CHAPTER 11

SPASTICITY IN THE PODIATRIC PATIENT

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The management of the podiatric patient presenting with spasticity can be both vexing and rewarding. Content with a seemingly good outcome at 1 year postoperatively must be tempered with an appreciation for the potential consequences of a growing limb and the complex interplay between muscles, tendons and osseous factors. The 10 year follow up outcome might be very different and the patient may have other as yet unrecognized functional aberrations.

So how does one treat these patients? Many questions come to mind. Do you transfer a spastic muscle? Is the neuromuscular lesion static or progressive? You are a podiatrist, what about the suprastructural deformity such as spastic hip adductors, what is their affect on the lower limb function? Is the foot reducible or rigid? Will a muscle tendon transfer shift the muscle power the wrong way causing a resultant different deformity? Are the extrinsic muscles affecting the foot and ankle under voluntary control by the patient? Maybe they could actively control tibialis anterior if it were not for the spastic gastrocsoleus complex. How does one know?

The author continues to encounter, mull over and search for the answers to these questions while treating children and adolescents with congenital or acquired spastic conditions, particularly during medical mission surgical trips. This chapter represents an effort to consolidate some thoughts, observations and information gleaned from our surgical forefathers while at the same time establish a personal blueprint for the successful management of these patients that is modifiable as clinical and didactic experience grows.

UNDERSTANDING THE NEUROMUSCULAR LESION

Upper motor neuron versus lower motor neuron, what does it mean? Is there pyramidal or corticospinal tract involvement or is it extrapyramidal? Is the patient with cerebral palsy spastic, athetotic or both? An organized clinical approach to these patients requires an intimate appreciation of normal neuromuscular pathways and structure.

In simplest terms the brain or cortex relays information to the anterior horn cells which then communicate with the motor unit and muscle itself. The anterior horn cells are the location of the lower motor neurons and diseases at this level such as polio effect the classic lower motor neuron syndrome.

This lower motor neuron condition exhibits in general that individual muscles may be affected, atrophy is pronounced as the muscles do not receive normal innervation, flaccidity and hypotonia of affected muscles with loss of tendon reflexes, plantar or babinski reflex if present is of the normal or flexor type, fasciculations may be present representing the sporadic discharge of muscle fibers due to diseased and irritable axons and electromyograms reveal reduced numbers of motor units and fibrillations representing isolated activity of individual muscle fibers. Sensorium is intact. No clonus is present. Diseases of lower motor neurons or peripheral nerves show distal limb weaknesses such as foot drop or a steppage gait.

In contrast the upper motor neuron pathway (think BRAIN versus spinal cord, cerebral palsy is a great example!) consists of central nervous system neurons and their associated descending pathways through the brainstem and spinal cord. There are also several indirect pathways operating through the basal ganglia and related subcortical nuclei that influence motor activity on a central level. These indirect pathways are the extrapyramidal system., while the corticospinal tract constitutes the pyramidal system.

Pyramidal involvement manifests as muscle groups that are affected diffusely, never individual muscles, atrophy is slight but strength is decreased, spasticity with hyperactivity of the tendon reflexes, an extensor plantar reflex or Babinksi's sign and fascicular twitches are not produced. The upper motor neuron syndrome is a motor neuron disorder characterized by a velocity dependant increase in tonic stretch reflex, (muscle tone) with exaggerated tendon jerks resulting from hyperexcitability of the released or disinhibited spinal motor neurons. Clonus is present. Pyramidal involvement is essentially a spastic motor dysfunction

Extrapyramidal involvement is characterized by increase muscle tone or rigidity (although may be hypotonic in infants) with disorders of movement such as chorea, athetosis or dystonia. Muscle strength is normal or mild decrease, tendon reflexes are normal or slightly increased and Babinski is normal or down going. The

involuntary movement disorders of extrapyramidal involvement are hallmarks. Dystonia is a fast and slow twisting movement of the trunk, head and extremities with very slow relaxation intervals. Athetosis is differentiated from dystonia by the smooth flow of posture from one position to another without sustained posturing of the limb. These movements are accentuated during purposeful movement and involve a peculiar, writhing, irregular movement with increased tone in the lower extremity. Chorea consists of rapid, involuntary and nonrhythmic, generalized jerks of various parts of the body. Movements occasionally dance from one joint to another and may involve facial grimacing and flexion-extension movements of the extremities.

If sensory changes coexist with a flaccid arreflexic paralysis this indicates anterior and posterior horn cell involvement or in mixed motor and sensory nerves. If only motor changes exist then the lesion must be at the level of gray matter of the spinal cord, anterior horn cell level, motor branches of peripheral nerves or motor axons alone.

Rigidity must be differentiated from spasticity and shows the distinction in quality of hypertonicity. In rigidity, there is a heightened discharge of alpha motor neurons and resistance to passive movement of the limb is constant and sometimes described as "lead pipe" or "plastic". This is an extrapyramidal exhibition and maintenance of a flexed position is common. "Cogwheel" rigidity is common in Parkinson's disease when hypertonic muscles show resistance to passive stretching by rhythmic jerky movements as though the resistance of the limb were controlled by a ratchet. Deep tendon reflexes are normal. Consistent with the extrapyramidal involvement is the disorder of involuntary movement.

Spasticity however is corticospinal or pyramidal and a hyperactivity of the stretch reflex (a tap on a tendon, stimulates muscle spindles, activates afferent neurons, transmits impulses to alpha motor neurons, resulting in brief muscle contraction or tendon reflex) secondary to central changes but without increased sensitivity of muscle spindles. Clinically this is seen as a free interval followed by a "clasp knife" or sustained phenomenon where the muscle doesn't contract until stretched a little and then during the stretch the augmentation in muscle tone quickly subsides. Deep tendon reflexes are increased.

CLINICAL EXAM

The clinical exam begins with an appreciation of the pathophysiology described above. I t proceeds not unlike the exam of any other patient with more attention to gait analysis for balance and movement issues, posture evaluation for general muscular control assessment and naturally range of motion exams to passive and muscle active testing to include deep tendon and superficial or cutaneous reflexes. Alterations of muscle tone such as hypertonicity, spasticity or rigidity are noted. Inspection for muscle atrophy and/or strength is performed.

Gait is facilitated in the neuromuscular patient by an extensor synergy thigh adduction and thigh and knee extension with plantarflexion of the feet and toes. This is more powerful then flexion synergy and this bias toward extension synergy assists weightbearing and walking. The limbs are checked for symmetry.

The presence of tissue contractures or not is noted and attempts are made to determine if certain muscle groups are being overpowered by spastic groups. Is an equinus present as a fixed condition or is it a dynamic equinus only noted during ambulation secondary to a spastic gastrocsoleus that demonstrates a posterior thrust of the knee.

It is important to remember that the muscles of the cerebral palsy patient show contracture and weakness due to neurological deficit as well as limited use. The atrophy of muscles in a clubfoot patient may be due to disuse and should be graded on their expected strength. Furthermore, spastic muscles undergoing short stretch show augmentation of tone while long stretch inhibits tone due to hypertonicity and resistance to passive movement. Athetotic muscles undergoing short stretch inhibits tone and long stretch augments it.

The determination of voluntary or involuntary muscle action is critical and muscle belly blockade with 1% lidocaine may be useful. The element of spasticity can not be determined under general anesthetic and therefore must be done preoperatively. It is particularly important to determine if tibialis anterior is under voluntary or involuntary control. If it functions with the "mass or confusion" reflex which is performed when the patient voluntarily flexes the hip and resistance is placed on the thigh and the anterior tibial muscle responds then it is functioning although under involuntary control. The patient utilizes the muscle to help dorsiflex the foot during gait and with hip flexion. Dosriflexion of the foot occurs despite the absence of voluntary action.

This maneuver has been termed synkinesia, "confusion or automatic" reflex or Strumpell test. It is essential to determine if tibialis anterior is under voluntary control and often this can not be elicited with the knee extended. Likewise, if under voluntary control, it is essential to develop active function to avoid recurrent equinus deformity postoperatively. Tachdjian has termed this the "cerebral zero" tibialis anterior and recognizes that

it may function after a tendo Achilles lengthening by 6 to 12 months postoperatively. This emphasizes the point that one of the goals of a tendo Achilles lengthening is a desire to not stimulate the stretch reflex or at least alter the point at which it is elicited while allowing or "freeing" up the tibialis anterior to function in dorsiflexion if indeed it is functionable.

One must have an appreciation for the classification of muscles as agonists or prime movers, antagonists or moderators, muscles of fixation that stabilize joints and create a firm base for muscle action and synergists that assist the agonists. Only the agonists are under voluntary or cortical control, the rest are controlled reflexively and subconsciously.

Simple actions such as dorsiflexion of the foot are indeed actually complex as no muscle forces act directly on the subtalar joint and represent a complex interaction of agonists, synergists, fixators and antagonists.

Foot mobility is also a critical determination of the clinical exam. Is the foot manually reducible? A very hypermobile foot may require tendon transfers and joint stabilizations while the more rigid foot may benefit from an isolated tendon transfer but a fusion may be less necessary if position is good. The goals of treatment are clearly to use the existing muscle to its best functional effect to counteract joint instability. Deformities in general depend on the mobility of the foot and especially the subtalar joint as there is a preponderance of spasticity and muscle strength imbalances on the medial side of the foot and ankle.

TREATMENT AND SURGICAL PEARLS

It must be understood that cerebral palsy is a static lesion that has a developmental progression as the child grows due to muscular imbalances. These imbalances continue throughout the years. The loss of selective motor control at the upper motor neuron level allows the lower motor neurons to take over at the lowest level and creates spasticity. The spasticity plus postural reflexes creates the muscle imbalance between agonists and antagonists. The muscle imbalances in the supple foot may be moderated by tendon transfers or lengthenings but may fail in the more rigid foot.

As noted above, evaluation of voluntary control of muscles including their power and the identification of spastic muscles or out of phase muscles will direct treatment. Choices for correcting muscle imbalances include removing the force, excising the tendon or transfer. If orthotic support is required to maintain upright posture then the muscle control is of little importance and may be removed. If a patient has enough muscle control and sensation to walk without orthotic support then appropriate tendon transfer with evaluation of power and phasic activity will be beneficial if it sufficient to control floor reaction forces during the stance phase of gait.

DeLuca has suggested there may be a role for tone reducing/inhibiting casts for spastic foot deformities or serial stretching casts. Central nervous system activity might be modifiable by peripheral management. In the failure or inadequacy of nonsurgical measures, surgical attempts are undertaken to restore muscle balance or correct fixed deformities.

A contracted or spastic muscle may be lengthened or released thereby weakening the motor unit and restoring length. It may be transferred to restore balance and phasic control. Its force may be negated by neurectomy or attempts to impact the abnormal reflexive pathway at the spinal root level via selective posterior dorsal rhizotomy may be attempted. General principles dictate it is better to undercorrect then over correct, a calcaneal gait is much worse than recurrent equinus. Likewise, residual heel valgus is more stable mechanically than excessive varus.

A muscle to be transferred ideally grades at least 4/5 and has a normal line of pull. It is best attached directly to bone and the bony deformity must be fixed. Tendon transfers do not change phase and can become antagonists out of phase to certain motions after transfer.

Surgery is best reserved until after the patient is 6-years old and an emphasis is on the preservation of effective toe off if possible. Gastroc recessions are favorable and decrease the chance of calcaneus deformity while removing some clonus that interferes with full foot loading and thereby preserves push off power. The soleus is persevered. Iatrogenic or progressive elongation due to uncorrected knee and hip flexion must be avoided. It will deny effective propulsion or push off. After a TAL the knee must be prevented from going into flexion or it will overstretch it.

The majority of hindfoot deformity is equinovalgus that in effect creates inappropriate prepositioning of the foot at heel contact and creates a loss of stance phase stability. During the clinical exam the varus alignment will be due frequently to tibialis posterior. This may occur in stance or continue on into the swing phase. If the activity of tibialis posterior is continuous and it is an active deformer in the swing phase and the deformity is passively correctable it is better to lengthen it or do a split transfer to peroneus brevis in patients older than 7 or consider an intramuscular tenotomy in those younger.

The advantage of intramuscular tenotomies is that a transfer may still be performed later. If it has shifted phase completely and is no longer assisting in normal stance stability it may be transferred. EMG's are essentially to determine this phasic activity. In the presence of uncontrollable hindfoot valgus an extraarticular arthrodesis may be warranted of the GreenGrice type. If using a fibular graft it should be taken from the middle to upper 1/3rd of the fibula to prevent ankle valgus. In the event of ankle valgus supramalleolar osteotomies may need to be considered.

Tibialis anterior transfers require specific thought as well. If deformity is mild and equinus moderate with inversion primarily in the swing phase and control is voluntary a split transfer may be performed. If tibialis anterior is only active with the confusion reflex than a complete transfer may be performed. If subtalar joint range of motion is limited or a fusion is planned and tibialis posterior is a severe deforming force than both tendons may be transferred to substitute for the dorsiflexors.

Naturally if deformities are not correctable passively or are rigid but can not be corrected by weakening or transferring muscles alone than realignment arthrodesis procedures are required. Foot plates and cast extensions past the toes are essential to maintain stretch on the toe flexors.

All cases will require individualized approaches based on specific deformities. It is hoped that this brief compilation of available thought and experience in the management of these complex case will serve as more groundwork for future coherent approaches to challenging patients at home and abroad.

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