Preface

Clinical Evaluation and Management of Spasticity aims to be an authoritative resource for practitioners interested in learning more about the management of spasticity. Given the recent advances in treatment, there has for some time been a need for a text devoted entirely to this topic. This is the first such volume.

This book is divided into three sections, each intended to allow one to refer quickly to management strategies for specific neurologic diseases as well as specific treatment modalities.

The first section reviews basic concepts in spasticity, including the physiology and pharmacology of spasticity, the clinical evaluation of spasticity, and an overview of the clinical features and principles in the management of spasticity.

The second section outlines current treatments for spasticity. It covers such topics as physical and occupational therapy approaches, splinting and orthotics, electrical stimulation, orthopedic interventions, nerve blocks, use of botulinum toxin, specific medical interventions, including novel treatments such as tizanidine, and the use of intrathecal medications and neurosurgical interventions.

The final section reviews a coordinated approach to the treatment of spasticity and specific neurologic diseases, including spinal cord injury, multiple sclerosis, stroke, cerebral palsy, and traumatic brain injury. The chapters herein provide a framework for physicians to develop a care plan for patients with these disorders. Spasticity is often a poorly treated manifestation of many neurologic disorders, and it is our hope that this book will become the definitive reference to the management and treatment of spasticity, and will ultimately lead to the improvement of patient care.

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Clinical Features of Spasticity and Principles of Treatment

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INTRODUCTION

Evaluating and treating the patient with spasticity is not easy. The clinician is typically presented with a patient who wants things that current treatments cannot usually achieve: more independence, more strength, and more coordination. Certain aspects, such as the control of painful spasms and the decrease of resistance to passive movement, can now be reliably addressed. However, the ever-increasing array of treatment choices forces the clinician to specify exactly what the goals of treatment are and to compare the possible benefits of treatment with the possible risks.

That entire books are written on the topic of spasticity testifies to the complexity and clinical importance of this disorder. The question of what, how and when to treat have been vigorously debated, and there remain few objective measures of the success of treatment. In this chapter, we will give a general overview of spasticity in neurologic disease and provide some guidelines for its rational treatment.

DEFINITION AND APPROACH TO THE PATIENT

> "Spasticity is a motor disorder characterized by velocity-dependent increase in tonic stretch reflexes ("muscle tone") with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome. . . [There are] other features of the upper motor neuron syndrome resulting from the release of flexor reflexes, such as flexor spasms. . .[and] the negative symptoms such as the pattern of weakness and loss of dexterity caused by withdrawal of the influence of descending motor pathways." *(1)*

Perhaps the most important thing that a clinician considering treating spasticity can remember is that what is really being addressed are the interlocking consequences of the upper motor neuron (UMN) syndrome. It is

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often difficult to dissect out what complaints of the patients are related to hypertonia and what symptoms are related to other consequences of the UMN injury. These other aspects of the UMN syndrome include muscle weakness, decreased dexterity, increased flexion withdrawal reflexes, and clonus. These symptoms can be seen following UMN damage from any cause *(2)*.

Another important consideration is that other neurologic impairments can also be seen in persons with the UMN syndrome. Many disease processes of the central nervous system (CNS) do not simply affect corticospinal tracts, but can also involve other sensorimotor subsystems. Central pain syndromes are common in persons with stroke or spinal-cord injury, and the pain in such cases may exacerbate the symptoms of spastic hypertonia. Hypertonia owing to spasticity may be accompanied by hypertonia owing to other causes, particularly dystonia. Dystonia, the co-contraction of agonist and antagonist muscles, has a mechanism and treatment that differs from hypertonia owing to spasticity. Tremor can also complicate both the diagnosis and treatment of hypertonia owing to spasticity.

A third consideration is that the patient may also be suffering from the complications of spasticity, particularly those involving the musculoskeletal system and skin. Often, these complications are quite treatable, and can form the focus of the efforts of the clinician. Fully addressing these complications may require the services of a multidisciplinary team of physicians, nurses, and therapists to optimally manage spasticity.

SYMPTOMS AND SIGNS OF THE UMN SYNDROME

Neurologic symptoms and signs are usually thought of as being "positive," that is, the presence of a new pathologic phenomenon, or "negative" the absence of some prior normal function. Table 1 outlines the symptoms of UMN injury, and their classification.

Positive Symptoms of the UMN Syndrome

The fundamental disruption in spasticity occurs in the stretch reflexes.

Phasic reflexes are those reflexes that are elicited by a tap on a muscle tendon or body. They are characterized by the force of tap needed to elicit a response, the speed and amplitude of the response, and the presence or absence of the spread of this response to adjoining or contralateral muscles. In UMN injury, tapping on a muscle tendon elicits a response that is faster, of higher amplitude, and that spreads to involve other muscle groups.

Tonic reflexes are manifested to the examiner as the resistance to passive movement across the joint. The classic impairment seen in spasticity is the "spastic catch" phenomenon. As discussed at length elsewhere, this velocity-dependent event causes triggering of muscle contraction, thus lead-

Table 1
Symptoms of Upper Motor Neuron Syndrome

Positive Symptoms
Increased passive resistance to stretch
Flexor spasms
Clonus
Negative symptoms
Weakness
Incoordination
Fatigability

ing to the passive resistance seen in spastic hypertonia. This phenomenon illustrates the properties of velocity dependency that are characteristic of hypertonia because of spasticity *(3)*. A patient with spastic hypertonia may demonstrate little or no hypertonia if the limb is moved through its range of motion very slowly. As the examiner increases the velocity of movement of the limb, the limb begins to manifest the spastic catch. The spastic catch is usually followed by a slow relaxation. If the examiner continues to increase the velocity of passive movements, the limb can display so much hypertonia that further movement becomes impossible—a phenomenon called "blocking." Much less common is the "clasp-knife" phenomenon, where there is initial resistance to movement with a sudden relaxation and reduction in resistance. The clasp-knife phenomenon can be seen in any spastic muscle.

The degree of spastic hypertonia can vary throughout the day. Many people with spasticity find that there are certain times of day when their tone is relatively good and other times when tone can be quite problematic. Most patients attribute this to fatigue, but other exacerbators may play a role. Many patients find that their spasms or hypertonia are made worse in some positions and lessened with other positions.

Painful spasms, both flexor and extensor, are commonly seen. These involuntary contractions of muscle can be provoked by many causes, including position, cutaneous stimuli, the onset of sleep, pain, and infection. Painful in themselves, they may also interfere with sleep, make wheelchair seating or splinting difficult, and even cause further spasms because of autoinduction of a pain-spasm-pain cycle. Although most dramatic in persons with spinal-cord injuries, spasms can occur with any type of UMN injury, including stroke and other forms of brain injury. Such spasms can be so severe that they can throw patients from wheelchairs and break splints.

The pattern of spasms can change as time passes after the injury. For example, in persons with spinal-cord injury, the initial flaccid period is

followed by a period of alternating flexor and extensor spasms. After 6–12 mo, the extensor spasms predominate *(4)*. These spasms can even be useful, because they can sometimes be induced by cutaneous stimulation of the groin or thighs, and be used by person with spinal-cord injury for standing during transfers.

Clonus is a series of rhythmic alternating contractions of agonist and antagonist muscles. This is a common involuntary movement after UMN injury *(5)*. Most commonly seen in the ankle, it can present in any skeletal muscle in the body. Clonus can be simply a curiosity to the patient and onlookers, or it can be disabling. It can prevent the functional use of an extremity, make splinting difficult, or be a distraction or embarrassment to the patient. Clonus is typically elicited only in certainly positions, usually a stretched position of the involved muscle. The contractions can usually be abolished by a change in position. It can be distinguished from a motor seizure by its positional nature, stereotyped appearance, and lack of spread to other areas of the body.

Co-contraction or synergy is another characteristic phenomenon in UMN injury. Persons with anything more than very mild spasticity often find it difficult to isolate the movements of a single muscle. Thus, when they attempt to flex the fingers, they find that they can do this only while simultaneously activating other muscles of the upper extremity. Most commonly in the upper extremity, finger flexion is accompanied by flexion at the wrist or elbow as well as internal rotation and adduction of the shoulder. Voluntary movements in the limbs can also be accompanied by contraction of trunk muscles, in the patient may often assume a twisted or flexed truncal posture. Notable but less troublesome are mirror movements, which are simultaneous movements of the same muscles on the contralateral side of the body. These are thought to represent changes in central motor function in response to acquired nervous-system injury.

Other causes of abnormal muscle tone can coexist with hypertonia owing to spasticity. Most troublesome and difficult to treat is dystonia, which is the simultaneous contraction of agonist and antagonist muscles. These contractions can be quite painful, and can thus exacerbate the hypertonia owing to spasticity. The combination of spasticity and dystonia can be the most challenging aspect of the clinical care of the patient.

Negative Symptoms of the UMN Syndrome

Weakness usually accompanies spastic hypertonia. Early after the injury, there is often a period of flaccidity, typically accompanied by areflexia. In persons with spinal cord injury, this is often referred to as “spinal shock” and in those with cerebrovascular disease, “cerebral shock.” Regardless of

cause, this period of flaccidity lasts from a few hours to a few weeks and then typically evolves into the hypertonic spastic syndrome.

Slowness and incoordination of voluntary movements are common complaints in persons with partially preserved motor function. It is important to remember that slowness and incoordination are not owing to the passive resistance to movement of spasticity, but rather the incomplete and disordered activation of motor units of UMN injury. In most cases, this condition is not treatable. One important exception can be when opposing muscle groups are unequally affected by spasticity (imbalance). For example, in the case when finger extensor tone is so severe that weekend finger flexors cannot overcome the resistance that the finger extensors generate, selective weakening of those finger extensors may allow the patient to develop a usable grip.

Fatigue is also a common complaint of persons with spasticity. Some of this may be because of the increased energy expenditures required to accomplish tasks such as walking or other motor activities. Some may be because of the mental fatigue associated with the requirement to more carefully consciously monitor movements. Patients, even those who seem to have made complete recovery from an UMN injury, often report that simple tasks require much more attention and concentration to accomplish. The contribution of decreased aerobic capacity because of reconditioning should not be overlooked. There may also be a central component, and the phenomenon of fatigue is particularly well known in multiple sclerosis (MS).

Clinical Consequences and Complications of Spasticity

While patients sometimes complain purely of stiffness in body parts affected by UMN injury, the hypertonia is typically embedded within multiple other phenomena.

Peripheral changes seen in the UMN syndrome may also contribute to the hypertonia of spasticity. The elastic and plastic properties of muscle also change with chronic spasticity, presumably making it more difficult for the patient with central weakness to execute muscle shortening and movement of a joint *(6)*. This change in the mechanical properties of the muscle can also contribute to examiner's perception of resistance to passive movement; it is not velocity-dependent.

Positioning of patients in braces, splints, and wheelchairs can be complicated by spastic hypertonia. The plantar flexor tone seen in the ankle may cause difficulties in proper seating of the heel into an ankle-foot orthosis. This tone may require the addition of unsightly straps or metal uprights, the use of more expensive custom-fitted orthoses, and even the use of

expensive highly rigid materials such as carbon fiber. Spastic hypertonia may dictate the use of expensive, complex wheelchair seating systems simply to prevent further contracture or skin breakdown.

Skin breakdown can be common in persons with spastic hypertonia. Poor positioning in wheelchairs, splints, or while lying in bed may lead to skin breakdown over pressure points. Common pressure points include heels, malleolli, sacrum, greater trochanters, and elbows. Loss of range of motion may the associated with skin breakdown in intertriginous areas, particularly the groin and axilla.

Contracture, the permanent loss of range of motion of a joint, is made more likely by the presence of spastic hypertonia. Contractures are areas of stiffness and shortening of soft tissues caused by joint immobilization in severe spasticity, and they can develop within a few days of the onset of weakness *(7)*. Difficult, painful, and time-consuming range-of-motion exercises can discourage the patient's caregivers from fully executing the daily exercises necessary to prevent permanent loss of range of motion.

Changes in mechanical properties of muscle also occur with disuse and the muscle shortening typically seen in persons with spasticity. As the increased tone forces extremities into postures that allow chronic muscle shortening, certain changes in the mechanical and physiological properties occur. When a muscle is in a contracted position, the length of the sarcomeres shorten. Biomechanically, the muscle becomes less efficient because of the shortened lever arm.

UMN injuries do not often cause atrophy of the affected muscle groups. However, some disuse atrophy does occur within a few weeks of the injury. Atrophy may lead to poorly fitting braces and splints. Palpation of muscles often reveals some increased tautness in the affected muscle groups. Fasciculations are not present, but clonus often is.

Differential Diagnosis of Hypertonia

Extrapyramidal disease or injury can cause rigidity, a form of hypertonia that is not velocity-dependent. Both agonist and antagonist muscles are involved simultaneously, thus there is resistance to passive movement in all directions. In some cases, limbs passively placed in a new position may maintain that position; this is called waxy flexibility or lead-pipe rigidity.

Gegenhalten is a manifestation of corticospinal or extrapyramidal system injuries. In this case, spontaneous movements can be quite normal, but when the limb is touched or passively moved, the antagonist muscles stiffen in proportion to the force being applied.

Reflex muscle rigidity is usually seen in response to local pain or irritation. This condition manifests as a muscle involuntarily contracted in a sustained fashion. It can occur with any local injury, including injury to the muscle or soft tissue, cold, or arthritis.

Myotonia is an uncommon condition usually seen in inherited disorders such as myotonic dystrophy, paramyotonia, and myxedema. In this case, increased tone is seen as a result of voluntary contraction. In the "handgrip" test, forceful squeezing of the finger flexors is followed by a very slow release of grip because the patient is unable to suppress muscle tone. A similar finding occurs if the muscle is struck with a reflex hammer: "percussion myotonia."

TREATMENT OF SPASTICITY

Goals of Treatment

The decision to treat spasticity should not be made lightly, and the need to prospectively specify the goals of treatment must be emphasized. All current treatments for spasticity entail costs in time, money, effort, or side effects. The clinician must be realistic about the ability of current treatments to improve the patient's complaints. To prevent misunderstanding, the limitations of current treatments should be reviewed with the patient before initiation.

The available data suggest that current treatments act primarily to ameliorate the positive symptoms of spasticity, including flexor spasms, passive resistance, and clonus. For patients whose symptoms concern primarily the positive symptoms of the UMN syndrome, current treatments can often bring relief, albeit at the price of significant side effects. For example, if the patient is having difficulty with nursing care or positioning of the limb owing to spasticity, then it is appropriate to attempt to decrease the spasticity to prevent complications such as contractures. Treatment of spasticity is also indicated if the patient is experiencing pain owing to spasticity or flexor spasms.

Frequently, the patient presents requesting improvements in the "negative" symptoms of the UMN syndrome, such as weakness and incoordination, which are not clearly related to hypertonia alone. One misconception about the weakness and incoordination seen with the UMN syndrome is that much of it is because of hypertonia, and that if the hypertonia were reduced, the patient would gain improved function. At best, this notion is controversial; most data suggests that weakness is owing to poor activation of motor units, not resistance of muscle to movement caused by hyperactivity of the stretch reflex *(8)*.

Control of Flexor Spasms

Flexor spasms are involuntary muscle contractions that are often painful. They occur in 15–60% of spinal-cord-injury patients, but also occur (less dramatically) in persons with cerebral injuries. The spasms of spinal-cord injury are said to respond better to certain treatments, particularly baclofen, than does spasticity of cerebral origin.

Facilitate Range-of-Motion Exercises and Splinting

One important reason to treat the passive resistance to stretch is to facilitate range-of-motion exercises. Range-of-motion exercises are important in preventing contracture, and anything that helps the patient execute these exercises can minimize or prevent the onset of contracture. Stretching may also decrease tone, at least for a few hours. Many people with spasticity encounter pain as a limiting factor in cooperating with range-of-motion exercises. Others find that simply getting the limb to relax to the point where stretching can actually begin is quite frustrating; people with acquired brain injuries often have low tolerance for frustration. Thus, interventions that reduce passive resistance can lead to better patient compliance with range-of-motion exercises, thus preventing the complications of contracture.

The goal of splinting is preservation of range of motion by maintaining a joint in a stretched position for several hours. Typically, the brace is designed to position the limb so that the joint in question is in the anatomically neutral or, if voluntary movement is preserved, the most functional position. The effectiveness of the splint is dependent on its ability to maintain the extremity in the splint, and oftentimes, straps are required to keep the limb properly seated in the splint. Reduction of hypertonia or decreases in the frequency and severity of spasms will facilitate the use of such splints and casts. Reduction of tone will also decrease the likelihood of skin breakdown at pressure points and decrease the pain associated with maintaining a prolonged stretch.

Appearance

Many persons with UMN injury object to some of the appearance changes associated with the UMN injury. In particular, ambulatory persons with UMN involvement of the arm often find that as they walk or engage in some other physical exertion, their arm will passively flex at the elbow. Some of these people feel that this passive flexion of the arm draws attention to their disability, and will request treatment for prevention. Both the clinician and the patient must consider risks and benefits of treatment.

CONSIDERATIONS IN TREATMENT

Systemic vs Local Treatment

One major choice the clinician faces is the use of local vs systemic treatments. Local treatments are those directed at a specific muscle or nerve. These local treatments include botulinum toxin, phenol neurolysis, or alcohol neurolysis. These local treatments avoid systemic side effects such as sedation or confusion, hepatoxicity, or diffuse weakness. Local treatments can also be preferred when there are focal imbalances of agonist and antagonist muscle tone, or where intervention is desired on only a few muscles. Systemic medications have the advantage of affecting large areas, and are therefore useful in treating widespread conditions such as paraplegia or hemiplegia. Duration of action is shorter, in that systemic medications are fully metabolized within a few days.

Team Approach

Only rarely can spasticity be clinically managed by a single clinician, because the condition usually requires the skills of several disciplines, not to mention the cooperation of the patient and family. While the diagnosis and pharmacologic management of spasticity is initiated by the physician, other interventions will be initiated by therapists, nurses, and others. Management of splints, wheelchair seating, bladder-training protocols, and assistive technology will all be executed by nonphysicians.

Timing of Treatment

Treatment choice will often depend on the length of time the spasticity has been present. Early after a precipitating injury, the motor exam is often changing quite rapidly. The patient's needs in spasticity treatment are also changing quite rapidly. Thus, investing in expensive carbon-fiber braces in the first few weeks after a stroke or spinal-cord injury may not be the best use of resources. Similarly, the use of invasive treatments, including implanted baclofen pump or other surgical procedures, is rarely warranted in the first few months.

On the other hand, even persons months or years out from their injury may have changes in their spastic hypertonia that will require changes in their treatment. Spastic hypertonia can increase over time, requiring treatment that is more aggressive.

Why Not to Treat

Sedation is the most common side effect of oral medication for spasticity. Many persons with spastic hypertonia find that the sedation associated

with most of the oral medications adversely affects their ability to function and leads to an overall reduction in their quality of life. In particular, persons with cerebral injuries are particularly prone to sedation. Dosages of medication must be quite low at first, often below the therapeutic threshold, and can only be increased slowly. Paradoxical agitation or confusion can result from mild to moderate sedation, limiting the total dosage. In such cases, peripherally acting treatments may be the best choice. Dantrolene is less sedating than the centrally acting benzodiazepines or gamma-amino butyric acid (GABA) agonists. Another attractive choice when sedation limits dosing is botulinum toxin.

Weakness is another common adverse event. Weakness is usually dose-dependent, and is particularly prone to occur with dantrolene. Of course, botulinum toxin has as its mode of action the weakening of muscles; generally the duration of toxin-induced weakness is several weeks to several months.

Cost of treatment can range from the rather inexpensive dantrolene or diazepam to the extremely expensive botulinum toxin. Particularly early after injury when motor function is changing rapidly, expenditures on expensive positioning devices or wheelchairs can be difficult to justify. Later, in the chronic phase, resources to pay for the newer, more expensive interventions may be lacking. Many persons requiring treatment for spastic hypertonia have chronic neurologic disease, and may have few resources to pay for ongoing daily, expensive medications. The cost of treatments such as intrathecal baclofen can run into the tens of thousands of US dollars each year. The physician must consider the benefits of treatment and for the alternative uses of these resources.

Hepatotoxicity is particularly associated with tizanidine and dantrolene. Particularly as doses escalate, regular monitoring of liver function must be maintained. In particular, the cerebrally injured patient on anticonvulsants and the hyperlipidemic person on HMGCoA reductase inhibitors should be regularly monitored.

Incontinence can result from systemic treatment of spasticity. Some persons maintain their continence through the maintenance of sphincter tone. The use of systemic drugs to reduce tone may adversely affect sphincter tone, causing or worsening incontinence.

Not All Spasticity is Bad

Some consequences of spasticity can be useful. For example, in many persons with stroke, the weak affected leg is transformed into a “cane” by the extensor tone of spasticity. The use of this “cane” allows for transfers and ambulation. The flexed, adducted position of the hemiplegic arm keeps the arm in a safer position; the position also moves the arm closer to the center of gravity, improving balance.

An even more dramatic example occurs in persons with paraplegia. Some paraplegics tap or pinch their thighs and groin to induce extensor spasms in their legs so that they can use their legs as "pillars of support." This maneuver allows the patient to bear weight for transfers even in the absence of voluntary leg-muscle activity. Abolishing such spasms would make the patient less mobile, not more.

Ablation of this tone will lead to the loss of the ability to transfer or ambulate. Similarly, persons with bladder-sphincter spasm may rely on that spasm to remain dry. Use of medications like dantrolene, which can cause sphincter weakness, may lead to urinary dribbling or incontinence where none existed before.

TREATMENT

Treatment options for spasticity will be discussed in detail throughout this volume, and only a bird's eye view will be described here.

A methodical, step-wise approach works well. Serial examination of the patient is essential. Tone can vary with position, and the patient should be examined in the same position at each assessment. An objective or semi-objective endpoint to assess treatment effect is helpful: such measurable things as range of motion, spasm frequency, pain scales, ability to seat properly in a brace or splint, or the Ashworth scale. Follow-up in the office or by telephone should be regular, and more frequent when treatment is being altered. Reports from caregivers are particularly important in persons with cerebral causes of spasticity who are cognitively impaired.

Exacerbating conditions should be treated aggressively. The first line of defense against spasticity is to remove those factors that exacerbate it. Table 2 outlines common exacerbators of spastic hypertonia. A careful history and physical examination can often elicit modifiable factors to improve the patient's clinical status. Perhaps the most common is pain, often related to the musculoskeletal complications of hypertonia. Contractures, prolonged positioning without frequent position changes, poorly fitting braces, and pressure points are all common causes of pain. Central pain syndromes can also contribute to exacerbating spasticity, and treatment of the syndromes can often reduce the symptomatic complaints of spastic hypertonia.

Wounds and infection can also worsen spastic hypertonia. Wounds owing to skin breakdown or other causes can increase hypertonia and spasms. Areas of skin breakdown or former intravenous catheter sites should be examined for infection. Urinary-tract infections are very common in persons with UMN injury, and often act to worsen hypertonia. Systemic infections, such as pneumonia or intra-abdominal infections, can worsen tone and spasms, and should be searched for carefully, particularly persons with quadriplegia

Table 2
Non-neurological Exacerbators of Spastic Hypertonia

Pain
Fatigue
Anxiety
Wounds
Fractures
Systemic infection
Ingrown toenail
Heterotopic ossification
Urinary retention
Constipation
Intra-abdominal processes: infection, obstruction, ischemia, inflammation

who may not be able to localize the location of infection. Similarly, other intra-abdominal processes such as constipation, gallbladder disease, urolithiasis, or intestinal obstruction can worsen spasticity.

Many patients report increases in tone, spasm, or clonus when they are tired or anxious. Most patients benefit from regular rest; advocates of complementary therapies also advocate relaxation regimens, meditation, and acupuncture.

Physical medicine and positioning interventions are a mainstay in the treatment of spasticity. Frequent and careful stretching maintains range of motion, reduces edema, and is said to reduce tone for several hours. Because of the temporary nature of stretching, further treatment is also instituted in most cases. Splints are used to preserve range of motion by maintaining anatomic position of joints for hours at a time. "Tone reducing" splints have not been systematically evaluated for efficacy superior to that of conventional splinting, but may play a role in certain individuals. Careful wheelchair seating minimizes pain and skin breakdown and therefore prevents exacerbation of spastic hypertonia. Cold compresses and ice bags are traditional treatments for tone, but probably useful only during therapy sessions in which range-of-motion exercises are performed. They may also be helpful for short-term exacerbations, but not longer-term management.

Systemic medications are useful for persons who do not respond to more conservative treatment, particularly persons with spinal-cord involvement. There are four major drugs used currently for spasticity management and each will be discussed elsewhere in detail. One of these, dantrolene, acts in the muscle membrane to decrease the force of muscle contraction. The other commonly used drugs, baclofen, diazepam, and tizanidine, work centrally.

Baclofen has been proven effective in spinal forms of spasticity where it reduces the frequency of flexor spasms and the pain caused by them. Diazepam is similarly effective but has the drawback of sedation in many patients. Tizanadine has similar efficacy to baclofen, and has the advantage of not causing weakness in clinical trials to date. Other drugs are occasionally still used for tone control, including anticonvulsants, antiarrhythmics, and clonidine. However, these second-line drugs have for the most part been replaced by tizanidine and botulinum toxin.

Intramuscular botulinum toxin and phenol neurolysis are useful for reducing tone in small numbers of specific muscles. They are not helpful for large-scale tone reduction in hemiplegia or paraplegia; systemic medications are cheaper, easier, and more effective. However, when the correction of a specific muscle imbalance can improve function, these interventions come to the fore. For example, an occasional patient will have persistent voluntary finger flexion, but this flexion is unable to overcome the spastic finger extensors. In such a case, selective weakening of the finger extensors may restore a gross grasp. For persons with inversion and plantar flexion posturing of the ankle, weakening of ankle invertors and plantar flexors may improve toe clearance and prevent "pistoning" of the heel out of an ankle brace.

Intrathecal medications are becoming more commonly used with the advent of implantable-pump technology and the demonstration of the effectiveness of intrathecal baclofen (ITB) in spinal-cord causes of spasticity. Use in persons with acquired brain injury such as traumatic brain injury or stroke is less well-studied, but is used occasionally by clinicians. Reserved for persons who fail oral medications and local interventions, intrathecal therapy requires a high level of commitment for monthly refills, monitoring for are infection, and adequate pump function.

Surgical procedures, especially dorsal rhizotomy, are being increasingly utilized, though data from randomized, controlled trials of these treatments are sketchy.

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