantarflexors, and peroneals. This produces a walking posture of hip flexion, adduction, and internal rotation, knee flexion, and ankle equinus with pes piano valgus. Interestingly, when these muscles are studied with dynamic electromyography in a spastic patient, they tend to be overactive, i.e., have prolonged activity during the gait cycle. Their antagonists, on the other hand, often show relatively normal electromyographic activity. Initially, only dynamic muscle contractures are present, but with time and growth they become fixed. Bones grow via epiphyseal plates. However, muscles grow by adding on sarcomeres at the musculotendinous junction.¹³

The stimuli for this growth are somatotropin (growth hormone) and stretch. Thus, as the normally growing child runs and plays each day, he stretches his muscles over the continually growing bone. If a muscle agonist is dominant over its antagonist, the latter will receive excessive stretch whereas the former will not be stretched adequately. This will produce inadequate growth of the agonist muscle and excessive growth of the antagonist which will shortly lead to growth contracture. It is probably the spastic tone in certain muscle groups which produces the relative agonist-antagonist imbalance across joints, since joint contracture is not a feature of athetoid cerebral palsy in which the tone pattern is one of dystonia as opposed to spasticity.

Deficient Equilibrium Reactions

The vestibular system is the seat of balance and equilibrium. It consists of receptors located in the inner ear, peripheral nerve fibers to mediate information from the vestibular nuclei in the midbrain, and ascending and descending pathways from these nuclei. The principal descending pathways are the medial and lateral vestibulospinal paths which terminate upon interneurons in the spinal cord. The medial vestibulospinal tract extends only to the upper thoracic level of the cord whereas the lateral vestibulospinal tract extends the entire length of the spinal cord. The tracts have strong facilitatory effects on motoneurons innervating antigravity muscles. The vestibular system reinforces the tone of the extensor muscles of the trunk and limbs enabling the muscles to support the body against gravity and maintain an upright posture. In an animal whose brain is transected at a midbrain level, a condition develops known as decerebrate rigidity. In humans this is characterized by extension of all the limbs with the arms adducted and internally rotated. Decerebrate rigidity is dependent on intact reflex arcs and the vestibular system. Therefore, it can be abolished by transection of the dorsal spinal cord roots and/or lesions of the central nervous system which interrupt the descending vestibular and reticular pathways.

Ascending pathways from the vestibular system synapse on the somatic motor nuclei of the cranial nerves that supply the extraocular muscles. The vestibular system is extremely important in controlling conjugate eye movements in response to head movement and to the position of the head in space. Without the vestibular system, the eyes could not remain fixed on stationary objects while the head and/or body are moving. Primitive reflexes which involve the vestibular system may become prominent when injury to the cerebral cortex unmasks the suppressive effect of the higher centers. Examples of these are the tonic reflexes. Tonic reflexes are those which maintain reflex contractions which are the basis of posture and attitude. As such the control of body position in space and the maintenance of balance are heavily dependent on these reflexes. The principal tonic reflexes are the symmetrical and asymmetrical tonic neck reflexes, and the tonic labyrinthine prone and supine reflexes.

It is clear that the presence of pathological movement can have a profound effect on functional ambulation. For the remainder of this text, discussion will be limited to the management of spasticity through the application of external biomechanical forces via orthotic systems.

Subtalar Instability

The spastic individual with subtalar instability can be very effectively managed with a UCBL orthosis provided the deformity is passively restorable to neutral alignment. The degree of imbalance between the peroneals and posterior tibialis tendons with the added complication of spasticity, requires the medial and lateral trimlines to be extended much higher dorsally. For the young adult and adult individuals with significant deformity and spasticity, a variant of the UCBL is very effective. This design is fabricated from 5/32" polypropylene which is stretched enough to result in 1/8" thickness (Figure 3). An overlap Velcro® closure is fashioned to provide total mid-foot control and a closed stabilization system. A Gillette modification is then added medially or laterally (depending on the deformity) with crepe filling in the medial longitudinal arch area (Figure 4). If this spasticity is severe or the extremity somewhat rigid, the control level must be extended to an AFO design as the UCBL or variants of the UCBL become marginally effective.

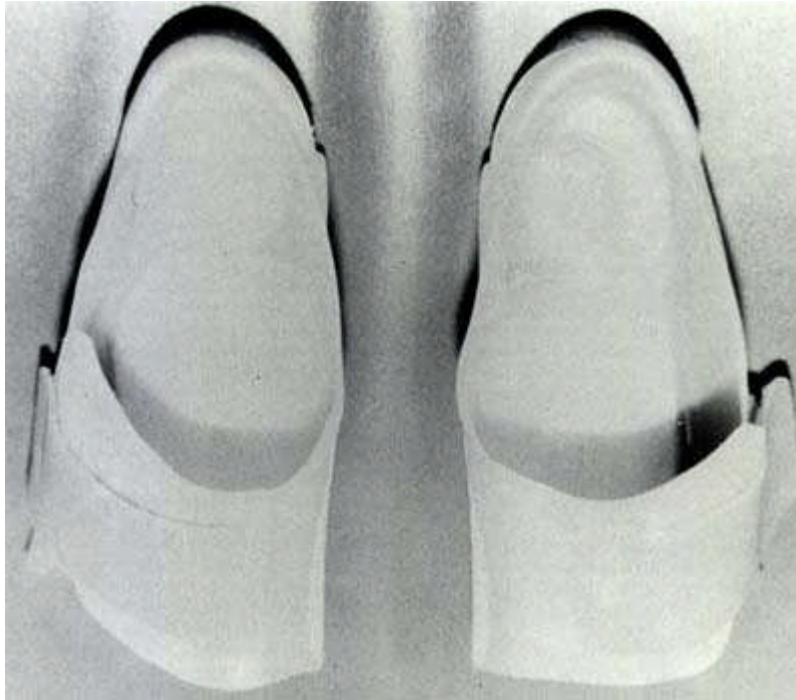


Figure 3. Dorsal UCBL variant for total control of midfoot and subtalar instability.



Figure 4. Dorsal UCBL with crepe filler in the area of the longitudinal arch.

Ankle-Foot Orthoses

AFOs have their most significant effect during stance phase of gait. This is the period in which the braced extremity has contact with the ground as it progresses forward. During this phase, the extremity is called upon to support a portion of the body weight, allow forward progression and resist unstable forces due to uneven terrain, impaired balance, weakness or pathological motion (in spasticity).

Stance phase of gait can be further divided into three rockers (Figure 5) . The first rocker begins at initial contact and ends at foot flat. The second rocker begins with the tibia advancing over the ankle foot complex from 15° plantarflexion to 15° dorsiflexion. Finally, third rocker is initiated with heel off and completed when the extremity pushes off to begin swing phase.

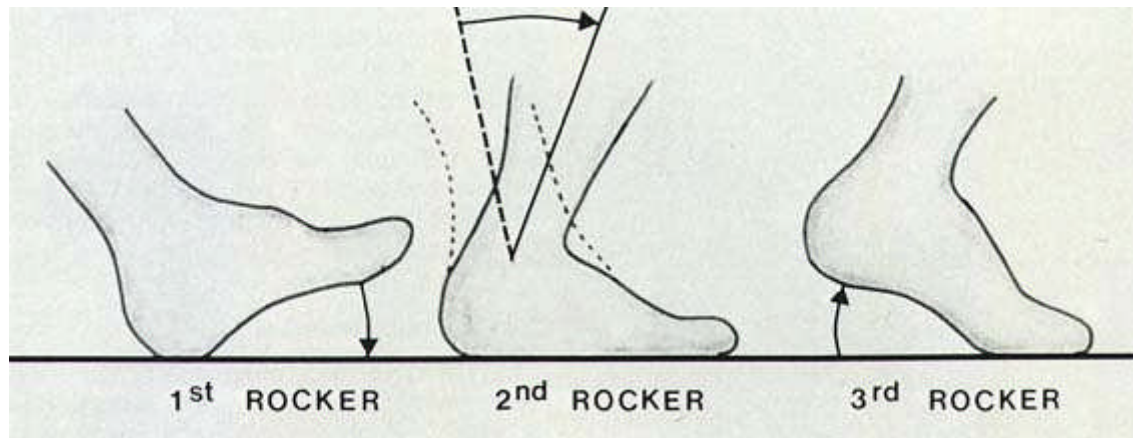


Figure 5. Three rockers in stance phase representing smooth transition from initial loading to push off.

The degree to which an orthosis affects the smooth occurrence of these three rockers negatively affects the efficiency of walking. Parameters of gait such as stride length, percentage of single support times and energy consumption are all affected by sagittal plane stabilization of the extremity. This compromise is often necessary and acceptable since the benefits derived from stabilization can outweigh the negative effects of restriction for the spastic individual.

In our experience, the athetoid patient functions better if controlled ankle movement is allowed. These individuals need to express the pathological movement inherent in their disorder. Rigid immobilization of the ankle foot complex frequently results in severely compromised balance in stance phase stability. The athetoid patients rely on the slow worm-like movement for equilibrium/ balance reactions and a proprioceptive assessment of where they are in space.

The optimum AFO design for these individuals are posterior leaf spring AFOs or articulated AFOs with elastic posterior check strap. Both these variants allow controlled movement with adjustability for future modification.

Solid Ankle Design

This configuration provides maximum stability in the transverse and sagittal planes. As a result, it is the design of choice when severe spasticity and medial lateral instability or deformity exists.

The solid ankle trimlines also enable translation of the ground reaction forces to the knee to promote a flexion or extension movement depending on the plantarflexion/ dorsiflexion angle of the ankle and the weight line relative to the knee axis.

Articulated Ankle-Foot Orthosis Design

The recent refinement of the articulated ankle-foot orthosis has resulted in numerous applications at Newington Children's Hospital over the past three years. This design has been theorized to be an improved, more versatile and dynamic AFO variant compared to its predecessors-the solid ankle or posterior leaf spring AFOs (Figure 6 and Figure 7).



Figure 6. Articulating AFO on an adult CBA patient. Fabricated from 3/16" polypropylene with Chicago screws at the ankle joints.

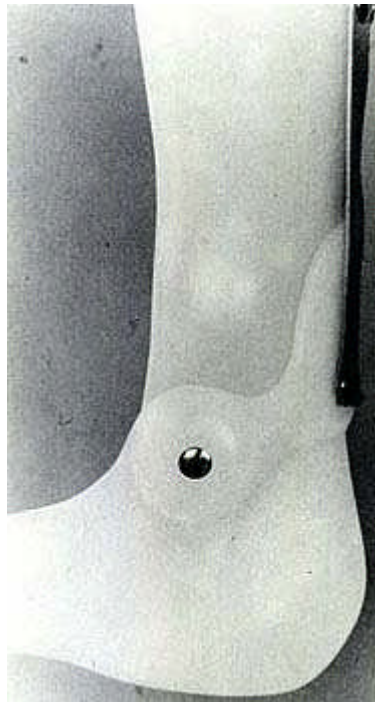


Figure 7. Posterior check strap from 1.5" double thickness dacron provides non-elastic dorsiflexion resistance.

Over this time period we have noted three primary disadvantages to using the articulated AFO. The first and most significant is the compromised ability to control severe subtalar varus or valgus. The slight spacing required to enable smooth joint movement provides enough slack to deter from achieving maximum medial lateral control. The second is the structural integrity of the AFO for the aggressive walker who puts enormous forces on the ankle joint area. We have experienced some plastic distortion and binding in these individuals.

Finally, this design has proven to be less cosmetically acceptable as it is bulkier, utilizes more plastic, and is much more

difficult to disguise.

Tone Reducing Modifications

While there is no scientific evidence that specific AFOs or AFO designs reduce tone, clinical observation and empirical data do show certain modifications to the articulated AFO to aid in diminishing tone and pathological reflexes. These include:

1. 15° to 200° of metatarsal phalangeal extension
2. Recessing area under metatarsal heads 2, 3 and 4 in order to reduce stimulation under the plantar surface of these anatomical structures, reduce the grasp, and decrease positive support reflex (Figure 8 and Figure 9).
3. . Build up along medial aspect of foot plates for foot valgus and recess slightly at lateral areas of calcaneus to elicit inversion reflex.
4. Build up along lateral side of foot plate for foot to be in varus (eliciting eversion reflex).
5. Aggressive control of medial lateral instability through the use of key contact points- medially at the deltoid ligament and laterally at the lateral aspect of calcaneus.
6. Internally mounted figure eight straps (Figure 10) .
7. Fixed 5 dorsiflexion stop angle for those patients with extensor tone. (The reverse for those with flexor tone.)

Incorporating these aforementioned modifications in the articulating AFO has resulted in clinical observation of tone reduction. Extensor tone in particular can be affected appreciably by these design considerations. In addition, other aspects of gait such as standing balance, stride length, step length, cycle time and walking velocity have been shown to improve. The question of how much of this improvement can be attributed to the tone reducing AFO designs is unanswerable at this point. Carefully executed scientific experiments with controls and a broad patient base will perhaps reveal some answers.

Pathological movement must be selectively controlled in the lower extremities to enable functional gait. The application of external biomechanical forces will invariably have positive and negative effects on stability and function. The balance between the two is extremely sensitive and requires critical analysis of post-orthotic application performance. It is only through the thorough comprehension of normal human locomotion, pathological gait and the understanding of the physiological basis of normal movement that we can maximize function for the spastic individual. Orthotic management of this population is often precarious and warrants scientific analysis of the effects of bracing on tone. Computerized gait analysis and EMG studies represent two potential methods of quantifying the results of our orthotic intervention. Hopefully, this will make us even more knowledgeable for our future encounters with abnormal movement and enhance the treatment of spasticity.

Robert S. Lin, CPO, is Director of Orthotics at Newington Orthotic and Prosthetic Systems Children's Hospital Center, 151 F. Cedar Street, Newington, Connecticut 06111.

James R. Gage, M.D., is Director of The Kinesiology Laboratory and staff orthopaedist at Newington Children's Hospital.

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