Spasticity

Management with a Focus on Rehabilitation

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Either injury to the brain or spinal cord can cause spasticity. The condition develops gradually, reaching its maximum extent long after the initial injury occurs. Spasticity can cause pain and abnormal posture, as well as difficulty with movement, self-care, and other activities of daily living.

In spasticity, like a car with faulty brakes, nerves that control muscle tone and motor control no longer exert the same level of inhibition that normally flows from the brainstem. As a result, muscles become hyperactive and reflexes are exaggerated.

Spasticity often occurs following spinal cord injury. Other causes include stroke, traumatic brain injury, or multiple sclerosis (MS). (1)

In recent years, this medical classification has been systematically studied regarding its pathophysiological mechanism, the onset time after the injury, the severity of occurrence, the impact on the individual's functioning and therapeutic intervention.

Following injury to the brain or spinal cord, a variety of clinical manifestations may appear that are described as negative or positive evidence of damage to the upper motor neuron. (2)

An upper motor neuron (UMN) lesion is characterized by both these positive and negative phenomena (Table 1), which differ in their pathophysiological basis and respond variably to treatment. The positive phenomena are "phenomena of presence" of involuntary focal or generalized muscle overactivity and expressions of generalized movement disorder. Their manifestation is sudden, unforeseeable, and characterized by intense symptoms. The negative phenomena are "phenomena of absence" that reflect an inability to move voluntarily (i.e. muscle hypoactivity), resist treatment, and result in a more severe neurological disorder. (3)

Spasticity has many causes and courses, so timing of therapy is important

Table 1 describes the positive and negative aspects of upper motor neurone Syndrome (UMNS).

TABLE 1
The positive and negative findings of upper motor neuron syndrome (UMNS)

Positive findings	Negative findings
Spasticity	Loss of dexterity
Spastic dystonia	Atrophy
Dystonia	Loss of coordination
Clonus	Loss of voluntary movement
Athetosis	Muscle weakness
Primary reflexes	Fatigue
Babinkski sign	
Rigidity	
Synergias	
Co-contractions	
Synkinesias	
Associated contractions	
Myelic automatisms	

Spasticity can be regarded as one of the positive features of the upper motor neuron syndrome and should not be confused with other positive features like clonus, automatic movement, etc. Every manifestation of upper motor neuron syndrome may be independent of another finding of UNMS. (4)

Since there are many causes and courses for development of the disease, to provide a patient with the right treatment at the right time, the medical provider will examine several things: (a) The nature of the spasticity, (b) how it differs from other muscle tone disorders, (c) its development related to the site and degree of injury, (d) its changes over time, (e) its course throughout the day and during sleep, (f) the presence of other symptoms such as pain, and (g) how the intensity changes due to touch or pressure, such as stroking a foot or having a full bladder.

Spasticity Definition and Specific Traits

The definition of spasticity has varied over time based on developments in laboratory and clinical investigations.

First, spasticity was described by neurologist James W. Lance, MD in 1980 as a motor disorder with increased muscle tone and involuntary jerking of tendons (the flexible cords that connect joints). In his words, "Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex, as one component of the upper motor neurone syndrome." (5)

Nine years later, Robert R. Young, MD introduced the term ‘‘spastic paresis,’’ which includes weakness, loss of dexterity, and trouble controlling muscles. He termed it "a motor disorder characterised by a velocity dependent increase in tonic stretch reflexes that results from abnormal intra-spinal processing of primary afferent input". (6)

The European working group EUSPASM defined spasticity as “disordered sensorimotor control resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles.” (7)

Since spasticity has sensory and motor aspects, the condition is a “sensorimotor” phenomenon in which automatic movements appear in response to sensory input. Muscle activity becomes overactive as the existing passive muscle stretch increases velocity. Changes occurring in the spinal cord also play a role. Diffuse injuries in the central nervous system (CNS) result in loss of descending inhibitory commands, and in abnormal impulses. This disordered activity arises at several points along the nervous system’s stretch reflex pathway. With loss of descending inhibitory (reticulospinal) influences, there is exaggerated excitability of dynamic gamma neurons and alpha motor neurons. Other spinal tracts such as the vestibulospinal and rubrospinal tracts become more active. Damage at more that one point in the central nervous system can lead to spasticity; essentially, spasticity can result from injury to the cortex, basal ganglia, thalamus, brainstem, cerebellum, central white matter, or spinal cord. (8)

Spasticity is not a static phenomenon but changes continuously throughout the day, even during sleep, depending on the presence of pain or other irritants such as inflammation, urinary stones, or infection, as well as general factors such as emotional states or a woman’s menstrual period.

Muscle overactivity can go beyond spasticity. One example is upper motor neurone (UMN) syndrome, where spasticity is present along with problems in normal movement, posture, and balance. The movement problems may include repeated rhythmic contractions (clonus); excessive muscle tone at rest that can lead to deformed joints and postures (spastic dystonia); exaggerated tendon reflexes; abnormal toe extension in response to touch; and muscle spasms. (9)

In its own right, spasticity is a chronic condition defined as “disordered sensory-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles” with postural limb changes. (10)

Even though spasticity should not be confused with movements that occur in upper motor neurone syndrome, such as clonus, their presence interferes with attempts to complete a normal motion. During a voluntary movement, unwanted spontaneous movements often appear, such as spasmodic flexing or extending of a limb, muscle stiffness, exaggerated movement, an unintended motion, and/or associated reactions. These movements are stereotypical, massive and irregular with no functional importance.

As spasticity develops or increases, these “immature” motor patterns tend to become activated, especially when a patient attempts to move, and they negatively affect normal movement, posture, and torso balance.

In injury to the central nervous system, there is a combination of involuntary muscle overactivity, i.e., "spasticity", and the emergence of immature movements leading to the so-called "pathological motor syndrome."

Pure spasticity may be seen when an arm or leg appears to "catch" momentarily when a relaxed limb is quickly moved – there will be a stiffening and then relaxation so the motion can be completed. Early in recovery from an injury to the central nervous system, this temporary "catching" symptom may appear as muscles gain tone and move from being flaccid to spastic. The degree of spasticity is usually mild, and full range of motion is retained. (11)

Stroke and Spasticity

In stroke, there are many abnormal pathological patterns in the upper limb postures due to the spasticity of certain muscle groups with respect to other muscle groups.
In the initial stage of stroke, abnormal movements of the hemiplegic upper and lower limbs are formed, which are a combination of muscles' flexion and extension in the effort of movement. (12 –14)

When spasticity is present following a stroke, the abnormal movements of the limbs are due to synergistic movements. They are characterized as primitive movements that dominate reflex and voluntary effort. Their presence interferes with coordinated voluntary movements such as eating, dressing and walking.

In these cases, synergy patterns of muscle flexion in the arm include scapular retraction, shoulder abduction and external rotation, elbow flexion, forearm supination, and wrist and finger flexion; in the leg, the patterns include hip flexion, adduction and external rotation, knee flexion, and ankle dorsiflexion.

Meanwhile, muscle extension synergy patterns in the arm include scapular protraction, shoulder adduction and internal rotation, elbow extension, forearm pronation, and wrist and finger flexion; in the leg, extension patterns include hip extension, adduction and internal rotation, knee extension, ankle plantar flexion and inversion, and toe flexion.

The most common pattern in stroke patients is the "typical arm posture" with characteristic antigravity postural patterns with shoulder adduction, elbow and wrist flexion, and the "typical leg posture" with hip adduction, knee extension, ankle plantar flexion, and foot inversion.

Spastic Dystonia

In contrast to what has been termed pure spasticity, when muscles at rest are overactive without any triggering factor, parts of the body assume abnormal positions, which are a major cause of disfigurement and social handicap. (15) This condition was first described as "spastic dystonia" in the 1960s by neurologist Derek Denny-Brown, MB, ChB. (16)

In time, during spastic dystonia, there are changes in muscle composition, so the muscle becomes shortened and the range of motion limited. Muscles may reach a state of permanent contraction, or joints may become completely immobile.

Secondary Spasticity

The shortening of soft tissues initiates chain reactions, such as triggering a rhythmic contraction during walking, so the lower limbs bend or extend farther than necessary, disrupting normal gait. That occurrence is called "secondary spasticity," or hypertonia. It includes a nervous system component triggered by spasticity, and a biomechanical component arising from changes in the soft tissue.

The constant contraction of "spastic" muscles renders opposing muscles inactive, and the latter grow weak due to disuse.

So, a vicious circle is created; in a typical example, when the muscle that flexes the elbow is permanently contracted, the inability to extend the elbow makes the opposing muscle become weak due to inactivity.

Secondary spasticity in one or more muscles is the commonest condition we encounter in patients with subacute spasticity – spasticity that lasts for a period of time, rather than being short-lived or chronic.

Hypertonia can be treated either with an injection of botulinum toxin to temporarily paralyze the spastic muscle, or an operation to lengthen or transfer tendons. The decision will depend on which of the two components (spasticity or tendon shortening) is prevalent and to what degree. The coexistence of spasticity, either local or generalized, must be taken into account. Usually the spasticity must be managed first.

As an example, a person who can flex their ankle up when the knee is bent, but not when the leg is straightened, will be suspected of having spasticity in gastrocnemius muscles

at the back of the shin. The problem is due to shrinkage of the Achilles tendon.

The Clinical Evaluation of Spasticity

The clinical signs of "spasticity" vary according to the injury type (sudden or progressive), site and extent of lesion (brain or spinal cord), and whether the spinal cord injury is complete or incomplete.

In localized brain injury, such as stroke, or an incomplete spinal cord injury, spasticity creates varying degrees of excess tone or stiffness in different muscles. The opposing, unaffected muscles also influence the position of the joint. Taken together, those factors increase the risk the joint will become deformed.

In contrast, when the brain injury is diffuse or the spinal cord injury is complete, spasticity is much more even throughout the body, causing relatively uniform muscle tone.

At the clinical level, there are similarities but also significant differences in the expression of spasticity due to whether the injury is complete or incomplete, whether sound antagonist muscles are involved during the execution of a movement, whether abnormal movement patterns are present or absent, and whether there is any coexistent damage of the peripheral nervous system or the extrapyramidal system.

Table 2 shows the differences in spasticity after a brain lesion and after damage to the spinal cord, respectively.

TABLE 2

Clinical findings in spastic paralysis of cerebral origin	Clinical findings in spastic paralysis of spinal origin
Presence of spasticity very soon after injury	**Spinal shock. Late appearance of spasticity**
Poor control of head and body movements – abnormal postures	**Massive automatic movements. Myelic automatisms**
Abnormal "models" of movements. Presence of primitive reflexes. Synergies and associated reaction	**Diffuse distribution of spasticity**
Co-contraction	**No co-contraction**
Possible coexistence with extrapyramidal damage	**Possible coexistence with peripheral nervous system damage**
Regional or focal spasticity	**Generalised spasticity**

Another important factor is duration of the condition. This is because repeated abnormal movement over time leads to the development of permanent contractions in adjacent normal muscles (dystonia). Normal muscles become involved in order to balance the body out in a motion that is safe, fast, and acceptable in appearance. (For instance, when spasticity causes "tiptoe" walking, the quadriceps muscle in the thigh may be continually flexed as a result).

There are four stages of clinical examination.

Stage 1: The clinician will note posture and motion as the patient enters the examination room, sits, and lies down. Any muscle atrophy (weakness from disuse) and muscle spasms will be noted.

Stage 2: While the patient lies down, the clinician will test the range of joint motion; note the degree of spasticity; observe the patient's active movement; and test reflexes. Motor control and muscle tone are noted.

Stage 3: While the patient sits, the clinician will test upper-body motor skills.

Stage 4: Body balance is checked while the patient is in an upright position and while the patient walks for a short and longer distance. Whether the patient tires or not is considered of major importance.

Timing and Type of Treatment

It is important to address the issue of proper timing of therapy to manage spasticity.

Early intervention with medication taken by mouth (oral antispasm compounds baclofen and tizanidine or dantrolene) is the method of choice. (17, 18)

Spasticity that is centered on one area or a region of the body may respond to treatment with botulinum toxin type A injected into stiff muscles to temporarily block the contraction. This should be started early after spasticity occurs and before soft tissues begin to shrink. (19, 20)

At a later stage, and even at the chronic stage when abnormal motor patterns are established (such as the typical arm posture of an immobile upper limb), a medical provider can inject botulinum toxin in selective "key muscles" to modify movements that amplify the disorder. (22)

Botulinum toxin injections can be repeated after three months or more, as long as the injections serve a predetermined goal each time. (23, 24)

Repeated injections of the botulinum toxin type A, appear to be safe for the patient if all the injection rules are maintained and the intervals between the new injection is extended beyond three months (25)

The time window (the time between the injury and the beginning of spasticity treatment) and the time plateau (when the treatment will end) will be considered.

If spasticity throughout the body does not respond to medication taken by mouth, or too many muscle groups are involved to safely use botulinum toxin injection, then an implanted catheter and pump are used to deliver baclofen to the spinal cord. The medication is infused into the intrathecal space around the spinal cord, so the treatment is called intrathecal baclofen or ITB.

ITB is a long-term treatment that offers continuous or programmable administration of medication to reduce spasticity, especially in spinal injury and MS patients.

Several assessments precede a final pump implantation. For instance, the patient may receive an injection (bolus test) in the lower back, or be fitted with a temporary drug delivery device. In the following 3-4 hours, the initial effectiveness will be noted. The usually recommended first test dose is 50 micrograms in adults, with a maximum dose of 150 micrograms that should be reached after three days.

While it has been standard practice to wait a year after injury for a pump to be implanted, there are cases when implantation is recommended earlier, if advantages outweigh disadvantages. (26)

For example, for an incomplete spinal cord injury in which significant spasticity limits recovery of voluntary movement, waiting may have the adverse effect of delaying physical therapy rehabilitation while other contractures develop. (27)

The time of intervention cannot be absolute, as it depends on the patient's clinical condition and overall therapeutic plan. (28)

A patient's condition may move through several stages following an injury; the stages of spontaneous neurological "recovery" have been described for both cerebral and spinal cord injuries, including transition from a flaccid to spastic phase.

Seven stages of recovery were described by the Swedish physical therapist Signe Brunnstrom in patients who had stroke-induced loss of movement on one side. In her model, early spasticity management begins during the second stage, which is marked by several factors: "Spasticity appears. Basic synergy patterns appear. Minimal voluntary movements may be present." (29)

In complete traumatic injury of the spinal cord, the transition from spinal shock to spasticity includes four stages, described by J. F. Ditunno, Jr., MD. Spasticity develops during the 4th stage, 1-12 months after the injury, when early management of spasticity will also begin. (30)

Any delays in treating the "spastic syndrome" create problematic motor patterns as the brain and spinal cord respond to an injury, thus increasing rehabilitation time and minimizing end results. The response could be called maladaptive and pathological, since it creates problems that form the basis of a health condition.

Spasticity Treatment and Rehabilitation

Neurorehabilitation comprises four main categories of spasticity management targets:

The first category involves nursing care: (a) Preventing or treating deformation contractures, (b) preventing or treating a tendency to slump over, (c) proper positioning of the body on the bed/wheelchair, (d) easy catheterization of the bladder, (e) easy fitting of mechanical aids, such as a brace, (f) facilitating caregiver work, (g) pain relief, and (h) improving sleep.

The second category centers on improving movement: (a) The unmasking of voluntary movements previously covered by significant spasticity in cases of incomplete lesions, (b) accelerating the "spontaneous" recovery process, (c) modifying the "immature" motor pattern, (d) using new recovery techniques to guide and encourage retraining of existing neural circuits, e.g. such as robotic, mechanized aids, and (e) a new functional pattern in moving and walking.

The third category includes daily life activities: transfers, getting around, dressing, personal hygiene, driving, etc.

The fourth category is about quality of life: (a) independent living, and (b) social and professional reintegration. (18)

The goals set by the rehabilitation team, in cooperation with patients and their families, should guide the therapeutic intervention for reducing spasticity and are a reliable index for a successful outcome. (19)

Please note: *This information should not be used as a substitute for medical treatment and advice. Always consult a medical professional about any health-related questions or concerns.*

For further information see:
WIKISTIM at http://www.wikistim.org – This free-to-use collaborative, searchable wiki of published primary neuromodulation therapy research was created in 2013 as a resource for the global neuromodulation community to extend the utility of published clinical research. The goals of WIKISTIM are to improve patient care and the quality of research reports, foster education and communication, reveal research needs, and support the practice of evidence–based medicine.

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