Laboratory analysis and dynamic polyEMG for assessment and treatment of gait and upper limb dysfunction in upper motoneuron syndrome

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Multiple muscle agonists and antagonists acting for all the joint movements in the upper and lower limb exist. This redundancy of motor control is very valuable in normal physiology, but when a central nervous system injury with resulting upper motor neuron syndrome takes place, the source of the functional impairment may be difficult to localize. In this paper we discuss the use of gait and motor control analysis studies as a tool particularly useful in determining the specific muscles that may be producing limb dysfunction. We present the most frequent patterns of upper motor neuron dysfunction that affect the upper and lower limb as a result of upper motor neuron syndrome. A case description of the features, the electromyographic patterns, and their functional implications are used. Our objective is to clarify the understanding of these patterns of dysfunction and their focal cause with the intent to improved care of the patient with upper motor neuron syndrome.

Key words: Extremities - Motor neuron disease - Gait.

Commonly, there are multiple agonists and antagonists for virtually any joint movement direction that takes place in our bodies. In order to match a required joint torque even across a single joint, the question regarding which muscles should be activated and at what levels of activity is likely to have a very variable answer without a unique solution. In the 1930s, Nikolai A. Bernstein suggested that a basic problem of motor control relates to overcoming redundant degrees of freedom in our multi-jointed skeletal system. Since we live in a 3 dimensional world but we have more than 3 degrees of freedom in our multi-jointed limb segments, the issue for Bernstein and many others in his tradition was to discover the coordinative rules or priorities used by the brain to constrain redundant degrees of freedom when performing motor tasks. Latash has pointed out that the Bernstein problem can occur even at the level of single-joint, single degree of freedom movements where there is seemingly no room for peripheral redundancy. Simply put, identifying muscles that produce deforming maladaptive joint movements and postures statically and dynamically is an important endeavor in aiding clinical interpretation of gait dysfunction and in rationalizing subsequent treatment interventions. For a given patient, however, there may be a unique solution in that equinovarus deformity may be solely attributable to an overactive tibialis anterior in one patient while in another it may be an overactive tibialis posterior or some other muscle.

Laboratory studies

For patients with central nervous system (CNS) injuries with resulting upper motoneuron syndrome...
(UMNS), kinesiologic multichannel dynamic electromyographic (EMG) studies, allow the examiner to evaluate relationships between agonist and antagonist muscles across individual joints and limb segments. Voluntary capacity and spastic reactivity are examined and interpreted in light of clinical and functional complaints. The laboratory examination supplements clinical examination because the latter alone may not be sufficient to identify all offending muscles interfering with active or passive range of motion. Dynamic EMG examination often helps to provide detail in the analysis of UMNS that provide direction and confidence for therapeutic interventions. In addition to dynamic EMG studies, gait laboratories may examine ground reaction forces; joint motion and foot pressures to further enhance movement analysis.

TREATMENT CONSIDERATIONS

There are 3 treatment considerations that are usefully kept in mind when dealing with movement related problems in the aftermath of UMNS.

1) The treatment approach will vary as a function of the period of recovery.

2) The treatment approach will be different for a focal problem versus a diffuse problem.

3) Many pharmacologic agents that are useful in treating movement related disorders also produce side effects, which are dysfunctional. A balance needs to be struck between benefits and adverse reactions and a useful way to do this is to target specific results that the drug agent is expected to produce.

In the period of motor recovery, temporizing interventions are used because permanent changes may result in chronic imbalance of forces across joints that likely will result in a contracture. Thus, chemodenervation agents such as botulinum toxin A and neurolytic agents such as phenol are used in this period or during rapid growth in children because their effect wear off in approximately 3-6 months. These agents are used when restricted motion occurs as a result of focal or multifocal muscle overactivity and spasticity. When these agents wear off, re-evaluation is performed to see whether additional physiological recovery has taken place and whether there is further indication for re-injection. Neurolytic blocks may be performed, but it is noted that repeated blocks might be less effective and more difficult because of fibrosis development after previous injections. Oral antispasticity agents, although usually accompanied by many adverse reactions such as sedating properties that interfering with attention and/or cognition, may be helpful in this period. Neuro-orthopaedic surgical corrections are generally not performed as an early intervention. But during the period of residual deficits and functional adaptation, neuro-orthopaedic interventions may be indicated in patients with restricted motion. Passive and active function are targeted as goals of surgical intervention.

Treatment approaches will vary as a function of focal or diffuse restrictions in motion secondary to muscle overactivity, spasticity or dystonia. In general, whether restricted motion can be attributed to a small number of muscles, focal blocks of these muscles is preferable if the problem is diffuse or generalized however, oral antispasticity agents may be considered.

More recently intrathecal delivery of medications such as baclofen has been introduced for the care of patients with generalized muscle overactivity with the intent to reduce potential adverse effects caused by systemic delivery of this medication. For some patients, bracing or splinting may be utilized to supplement the effects of this interventions.

In our opinion, focal injections with chemodenervation agents is the most suitable approach for treating restricted motion secondary to muscle overactivity and spasticity if the functional problems of the patient can truly be ascribed to specifically identified muscles. Even if many muscles in a limb are involved a number of focal injections are possible and by doing so CNS side effects can be avoided. When it comes to focal injections, the technique of botulinum toxin injection overall appears to be safer, quicker and easier than phenolization techniques. Both types of injection techniques overall seem more easily accomplishable than having to take oral medications on a day in - day out basis, at least with patients with cognitive deficits.

Problems of movement control and limb deformities are common consequences of the UMNS. The genesis of limb deformity is based, in large part, on the concept that UMN negative and positive features such as paresis, spasticity, and impaired motor control generate an imbalance of muscular forces affecting joint position statically and joint movement dynamically.

Typical patterns of UMN deformities are diagnostically evaluated to identify which muscles contribute dynamically and statically to the observed deformity and impaired motor control. Dynamic EMG, gait, motion analysis and diagnostic nerve blocks frequently provide the necessary detailed information about
specific muscle groups that will guide decision making for treatment. Prior to selecting interventions, the clinical team and the patient should explicitly develop functional goals. Functional goals may be classified as symptomatic, passive or active in nature. Symptomatic refers to pain, flexor spasms, extensor spasms and clonus as some of the presenting problems. Passive function refers to the passive manipulation of limbs to achieve functional ends, typically performed by caregivers, though patients may also manipulate their limbs passively with their non-involved limbs. Active functions, on the other hand, refer to the patient's direct use of the limb to carry out a functional activity. Identifying muscles with volitional capacity is important to the achievement of this goal. Another requirement is behavioral compliance and the ability to incorporate the newly gained increases in motion into the daily routine.

In broad terms, clinical evaluation focuses on the identification of 3 factors:

1) The clinical pattern of motor dysfunction.
2) The patient’s ability to control muscles involved in the pattern of dysfunction.
3) The role of rheologic properties of muscles, including contracture, and negative features such as weakness as they relate to the functional problem.

The treatment interventions suggested in this paper are usually muscle-specific and the outcome of a detailed evaluation using 3-D gait analysis and dynamic EMG techniques is to identify muscle-specific motor behaviors that will be linked to specific interventions. This implies that the unit level of evaluation is typically a joint and individual muscle affecting this joint.

Selected patterns of UMN dysfunction

Patterns of UMN dysfunction affecting the upper and lower limb have been described in the past and are listed in Table I.

For this paper we have described selected patterns for the upper and lower limbs on the basis of frequency of presentation in the UMNS: the adducted and internally rotated shoulder, flexed elbow, flexed wrist, clenched fist and thumb in palm. For the lower limb the selected patterns for discussion include equinovarus foot, hyperextended great toe, flexed knee, stiff knee, adducted (scissoring) thighs.

These patterns of dysfunction are amenable to strategies of focal evaluation and localized treatment. Clinical examination supported by laboratory studies are the mainstays of evaluation and clinical questions of interest regarding a given muscle (that might be targeted for localized intervention) include the following: does the patient have selective voluntary control over the given muscle? Is the muscle activated dyssynergically (i.e., in antagonism to movement) when the patient attempts to move the relevant joint? Is the muscle resistive to passive stretch? Does the given muscle have fixed shortening (i.e., contracture: limited range of motion that is attributed, in large measure, to fixed shortening of the given muscle crossing its joint)? Given the degree of clinical effort, patient comorbidity and complicated movement dysfunction in patients with UMNS, clinical examination alone may not be sufficient to answer these questions with a high degree of confidence. Technology driven laboratory assessments that may include formal gait and motion analysis, dynamic EMG studies and nerve blocks may be helpful. Dynamic multichannel EMG is acquired with simultaneous measurements of joint motion (kinematics) in the upper and lower extremities and with ground reaction forces (kinetics) obtained from force plate measurements in the lower extremities. Kinetic, kinematic, and dynamic EMG data assist the clinician in interpreting whether voluntary function (effort-related initiation, modulation, and termination of activity) is present in a given muscle and whether that muscle’s behavior is also dyssynergic (sometimes referred to as out of phase behavior). In addition, responses to different rates of passive stretch of muscle before and after local anesthetic nerve block can help the clinician distinguish between the dynamic, velocity-sensitive reflex resistance of spasticity versus passive muscle tissue stiffness and contracture. Combined with clinical information, laboratory measurements of muscle function often provide the degree of detail and confidence necessary for

| Table I.—Patterns of UMNS limb dysfunction. |
|----------|----------|----------|----------|----------|
| Upper limb | Lower limb |
| Adducted and internally rotated shoulder | Equinovarus foot |
| Flexed elbow | Equinovalgus foot |
| Pronated forearm | Hyperextend hallux |
| Flexed wrist | Stiff knee |
| Clenched fist | Flexed knee |
| Thumb in palm | Adducted thigh |

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making appropriate treatment decisions. In the section below, we illustrate how to apply strategies of focal evaluation and localized treatment, using a joint by joint approach to patients with frequently seen patterns of UMN dysfunction.10

The adducted and internally rotated shoulder

The adducted and internally rotated shoulder, with the arm tightly against the lateral chest wall and shoulder internal rotation, causes the forearm to lie against the middle of the chest anteriorly (Figure 1). The elbow is often flexed but decerebrate posturing is associated with an extended elbow. The tendon of pectoralis major is often prominent when the examiner attempts to abduct and externally rotate the shoulder, but other muscles contribute as described below. Recall that the glenohumeral joint functions as a universal joint, enabling the hand to reach an almost spherical volume of locations in 3-D space. When patients attempt to reach forward, spastic adductors and internal rotators can severely restrict acquisition of targets in the environment and on the body. The patient’s ability to stabilize, push or apply force to an object is also compromised. From the perspective of passive function goals such as skin care and axillary hygiene, spastic adductors and internal rotators hinder efforts of caregivers to gain access to the axilla to provide needed care. Restricted motion may impair dressing, washing and bathing and promote skin irritation and maceration. Passive manipulation of the shoulder during personal care may cause pain when motion and contact trigger spastic resistance in reactive muscles.

Muscles that often contribute to spastic adduction/internal rotation dysfunction of the shoulder include latissimus dorsi, teres major, the clavicular and sternal heads of pectoralis major and subscapularis. Consider involvement of latissimus dorsi and teres major when hyperextension posturing of the shoulder is observed, especially during standing transfers and gait. Antagonistic activity in these muscles may be masking a patient’s potential for active flexion. Diagnostic lidocaine block to the thoracodorsal nerve and/or lower subscapular nerve may help to distinguish whether one or both heads are pathophysiologically active. Some reports have indicated that subscapularis may be an important source of spastic internal rotation.1,5 This muscle is difficult to study electromyographically and to chemodenervate. Diagnostic lidocaine blocks to the innervations of pectoralis major, latissimus dorsi and teres major may remove their influence and allow clinical assessment of the remaining potential contribution of subscapularis. During the period of motor recovery, large spastic shoulder muscles are amenable to phenol motor point neurolysis or botulinum toxin. During the period of functional adaptation, orthopaedic surgical approaches are typically considered.

The flexed elbow

Upright posture favors hypertonia in the antigravity elbow flexors of the upper limb. In the patient without motor control, severe flexion posturing can lead to skin maceration in the antecubital fossa, malodor and skin breakdown. Many patients complain
treatment such as serial casting, effectively supported by a block that addresses relief of the deformity. Dynamic EMG and diagnostic lidocaine or bupivacaine blocks help determine voluntary capacity and spastic reactivity of elbow flexors and extensors along with the presence of contracture. When patients have little or no ability to voluntarily activate and control extensors and flexors of the elbow, surgical efforts are aimed at improving passive function related to antecubital skin care, malodor, dressing, flexion deformity that affects standing and walking balance.

The flexed wrist

A flexed wrist is most common but hyperextension deformity may also be seen. Patients complain of difficulty inserting their hand into shirts, jackets and other narrow openings and they frequently have pain on passive motion. They may also have symptoms of carpal tunnel syndrome secondary to compression of the median nerve against the transverse carpal ligament by taut flexor tendons. In severe cases, wrist subluxation may be present. Radial or ulnar deviation and a clenched fist are often concomitant (Figure 3).

Muscles that potentially contribute to wrist flexion include flexor carpi ulnaris (FCU), flexor carpi radialis (FCR), palmaris longus, flexor digitorum sublimis (FDS) and flexor digitorum profundus (FDP). FCU, FCR or both may bowstring across the wrist and radial or ulnar deviation suggests their respective involve-
A clenched fist points to extrinsic finger flexors as playing a role. If fingernails dig into the palm, FDP is likely to be involved. If the proximal interphalangeal (PIP) but not the distal interphalangeal (DIP) joint is markedly flexed, involvement of FDS is likely. Distinguishing between limitations attributable to wrist versus finger flexors is one aim of passive range of motion testing. By allowing the fingers to remain flexed in the palm, passive extension of the wrist provides preferential information about wrist flexors. When finger flexors are tight, simultaneous passive stretch of the wrist and finger flexors markedly restricts wrist motion.

Smoothness of motion, speed, effort, decrement in movement amplitude over time and fatigue may be observed. Singly or in combination, the wrist and finger flexors may have variable findings of spasticity, contracture and voluntary control. Because they have a larger cross sectional area, wrist flexor muscles are generally stronger than their extensor counterparts. Despite a net balance of forces favoring flexion, the extent to which a patient may have voluntary control over wrist extensors should be investigated. Dynamic EMG studies and temporary diagnostic motor point blocks are helpful in this regard and diagnostic nerve blocks are often helpful in unmasking movement. Temporary chemodenervation with botulinum toxin of a dysynergic wrist flexor may unmask strength in the wrist extensors sufficient to improve active wrist movement. A similar hypothesis can be advanced about the extrinsic finger flexors after dynamic EMG reveals whether FDS and/or FDP are generating activity in antagonism to wrist extension. Motor point block of the target muscle group or median and/or ulnar nerve blocks at the elbow may be performed to examine for active wrist extension during reach. Combined median and ulnar nerve blocks at the elbow will also reveal the presence of muscle contracture.

During the period of motor recovery, botulinum toxin may be injected into wrist flexors and/or the various muscle slips of FDS and FDP, depending on clinical and laboratory identification of the offending muscles. Surgical options include muscle releases and myotendinous lengthening.

**The clenched fist**

The fingers are flexed into the palm. Access to the palm for washing is typically compromised, the fingernails may dig into palmar skin. When access is chronically restricted, skin maceration, breakdown and malodor develop (Figure 4). Patients complain of pain when they or their caregivers attempt to open the fingers in order to gain palmar access. Some relaxation of finger flexor tightness may occur if the wrist is positioned by the examiner in flexion.

Muscles that contribute to the clenched fist deformity include FDS and FDP. The intrinsics may be also be spastic but an intrinsic plus posture (i.e. combined metacarpophalangeal flexion and PIP extension) is not seen because spastic extrinsic flexors dominate by flexing all joints. Some degree of contracture of the extrinsics is typical of patients with a chronic clenched fist.

Dynamic EMG and differential lidocaine blocks may be helpful in the assessment of the spastic finger flexors. Sometimes a patient presents with spasticity in just 1 or 2 muscle slips of either FDP or FDS. For example, isolated finger flexion is not uncommonly seen secondary to spastic FDP and/or FDS muscle slips to a particular finger. These patients complain that their flexed finger gets in the way of picking up objects.

During the period of motor recovery, focal chemodenervation with botulinum toxin is a useful intervention. It should also be pointed out that reduction of spasticity in the extrinsics may unmask spasticity in the intrinsics, potential converting an extrinsic deformity into an intrinsic plus deformity. Chemodenervation with botulinum toxin is an excellent remedy for treating
spasticity of the intrinsics because these small muscles of the hand are readily accessible for injection and require only small amounts of toxin to be very effective.

During the period of functional adaptation, a variety of orthopaedic options is available to remediate active function of the fingers.

The thumb-in-palm deformity

The thumb is held within the palm, the DIP joint of the thumb is commonly flexed and the thumb is unable to function during 3-jaw chuck type of grasp or in key grasp pattern (Figure 5). Some patients may be able to extend the thumb if the wrist is flexed, suggesting that a spastic flexor pollicis longus (FPL) may be impeding active thumb extension when the wrist is more extended and FPL is tight. The thumb-in-palm deformity may result from spastic activity in adductor pollicis (AP), FPL and/or the thenar muscles (particularly flexor pollicis brevis). Contracture of the web space skin and interphalangeal joint contracture of the thumb may also develop over time. If some volitional potential in thumb extensors or thumb abductors is present, treatment of spastic FPL and AP may facilitate active grasp, usually in the form of a modified type of key grasp. Dynamic EMG and lidocaine blocks may be helpful in this regard. During the period of motor recovery, treatment of spastic muscles by chemodenervation may allow application of hand orthoses for passive or active purposes.

The 3 main functional goals of human ambulation are to move from one place to another, to move safely and to move efficiently. These 3 goals are frequently compromised in the patient with residual effects of acquired or congenital brain injury. The great majority of patients will be able to perform limited ambulation, but they will often have problems because of inefficient movement strategies, the presence of pain due to abnormal limb postures and decreased safety. Often, the compensatory movements necessary for ambulation produce exaggerated displacements of the center of gravity, which result in increased energy expenditure. Impaired balance, sensation and problems with limb clearance can contribute to the anxiety of ambulation and may increase the frequency of loss of balance and falls.

Some generalizations can be made about the gait of a patient with residual effects of UMNS. These include a decrease in walking velocity with a reduction in the duration of stance phase, impairment of weight bearing in the affected limb with an increase in the duration of stance time of the less affected limb. Ochi et al. reported on differences in temporospatial parameters of locomotion among patients with residual stroke and traumatic brain injury. From a functional perspective, gait deficiencies can be categorized with respect to the gait cycle. In the stance phase, an abnormal base of support can be caused by equinovarus, toe flexion, or ankle valgus. Limb instability can occur due to knee buckling (sudden flexion) or hyper-extension, which may result in knee joint pain. This may make walking unsafe, energy inefficient, and painful.

During the swing phase, inadequate limb clearance caused, for example, by a stiff knee and inadequate limb advancement caused, for example, by limited hip flexion or knee extension may interfere with the safety and energy efficiency of walking. To focus more appropriately on the essence of mul-
is stationary. The tibia ordinarily progresses forward by means of ankle dorsiflexion but the lack of available dorsiflexion in the equinovarus foot results in hyperextension thrust of the knee and restrained forward translation of the body’s center of gravity. Walking on uneven ground is difficult and overall balance is compromised. During early swing phase, toe drag associated with equinovarus may make floor clearance and limb advancement difficult. Equinovarus affects the priority of c.g. translation during stance limb advancement. It affects the priority of c.g. stability during swing phase clearance and during stance phase loading and single limb support. Functional products of gait affected by equinovarus include a reduced range of: step and stride lengths, velocities, base widths, single limb postures, elevations and descents, articulation with uneven surfaces and clearance stability. A patient’s ability to change direction is more difficult as well.

A number of muscles may generate unbalanced forces with respect to the equinovarus pattern. Deforming muscles may include tibialis anterior, tibialis posterior, gastrocnemius, soleus, long toe flexors and extensor hallucis longus (EHL). Weak muscles may include peroneus longus and peroneus brevis in stance phase and the long toe extensors in swing phase.

Hyperextended great toe

Hyperextended great toe is a deformity that is characterized by toe extension throughout the gait cycle. Ankle equinus and varus may accompany this abnormal foot posture (Figure 7). When wearing shoes, the patient complains of pain at the tip of the toe and under the first metatarsal head during stance. During gait, toe extension during early and mid stance complicates weight bearing. The patient will shorten stance time of the affected limb and there is interference with push off and forward propulsion in terminal stance and pre-swing. During swing phase, there may be sustained hallux extension. Thus, hyperextended great toe has an impact on c.g. translation during stance limb advancement. It also has an impact on c.g. stability during stance phase loading and single limb support. Functional products affected by hyperextended great toe include a reduced range of the following: step and stride lengths, velocities, single limb postures, elevations and descents and articulation with uneven surfaces. A number of muscles may generate unbalanced forces with respect to the hyperextended great toe pattern.
Extensor hallucis longus provides a deforming force causing great toe hyperextension. A weak flexor hallucis longus may not be able to compensate and offset the hyperextension force of EHL. When equinovarus is present, analysis of the contributions of tibialis anterior, tibialis posterior, gastrocnemius, soleus and the long toe flexors are taken into account as well.

**Flexed knee**

In the flexed knee deformity, the knee remains flexed throughout swing and stance. A flexed knee during swing phase requires compensatory hip flexion on the same side and increased hip and knee flexion during swing contralaterally. Bilateral knee flexion deformity results in a crouch pattern (Figure 8). The impact of a flexed knee on translation of the center of gravity may be seen in the operational feature of swing limb advancement. Lack of full knee extension in terminal swing limits limb advancement and results in a shortened step length. The flexed knee also has an impact on stance phase advancement since knee extension from mid to terminal stance is absent e.g. stability is affected by a flexed knee deformity during...
the operational features of limb loading and single limb support. The impact of flexed knee on the functional products of gait includes a reduction in the range of step and stride lengths, velocities, base widths, single limb postures, elevations and descents, articulation with uneven surfaces and changes in direction.

Muscles that may generate deforming forces with respect to the flexed knee pattern include medial and lateral hamstrings in both phases of the gait cycle, gastrocnemius in stance phase and iliopsoas in swing. A weak quadriceps may be unable to counterbalance the hamstrings. In addition, a weak gastrocnemius and soleus group may lead to drop off pattern of gait in terminal stance (i.e. sudden and simultaneous ankle dorsiflexion and knee flexion).

**Stiff knee**

The stiff knee is a swing phase deformity by definition. The term stiff knee is often placed in quotation marks to signify that it refers to a dynamic deformity created by muscle contraction rather than an intrinsic structural deformity of the knee causing loss of passive motion. In stiff knee gait, the knee typically remains extended during swing phase and frequently in stance phase as well (Figure 9). A stiff knee lengthens the limb and poses problems particularly during the pre-swing and swing phases of gait. Toe drag and early swing may cause tripping and falling if there is an associated equinovarus. The limb appears to be functionally longer because it remains extended throughout swing phase. In order to achieve clearance of the floor during swing phase, the patient may attempt to compensate for this relative leg length discrepancy by ipsilateral limb circumduction, by hiking the pelvis or by contralateral vaulting that consumes increased energy. The impact of stiff knee includes an increase in the inertial load of the swinging limb (by virtue of its longer moment arm when the knee is extended compared with a flexed knee), thereby potentially influencing c.g. translation during swing limb advancement. An extended knee may become problematic during swing phase clearance and its associated effect on c.g. stability. The impact of stiff knee on functional products of gait includes a reduced range of velocities, single limb postures, elevations and descents, articulation with uneven surfaces and on clearance of a cluttered environment.

Muscles that may generate deforming forces with respect to the stiff knee pattern include rectus femoris, vastus lateralis, intermedius and medialis, gluteus maximus and the gastrosoleus group. An overactive gluteus maximus in swing phase may act to restrain hip flexion and swing limb advancement resulting in a nonswinging extended knee. An overactive calf muscle group, especially if contractured in equinus, promotes compensatory hip flexion in stance...
phase. Ankle equinus causes the knee to hyperextend by preventing forward progression of the tibia during stance phase and the hip flexes to bring body mass forward and compensate for the posterior oblique angling of the tibia. Knee hyperextension of stance phase typically carries over into the sub phase of pre-swing and often into the other sub phases of swing as well. When iliopsoas is weak in swing phase, weak hip motion is accompanied by weak knee flexion as well.

**Adducted (scissoring) thigh**

The patient with adductor spasticity sits with scissoring thighs or walks with a scissoring pattern characterized by both lower limbs exhibiting medial thigh contact throughout the gait cycle. Nevertheless, as a deviant pattern of gait, the scissoring thigh pattern is largely considered to originate in swing phase. Scissoring thighs may interfere with hygiene, dressing, sexual intimacy, sitting and transfers, standing and walking. During swing phase, severe hip adduction interferes with limb advancement (Figure 10). The adducted posturing at the end of swing phase generates a narrow base of support during stance, ultimately making upright balance precarious. When adductor spasticity is complicated by hip flexor spasticity, toileting functions and perineal access are markedly hampered and frequent repositioning of the patient sitting in a chair is required and usually does not work well. The impact of scissoring thighs on translation of the center of gravity may be seen during swing limb advancement and stance limb advancement. Stability of the center of gravity may be challenged by scissoring thighs, which can pose a problem of clearance in swing phase. The adducted position of the lower limb at the end of terminal swing has an impact on subsequent loading and single support in stance. Functional products affected by scissoring thighs include a reduced range of the following: step and stride lengths, velocities, base widths, single limb postures, elevations and descents, articulation with uneven surfaces and changes in direction.

A number of muscles may generate unbalanced forces in swing phase with respect to the adducted (scissoring) thigh pattern. Muscles that may contribute deforming forces include adductor longus, brevis, and magnus, gracilis and pectineus. Muscles that may generate unbalanced forces. Weak muscles may include iliopsoas and sartorius. The patient may adduct the thigh in swing phase by using adductors to compensate for these weak hip flexors. The patient may develop stance phase difficulties after an adduction deviation in swing phase because of the subsequently narrow base width. Control of stance phase stability with a narrow base width of support requires strong hip abductors. If gluteus medius and tensor fascia lata are weak, stance phase stability may be impaired.

**Conclusions**

Gait dysfunction resulting from spasticity, contrac-
ture, and impaired motor control after UMN injury can be quite complex. Kinematic, kinetic, and dynamic polyEMG analysis along with diagnostic selective temporary blocks can help optimize rehabilitation planning and treatment interventions. It is important to clearly identify the muscles involved by a combination of focused clinical examination supplemented by evaluation in the Gait and Motion Analysis Laboratory. The imbalance of muscles can be improved by rehabilitation interventions aided by the use of chemodenervation with botulinum toxin and motor point blocks with phenol.

Patterns of lower limb dysfunction in the UMNS have an impact on the core priorities of gait and upper limb functional use. A number of muscles typically cross major joints of the lower extremity and identifying the actual muscles that contribute dynamically and statically to a UMN deformity is an important key to clinical management of the resulting dysfunctions. Clinical evaluation contributes to the analysis of dysfunction but laboratory evaluation using dynamic EMG is often necessary to identify the particular contributions of offending muscles with confidence. The correct selection of target muscles that contribute to any pattern of dysfunction may serve as a rational basis for interventions that focus on specific muscles including chemodenervation with botulinum toxin, neurolysis with phenol and surgical lengthening, transfers and releases of individual muscles. In a sense, the Bernstein degrees of freedom problem discussed earlier for any given patient is solved through clinical hypothesis development based on the combined findings of the clinical examination and gait laboratory data. Treatment interventions may be considered as empirical tests of these clinically formulated hypotheses.

References