Module 1: Pathophysiology and Assessment of Spasticity; Goal Setting

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Abstract

This module discusses the pathophysiology of spasticity and the lesions underlying the condition. It considers the clinical presentation of spasticity and outlines the relevant clinical history that should be documented. The positive and negative signs of spasticity are explained. Clinical presentations of spasticity are discussed, and an illustrated table of spastic limb postures details how the muscles involved in each individual's condition may be identified. The main systems for assessing the severity of the condition, the Ashworth Scale, the modified Ashworth scale, and the Tardieu Scale, are explained. The likelihood of spasticity developing following a stroke and the probable long-term outcomes are considered. The value of involving patients in their own treatment regimens, by defining and setting goals, using the SMARTER system is explained, and the need to continually assess and refine treatment with time as the condition progresses is also discussed.

Keywords: Spasticity, pathophysiology, assessment

Learning Objectives

On completion of this module, the learner will be able to:

1. Describe the pathophysiology giving rise to spasticity based on disease, condition, and the location of the underlying lesions
2. Compare and contrast the positive and negative signs of spasticity and predict likely occurrence of spasticity
3. Verbalize key elements in the assessment of a patient's history and current condition
4. Explain different presentations and various spastic limb postures and identify the specific muscles involved in postural abnormalities
5. List the impact of spasticity on patients' quality of life
6. Outline the steps in performing the modified Ashworth scale (MAS) or modified Tardieu scales
7. Demonstrate treatment goal setting with the patient using goal attainment scaling (GAS).

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Pathophysiology

Pathophysiology of spasticity based on disease condition and location of the lesion

The development of muscle over-activity, or hypertonia, is a well-known consequence, resulting from a lesion to the upper motor neuron (or motoneuron) (UMN) pathway. There are various types and clinically distinct presentations of hypertonia. The clinical pattern of motor over-activity is primarily (along with other factors to be discussed) determined by the location and extent of the lesion to the UMN.

An understanding of the basic neuroanatomy and pathophysiology pertaining to the UMN will assist clinicians in providing the most effective treatment options. The focus of this learning objective is one particular positive sign of UMN injury: spasticity.

Over the years, the term "spasticity" has been attributed to a number of signs and symptoms seen as part of the UMN syndrome (UMNS). Its definition has been debated and discussed repeatedly over the past 40 years. The debate has centered on a definition where a precise description is based solely on physiology versus one that is more aligned with the clinical presentations and residual sequelae. Examples of the most commonly accepted definitions are:

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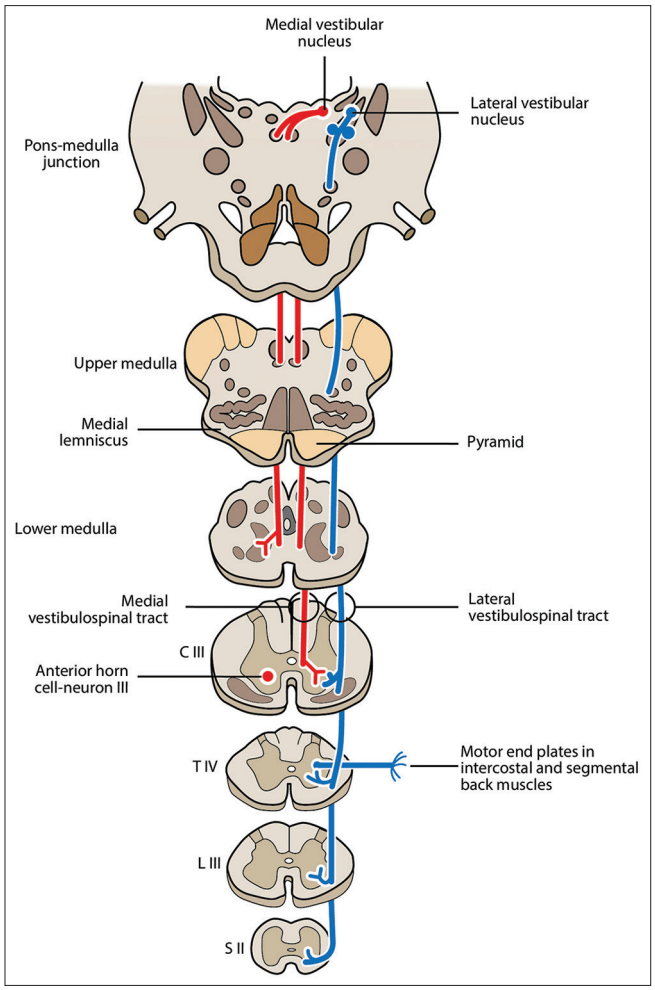


Figure 1: Vestibulospinal and reticulospinal tracts

1. A motor disorder characterized by a 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 UMNS[1]
2. Enhanced excitability of velocity-dependent responses to phasic stretch at rest[2]
3. A disordered sensorimotor control, resulting from an UMN lesion, presenting as intermittent or sustained involuntary activation of the muscles. SPASM project, 2005 – the Support Program for Assembly of a database for Spasticity Measurement.[3]

Being the final motor pathway, the UMN plays an important role in the control of muscle tone and activity. It receives descending supraspinal inhibitory and excitatory fibers that exert a balanced control on spinal reflex activity. "An UMN lesion disturbs the balance of supraspinal inhibitory and excitatory inputs, producing a state of net disinhibition of the spinal reflexes."[4] Spasticity results from this net hyperexcitability of the stretch reflex.

Muscle tone is maintained by a controlled balance on the stretch reflex arc by the inhibitory influence of corticospinal tract (CST) and dorsal reticulospinal tract (RST), and a facilitatory influence (on extensor tone) by the medial RST and, to a lesser extent in humans, by the vestibulospinal tract (VST).

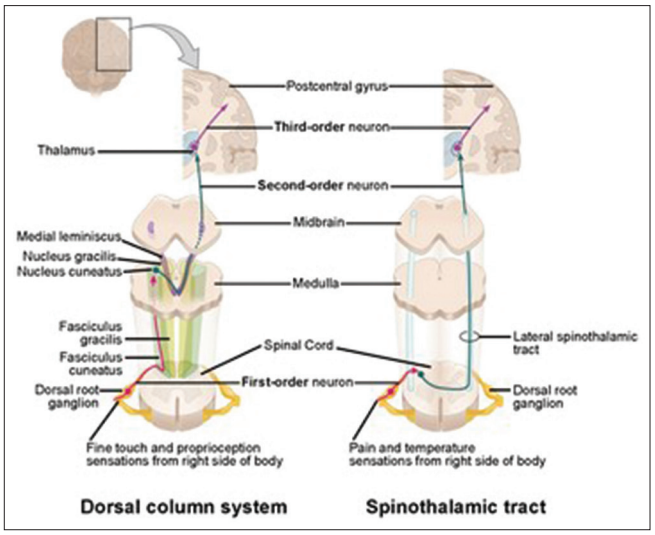


Figure 2: The dorsal column system and spinothalamic tract. This figure is taken from Open Stax Leaning. Download for free at http://cnx.org/content/col11496/latest/

The four descending pathways that are important in spastic paretic syndrome are arranged as follows in the spinal cord: lateral funiculus contains CST and dorsal RST, while anterior funiculus contains VST (that has lesser role in human spasticity)[5] and medial RST (in proximity with medial longitudinal fasciculus).[6]

The positive features of UMNS are probably more related to damage to the parapyramidal motor pathways with brainstem origin than to the pyramidal tracts.[5]

Spasticity occurs due to a hyperexcitability of the stretch reflex. Hyperexcitability occurs from an imbalance of descending inhibitory signals from the dorsal RST and the excitatory signals from the medial RST and VST. Spinal stretch reflexes are mediated by Ia afferents and involve muscle spindles whose excitability is controlled by the gamma efferents. No evidence of muscle spindle hypersensitivity due to increased gamma efferent drive has been found; however, altered intraspinal processing and peripheral muscular changes can also contribute to spasticity [Figures 1 and 2].[6]

Positive and negative signs and symptoms of the upper motor neuron syndrome

The functional impairments seen in patients with spasticity occur due to three main processes: weakness, biomechanical changes (soft tissue stiffness, muscle shortening, tendon contracture), and muscle over-activity through hyperexcitability or loss of inhibition. These can be grouped as positive and negative signs and symptoms [Table 1].

Spasticity is characterized by velocity-dependent increase in stretch reflexes along with exaggerated tendon jerk responses and increased muscle resistance to passive stretch. These become more pronounced as the speed of the applied stretch increases.

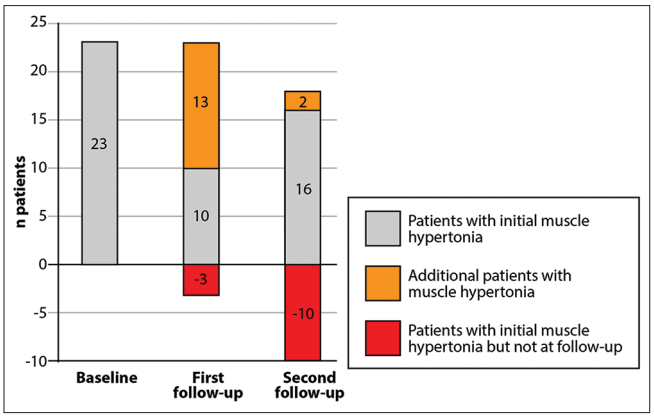


Figure 3: Development of muscle hypertonia during follow-up[8]

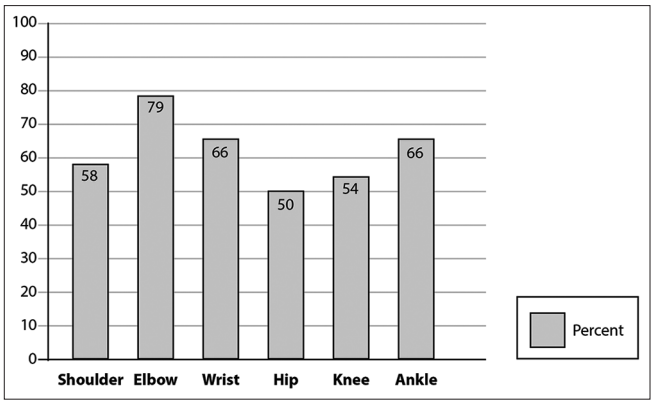


Figure 4: Localization of spasticity 6 weeks poststroke[8]

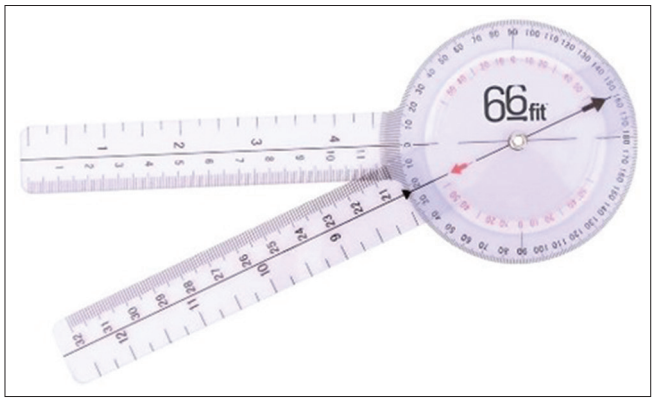


Figure 5: A goniometer

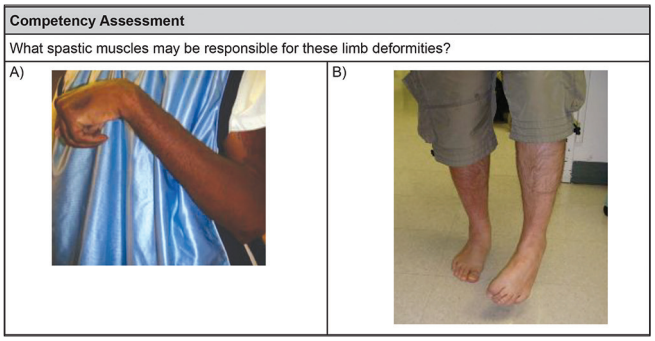


Figure 6: Patient images for Competency Assessment 2, question 1

Table 1: Positive and negative signs of functional spasticity

Positive signs (abnormal behaviors)	Negative signs (performance deficits)
Spasticity	Weakness
Spastic dystonia	Paralysis/paresis
Co-contraction	Decreased dexterity
Clonus	Fatigability
Hyper-reflexia	
Release of primitive reflexes	
Dystonia	
Increased cutaneous reflexes	
Associated reactions	

Spastic dystonia displays tonic muscle contraction at rest and is present in the absence of passive stretch, spinal reflex activation, or voluntary effort. Spastic dystonia is sensitive to stretch and length of muscle (although not dependent on stretch reflex), as described by Denny-Brown.[7] It provides a significant contribution to limb deformities, muscle shortening, and disfigurement.

Co-contraction is due to simultaneous activation of agonist and antagonist muscle groups during voluntary movement. It results from the failure of reciprocal inhibition at the level of either the spinal cord or the cortex.

Clonus is a low-frequency (6–8 Hz) rhythmic oscillation generated as a result of a rapid stretch of a muscle, which may also be triggered by cutaneous stimuli or voluntary effort.

Mass synergy patterns are primitive movements that dominate reflex and voluntary effort and interfere with coordinated voluntary movements, for example, flexion of the upper limb and extension of the lower limb in a stroke patient.

Associated reactions include involuntary activity in one limb that is associated with a voluntary movement effort made by other limbs. Associated reactions may be due to disinhibited spread of voluntary motor activity into a limb affected by a UMN lesion.

- Examples
  - Progressive flexion of the hemiplegic elbow seen as a stroke patient walks
  - Action-induced spastic dystonia: an overflow phenomenon associated with voluntary movements, for example, knee extension and ankle plantar flexion seen on the hemiparetic side in a stroke patient which occurs upon standing from a seated position or with walking, although this posture is not present at rest
  - Imitation synkinesis: a motor response performed in the unaffected extremity will elicit the same motor response in the hemiparetic limb
  - Flexor synergy of a hemiparetic arm during yawning.

Flexor and extensor spasms are caused by abnormal sensorimotor reflexes where a decreased inhibitory stimulus

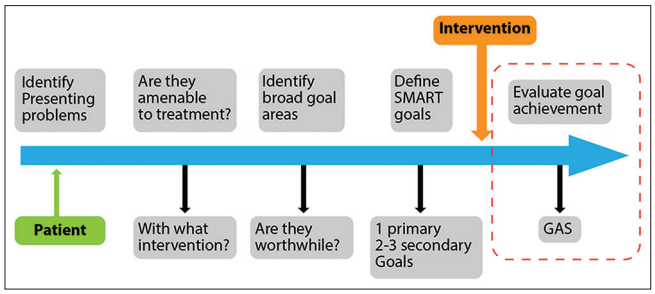


Figure 7: The goal attainment scaling model

results in a disinhibited reflex with increased afferent stimulation. For example, flexor spasms seen after spinal cord lesions result from the disinhibited flexor reflex with flexor muscle contraction across multiple joints. It is also termed "release of flexor reflex afferents."

Prediction of occurrence of spasticity within the first few months after injury or disease onset

Being able to predict the development of spasticity may help clinicians to be more vigilant of complications that need to be assessed and managed in order that further complications can be avoided. It can also assist in long-term planning and resource allocation. Given individual patient and disease variability, a universal prediction model does not exist. Perhaps, the evolution of spasticity is best described in the months subsequent to a stroke. Wissel *et al*. conducted a prospective, observational study in two stroke units and one rehabilitation facility in and around Berlin. A total of 103 stroke survivors were observed. Spasticity, defined as a MAS score of 1 or more, was assessed at 6 days, 6 weeks, and 16 weeks poststroke. They reported a prevalence of spasticity in 24.5% (23 of 94 survivors) at 6 days, 26.7% (23 of 86 survivors) at 6 weeks, and 21.7% (18 of 83 survivors) at 16 weeks.[8]

There were 13 stroke survivors who had spasticity at 6 days but not at 6 weeks. During the same timeframe, three who did not have spasticity initially were found to have spasticity at 6 weeks. At the 16-week follow-up, there were two who did not have spasticity initially but had the condition and 10 whose initial spasticity resolved. It appeared that, in 98% of subjects with poststroke spasticity (PSS), hypertonia emerged about 6 weeks poststroke [Figures 3 and 4].[8]

In considering the factors predictive of PSS, the strongest predictor of moderate-to-severe spasticity (defined as Ashworth >2) is severe proximal and distal limb weakness on acute hospital admission. Reduced sensorimotor function was the most important predictor both for any and severe spasticity 12 months poststroke.

A best predictor model suggests that any spasticity can be predicted by 10 days poststroke and that spasticity 4 weeks poststroke is a significant predictor of severe spasticity.

Factors predictive of PSS are summarized in Table 2.[8-12]

When instigating early treatment, clinicians should consider the nature of the spasticity and the likelihood that it will resolve or be a long-term problematic condition. Decisions should always take into consideration the evolution of spasticity.

Competency Assessment 1

The answers to these questions can be found at the end of this module before the references.

1. What are the three main processes that lead to functional impairments seen in an UMN injury?
2. A patient had a left subcortical stroke 4 weeks ago and is now ready for discharge. He has spasticity of the right elbow flexors and plantar flexors (both scored as "3" in the MAS) and is nonambulatory. Hemiplegia has persisted. His spouse asks you what will happen to the elbow and ankle spasticity in the near future.
3. Describe the clinical features being exhibited by this patient. [https://www.jisprm.org/articles/2022/5/5/images/IntJPhysRehabilMed_2022_5_5_3_347807_sm21.mp4].
4. A 75-year-old male presents for rehabilitation 2 weeks after sustaining an ischemic right middle cerebral artery (MCA) artery cerebrovascular accident (CVA) with left hemiparesis. His examination is noted as having left arm movement at the elbow limited to a flexor synergy pattern and hyper-reflexia at the biceps tendon. His tone using the Modified ashworth scale (MAS) in the left arm is significant for a 2 for the elbow flexors and 1+ each for the wrist and finger flexors. As you are formulating a treatment plan, what other signs of the UMNS do you anticipate to encounter which may interfere with functional improvements as motor recovery progresses?
5. A stroke patient reports that every time, he yawns his hemiparetic elbow is able to flex, although he is unable to flex the elbow on command. The patient is describing what phenomenon?
    1. Spastic dystonia
    2. Associated reaction
    3. Spasticity
    4. Mass synergy pattern.

Assessment

Historical and medical information specific to spasticity across diagnoses

Evaluation of the patient with spasticity should start with a full history.[13] The items to be covered should include:

- History of present illness
    - Detailed description of symptoms of spasticity, such as characteristic posture, temporal nature, triggering and relieving factors, and significance and severity as measured by its impact on mobility and activities of daily living (ADLS)
    - Spasticity onset and progression[14]
    - Associated symptoms, such as pain.
- Review of systems and medical history
    - In addition to obtaining information on medical comorbidities, other important elements that may

Table 2: Factors predictive of poststroke spasticity

Risk factor	Time of onset	Time and degree of spasticity development
Severe arm paresis[9]	Baseline (2-10 days poststroke)	Spasticity by 1 month
Increased muscle tone (MAS >1)[8,10]	Baseline (1-14 days poststroke)	Spasticity by 12-24 weeks poststroke
Low BI score[11,8]	Baseline (1-4 days poststroke)	Severe spasticity (MAS >3) by 12-24 weeks poststroke
Moderately increased muscle tone (MAS >2)[8]	Baseline to 6 weeks poststroke	Severe spasticity by 12–24 weeks poststroke
Hemihypesthesia[11]	Baseline (1-5 days poststroke)	Spasticity by 6 months
Severe paresis[11]	Baseline (1-5 days poststroke)	Spasticity by 6 months
Low EQ-5D score	Baseline (1-5 days poststroke)	Spasticity by 6 months
Paresis[8,11]	Any time point poststroke	Spasticity by 6 months
Low day 7 BI score with early arm or leg weakness[12]	Baseline (7 days poststroke)	Spasticity by 12 months
Low day 7 BI score with left-sided weakness and positive smoking status[12]	Baseline (7 days poststroke)	Severe spasticity by 12 months
Hemispasticity[12]	4-12 weeks poststroke	Permanent spasticity

MAS: Modified Ashworth scale, BI: Barthel index, EQ-5D: Standardized instrument for health-related quality of life

affect assessment and treatment include, but are not limited to, the following:

- Cognition (i.e., cognitive impairment)
- Mood disorder
- Liver disease
- Bowel disorders, such as constipation
- Bladder continence
- Coagulopathy.[15]

- Functional limitations influenced by spasticity
  - Relevant family history
    - A history of neurological disease, such as hereditary spastic paraplegia
  - Relevant general information
    - Residence (domicile, facility-based long-term care services including assisted living, nursing homes, and continuing care community)
    - Patient's family support (i. e., caregivers)
    - Economic status
    - Health insurance.

The radiological features of the lesions leading to spasticity must be documented since a better understanding of the relationship between the brain lesion profile (lesion location and volume) and the presence and severity of spasticity may help early identification of those patients with higher risk of developing spasticity and those who may particularly benefit from preventative and therapeutic strategies.

Brain lesion characteristics correlate with poststroke functional outcome, motor recovery,[16,17] and gait.[18] Damage to the corona radiata and internal capsule has been associated with poor recovery, whereas recovery was linked with lesions sparing the motor cortex.[16,19]

Different studies have reported an association between poststroke upper limb spasticity and lesions involving subcortical structures.[20,21] Injuries to the insula, thalamus, basal ganglia, and white-matter tracts (i.e., internal capsule, corona radiata, external capsule, and superior longitudinal fasciculus) were found to be significantly associated with severe spasticity.[20,21] Lesion volume was found to be positively correlated with spasticity severity.[21]

Damage to the basal ganglia might contribute to spastic dystonia,[17] while Bertoni *et al*. showed that subjects with multiple sclerosis (MS) developing spasticity have three main lesion patterns: small lesions in the genu or posterior limb in the internal capsule, lesions in the rostral brainstem, or extensive lesions in the callosal radiation.[22]

Previous or current treatments for spasticity should be considered, including:

- Rehabilitation interventions – stretching: passive, active, static/dynamic splinting, serial casting;[23] vibrotactile stimulation: whole-body vibration technique,[24] segmental or focal vibration;[25] electrical stimulation: transcutaneous electrical nerve stimulation,[26,27] functional electrical stimulation,[28] extracorporeal shock wave therapy[29]
- Medications – oral medications, botulinum toxin (BoNT), chemical neurolytic agents, and intrathecal drugs
- Surgical treatments (orthopedic procedures: e.g., tendon transfers, muscle/tendon lengthening, tenotomy, joint stabilization; neurosurgical techniques: e.g., rhizotomy, peripheral neurotomy).

For patients in whom spasticity is worsening, it is important to look for the triggers that can increase spasticity [Table 3].

Differences in clinical presentations of spasticity of cerebral versus spinal origin

Clinical presentation

Clinically, spasticity may be of different types due to involvement of descending pathways.[30] There are clinical differences between spasticity of supraspinal (or cerebral) and spinal origin, most of which can be understood by the location and the extension of the UMN lesion. It is the mixing and matching of lesions that leads to a variety of clinical syndromes.[4]

The problem is made difficult by the fact that individual patients have lesions affecting different pathways to different extents and that the subsequent adaptations in the spinal networks may vary considerably. It is likely that spasticity is not caused by a single mechanism, but rather by an intricate chain of alterations in different interdependent networks.[30]

Table 3: Triggers that can increase spasticity

Physiological	Psychological	Environmental	Pathological	Iatrogenic
Pregnancy, posture, circadian rhythm, menstrual cycle	Mental and emotional stress	Cold weather	Disease progression (e.g., MS or development of traumatic syringomyelia after spinal cord injury)	Removal of antispasticity medications
		Tight clothing or braces	Bladder-related issues (i.e., urinary tract infections or calculi), bowel-related issues (e.g., constipation), hemorrhoids, deep vein thrombosis, fever, skin conditions (i.e., pressure ulcers or skin infections) or chest infections	Failure of intrathecal baclofen pump
			A new disease process that may present initially with spasticity	
			Other example – heterotrophic ossifications and painful joints	

MS: Multiple sclerosis

Spasticity of cerebral origin

In cortical and internal capsular lesions, the controlling drive (corticoreticular pathway) on the inhibitory center in the medullary brainstem (ventromedial bulbar reticular formation) is lost and so, in the absence of the inhibitory influence of the dorsal RST originating from this center, facilitatory action of medial RST becomes unopposed. This results in spastic hemiplegia with antigravity posturing, but flexor spasms are unusual.[30]

Damage to the basal ganglia might contribute to the spastic dystonia component, which is common in patients with hemispheric lesions. The basal ganglia play an important role in motor control: they have bidirectional connections with the primary motor cortex, premotor areas, and supplementary motor areas through basal ganglia–thalamocortical circuits.[20]

Spasticity of spinal origin

Incomplete (partial) myelopathy involving lateral funiculus (e.g., early MS)[31] may affect CST only to produce paresis, hypotonia, hyporeflexia, and loss of cutaneous reflexes. If dorsal RST is involved, in addition, unopposed medial RST activity then results in hyper-reflexia and spasticity (similar to cortical or capsular lesions), the latter being marked in antigravity muscles to produce paraplegia in extension. Extensor and flexor spasms may occur (due to hyperexcitability or disinhibition of flexor withdrawal reflex and extensor reflex, respectively), the former being more common.[30] Paraplegia in flexion is also possible if flexor reflex afferents get stimulated by factors such as pressure sores.

Severe myelopathy with involvement of all the four descending pathways produces less marked spasticity compared to isolated lateral cord lesion because of lack of unopposed excitatory influences of medial RST and VST. The latter factor is also responsible for lack of extensor hypertonia, and in the presence of release of flexor reflexes by dorsal RST lesion, it helps to produce paraplegia in flexion.[30]

Isolated dorsal RST involvement with CST sparing (proved pathologically and electrophysiologically)[32,33] may explain marked spasticity and spasms with little weakness in many cases of spastic paraparesis.

Isolated anterior cord lesions may produce hyper-reflexia with normal tone.

In patients with chronic motor complete spinal cord injury, significant relationships were noted between spasticity and variables of body composition and metabolic profile. This suggests that spasticity may play a role in the defense against deterioration in these variables years after injury; however, the exact mechanism is yet to be determined.[34] Both types of spasticity may be treated with intrathecal baclofen. One study showed that cortical spasticity and spinal spasticity appear to parallel each other with no significant differences in daily dosing, dosing changes, and mode of delivery of intrathecal baclofen. The significant difference noted within groups for daily dosing over the first 3 years challenges the notion of stable dosing over time.

Focal injections of BoNT/phenol in the upper extremities are an important adjunct therapy for patients with cortical spasticity, even after the placement of an intrathecal baclofen pump.[35]

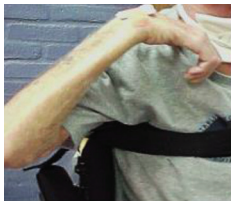
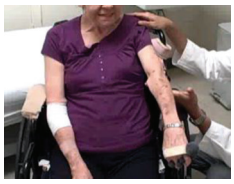
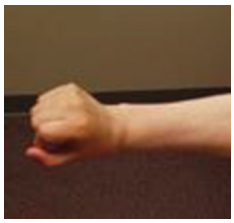
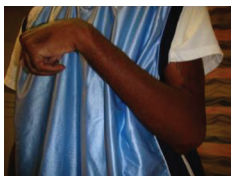
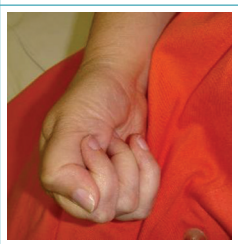
Different clinical presentations and various limb spastic postures

It is important for clinicians to be knowledgeable of functional anatomy to make the best decision regarding which spastic muscles are responsible for common postural abnormalities of the limbs. While instrumented analysis provides more conclusive data, only a few clinicians have access to these sophisticated devices. Hence, the clinician has to assess using examination skills with a foundation of knowledge of functional anatomy. Because several muscles cross limb joints, typically more than one muscle is responsible for a postural abnormality of a limb. Table 4 lists muscles potentially responsible for postural abnormalities.[36]

Muscles involved in various limb spastic postures

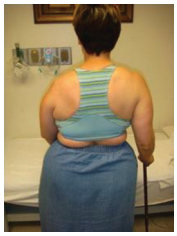
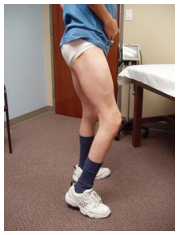
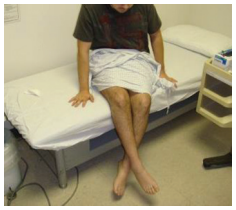
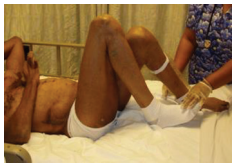
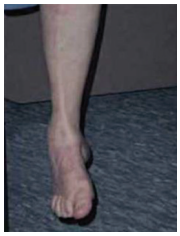
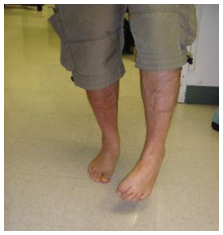
Identifying the muscles involved in any spastic limb posture is crucial to planning treatment. It is important to differentiate between spasticity and weakness since, although they both cause limb deformity, their treatment vary considerably.[37] Spasticity usually involves several muscles and may occur in common postural patterns, whereas weakness may be more generalized.

Table 4: Muscles potentially responsible for postural abnormalities Photographs taken from Francesco and Li 2015.[36]

Postural abnormality		Muscles potentially involved	Benefits of correcting postural abnormality
Shoulder adduction		Pectoralis major Latissimus dorsi Coracobrachialis (especially when shoulder is forward flexed)	Sitting posture Ease of dressing Axillary hygiene Improve balance and symmetry of gait and can sometimes help to reduce unwanted spasticity in the elbow and hand
Shoulder internal rotation		Subscapularis Teres major Pectoralis major and minor	
Elbow flexion		Brachialis Biceps Brachioradialis Pronator teres	Improve flexion deformity Improve reach/retrieve
Elbow extension		Triceps Anconeus	Improve extension deformity Improve ability flex elbow and bring hand close to body axis
Forearm pronation		Pronator teres Pronator quadratus	Improve ability to supinate the forearm Improved functional use of arm and hand
Wrist extension		Extensor carpi radialis Extensor carpi ulnaris	Improve wrist flexion Prevent worsening of finger flexion (tenodesis phenomenon)
Wrist flexion		Flexor carpi radialis Flexor carpi ulnaris Palmaris longus	Maintain palmar skin hygiene
Metacarpophalangeal (knuckle) flexion		Lumbrical	Maintain palmar skin hygiene Improve grasp and release
Finger flexion		Flexor digitorum superficialis (proximal phalanx) Flexor digitorum profundus (distal phalanx)	Maintain palmar skin hygiene Improve grasp and release
Thumb flexion Thumb adduction		Flexor pollicis brevis (proximal) Flexor pollicis longus (distal phalanx) Adductor pollicis	Maintain palmar skin hygiene Improve grasp and release

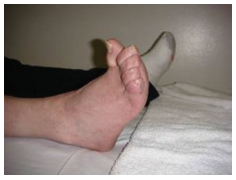
Contd...

Table 4: Contd...

Postural abnormality		Muscles potentially involved	Benefits of correcting postural abnormality
Trunk flexion, lateral		Quadratus lumborum Latissimus dorsi	Improve trunk position and comfort decrease trunk asymmetry during gait
Hip flexion		Psoas Iliacus Rectus femoris	Improve weight bearing Improve gait pattern and seating posture
Hip extension		Gluteus maximus Semitendinosus, semimembranosus, bicep femoris	Increase pelvic mobility and facilitate hip advancement (flexion) during gait
Hip adduction		Adductor magnus Adductor longus Adductor brevis Sartorius Gracilis	Improve “scissor gait” Ease of perineal hygiene and urinary catheterization Easier sexual intercourse
Knee extension		Recturs femoris Vastus medialis Vastuc intermedius Vastus lateralis Gastrocnemius (at certain phases of gait)	Improve knee flexion ability Increase knee flexion during gait Decrease genu recurvatum and associated pain and overloading of knee joint
Knee flexion		Semimembranosus Semitendinosus Bicep femoris Gracilis Gastrocnemius Tensor fascia lata	Seating posture (note potential to worsen sit and stand and standing) Improve knee extension Improved knee stability during stance
Ankle plantar flexion		Gastrocnemius Soleus Tibialis posterior Flexor hallucis longus Flexor digitorum longus	Correct equinus deformity, and foot inversion to allow heel strike Improve fit and comfort of AFO and shoes
Ankle inversion		Tibialis posterior Tibialis anterior Extensor hallucis longus Flexor hallucis longus Flexor digitorum longus	Correct varus deformity, and foot inversion to allow heel strike Improve fit and comfort of AFO and shoes

Contd...

Table 4: Contd...

Postural abnormality		Muscles potentially involved	Benefits of correcting postural abnormality
Small toe flexion		Flexor digitorum brevis (proximal) Flexor digitorum longus (distal)	Decrease pain during toe off phase of gait cycle (brevis and longus) and secondarily decrease foot inversion (longus) Improve fit and comfort of AFO and shoes
Great toe hyperextension		Extensor hallucis longus	Ease of donning footwear and comfort

AFO: Ankle–foot orthosis

Table 5: Aims in treating spasticity

Aims	Examples
Relieve symptoms or reduce impairments	Pain/spasm reduction
	Reducing sleep disturbances
	Reducing disfigurement and improving body image
	Prevention of contracture
	Prevention of subluxation
	Pressure sore reduction
	Increased tolerance for orthotics/shoes/splints
	Reduce abnormal bone growth in children
Improve passive function	ADLs: LE dressing, hygiene, bathing
	Toileting and perineal care
	Wheelchair and bed positioning
	Transfers
	Application of splints, orthoses, and footwear
	Promotion of physical and occupational therapy programs
Improve active function	Mobility (transfers, improved gait pattern)
	Improved balance
	Energy demand reduction
	Wheelchair management and mobility
	ADLs: LE dressing, hygiene, bathing, toileting
	Use of UEs

ADL: Activities of daily living, LE: Lower extremity, UE: Upper extremity

It is important to consider the predominant active muscles in relation to the intended goals of treatment.

Detailed descriptions of the location, origin, and insertion of these muscles are given in "Spasticity Early and Ongoing Management" by Bavikatte *et al*.[38]

The impact of spasticity on quality of life

The disadvantages of spasticity include:

- Body
  - Painful spasms
  - Impeded ambulation
  - Contractures or dislocations
  - Abnormal bone growth
  - Skin breakdown
  - Impairment of respiratory function.
- Activities
  - Interference with ADLS
  - Masked volitional movement.
- Social/societal
  - Sexual dysfunction
  - Fatigue/depression
  - Social isolation
  - Decreased productivity.

It is important that patients and physicians also consider other factors that may be contributing to or exacerbating the spasticity. These include urinary tract infection, kidney stones, menses, bowel impaction or gas, deep vein thrombosis (DVT), pneumonia, wounds or infections, progression of disease, stress, ingrown nails, restrictive clothing, fatigue, psychological factors, and change in temperature or humidity.

The benefits of treating spasticity include an increased stability in sitting or standing, assisting with transfers, prevention of edema, prevention of DVTs, awareness of noxious stimuli, improvement in cough strength, and improvement in venous return.

Effective spasticity treatment relies on contributions from a multidisciplinary team. The patient and family/carers are central to management strategy. Physician input should be provided by a physiatrist, neurologist, neurosurgeon, and orthopedic surgeon. Nurse/nurse practitioners, social workers, physical therapists, occupational therapists, speech/language pathologists, dieticians, and psychologists can all make valuable contributions to the patient outcome.

An important step in planning treatment is goal setting (see the section on "Goal Setting" below) where the patient and the team define the aims of treatment and goals to be achieved.

Some examples of aims are given in Table 5.

Comprehensive spasticity management involves rehabilitation treatments, reduced nociceptive input, focal/segmental treatments (nerve/motor point blocks, tendon transfer/lengthening), and generalized treatments (oral/intrathecal medications, rhizotomy).

Performing the Ashworth or modified Ashworth scale

History

The Ashworth Scale (AS) was originally described by Ashworth in 1964[39] in assessment of carisoprodol in MS. It

consisted of an assessment of resistance to passive stretch. In 1987, Bohannon and Smith[40] developed the MAS which is performed by testing functional muscle groups starting from a shortened position to a lengthened position. They used the scale to assess interrater reliability of assessment of elbow flexor spasticity, testing muscle groups by moving through a range of motion (ROM, from flexion to extension) over approximately 1 s. The scale is given in Table 6.[41]

Table 6: The Ashworth and modified Ashworth scales

Score	AS	MAS
0	No increase in tone	No increase in muscle tone
1	Slight increase in tone giving a catch when the limb was moved in flexion or extension	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part (s) is moved in flexion or extension
1+		Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
2	More marked increase in tone but limb easily flexed	More marked increase in tone through most of the ROM, but affected parts easily moved
3	Considerable increase in tone, passive movement difficult	Considerable increase in tone, passive movement difficult
4	Limb rigid in flexion or extension	Affected part (s) rigid in flexion or extension

Taken from.[41] ROM: Range of movement, AS: Ashworth scale, MAS: Modified Ashworth scale

Table 7: Tardieu scale

- Tardieu scale principles
  - Muscle assessment always performed
    - On a muscle at rest before the stretch maneuver
    - At a reproducible velocity of stretch
    - At the same time of day
    - In a consistent body position for a given limb (seated vs. supine)
    - Other joints, particularly the neck, must also remain in a consistent position during the assessment
  - Velocity of stretch
    - V1 - As slow as possible (slower than the rate of natural drop of the limb under gravity)
    - V2 - The speed with which the limb falls under gravity
    - V3 - As fast as possible (faster than the natural drop of a limb with gravity)
  - X=spasticity angle (threshold)
    - End angle at slow speed X_{v1} minus angle of catch at fast speed Y_{v3}
  - Y=Spasticity grade (gain)
    - 0 - No resistance throughout passive movement
    - 1 - Slight resistance throughout passive movement
    - 2 - Clear catch at a precise angle, followed by a release
    - 3 - Fatigable clonus (<10 s) occurring at a precise angle followed by release
    - 4 - Unfatigable clonus (>10 s) occurring at a precise angle
      - Catch without release: Graded 0 if $X_{v1}=X_{v3}$
      - Catch with "minimal" release: Graded 2 if X_{v3} is consistent and consistently less than X_{v1}
      - Angle 0°=Position of minimal stretch of the tested muscle
      - For Grades 0 and 1, spasticity angle X=0° by definition

From[47]

Several studies have investigated the interrater reliability of the AS and MAS. Meseguer-Henarejos *et al*.[42] performed a systematic review of the MAS and demonstrated that upper extremities had good-to-moderate interrater reliability while the lower extremities had fair-to-moderate interrater reliability.

Looking at specific populations, the AS has shown good reliability in poststroke upper limb spasticity.[43] However, the MAS only demonstrated moderate reliability in hemiplegic patients – both upper and lower extremities.[44]

In spinal cord injury patients, there was fair-to-moderate agreement for AS and generally fair agreement for MAS when assessing the lower extremities.[45] However, moderate-to-substantial interrater reliability of the MAS was shown in the elbow flexors of stroke patients; this was better than seen in assessments of the ankle plantar flexors in these patients.

A drawback of the AS and MAS is that these scales to not account for differences in velocity. They describe resistance to passive stretch of a joint but do not differentiate spasticity from soft tissue or joint changes.[46]

The American Spinal Injury Association has developed a Spasticity Assessment Training e-Program that includes a module designed to educate treaters on how to examine and score a patient's spasticity using the AS and Tardieu method, based on a consensus panel convened and surveyed in 2013. This course can be accessed at: https://asia-spinalinjury.org/learning/.

Performing Tardieu assessment

In 1954, Tardieu *et al*. described the spastic reaction of the limb, which was velocity dependent based on the speed at which the limb was moved. In 2010, Gracies *et al*. translated and compiled Tardieu's work describing the four basic principles of Tardieu.[47]

The first principle is ensuring that the muscle that is being assessed is completely relaxed. This is thought to be theoretically obvious, however, commonly not maintained in practice.

Second was the principle of maintaining a constant position of proximal segments, especially on testing of two joint muscles. As an example, this is important while testing the gastrocnemius at the ankle, ensuring a constant length of the muscle by maintaining a constant angle of the knee, which it also crosses.

The third principle is to identify the angle where passive stretch is arrested, followed by the fourth principle which is to use this angle to differentiate between spasticity and contracture.

The Tardieu scale is described in Table 7.

Table 8: Performing a Modified Ashworth Scale assessment or Tardieu assessment

Principal muscles	Patient positioning	Examiner positioning	Examiner action	Range of motion
Shoulder adductors				
Latissimus dorsi, pectoralis major Secondary movers: Subscapularis, teres minor, infraspinatus, triceps brachii, coracobrachialis	0° of shoulder flexion, extension and abduction, forearm and hand in a neutral, relaxed position	Glenohumeral joint stabilized while grasping the arm just above the elbow	Abduct the shoulder while maintaining 0° of shoulder flexion	90°
Shoulder internal rotators				
Subscapularis, teres major, latissimus dorsi, pectoralis major Secondary movers: Deltoid	0° of shoulder abduction, minimal flexion and full internal rotation	Stabilize elbow in mid-flexion while grasping the forearm at the wrist	Externally rotate shoulder while maintaining elbow mid-flexion and shoulder abduction at 0°	80°
Elbow flexors				
Biceps brachii, brachialis, brachioradialis Secondary movers: Pronator teres, extensor carpi radialis longus, flexor carpi radialis, flexor carpi ulnaris	0° shoulder abduction and flexion, elbowed fully flexed Forearm in full supination to isolate the biceps brachii Forearm in neutral to isolate the brachialis Forearm in full pronation to isolate the brachioradialis	Stabilize patient's anterior shoulder while grasping the arm at the elbow	Fully extend the elbow	150°
Elbow extensors				
Triceps brachii (all 3 heads) and anconeus	0° shoulder abduction, enough shoulder flexion to be able to fully extend the elbow	Stabilize the elbow while grasping the forearm at the wrist	Fully flex elbow	150°
Forearm supinators				
Biceps brachii (long and short heads), supinator	0° shoulder abduction, flexion, and neutral rotation, 45°–90° of elbow flexion and full pronation	Stabilize the arm just above the elbow while grasping the hand	Full elbow supination	80°
Forearm pronators				
Pronator quadratus (humeral and ulnar heads) pronator teres muscle Secondary movers: Flexor carpi radialis	0° shoulder abduction, flexion, and neutral rotation, 45°–90° of elbow flexion and full pronation Elbow fully flexed to isolate the pronator quadratus	Stabilize the arm at the elbow while grasping the hand	Full supination of the forearm	80°
Wrist extensors				
Extensor carpi radialis (brevis and longus), extensor carpi ulnaris (both heads) Secondary movers: Extensor digitorum, extensor digiti minimi, extensor indicis	0° shoulder flexion, abduction and rotation, forearm in in full supination, wrist in full extension	Stabilize elbow while grasping the hand	Full wrist flexion	70°
Wrist flexors				
Flexor carpi radialis, flexor carpi ulnaris (both heads) Secondary movers: Palmaris longus, flexor digitorum superficialis, flexor digitorum profundus, abductor pollicis longus, flexor pollicis longus	0° shoulder flexion, abduction and internal rotation, forearm in in full pronation, wrist in full flexion	Stabilize elbow while grasping the hand	Full wrist extension	80°
Finger flexors (interphalangeal joints)				
Flexor digitorum superficialis, flexor digitorum profundus	90° elbow flexion, forearm in pronation, wrist in neutral/20° extension, all finger joints in full flexion	Stabilize wrist while grasping index, long, ring, and small fingers	Stabilize wrist while grasping index, long, ring, and small fingers	135°

Contd...

Table 8: Contd...

Principal muscles	Patient positioning	Examiner positioning	Examiner action	Range of motion
Finger flexors (MCP joints)				
Lumbrical, dorsal, and palmar interossei Secondary movers: Flexor digitorum superficialis, flexor digitorum profundus, flexor digiti minimi brevis, opponens digiti minimi	90° elbow flexion, forearm in pronation, wrist in full extension, MCP joints at 90° flexion, full extension at interphalangeal joints	Stabilize wrist while grasping index, long, ring, small fingers just distal to the MCP joint	Full MCP extension	90°
Thumb adductors				
Adductor pollicis (both heads) Secondary movers: 1st dorsal interosseous	90° wrist flexion, forearm pronation, thumb in full adduction	Stabilize hand while grasping the thumb	Full thumb abduction	70°
Thumb flexors				
Flexor pollicis brevis and flexor pollicis longus	90° elbow flexion, neutral forearm rotation, full thumb flexion at interphalangeal joints	Stabilize wrist and hand while grasping the thumb distal to the distal interphalangeal joint	Full thumb extension	130°
Hip extensors (isolating gluteus medius)				
Gluteus maximus muscle, long head of biceps femoris, Semimembranosus muscle, Semitendinosus muscle Secondary movers: Adductor magnus, gluteus medius	Patient on side, 0° hip abduction, hip in full extension, knee flexed	Stabilize patient and pelvis while grasping the thigh above the knee	Hip flexion	120°
Hip extensors				
Gluteus maximus muscle, long head of biceps femoris, Semimembranosus muscle, Semitendinosus muscle Secondary movers: Adductor magnus, gluteus medius	Patient on side, 0° hip abduction, hip in full extension, knee extended	Stabilize patient and pelvis while grasping the leg below the knee	Hip flexion	120°
Hip flexors				
Psoas major, iliacus Secondary movers: Rectus femoris, sartorius, tensor fascia lata, pectineus, adductor brevis, adductor longus, adductor magnus, gluteus medius	Patient on side, 0° hip abduction, hip in full flexion, knee flexed	Stabilize patient and pelvis while grasping the thigh above the knee	Hip extension	120°
Hip adductors				
Adductor longus, adductor brevis, adductor magnus, pectineus, gracilis	Supine, 0° hip flexion and abduction, knee in neutral	Grasp foot of lower extremity being tested and stabilize below knee on contralateral lower extremity	Abduct hip	45°
Knee extensors				
Rectus femoris, vastus intermedius, vastus lateralis, vastus medialis	Lying on side with neutral hip and knee in full extension	Grasp foot while supporting thigh under knee	Full knee flexion	135°
Knee flexors				
Biceps femoris, semimembranosus, semitendinosus Secondary movers: Gastrocnemius, gracilis, sartorius	Supine, 90° hip flexion, knee in full flexion	Grasp foot while stabilizing thigh slightly above the knee	Full knee extension	135°

Contd...

Table 8: Contd...

Principal muscles	Patient positioning	Examiner positioning	Examiner action	Range of motion
		Ankle plantar flexors (isolating soleus)		
Gastrocnemius, soleus Secondary movers: Plantaris, flexor hallucis longus, flexor digitorum longus, tibialis posterior, fibularis longus, fibularis brevis	Supine, flex hip and knee 90°, ankle in full ankle plantar flexion	Grasp foot while stabilizing leg at the knee	Full ankle dorsiflexion	70°
		Ankle plantar flexors		
Gastrocnemius, soleus Secondary movers: Plantaris, flexor hallucis longus, flexor digitorum longus, tibialis posterior, fibularis longus, fibularis brevis	Supine, hip and knee in neutral, ankle in full ankle plantar flexion	Grasp foot while stabilizing leg at the knee	Full ankle dorsiflexion	70°
		Ankle invertors		
Tibialis anterior, tibialis posterior Secondary movers: Peroneus tertius, extensor digitorum longus and extensor hallucis longus	Supine, 0° hip, knee, ankle flexion, foot inverted	Grasp distal foot and stabilize leg at the ankle	Full foot eversion	40°

MCP: Metacarpophalangeal

Table 9: Recording GAS without numbers (GAS-light)

	Verbal Rating			Numerical conversion	
At baseline	With respect to this goal do they have?	Some function	☐	-1	
		No function (as bad as they could be)	☐		-2
At Outcome Was the goal achieved?	**Yes**	A lot more	☐	+2	+2
		A little more	☐	+1	+1
		As expected	☐	0	0
	No	Partially achieved	☐	-1	-1
		No change	☐	-1	-2
		Got worse	☐	-2	

Table 10: Goal attainment scaling recoding before treatment

Goal	Category	Subcategory	Baseline	Expected
1ary	Symptoms	Pain	9/10	6/10
2ary	Passive function	Dressing	7/10	3-4/10
2ary	Passive function	Hygiene	7/10	3-4/10
2ary	Passive function	Use of orthosis	1.0 h daily	1.5-3.0 h daily

The advantages of the Tardieu scale over the MAS are that the Tardieu:

- May be able to identify the presence of spasticity better than AS
- May be able to differentiate spasticity from contracture, whereas the AS does not.

In assessing the interrater reliability of the Tardieu scale in cerebral palsy (CP), good-to-excellent agreement between inexperienced and experienced raters across all joints.[47] It was also noted that a 1-day training session substantially improved reliability and there was high agreement between goniometric and visual angle assessments, suggesting that it can be reliably administered without a goniometer.

In spinal cord injury patients, excellent interrater reliability was found for the R1–R2 (spasticity angle) for all muscles tested. This group also found that the assessment of R1 was excellent in terms of interrater reliability in the hip adductors, hip extensors, knee flexors, and knee extensors. However, only fair interrater reliability was seen when assessing at the ankle plantar flexors.[48]

The limitations of the Tardieu are that the spasticity grade (Y) describes a quantifiable reaction of muscle, not necessarily increasing severity of spasticity. The data are nominal rather than ordinal and it is not as extensively studied as the AS and MAS.[49]

Performing the modified Ashworth or Tardieu assessment

Although the two scales rate the patient's spasticity in slightly different ways, the physical examination by the clinician is the same for both.

A summary of important information on performing the AS or Tardieu assessments for different muscle groups is given in Table 8.

Video

Please view this video for more information on performing the MAS measurement. [https://www.jisprm.org/articles/2022/5/5/images/IntJPhysRehabilMed_2022_5_5_3_347807_sm22.mp4].

Available on: https://www.dropbox.com/sh/jl6xudc3evrkkvc/AADvQht-SxMvV1IrxDzKwgcma/Part_2.mp4?dl=0.

ACTIVE RANGE OF OPTION

Perform goniometric measurement of limb spasticity

Goniometers are simple plastic devices that allow measurement of joint angles. By having two rulers joined in way that the angle between them can be altered and measured allows the two ruler scales to be aligned with a joint and the angle formed by the bones determined. By moving the joint and repeated measurements, the ROM may be determined.

Goniometric measurements are frequently used for joints such as elbow, shoulder, and hip [Figure 5].

To perform a goniometric assessment, the clinician should follow this protocol:

- Position the joint in zero position and stabilize the proximal joint component
- The joint should be moved to the end of the ROM (to assess quality of movement)
- The end-feel at the limit of the ROM should be sought and the joint rested at this angle
- The bony landmarks should be palpated
- The goniometer should be aligned with bony landmarks while holding joint at the end of range
- The goniometer reading should be taken and the measurement recorded.

Classically, a standard goniometer for measuring joint ROM is the gold standard in clinical settings because it is portable and relatively inexpensive. However, it has several limitations, making it difficult for clinicians to use. Clinicians need both hands to use a goniometer, making limb stabilization difficult. Thus, the risk of a high measurement error increases.[50]

Video

Please view this video for more information on how to perform goniometric measurements. [https://www.jisprm.org/articles/2022/5/5/images/IntJPhysRehabilMed_2022_5_5_3_347807_sm23.mp4].

https://www.dropbox.com/sh/jl6xudc3evrkkvc/AADuTF7whVT0t96_IDBbwR0Za/Part_1.mp4?dl=0.

Describing the role of diagnostic nerve block in assessing spasticity versus contracture

A nerve block is the application of a chemical substance to a nerve that will interfere temporarily or permanently with conduction along the nerve.[51] There are two types of nerve blocks: diagnostic nerve block (DNB) with anesthetics and therapeutic nerve block with alcohol or phenol. The technique of injection is the same while the drugs injected and the indications are different.

Therapeutic nerve blocks are considered in more detail in Module 2.

The DNB is performed with anesthetics[52] which allows evaluation of how much the lack of ROM, joint and muscle tightness, and joint deformity can be attributed to spasticity instead of muscle or soft tissue rheologic changes. Consequently, DNB can also assist the clinician in diagnosing contractures (that will not respond to chemodenervation or neurolysis) on top of underlying spasticity, identifying potentially undesirable outcomes (e.g., excessive muscle weakness), and appreciating the beneficial effects of pain reduction and improved limb posture on function and hygiene.

The benefits of anesthetic blocks in the evaluation of spastic patients are that they allow differentiation between muscular hyperactivity and contractures and facilitate evaluation of the antagonists in terms of strength, dystonia, and co-contractions. It allows the muscles involved in motor problems to be identified, thus helping identify the muscles to be targeted for treatment with BoNT-A. An anesthetic nerve block can provide information on the likely outcome of treatments such as BoNT-A or selective neurotomy.

Performing a diagnostic nerve block

The DNB procedure consists of injecting a local anesthetic (usually lidocaine 1%–2%) on a motor nerve innervating a spastic muscle. It is performed using a disposable needle for conduction anesthesia coupled to an electromyography (EMG) apparatus or an electrical stimulator. Once the needle is inserted, the nerve is located according to anatomic landmarks, electrical stimulation, or by ultrasonography.[52,53]

When a clinical muscular contraction is still obtained with a low stimulation intensity (1 ms duration and 0.1 mA intensity with a portable stimulator or 0.01 ms duration and 4–10 mA intensity with an EMG apparatus), meaning a close contact of the needle with the nerve, the anesthetics is injected.

A DNB eliminates spasticity after few minutes and lasts for some hours, allowing assessment of the respective contribution of the spastic muscles, the degree of muscle shortening, and the weakness of the antagonistic muscles. It will allow the clinician to determine the potential benefits of performing longer lasting interventions such as chemodenervation or surgery. It also allows the patient to experience the potential benefit of reduced muscular hyperactivity and have a better understanding of what to expect from more definitive procedures.

Sensorimotor (mixed) nerve block (i.e., tibial nerve in case of spastic foot, musculocutaneous nerve in case of elbow flexion, and median and ulnar nerve in case of spastic hand) is the easiest to perform. Due to their size and well-known anatomic location, these nerves are easy to find and target and require a 3 mL dose of anesthetic. However, as such nerves innervate many muscles (i.e., the tibial nerve innervates both soleus,

gastrocnemius, tibialis posterior, and flexor digitorum muscles), it does not provide information about the precise spastic muscles that are the most involved in the deformity. Furthermore, the procedure may induce sensory disturbances (i.e., the tibial nerve includes the sensory fibers innervating the sole of the foot), which may interfere with function and assessment (i.e., after tibial nerve, the induced foot anesthesia interferes with gait and balance). Therefore, such sensorimotor nerve DNB is mainly used to simply differentiate increased muscle tone from fixed contracture. A small volume, e.g., 1–2 mL, can be used to help differentiate between a true contracture and a spastic muscle.

Selective motor nerve blocks (i.e., individual motor branches of the tibial nerve innervating the soleus, gastrocnemius, and tibialis posterior) are more complex to perform due to manifold steps, but of great value in more complicated clinical presentations. Considering their small size, they are more difficult to find but require smaller dose of anesthetics (0.5–1.5 mL). However, such selective DNB allows the spastic muscle(s) involved in the deformity to be identified (e.g., soleus muscle is usually responsible for the triceps clonus; disappearance of the clonus after selective DNB of the soleus motor nerve branch confirms it) and avoids sensory disturbances (as motor nerves branches are separated from the sensory nerves). Several selective DNB (e.g., soleus motor branch followed by gastrocnemius motor branch) can be performed during the same session.

Interestingly, DNB can be used as a valuable screening tool before surgery such as neurotomy. The spasticity reduction and gait kinematics improvement obtained after DNB is consistent with the one obtained after surgery.[54] At last, DNB is a safe technique with clinical guidelines devoted to increase the security was recently published.[55]

To summarize the procedure:

First, prepare the appropriate material and equipment for the procedure:

- Needle of 1”–2” length
- Syringe (3cc or 5cc)
- Gel electrodes
- Internal ± external nerve stimulator
- Gauze and alcohol or betadine
- Desired medication for injecting
- Draw needle.

The procedure and goals of treatment must be explained to the patient who must give informed consent to proceed. The patient must be positioned appropriately depending on the nerve or motor point to be treated (e.g., for treating a tibial nerve, then patient should be prone with knee extended).

The injection site can be selected based on anatomical landmarks. Gel electrodes should be applied and the stimulator turned on to observe contractions; the site can then be marked. Potential anatomical pitfalls should be identified.

The technique for administering the injection can be summarized as:

- Clean site with chlorhexidine, alcohol, or betadine
- Draw up injectant
- Attach syringe to needle
- Connect electrode
- Insert special device needle for conduction anesthesia into site and adjust output of stimulator for maximum contraction </=1.0 mA
- Aspirate syringe prior to injection to look for blood return
- Inject as needed monitoring for loss of contraction (up to 3cc)
- Ultrasound can be used for localization of injection needle and for nerve stimulator for muscle contraction.

Aftercare must be explained to the patient, the site monitored for redness, pain, or sensory complaints. Ice should be applied as required and if any excessive adverse changes occur the physician should be contacted. The response to the injection should be evaluated in terms of tone and ROM. Future plans and follow-up schedules should be discussed with the patient.

Competency Assessment 2

The answers to these questions can be found at the end of this module before the references.

1. What spastic muscles may be responsible for these limb deformities?
2. A 65-year-old patient admitted following the right MCA stroke. On 5th day following the stroke, examination findings demonstrated cognitive deficits, visual inattention to the left side, MAS 2 around his left wrist and elbow, and muscle power of 1/5. What are the features that indicate higher risk of developing spasticity in this patient?
3. What scales would you consider for measuring the outcome of treatment?
4. An 80-year gentleman admitted following brain injury. On examination, he has no power on his right side with spasticity (MAS 3 over his right wrist and elbow flexors). He also was diagnosed with a urinary tract infection 2 days ago and on treatment. He has an indwelling catheter and blood was found in the urine bag. He appears agitated and restless. He shouts out when touched. He has ingrown toenails. What are the aggravating factors (triggers) of spasticity in this patient?

Management

Goal setting

Most human behavior is arguably goal directed; people generally act for a reason even if it is nebulous or unconsidered. Hence, a goal is an end or result toward which behavior is consciously or unconsciously directed. In the context of spasticity, a goal may be defined as the intended consequence of actions undertaken by the patient and rehabilitation team.[56]

Goal setting may be defined as the process during which patient and clinical members of the multidisciplinary team make a collective decision, following an informed discussion, of how and when to carry out rehabilitation activities. The goal setting process should lead to the explicit and comprehensive identification of the reasons for all actions to be taken.[56]

The importance of treatment goal setting

Adequate goal setting should derive a set of goals that motivates patients, caregivers, and the team, ensuring that the same goals are desired, important actions are not overlooked, and there is adequate monitoring of change and quick cessation of ineffective actions.[56]

Setting goals for a person increases their behavior changes, presumably through increasing their motivation. The team effort in goal setting should facilitate both the efficiency and effectiveness of rehabilitation and allow the rehabilitation process to be monitored objectively. In this context, it is very important that actions which are clearly ineffective should be stopped as soon as their lack of desired effect is apparent and an alternative way of achieving the goal can then be started in a timely fashion.

When setting goals, it is important that the team do not make assumptions about the wishes and expectations of individual patients in any situation; even when they seem obvious. Primary goal setting should take into account the patient's wishes. If the patient is unable to decide on their own, then the wishes and expectations of other important parties, such as family, caregivers, peers, funding bodies, and the rehabilitation team, may also be considered. Goals should be recorded using "patient-friendly" wording wherever possible.

Despite setting common goals, patient and clinician's evaluations of benefit of an intervention are not always aligned. In a study of BoNT-A injections, treatment effect was rated as excellent or good by 76% of neurologists but only by 52% of patients.[57]

Treatment satisfaction may change over the course of their treatment. In a study assessing patient satisfaction at different intervals after BoNT-A treatment, it was shown that satisfaction dropped significantly with time after the peak effect of the toxin.[58] It is important to emphasize the need to make attainable, patient-centric goals and manage the patient's expectations.

An algorithm for developing an individual strategy is provided by Turner-Stokes.[59]

SMARTER goals are goals identified on an individual basis:

- **S**pecific
- **M**easurable
- **A**chievable
- **R**ealistic
- **T**imed
- **E**thical
- **R**ecorded.

It is recommended that one primary and several secondary goals (about 3–4) are defined[59] with a defined expected outcome and the time frame in which this is potentially achievable; the combination of the objective goal and action plan is important for success.

Goal attainment scaling

GAS is a method of assessing the degree of achievement in reaching established rehabilitation goals. Each person has their own measure for each goal expected outcome. It allows the achievement to be scored in a standardized way, ascribing a numerical value which can then be used for statistical analysis.

The most frequent goal categories are divided into functional activities, such as active function, passive function and mobility, and symptoms of impairment, such as joint ROM, pain, discomfort, involuntary movements, and associated reactions. One parameter should be chosen for each goal, and after identification of 3–4 goals, a primary goal should be identified.

GAS may be used to monitor an individual patient over consecutive treatment cycles and also to compare different patients or groups of patients or different treatment strategies.

Goal attainment is scored by giving points to the outcome where the goal achievement is:

- Much better than expected: 2
- A little better than expected: 1
- As expected: 0
- Only partial: −1
- No change: −1
- Worse: −2.

The GAS can be calculated via a formula or downloadable Excel spreadsheet from https://www.kcl.ac.uk/cicelysaunders/resources/tools/gas.

The formula is:

$$GAS = 50 + \frac{10 \sum (w_i x_i)}{\left[(1 - \rho) \sum w_i^2 + \rho (\sum w_i)^2\right]^{½}}$$

Where w_i = goal weight, X_i = result (−2 to +2), and ρ = expected correlation for goal scales (ρ = 0.3).

Goal attainment scaling-light

GAS-light is a simplified version of GAS designed to be used in routine clinical practice. It provides a verbal rating scale for clinicians who prefer verbal descriptors to numbers.

Clinicians often think in terms of change from baseline. A problem with the five-point GAS score is that it does not allow "partial achievement" of a goal to be recorded if the baseline score was −1. On the other hand, if all baseline scores are recorded at −2, this does not allow for worsening.

The following algorithm allows clinicians to record goal attainment without reference to the numeric scores and so avoids the perceived negative connotations of zero and minus scores.

Clinical Case Example

The patient was a 58-year old man who had suffered a stroke 6 months ago. He had a right spastic hemiplegia (muscular strength 0/5; global MAS score 3). He did not have any spasms or clonus. Because of spasticity, he had difficulty to put on his wrist–hand orthosis.

The main problem of the patient was pain, both at rest and during passive movement. He also had difficulty with dressing and hygiene. The patient and clinician agreed that the main goal of treatment was to decrease pain from 9 to 6 (visual analog scale), mainly in the shoulder (he also had axillary candidiasis).

Other objectives were to decrease spasticity in the elbow (to facilitate dressing) and in the hand (to facilitate palmar hygiene). A treatment program consisting of BoNT-A injection, followed by physical therapy (1 hour a day, from Monday to Friday) was planned. In addition, a wrist–hand orthosis (splint) to maintain the expected spasticity reduction in the hand was contemplated. The target is to achieve the goals in 6 weeks.

Pretreatment video

The video shows how painful passive movement in the upper limb was, mainly in the shoulder. [https://www.jisprm.org/articles/2022/5/5/images/IntJPhysRehabilMed_2022_5_5_3_347807_sm24.mp4].

It was difficult to examine the range of movement in each joint because of the spasticity and the associated pain. BoNT-A (Incobotulinum toxin A) was injected into the following muscles:

- Pectoralis major 100 U (2 points)
- Subscapularis 50 U
- Brachioradialis 50 U
- Brachialis 50 U
- Flexor digitorum superficialis 50 U
- Interossei 40 U
- Opponens pollicis 10 U.
- Total dose: 350 U.

Posttreatment video

The pain decreased more than what was previously expected. The video shows that physical examination was easier to perform. Passive mobilization of the shoulder and the other joints of the upper limb was easier than before the injection. [https://www.jisprm.org/articles/2022/5/5/images/IntJPhysRehabilMed_2022_5_5_3_347807_sm25.mp4].

The GAS-eous tool is a system for using GAS in the **E**valuation of **O**utcome for **U**pper limb **S**pasticity. It consists of a semi-structured framework for goal setting and outcome measurement. It is divided into two domains: symptom/impairment and activities/function. There are with six main goal areas: pain/discomfort, involuntary movements, ROM/contracture prevention (domain 1); and passive function (care tasks), active function, mobility (domain 2) with additional goals of cosmesis/body image and therapy facilitation.

It is important to note that there may be cultural and geographic variations in the meaning of the terms Cosmesis and Esthetic. In the current context, the term "cosmesis" is referring to the preservation, restoration, or bestowing of physical beauty to the human body, whereas "esthetic" is concerned with the idea of beauty or appreciation of beauty.

Table 11: Goal attainment scaling recording after treatment

Goal	Category	Subcategory	G. parameter	Baseline	Expected	Achieved	GAS
1ary	Symptoms	Pain	VAS	9	5-6	4	+1 (a little more than expected)
2ary	Passive function	Dressing	VAS	7	3-4	3	0 (as expected)
2ary	Passive function	Hygiene	ROM (ease, less pain)	90°, painful 7/10	90°, less pain 4/10	4	0 (as expected)
2ary	Passive function	Use of orthosis	Time (hours)	1.0 h	1.5-3.0 h	2.0 h	0 (as expected)

P=0.3. GAS: Goal attainment scaling, ROM: Range of motion, VAS: Visual analog scale

Table 12: The GAS-eous system

Domain 1	Symptoms/impairment	Parameter
Pain/discomfort (b280)	Spasticity-related pain or discomfort	Pain rating/10
Involuntary movements (b735, b765)	Unwanted involuntary movements during use of other limbs (spasms/associated reactions)	Carry angle (spasm frequency)
Range of movement/contracture prevention (b710, b735)	Range of movement, or splint tolerance prevention of contractures/deformity	Percentage joint range
Domain 2	**Activities/function**	**Parameter**
Passive function (care tasks) (d520)	Ease of caring for the affected limb	Ease of care rating/10
Active function (d440, d445)	Using the affected limb in some active motor tasks	Able to do defined task (time taken/control)
Mobility (d450)	Improved mobility - Transfers/standing/walking	Confidence rating/10 (gait speed//endurance)
	Other	
Cosmesis/body image	Patient's perception of body image, aesthetic appearance	Rating/10
Therapy facilitation	Team's perception of interference with therapy	Team rating/10

Modified from: Turner-Stokes *et al*. The GAS-eous tool Goal Attainment Scaling – Evaluation of outcomes for upper limb spasticity version 1.1. 30.12.13. http//www.csi.kcl.ac.uk/Gaseous_tool3 pdf. The Domains are mapped onto the WHO ICF, disability and health, ICF, disability and health ICF (who.int). GAS: Goal attainment scaling, ICF: International Classification of Functioning

A worksheet for using GAS-eous is available online at: tools-gaseous-gaseous-tool.pdf (kcl.ac.uk).

Table 12 summarizes the Gas-eous system.

Free tools are available on the internet for calculating GAS, GAS-light, and GAS-eous. A good selection of tools is available from King's College UK in: https://www.kcl.ac.uk/cicelysaunders/resources/tools/gas.

Competency Assessment 3

The answers to these questions can be found at the end of this module before the references.

1. Attributes of SMARTER goals are:
   a. Specific, measurable, realistic, timed
   b. Specific, measurable, aspirational, realistic
   c. Specific, achievable, elusive, realistic
   d. Specific, ambiguous, realistic, timed.
2. According to the GAS, when a outcome of an intervention is "a little better than expected," the score assigned is:
   a. −2
   b. −1
   c. 1
   d. 2.
3. Domains in the GAS-eous tool in the evaluation of outcome for upper limb spasticity include:
   a. Pain, agraphia, care tasks, cosmesis
   b. Pain, involuntary movement, mobility, cosmesis
   c. Discomfort, dystonia, mobility, cosmesis
   d. Involuntary movements, contracture prevention, motivation, therapy facilitation.
4. Spasm frequency is a parameter for what GAS-light system domain?
   a. Pain
   b. Passive function
   c. Involuntary movement
   d. Cosmesis.

Competency Assessment Answers

Competency Assessment 1 Answers

1. What are the three main processes that lead to functional impairments seen in an UMN injury?
   Expected content of answer
   The functional impairments seen in patients with spasticity occur due to three main processes: weakness, biomechanical changes (soft tissue stiffness, muscle shortening, tendon contracture), and muscle over-activity through hyperexcitability or loss of inhibition.
2. A patient had a left subcortical stroke 4 weeks ago and is now ready for discharge. He has spasticity of the right elbow flexors and plantar flexors (both scored as "3" in the MAS) and is nonambulatory. Hemiplegia has persisted. His spouse asks you what will happen to the elbow and ankle spasticity in the near future.
   Expected content of answer
   In some people with a stroke, spasticity may disappear after about 4 months (16 weeks), but in others, it may persist. Your husband is at high risk of having persistent and severe spasticity because continues to have no movement at all on his right side. Therefore, it is important that he will be followed up to manage the limb tightness.
3. What clinical features was the patient exhibiting?
   Expected content of answer
   This video displays an example of finger muscle co-contraction. Co-contraction is the simultaneous activation of agonist and antagonist muscles groups during voluntary movement. It results from failure of reciprocal inhibition at either the spinal cord or cortical level. In this video, the patient's finger flexors are co-contracting while the patient is attempting to extend the fingers.
4. A 75-year-old male presents for rehabilitation 2 weeks after sustaining an ischemic right MCA artery CVA with left hemiparesis. His examination is noted as having left arm movement at the elbow limited to a flexor synergy pattern and hyper-reflexia at the biceps tendon. His tone using the MAS in the left arm is significant for a 2 for the elbow flexors and 1+ each for the wrist and finger flexors. As you are formulating a treatment plan, what other signs of the UMNS do you anticipate encountering which may interfere with functional improvements as motor recovery progresses.
   Expected content of answer
   Using the models of motor recovery described by Twitchell and Brunnstrom, it is expected that the spasticity will increase over the next few weeks. The patient may also develop other positive signs associated with the UMNS including increased reflex activity, clonus, co-contraction, and dystonia. It is important to minimize the impact these findings will have on the early motor recovery. In addition, efforts to maintain the normal elastic properties of the muscle and tendons are important since the combination of the above postures and resistance to stretch will lead to muscle stiffness and fibrosis.
5. A stroke patient reports that every time he yawns his hemiparetic elbow is able to flex, although he is unable to flex the elbow on command. The patient is describing what phenomenon?
   1. Spastic dystonia
   2. Associated reaction
   3. Spasticity
   4. Mass synergy pattern.

   Expected content of answer
   Associated reaction. Associated reactions may be due to disinhibited spread of motor activity into a limb affected by a UMN lesion.

Competency Assessment 2 Answers

1. What spastic muscles may be responsible for these limb deformities?
   Expected content of answer

Figure A

   Elbow flexion – Biceps, brachialis, brachioradialis
   Forearm pronation – Pronator teres and quadratus
   Wrist flexion – Flexor carpi radialis and ulnaris

Proximal finger flexion – Flexor digitorum superficialis and profundus (because it crosses the proximal interphalangeal joint).

Figure B

Equinovarus – Tibialis posterior, triceps surae, flexor hallucis longus, flexor digitorum longus

Toe flexion – Flexor hallucis longus, flexor digitorum longus, and brevis.

2. A 65-year-old patient admitted following the right MCA stroke. On 5th day following the stroke, examination findings demonstrated cognitive deficits, visual inattention to the left side, MAS 2 around his left wrist and elbow, and muscle power of 1/5. What are the features that indicate higher risk of developing spasticity in this patient?

   Expected content of answer

   Cognitive deficits, visual inattention, reduced motor power, and already present high tone across 2 joints.
3. What scales would you consider for measuring the outcome of treatment?

   Expected content of answer

   GAS, MAS, Tardieu scale, goniometer (improved range of movement), patient/caregiver satisfaction rates, improved walking speed.
4. An 80-year-old gentleman admitted following brain injury. On examination he has no power on his right side with spasticity (MAS 3 over his right wrist and elbow flexors). He also was diagnosed with urinary tract infections 2 days ago and on treatment. He has an indwelling catheter and blood was found on the urine bag. He appears agitated and restless. He shouts out when touched. He has growing toe nails. What are the aggravating (triggers) factors of spasticity in this patient?

   Expected content of answer

   Urinary tract infection, possible trauma from catheter, ingrown toenail, and pain.

Competency Assessment 3 Answers

1. Attributes of SMARTER goals are:
   a. **Specific, measurable, realistic, timed**
   b. Specific, measurable, aspirational, realistic
   c. Specific, achievable, elusive, realistic
   d. Specific, ambiguous, realistic, timed.
2. According to the GAS, when a outcome of an intervention is "a little better than expected," the score assigned is:
   a. −2
   b. −1
   c. 1
   d. 2.
3. Domains in the GAS-eous tool in the evaluation of outcome for upper limb spasticity include:
   a. Pain, agraphia, care tasks, cosmesis
   b. **Pain, involuntary movement, mobility, cosmesis**
   c. Discomfort, dystonia, mobility, cosmesis
   d. Involuntary movements, contracture prevention, motivation, therapy facilitation.
4. Spasm frequency is a parameter for what GAS-light system domain?
   a. Pain
   b. Passive function
   c. **Involuntary movement**
   d. Cosmesis.

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