Spasticity
Spasticity

Diagnosis and Management

SECOND EDITION

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We dedicate this book to our families for their unconditional support, and to our professors, colleagues, students and patients who continue to humble us with their strength and challenge us to improve the care of those with spasticity.
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Preface

Spasticity: Diagnosis and Management is the first book solely dedicated to the diagnosis and treatment of spasticity. This second edition has been substantially revised to reflect the significant advances in the treatment of spasticity since the first edition. Our objectives in the development of this second edition were to outline the still-evolving process for the diagnosis of spasticity and the basic science behind its pathophysiology, and to provide updated information on both the measurement tools used for spasticity evaluation and the newest available treatment options. This book remains the most comprehensive guide to diagnosis and management of spasticity.

Over the past 5 years, the focus of spasticity management has moved from interventions on tone to the impact of the spasticity on the lives of patients and caregivers. Additional drugs, including new forms of botulinum toxin, have been reported in large clinical trials and are changing or will, in the future, change treatment paradigms. Comprehensive programs in spasticity management increasingly focus on special populations including children, cancer survivors, and patients in long-term care programs. As a result, this edition addresses new treatment pathways, outcomes, and economics of spasticity care within the larger context of the rapidly changing health care environment.

Divided into four sections, this book is intended to provide both clinicians and researchers up-to-date access on the latest comprehensive treatment of spasticity. Part I includes a general overview with four chapters highlighting why spasticity is important, epidemiology of spasticity and other signs of the upper motor neuron syndrome, and finally ancillary findings associated with caring for the patient with spasticity.

Part II focuses on the assessment tools in diagnosis and management of spasticity. Five chapters include an outline of general overview measurement tools, specific techniques and scales, assessment of the upper and lower extremity, and setting realistic goals for treatment. The revised chapter, “Measurement Tools and Treatment Outcomes in Patients With Spasticity,” includes the Goal Attainment Scale, which is specifically designed to focus on patient-specific outcomes. The newly added chapter, “Techniques and Scales for Measuring Spastic Paresis,” details the use of scales such as the Tardieu. The use of such scales is more common in both patient care and clinical trials. These chapters provide details on the administration of these scales. Taken together, these five chapters provide a comprehensive review of assessment and measurement of spasticity.

Part III provides 11 comprehensive chapters on treatment of spasticity. New chapters include the role of the physical and occupational therapist in spasticity management, the use of ultrasound in guidance of botulinum toxin management, and emerging technologies in the treatment of spasticity. Part III is designed to highlight the changes in the field in the past 5 years.

The final section, Part IV, is devoted to individual diseases involving spasticity and treatment within the context of these conditions. In addition to updated chapters on evaluation, genetics, and spasticity in adults and children with spinal cord injury, multiple sclerosis, stroke, traumatic brain injury, and cerebral palsy, we have added new chapters on more specialized areas including spasticity in patients with cancer, treatment of spasticity in patients in long-term care facilities, and the economics of spasticity treatment.

With the development of effective therapies for spasticity, we originally sought to address the diagnosis and treatment of spasticity in an integrated, clinically useful text. This revised second edition builds on that foundation and integrates recent advances in the field for diagnosis, treatment, and outcomes. The real focus of this book is on providing the most up-to-date, effective, comprehensive, and economical therapy for patients with spasticity. We invite you to explore these pages and join us in our mission to improve the care for our patients with spasticity.

Allison Brashear, MD, MBA

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Thank you to our patients, their families, our colleagues and staff, and our families for their many contributions to this text. This second edition challenges us to improve the diagnosis and care of spasticity in our patients. Thank you to you, the reader, for joining us on this journey. We hope this book inspires you to continue to improve the diagnosis and management of spasticity.

Allison Brashear, MD, MBA
General Overview
Why Is Spasticity Treatment Important?

Allison Brashear and Elie Elovic

Spasticity treatment is important because the increased tone may interfere with the physical functioning of patients. The overarching goal of spasticity management should be to improve the ability of patients to perform active and passive ranges of motion and improve the ability of caregivers to assist patients with disabilities. Increased tone or spasticity is the tightness that patients and/or caregivers report with passive movement of the limb. In more scientific language, spasticity is a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflex. A clinical finding on the neurologic examination, spasticity, together with increased tone, brisk reflexes with incoordination, and weakness, represents the upper motor neuron syndrome.

Regardless of the cause, spasticity causes significant disability. An estimated 4 million individuals are stroke survivors in the United States, and as many as one third may have spasticity with sufficient disability to require treatment. According to the Centers for Disease Control and Prevention, 1.4 million people in the United States sustain a traumatic brain injury each year, and additional patients develop spasticity after spinal cord injury. The result of any brain or spinal cord injury is a variable pattern of increased tone with weakness and discoordination that leads to significant disability in many patients.

The treatment of spasticity relies on the physician’s assessment of the individual together with conversations with the caregiver. Patients’ inability to perform simple activities of daily living for themselves and the adverse effects on the caregiver drive physicians to find ways to decrease tone, build strength, and improve coordination. The team approach is a cornerstone of a successful treatment, and interaction of the patient, the caregiver, the therapist, and the physicians works best to provide a care plan that addresses functional impairment and plots a course to treat the problems.

Spasticity is a clinically relevant medical problem when it interferes with function or care of patients. The evolution of upper motor neuron syndrome may take days to months after a central nervous system injury. Moreover, the presentation in one patient may differ from that of another despite both having similar central nervous system lesions. The lesion alone does not predict the amount or impact of the spasticity. Other factors such as medications, stress, medical illness, timing of therapy, and so on impact the clinical presentation. As a result, each patient must be assessed individually with his or her caregiver, noting the concerns that impair the performance of activities of daily living or other deficits. No matter how much we learn about stroke, traumatic brain injury, multiple sclerosis, and spinal cord injury, the assessment of spasticity and the effect of tone on function will remain unique to each individual patient’s circumstance.

Although neurologic examination is essential for the diagnosis of spasticity, the management of spasticity has many paths for treatment depending on the disability and goals of the patient and caregiver. One patient may benefit from a combination of tools for spasticity, including interventions such as botulinum toxin injections and intrathecal baclofen, whereas others may require a more conservative route such as splinting or oral medications. The informed physician should know how to assess the amount of spasticity, determine the functional limitations it creates, and then be able to develop a management plan for that individual patient.

How to assess the complicated picture of spasticity and when to intervene are the focus of this text. Our coauthors define for you why spasticity is important and detail the diagnosis and management options, but the goal is to provide the reader with the best options for the physician’s individual patient. As editors, we aim to explore the diagnosis and management of the
many different types of patients with spasticity and to open the door to the different treatment paradigms for patients with spasticity. This second edition has been updated to reflect the newest assessments and treatments.

So why is spasticity important? The answer is because it often causes disability and impairs function in our patients. The goal of this book is to provide the foundation for excellent care of our patients facing these disabilities.
Botulinum Toxin in the Treatment of Lower Limb Spasticity

Alberto Esquenazi

Approximately 700,000 people are affected by a stroke each year in the United States, and there are more than 1,100,000 Americans surviving with residual functional impairment after stroke (1,2). Traumatic brain injury (TBI) is another form of acquired brain injury and continues to be an enormous public health problem in the 21st century even with modern medicine. Most patients with TBI (75%–80%) have mild head injuries; the remaining injuries are divided equally between moderate and severe categories. The cost to society of TBI is staggering, both from an economic and an emotional standpoint. Almost 100% of persons with severe head injury and as many as two thirds of those with moderate head injury will be permanently disabled in some fashion and will not return to their premorbid level of function. In the United States, the direct cost of care for patients with TBI, excluding inpatient care, is estimated at more than $25 billion annually. The impact is even greater when one considers that most severe head injuries occur in adolescents and young adults with long survival rates.

Acquired brain injury affects a person’s cognitive, language, perceptual, sensory, and motor functions (3). Recovery is a long process that continues beyond the hospital stay and into the home setting. The rehabilitation process is guided by clinical assessment of motor abilities. Accurate assessment of the motor abilities is important in selecting the different treatment interventions available to a patient.

Spasticity is a term that is often used by clinicians, and although used frequently, it can have different meanings in its interpretation and presentation. Spasticity is just one of the many positive signs of the upper motor neuron syndrome (UMNS), yet, under the heading of “spasticity,” clinicians often group all positive signs together and sometimes include negative signs as well. Many of these frequently misidentified phenomena fall under the broader heading of the UMNS—a condition that has classically been partitioned into a syndrome of positive and negative signs, including weakness, loss of dexterity, increased phasic and tonic stretch reflexes, clonus, cocontraction, released flexor reflexes, spastic dystonia, and associated reactions or synkinesias.

The issue of terminology is more than semantics and of great clinical importance because, for example, treatment of cocontraction, a phenomenon likely to be of supraspinal origin, will differ from treatment of clonus, a phenomenon of the segmental stretch reflex loop. If clinicians desire a concise, descriptive, utilitarian term that captures the essence of positive UMN phenomena, “muscle overactivity” may be a more suitable term than “spasticity,” especially because the phrase “muscle overactivity” evokes an image of dynamic muscle contraction, the general hallmark of all positive signs of UMNS (4).

Spasticity has classically meant increased excitability of skeletal muscle stretch reflexes, both phasic and tonic, that are typically present in most patients with a UMN lesion. After a UMN lesion, a net loss of inhibition impairs direct descending control over motor neurons. There is also a loss of inhibitory control over interneuronal pathways of the cord that ordinarily regulate segmental spinal reflexes, including stretch reflexes, especially those concerned with antigravity muscles.

Lance characterized spasticity as an increase in velocity-dependent tonic stretch reflexes with exaggerated tendon jerks (5). In Lance’s consensus definition, tonic stretch reflexes referred to the output response of a muscle group that was stretched at different velocities. “Exaggerated tendon jerks” were examples of “phasic” stretch reflexes. In routine practice at the bedside, the two ways of assessing phasic...
and tonic stretch reflexes are tendon taps and passive stretch of a muscle group at different velocities (6,7).

Although positive signs are a common source of clinical concern and are frequently treated, negative signs may be at times more functionally disabling and difficult to address. Negative signs signify loss or impairment of voluntary movement assembly and production, a kind of “muscle underactivity” that, in effect, can be described as phenomena of absence (5,8).

The clinical picture is made more complex by another phenomenon that has not been classically positioned among the positive signs, namely, contracture or what is better described as the physical changes in the rheologic properties of muscle tissue. Contracture is well recognized by rehabilitation clinicians as a major source of disability for patients with UMNS. Ironically, phenomena of absence and phenomena of presence can both provide a context for the development of contracture (9).

**FUNCTIONAL IMPLICATIONS OF SPASTICITY**

Fifty years ago, Nikolai A. Bernstein suggested that the basic problem of motor control relates to overcoming redundant degrees of freedom in our multi-jointed skeletal system, the multijointed limb segments that allow us to interact with the three-dimensional (3-D) world we live in. Commonly, there are multiple “agonists” and “antagonists” for virtually any movement direction. 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. 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, whereas in another, it may be an overactive tibialis posterior (9).

Patterns of limb dysfunction in the UMNS have an impact on the limb utilization for gait or other functional use. A number of muscles typically cross major joints of the extremities, and identifying the actual muscles that contribute dynamically and statically to a UMNS deformity is an important key to clinical management of the resulting gait or upper limb dysfunctions (10,11). Clinical evaluation is useful to the analysis of movement dysfunction, but gait and motor control assessment laboratory evaluation using dynamic electromyography (EMG) and other assessment techniques is often necessary to identify the particular contributions of offending muscles with confidence. The correct selection of target muscles that contribute to any one pattern of dysfunction may serve as a rational basis for interventions that focus on specific muscles, including chemodenervation with botulinum toxin (BoNT); neurolysis with phenol; and surgical lengthening, transfers, and releases of individual muscles.

This concept, namely, identifying which muscles contribute dynamically and statically to upper motor neuron dysfunction, serves as a conceptual basis for this text. 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 (12,13).

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. Before selecting treatment interventions, the clinical team and the patient should explicitly develop functional goals. Functional goals may be classified as symptomatic, passive, or active in nature (9). A symptomatic goal refers to the intent to address clonus, flexor, or extensor spasms, and pain, among others, as some of the targeted goals. Active functions refer to a patient’s direct use of the limb to carry out a functional activity. Passive function has a different context and refers to the passive manipulation of limbs to achieve functional ends, typically through patients’ passive manipulation of the affected limb with the noninvolved limbs or having their caregivers perform the manipulation. Identifying muscles with volitional capacity is important to the achievement of this goal. In broad terms, clinical evaluation focuses on the identification of several factors: Is there selective voluntary control of a given muscle? Is the muscle activated dyssynergically (ie, as an antagonist in movement)? Is the muscle resistive to passive stretch? Does the muscle have fixed shortening (contracture)? In the Gait and Motion Analysis Laboratories, dynamic EMG is acquired and examined in reference to simultaneous measurements of joint motion (kinematics) and ground reaction forces (kinetics) obtained from force platforms. Kinetic, kinematic, and dynamic EMG data augment the clinician’s ability to interpret whether voluntary function is present in a given muscle and whether that muscle’s behavior is also dyssynergic (Figure 11.1). Combined with clinical information, the laboratory measurements of muscle function often provide the degree of detail and confidence necessary to select, aim, and optimize the rehabilitation interventions. In addition, evaluation under the effect of temporary diagnostic nerve or motor point blocks can help the clinician distinguish between obligatory and compensatory limb postures and gait patterns (14).
CLINICAL ASSESSMENT OF SPASTICITY

There are many assessment techniques used in routine clinical examination of the patient with spasticity. Motor control, passive range of motion, manual muscle strength, Ashworth, and Tardeau are examples of such techniques that are frequently used.

For more information, the reader is encouraged to review Chapter 7 of this text. Passive range of motion can be used to determine the available movement for each joint but does not provide information on the cause of limitations if present. Spasticity, muscle overactivity, contracture, or pain can all play a role in limited joint passive range of motion.

Manual muscle testing allows grading of available strength if normal control is present; the grading is done using a 6-point scale, where 5 is a normal rating with ability to resist significant force and 0 is unable to move. In the UMNS, testing of strength may be affected by impaired motor control, the presence of synergistic patterns, and cognitive deficits.

The Ashworth Scale allows assessment of muscle tone; in the Modified Ashworth, the rating uses a 5-point scale. The scale has only been validated for the elbow and requires the movement of the joint through its available range in 1 second. Ideally, the test should always be done in the same position and under similar conditions (15). One disadvantage is that this test does not take into consideration the presence of contracture or other factors that may limit joint motion.

The Tardeau Test was developed in the pediatric population in the mid-1960s. It attempts to assess spasticity by varying the speed of joint motion available from very slow (V1) to as fast as possible (V3). The difference between the parameters permits an estimation of the effect of spasticity (16) (Figure 11.2).

Unfortunately, none of these assessments provides a functional perspective, such as during walking, and cannot precisely determine the source of the problem. Based on our clinical experience, methods based on a functional perspective such as those described in the following can be more helpful in this regard.

The Impact of Gait

Gait is a functional task performed by most humans. The three main functional goals of ambulation are to move from one place to another, to move safely, and to move efficiently. These three goals are frequently compromised in the patient with residual UMNS. Most patients will be able to perform limited ambulation, but they will often have problems because of inefficient movement strategies, the presence of instability or pain due to abnormal limb postures, and decreased safety. Some generalizations can be made about the gait of patients with acquired brain injury. These include a decrease in walking velocity with a reduction in the duration of stance phase and impairment of weight bearing in the affected limb with an
increase in the duration of stance time of the less affected limb (17). Ochi et al (18) reported on differences in temporo-spatial parameters of locomotion among patients with residual stroke and TBI. 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 hyperextension, which may result in knee joint pain or lack of trunk control. This may result in unsafe, inefficient, or painful walking.

During the swing phase, inadequate limb clearance caused, for example, by a stiff knee and inadequate limb advancement caused by limited hip flexion or knee extension may interfere with the safety and energy efficiency of walking. To identify the potential source of the problem and to focus more appropriately on the essence of multifactorial gait dysfunction, formal gait analysis in a laboratory may be required. Combining clinical evaluation with laboratory measurements will increase the degree of resolution needed to understand the common patterns of gait dysfunction in the UMNS (17).

Patterns of UMN Dysfunction

Because of scope and space limitations, only the most common patterns of UMN dysfunction in the lower limb that affect walking have been selected for review in this chapter, and they include: (a) equinovarus foot, (b) hyperextended great toe, (c) stiff knee, (d) adducted (scissoring) thighs, and (e) flexed hip (9,12). The first two patterns are considered to be problematic throughout the gait cycle, meaning that they may interfere with both swing and stance phases. Stiff knee and adducted thigh are predominantly deviations of the swing phase, and both can interfere with limb clearance and advancement. The flexed hip is considered a primary stance phase deviation.

Equinovarus. Equinovarus foot is the most prevalent UMN posture affecting walking and requiring intervention after an acquired brain injury. The foot and ankle are turned down (Figure 11.3A), and toe curling or toe clawing may coexist. The lateral border of the foot is the main weight-bearing surface. Skin breakdown over the metatarsal head may develop from concentrated pressure particularly over the fifth metatarsal head; weight bearing typically occurs when walking but may take place against the footrest of a wheelchair in the nonambulatory population. In walking, equinovarus is frequently maintained throughout stance phase and inversion may increase, causing ankle instability during weight bearing. Limited ankle dorsiflexion during early and midstance prevents the appropriate forward advancement of the tibia over the stationary foot, promoting knee hyperextension. Impairment in dorsiflexion range of motion in the late stance and preswing phases interferes with push-off and forward propulsion of the center of mass, and, combined with reduce walking velocity, results in marked reduction in joint power generation. During the swing phase, the equinus posture of the foot may result in limb clearance problem, whereas the lack of appropriate posture of the foot in the stance phase may result in instability of the whole body. Under the latter presentation, correction of this problem is essential even for limited ambulation or those performing standing transfers.

A number of muscles may generate the abnormal forces with respect to the equinovarus pattern (19). Muscles that can potentially contribute to the equinovarus deformity include the tibialis anterior, tibialis posterior, long toe flexors, gastrocnemius, soleus, extensor hallucis longus (EHL), and the weakness of the peroneus longus, peroneus brevis, and the long toe extensors. As mentioned, dynamic polyelectromyographic (poly-EMG) recordings of the aforementioned muscles in combination with clinical examination provide a more detailed understanding of the genesis of this deformity. Dynamic poly-EMG recordings often demonstrate prolonged activation of the gastrocnemius and soleus complex, as well as the long toe flexors as the most common cause of plantar flexion. Occasionally, the gastrocnemius and soleus may activate differentially, and treatment interventions must take this into consideration. Ankle

FIGURE 11.2 Demonstrating the Tardeau measurement using superimposed images of very slow and very fast PROM. A difference of approximately 20° can be seen between the two measures and indicative of the degree of spasticity.

PROM, passive range of motion.
inversion is the result of the overactivation of the tibialis posterior and anterior in combination with the gastrocnemius and soleus and, at times, the EHL (Figure 11.3B). If the tibialis posterior and anterior are both suspect of contributing to the ankle varus deformity, a decision has to be made about which one of the two muscles is the main contributor. Two approaches are possible for this differentiation. The first one is to use the EMG data and the joint powers obtained as part of the kinematic data in routine gait analysis. The second possibility is a diagnostic tibial nerve block with a short-acting anesthetic. One has to be mindful that reducing the activation of the gastrocnemius–soleus complex will tend to increase ankle dorsiflexion and that tightness of the toe flexors usually becomes more apparent as a result of the

**FIGURE 11.3** (A) Equinovarus left foot posture after cerebrovascular accident. Patient has a large bursa under the base of the fifth metatarsal with complaints of pain and instability during the stance phase. (B) Dynamic EMG data of the subject seen in panel (A) with equinovarus foot posture after cerebrovascular accident. Data are normalized, and vertical line at 62% indicates the initiation of the swing phase. Note overactive tibialis anterior, EHL, and gastrocnemius and soleus complex during the swing phase.

EHL, extensor hallucis longus; EMG, electromyography.
increased dorsiflexion.

Hyperextended Great Toe. Hyperextended great toe is a deformity that is characterized by toe extension throughout the gait cycle, sometimes referred to as striated toe or “hitchhiker’s toe.” Ankle equinus and varus may accompany this foot deformity (Figure 11.4). When wearing shoes, the patient may complain of pain at the tip of the big toe, and during stance phase, abnormal concentration of forces under the first metatarsal head can also produce pain. Toe extension during early and midstance affects weight bearing and can impair gait due to inefficient translation of the center of pressure during late stance phase. It also has an impact on center of gravity stability during stance phase single limb support. EHL hyperactivity is the main deforming force causing great toe hyperextension. A weak flexor hallucis longus may not be able to compensate and offset the extension force of EHL. When equinovarus is also present, analysis of the contributions of tibialis anterior, tibialis posterior, gastrocnemius, soleus, and the long toe flexors needs to be taken into consideration as well. Chemodenervation with BoNT to individual heads of the quadriceps may be considered; caution in dosing is suggested to avoid overweakening of the knee extensor mechanism that may result in stance phase knee instability. If there is uncertainty of the quadriceps’ force-generating capacity during walking, it may be advisable to perform a diagnostic block of the motor branch of the femoral nerve to the knee extensors with a short-acting anesthetic to better determine it. If involvement of the gluteus maximus is evident, this can also be treated with chemodenervation with BoNT (Figure 11.5B). Treatment should also incorporate marching exercises to strengthen hip flexors and stretch quads, and if the patient exhibits an abnormal ankle posture, appropriate interventions for this problem should be implemented.

Adducted (Scissoring) Thigh. This deformity is characterized by adduction of the hip during the swing phase of locomotion. Hip adduction posturing at the end of the swing phase generates a narrow base of support during stance, ultimately making upright balance uncertain. It can also interfere with limb advancement because the adducting swing phase limb may collide with the contralateral stance limb. When adductor spasticity is complicated by hip flexion, other functional activities such as toileting and perineal access can be affected and posture in a

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**FIGURE 11.4** Hyperextended hallux after cerebrovascular accident. The patient complains of pain at the tip of the big toe and pressure under the first metatarsal base.

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11 **BOTULINUM TOXIN IN THE TREATMENT OF LOWER LIMB SPASTICITY**

Chair requires frequent repositioning of the patient (Figure 11.6). Dynamic poly-EMG recordings will frequently demonstrate overactivation of the hip abductors, medial hamstrings, and pectineus. Weakness of the hip abductors and the iliopsoas may also contribute to this deformity because the patient may be attempting to use the hip abductors during walking in a compensatory manner to advance the limb forward during the swing phase.

For the patient with walking capacity, it is essential to ascertain if the hip adductor deformity is obligatory (the result of adductor overactivity) or compensatory (the result of weak hip flexors) because treatment will differ. If the clinician is uncertain, a diagnostic temporary obturator nerve block can be helpful to differentiate the role of hip adduction in an obligatory-versus-compensatory pattern. Longer term interventions, such as chemodenervation with botulinum neurotoxin (BoNT), can be easily carried out after that. Other treatment options, such as a percutaneous phenol obturator nerve block, exist. After the intervention, aggressive stretching of the hip abductors and exercises to strengthen the hip flexors and abductors should be implemented. Electrical stimulation to the hip abductors may be used to promote strengthening (14,20).

**Flexed Hip.** The patient with excessive hip flexion potentially experiences difficulty during walking with negative impact during both phases of the
III TREATMENT OF SPASTICITY

gait cycle (Figure 11.7). In normal gait, the hip is flexed 30° at initial contact but thereafter extends throughout stance phase to about 10°. This deformity can also interfere when standing up from a seated position and during perineal care and sexual intimacy. The UMN pattern of hip flexion is defined as persistent hip flexion throughout stance. Knee flexion deformity may develop as a consequence of severe hip flexion deformity, because in the supine position, the knee flexes to allow the heel to touch the bed. During walking, a shortened contralateral step results from stance phase excessive hip flexion. Excessive hip flexion may also affect single limb support stability of the center of gravity. Dynamic poly-EMG recordings during walking may identify overactive iliopsoas, rectus femoris, hip adductors, or lack of activation of the hip extensors and paraspinals. Interventions to reduce overactive hip flexors (iliopsoas and rectus femoris), chemodenervation with BoNT, to these two muscles can be easily performed guided by electrical stimulation or ultrasound and followed by appropriate rehabilitation techniques including the implementation of hip stretching and attempting long step walking (14).

THE ROLE OF BoNT IN THE TREATMENT OF SPASTICITY

Intramuscular injection of BoNT inhibits the release of acetylcholine at the neuromuscular junction causing muscle weakness. Three steps are involved in the toxin-mediated paralysis: (a) internalization, (b) disulfide reduction and translocation, and (c) inhibition of neurotransmitter release. The toxin must enter the nerve ending to exert its effect. OnabotulinumtoxinA injection is currently approved by the U.S. Food and Drug Administration (FDA) for the treatment of blepharospasm, facial spasm, strabismus, cervical dystonia, hyperhidrosis, and upper limb spasticity. AbobotulinumtoxinA is approved for cervical dystonia and upper limb spasticity by the FDA and IncobotulinumtoxinA is only approved by the U.S. FDA for the treatment of cervical dystonia and blepharospasm. In Europe, Canada, and several countries in Latin America, BOTOX, Dysport, and Xeomin are also approved for the management of cerebral palsy-related and stroke-related spasticity. BoNT-B (formulated as Myobloc® in the United States and NeuroBloc® elsewhere) is approved by the U.S. FDA only for the treatment of cervical dystonia. The reader is encouraged to read other chapters of this text for further information on the topic (21,22).
The purpose of BoNT injections in the management of the UMNS is to reduce force produced by a contracting overactive muscle or muscle group. A reduction in muscle tension can lead to improvement in passive and active range of motion and allows for more successful stretching of tight musculature. More subtly, and more importantly as well, improved motor control and posture may provide the patient with the opportunity to develop compensatory behaviors during functional activities (9). A reduction in muscle overactivity in one muscle or muscle group may have consequences for tone in other muscle groups of the limb through a reduction in the overall effort required to perform movement and/or through changes in sensory information going to the central nervous system from that limb, and may influence more distant muscles or benefit function (21). Finally, the application of external devices such as braces, splints, casts, and even shoes can be facilitated by interventions with chemodenervation.

BoNT is injected directly into an offending muscle. The major advantages in its use are the ease of application that permits its injection without anesthesia and its predictable effect. The most common adverse effect is excessive weakness of injected muscles, which occasionally spreads to nontarget muscles. Given sufficient time, when the patient has a strong response to the paralytic effect of the toxin with excessive weakening, strength will gradually return. No adverse effect on the sensory system are evident with botulinum toxin A (BoNT-A), but pain relief when pain is present has been reported in some patients (22,23). In rare cases, nausea, headache, and fatigue have also been reported. No anaphylactic response has ever been reported due to BoNT-A injection. Depending on the size of the muscle being injected, therapeutic doses of BOTOX have ranged between 10 and 400 U. Because of the potential risk of migration out of the muscle and the possibility of antibody formation, usually doses not greater than 600 U of BOTOX and Xeomin or 1,500 U of Dysport are administered in a single-treatment session (24). This may be sufficient, however, to treat a number of muscles in that one session (22,25). In cases of accidental poisoning, an antitoxin is available. Based on clinical experience and prospective randomize trials, the development of resistance to BoNT-A therapy does not impact the management of patients with muscle overactivity. However, to minimize the risk of immunoresistance, it is recommended that clinicians use the smallest possible effective dose, extend the interval between treatments for at least 3 months or longer, and avoid the use of booster injections in between treatment or mix different toxin brands. Careful documentation of muscle selection, dose, and effects is encouraged to allow for dose or muscle selection adjustment in future treatment cycles if necessary. In our practice, if multiple large muscles are to be injected, we try to concentrate the available dose to a few of them and we may increase dilution and use electrical stimulation before the treatment to enhance the effect and consider using other agents such as phenol injected to other muscles or motor nerves to achieve a complete treatment strategy. With the currently available information, we recommend not injecting BoNT in patients who are pregnant or lactating or have significant medical comorbidities (22,25,26).

Before using BoNT for the clinical management of spasticity, the physician should be knowledgeable about the diagnosis and medical management of the condition producing the UMNS. The physician should be proficient in the relevant anatomy and kinesiology and have a clear understanding of the potential benefits of unmasking function and of the limitations of this therapeutic intervention. Unlike the patient with dystonia where voluntary capacity is not an issue, spastic muscles may very well have evidence or potential for voluntary capacity, which the clinician would like to preserve or unmask, and, therefore, titration of the paralytic effect of the toxin becomes a much more critical factor in its administration (5). The duration of toxin effectiveness ranges between 10 weeks and 4 months. In our experience, patients have received doses greater than 600 U of BOTOX or 1,500 U of Dysport at 3-month intervals for more than 3 years without evidence of loss of effectiveness of the medication. Esquenazi et al (26) have reported an increase in duration of effect over time under a similar treatment paradigm.

The toxin might be an effective tool to “simulate” the effects of surgery to the benefit of the surgeon and patient alike (24).

The strategy of performing a BoNT-A injection is as follows: the skin is prepared by cleaning it with alcohol before insertion of a Teflon-coated, 25-gauge stimulating injecting needle. The electrically conductive inner core of the tip of the needle is used to pass current to the tissues or to record EMG activity; alternatively ultrasound can be used to locate the needle position within the desired muscle. Before or soon after injection, muscle activation should be encouraged to increase the availability of Synaptobrevin 2, a major factor in the uptake and internalization of BoNT-A. As the paralytic effect appears evident, aggressive stretching, muscle reeducation, and functional training are important parts of the treatment protocol (17) (Table 11.1).

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TABLE 11.1

<table>
<thead>
<tr>
<th>Clinical Pattern</th>
<th>Potential Muscle Involved</th>
<th>BOTOX Dose U/Session</th>
<th>Dysport Dose U/Session</th>
<th>No. of Injection Sites</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equinovarus foot</td>
<td>Gastrocnemius</td>
<td>50–250</td>
<td>150–300</td>
<td>2–4</td>
</tr>
<tr>
<td></td>
<td>Soleus</td>
<td>50–200</td>
<td>150–300</td>
<td>2–4</td>
</tr>
<tr>
<td></td>
<td>Tibialis posterior</td>
<td>25–150</td>
<td>50–250</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Flexor hallucis longus</td>
<td>25–75</td>
<td>50–150</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Flexor digitorum longus</td>
<td>25–100</td>
<td>50–200</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Flexor digitorum brevis</td>
<td>20–40</td>
<td>50–100</td>
<td>1</td>
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<tr>
<td></td>
<td>Tibialis anterior</td>
<td>20–120</td>
<td>50–200</td>
<td>1–3</td>
</tr>
<tr>
<td>Flexed hip</td>
<td>Iliacus</td>
<td>50–150</td>
<td>150–250</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Psoas</td>
<td>50–150</td>
<td>150–250</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Rectus femoris</td>
<td>75–200</td>
<td>200–450</td>
<td>2–4</td>
</tr>
<tr>
<td>Flexed knee</td>
<td>Medial hamstrings</td>
<td>50–200</td>
<td>150–450</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Lateral hamstrings</td>
<td>50–200</td>
<td>150–450</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Gastrocnemius (as knee flexors)</td>
<td>50–150</td>
<td>150–250</td>
<td>2–4</td>
</tr>
<tr>
<td>Extended (stiff) knee</td>
<td>Rectus femoris</td>
<td>50–200</td>
<td>150–450</td>
<td>2–4</td>
</tr>
<tr>
<td></td>
<td>Vasti</td>
<td>50–150</td>
<td>150–250</td>
<td>2–4</td>
</tr>
<tr>
<td>Hyperextended toe</td>
<td>EHL</td>
<td>20–100</td>
<td>50–200</td>
<td>1–2</td>
</tr>
<tr>
<td>(striatal)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adducted thigh</td>
<td>Adductor longus/magnus/brevis</td>
<td>75–300</td>
<td>200–500</td>
<td>4–6</td>
</tr>
</tbody>
</table>

EHL, extensor hallucis longus.


**CONCLUSION**

This chapter reviewed the most salient points related to the clinical presentation of UMNS in the lower limb especially as it affects walking. Negative signs of the UMNS include weakness and loss of dexterity. Positive findings such as spasticity, increased phasic and tonic stretch reflexes, clonus, cocontraction, released flexor reflexes, spastic dystonia, and associated reactions or synkinesias can all be summed up in the term “muscle overactivity,” with resulting gait impairment. The clinical picture is made more complex by changes in the viscoelastic properties of muscle and other soft tissues in the form of a contracture. The combined effects of these phenomena are well recognized by rehabilitation clinicians as a major source of disability for patients with UMNS. This syndrome produces upper and lower limb patterns of dysfunction that commonly affect more than one joint at a time and that need to be correlated with their clinical presentation and resulting impairment. Identifying the specific possible source of the deforming force is of the essence for proper treatment planning and intervention. Dynamic poly-EMG and motion analysis can be used to identify the contributors to the specific pattern, and when the technology is not available, thorough careful clinical assessment and selected use of diagnostic nerve blocks can be used to develop a successful BoNT chemodenervation management strategy for this patient population.
REFERENCES


