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Is Spasticity a Syndrome? A historical perspective on spasticity definitions and descriptions

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Abstract

"Spasticity" has long been used as a generalized term for a constellation of distinct positive motor signs arising from a central nervous system lesion, creating ambiguity in identifying individual impairments. These include spasticity *sensu stricto*—the velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as defined by Lance in 1980—as well as spastic dystonia, spastic co-contraction, and spasms. Drawing on our clinical experience and collaborative work within the Toxnet group (www.toxnet.net), we believe that using spasticity as a broad diagnostic label creates ambiguity and may obscure the specific impairments contributing to the clinical presentation. Such generalization may compromise treatment decisions and outcomes and is compounded by using manual tools like the Tardieu and modified Tardieu Scales, which lack evidence of validity and reliability, and the Ashworth and modified Ashworth Scales, which do not measure the velocity-dependent nature of spasticity or provide dependable insights into the mechanisms driving the torque changes. To optimize clinical assessment precision and treatment outcomes, we propose "spasticity syndrome" as a new umbrella term encompassing the spectrum of clinical manifestations and underlying pathologies associated with upper motor neuron injury, alongside non-neural peripheral changes and patient-reported symptoms.

Introduction

When we consider the term "spasticity" and its long-standing use as an umbrella term to describe various distinct positive motor signs that occur following a central nervous system (CNS) lesion, we are reminded of the expression often attributed to Søren Kierkegaard: “By giving me a name, a label, you negate all the other things I could possibly be” (n.d.).

The positive motor signs associated with upper motor neuron (UMN) injury include spasticity *sensu stricto*, the velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex,[1,2] and spastic dystonia, spastic co-contraction, spasms, and resistance to passive stretch due to muscle changes. In addition, patients can exhibit negative motor signs—such as weakness and incoordination—which further contribute to the presenting impairments and functional disabilities. Using spasticity as a broad term to encompass all motor signs and postural changes that present following a UMN lesion creates ambiguity in identifying specific impairments. It effectively reduces a set of consistently co-occurring elements to a single label, which ultimately undermines the complexity of the patient’s presentation.

This paper is authored by members of the Toxnet group (www.toxnet.net), a global network of leading experts and specialists promoting education, research, and clinical innovation to enhance the management of people with “spasticity”. Our observations in clinical practice indicate that using spasticity as an umbrella term may indiscriminately hinder the ability to pinpoint the spectrum of impairments that contribute to the clinical problem; thus, not planning for the appropriate treatments or combination therapies may lead to less effective outcomes. Accurate identification and assessment of impairments are crucial for selecting the proper treatment.[3] In

this regard, broadly classifying these signs as spasticity is problematic, as it remains unclear if the reference is to spasticity *sensu stricto* or to one or more of the other motor dysfunctions resulting from UMN pathology. In our opinion, this may lead to suboptimal recognition of impairments that may coexist with spasticity *sensu stricto*, thus contributing to the presenting problem and ultimately resulting in incomplete treatment.

The indefinite clinical meaning of the term spasticity is compounded by using manual measurement scales such as the Ashworth Scale, (AS), modified AS (MAS), Tardieu Scale (TS), and modified TS (MTS). The AS was developed to rate resistance to passive movement in the rest position but has been widely used to evaluate spasticity.[4-6] Notably, the AS was developed before Lance defined spasticity [*sensu stricto*] as "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"[1]. The AS and MAS measure resistance to passive movement in the rest position at one speed, while the TS and MTS measure it at various speeds.[5] The TS and MTS are considered superior to the AS and MAS in terms of differentiating between neural and peripheral contributors to spasticity.[6,7] The AS and MAS have been criticized for not measuring the velocity-dependent aspect of spasticity or providing reliable information around the origin of the associated torque changes, and for a lack of inter-rater reliability.[8-10] Furthermore, being passive by nature, the AS and MAS cannot assess spastic co-contraction, which only occurs during active movement. The paucity of evidence regarding the validity and reliability of the TS and MTS may be attributable, in part, to the absence of a universally accepted definition of spasticity and an incomplete understanding of its underlying pathophysiology.[9] These limitations hinder the establishment of optimal methodologies for its measurement.[9] This has given rise to a range of distinct definitions and descriptions of spasticity,

resulting in varying interpretations among clinicians and within the published literature.[5,11] Moreover, these commonly used clinical scales do not consider the different components of "spasticity" .

As clinicians working in spasticity management, we recognize that while explicitly describing each impairment promotes accuracy of diagnosis, documenting this in high-demand clinical environments may be challenging. We acknowledge that a single label to describe a clinical phenomenon may be the most practical option, on the condition that it implies the presence of multiple underlying impairments responsible for a patient's clinical presentation. Accordingly, we propose the inclusive construct "spasticity syndrome" to more accurately reflect the spectrum of clinical signs and symptoms associated with the upper motor neuron syndrome (UMNS) in addition to non-neural peripheral abnormalities. This aligns with Stedman's Medical Dictionary, which defines a syndrome as an "aggregate of symptoms and signs associated with any morbid process, together constituting the picture of the disease."[12] Our goal is to establish a term based on clinical phenotype.

This paper introduces spasticity syndrome as a new umbrella term for the clinical phenomenon associated with the UMNS, which is often referred to as spasticity. The studies cited in this paper have already provided comprehensive accounts of the various pathophysiologic processes underlying the clinical presentation of the UMNS.

Overview of well-known definitions and descriptions of "spasticity"

Many definitions and descriptions of spasticity have emerged over the years and are considered well-known among clinicians working in the field of neurorehabilitation. Several of the most recognized examples are outlined in terms of neural and non-neural contributors to spasticity in **Table 1.** The differences between them, and the fact that none fully captures the range of motor signs of neural origin that arise following a UMN lesion, are clear. Discrepancies in the potentially coexisting peripheral muscle and soft tissue changes of non-neural origin—and in patient-reported symptoms—are also apparent.

In its initial conceptualization, spasticity was characterized as a pathological neural muscle activity. In 1843, Little[13] described how hemiplegia, as a neural contributor, led to "spastic rigidity of muscles." Burke & Ashby[14] later attributed this to the suppression of the presynaptic inhibitory mechanisms. In 1974, Landau[15] described spasticity according to its neural contributors and linked the positive symptoms of neurological syndromes to lowered threshold and increased responsiveness of released lower-level mechanisms caused by disrupted neural integrations. He emphasized that rehabilitation should consider evaluation of all symptoms, whether disabling or not.

Lance's 1980 definition of spasticity [*sensu stricto*][1], described above, is currently the most widely cited definition. Gracies defined spasticity as an increase in the velocity-dependent reflexes to phasic stretch, detected and measured at rest.[5,16,17] However, O'Dwyer et al.[19] hypothesized that, in some patients, shortening of the muscle and its spindles (muscle contracture), rather than reflex hyperexcitability, could potentiate spasticity/stretch reflex, and ultimately hypertonia. Mirroring this, Pandyan[3] and Dressler[2] noted that spasticity is not

exclusive to stretch reflex hyperexcitability. In addition, Nielsen and colleagues[20] argued that hyperexcitable reflexes do not have a central role in the pathophysiology and clinical manifestations of spasticity. Rather, they proposed that spasticity is characterized by a reduced ability to selectively activate muscles with significant co-activation of antagonist muscles.

In 2021, Li et al.[18] questioned whether spasticity *sensu stricto* truly interferes with motor recovery following stroke, noting that reducing poststroke spasticity with botulinum toxin treatment does not consistently improve volitional movement and function, and *vice versa*. This may reflect limitations in current tools for detecting functional improvements, rather than an absence of therapeutic response. Li et al.[18] redefined spasticity as a velocity- and muscle length–dependent increase in resistance to passive stretch driven by hyperexcitable descending excitatory brainstem pathways and the resultant exaggerated stretch reflex responses. They emphasized that spasticity occurs in resting muscles, while other impairments—such as abnormal synergies and inappropriate muscle activation—coexist with spasticity and share similar pathophysiological origins.

All these definitions and descriptions, except for those proposed by Gracies[16] and Nielsen et al.,[20] are limited to abnormal muscle activity during passive movement. Gracies[16] highlighted the various positive signs of neural origin that occur spontaneously and/or during passive and active movement. Others have drawn attention to the influence of non-neural components such as muscle, soft tissue, and joint changes on the clinical presentation under assessment.[21,22] Furthermore, Pandyan[3] introduced the role of the sensory system by describing spasticity as a sensorimotor disorder. Nielsen et al.[20] proposed that uncoordinated co-activation of antagonist muscles and stiff posture and gait may be adaptations to maintain joint and postural stability

because of insufficient muscle strength; thus, involuntary muscle activity may be linked to an inaccurate prediction of the sensory consequences of movement.

Gracies[17] emphasized that the Lance definition, specifically spasticity *sensu stricto,* does not sufficiently address the core issues impacting function and quality of life in patients with paresis. They proposed the umbrella term "spastic muscle overactivity," which includes varying forms of neural muscle overactivity.[16,17] In contrast to spasticity *sensu stricto*, spastic muscle overactivity includes other positive signs of neural origin that are distinguished by their primary triggering factor: spasticity *sensu stricto* is triggered by phasic stretch reflexes; spastic dystonia by tonic muscle stretch; spastic co-contraction by volitional command; and spasm by sensory input. Gracies[16,17] noted that spastic dystonia (observed at rest) and spastic co-contraction (observed during active movement) are usually considered the main causes of passive and active functional limitations, while spasticity *sensu stricto* does not appear to be the main barrier to active movement in deforming spastic paresis, except during fast or ballistic movements.

In 2017, following European consensus meetings with field experts, van den Noort et al.[23] developed a conceptual model of the pathophysiological neuromuscular responses to passive muscle stretch. They suggested using the term "hyper-resistance" to describe the impaired neuromuscular response during passive stretch instead of terms like spasticity. van den Noort and colleagues[23] highlighted the importance of clearly differentiating between two different sources of hyper-resistance: (i) neural (CNS-related), which includes velocity-dependent stretch hyperreflexia and non-velocity-dependent involuntary background activation; and (ii) non-neural (tissue-related), involving factors such as muscle shortening and elasticity, and connective tissue viscosity. They emphasized that the term "spasticity" should be used specifically for stretch hyperreflexia, while "stiffness" should refer solely to passive tissue contributions.

More recently, the Spasticity Study Group of the International Parkinson and Movement Disorders Society (IPMDS) published a manuscript in which they described spasticity as "the symptomatic hallmark of the syndrome of spastic paresis following lesions that involve pyramidal pathways."[24] In the same manuscript, the authors proposed that "spastic paresis" is a movement disorder, echoing prior work suggesting that the pathophysiologic and clinical features of spasticity qualify it as a movement disorder.[20,22,25] Acknowledging the contribution of non-neural muscle changes and paresis, the umbrella term "spastic paresis" proposed by the authors encompasses all neural and non-neural abnormalities, in addition to paresis and the passive and/or active limitation of movement after a CNS lesion. Within this construct, paresis, muscle stiffening, spastic co-contraction, synkinesis, spastic dystonia, clonus, and spasms integrate to produce a composite hypokinetic and hyperkinetic spastic movement disorder—asymmetric around the joints—causing cosmetic and functional limitations. On one side of the joint, the muscle becomes shortened and stiffened. Once this reaches a certain threshold, it can elevate the excitatory spindle afferent activity to the homonymous motor neuron in the brain, leading to increased muscle tone and impairment. Meanwhile, the muscle on the opposite side of the joint exhibits signs of chronic relaxation—due to prolonged reciprocal inhibition from the stiffened muscle on the other side—contributing to worsened paresis.

In response to the lack of a consensus definition and classification of spasticity, and the overlap between the classification and definition of dystonia and spasticity, a recent international expert panel has contributed further to the literature by offering a simplified classification of spastic movement disorders as "involuntary muscle overactivity in the presence of a CNS lesion, most often in conjunction with paresis"[26]. In their publication, the expert consensus group acknowledges that non-neuronal components, such as contractures and rheological changes,

should be assessed separately, and that when the spastic movement disorder hinders body function, activity, and/or participation, it should be defined as disabling spasticity. Despite the distinctions outlined in the definitions and descriptions that have evolved over the years, the term "spasticity" continues to be applied inappropriately to both the neural and non-neural contributions to the clinical presentation. This misapplication persists even though spasticity *sensu stricto* is not consistently the most prominent impairment observed during clinical evaluation.

The need for a new comprehensive term

The current definitions and descriptions of spasticity offer valuable insights into this condition; however, it is not always clear whether the reference pertains solely to spasticity *sensu stricto* or to the broader motor impairments resulting from UMN injury. Additionally, they do not capture the complete clinical picture, such as the concomitant non-neural peripheral changes in the muscles and soft tissues. As defined by Lance,[1] spasticity describes a clinical condition—"a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks"—and indicates "hyperexcitability of the stretch reflex" as the likely underlying cause. In routine clinical practice, however, spasticity has evolved to encompass all co-occurring impairments in an individual patient, and at times, it is used as a clinical diagnosis.

In some cases, the clinical features and possible causes of related impairments may differ from those of spasticity *sensu stricto*. For instance, in addition to spasticity *sensu stricto*, a single patient might exhibit muscle weakness due to corticospinal involvement; abnormal co-contraction during active movement—likely due to loss of reciprocal inhibition; spastic dystonia from sustained muscle contraction independent of passive stretch—probably resulting from cortical involvement; and muscle contracture that may be attributable to loss of sarcomeres and other changes in soft tissue. Referring to these impairments as "spasticity" will lead to ineffective treatment. In that regard, if spasmolytic medication alone is prescribed in the scenario above, the drug will relieve the presenting spasticity *sensu stricto* and muscle hyperactivity but will not improve muscle contracture and weakness. Interestingly, the same patient may present

with various impairments: a clenched fist due to spastic dystonia, a stiff knee gait due to spastic co-contraction, and a fixed *equinus* due to muscle shortening.

Therkildsen et al.[27] acknowledged the wide range of signs and symptoms commonly associated with the term spasticity and noted inconsistencies in its definitions. They expressed doubt about the feasibility of reaching a consensus on any definition, let alone proposing a new one. The Toxnet group members agree that misuse of spasticity as a diagnosis has led to incomplete identification of all impairments contributing to a patient's presenting issues. Consistent with the conclusions drawn by Therkildsen and colleagues,[27] we recognize that a new definition for spasticity might not be helpful and instead have chosen to propose a new term that more comprehensively describes the clinical experience of both patients and clinicians: spasticity syndrome. This broad term aims to include the neural contributors to motor signs linked to the UMNS, as well as patient-reported symptoms and non-neural changes.

To further explore and reach a consensus on the components of spasticity syndrome, all 23 Toxnet group members participated in a survey and several face-to-face discussions. At an in-person meeting in October 2024, members of the group collaboratively generated a preliminary list of potential features of spasticity syndrome. The list was subsequently refined to its core components and incorporated into an electronic questionnaire, which was distributed later that year. Although no two respondents selected an identical set of components—underscoring the inherent complexity of the topic, even amongst experts in the field—multiple participants endorsed several components, indicating areas of emerging consensus **(Table 2)**. A pre-defined cut-off percentage for identifying the components of spasticity syndrome was not established prior to the questionnaire's distribution. Consequently, rather than selecting components based solely on response rates, the questionnaire findings were used to inform a face-to-face discussion

held in April 2025 to determine which components should be included in the definition of spasticity syndrome.

Through the course of the meeting discussions, the Toxnet group reached consensus that non-neural peripheral changes should be included as components of spasticity syndrome, acknowledging that these components may or may not be present in individual patients and are not required for a diagnosis of spasticity syndrome. They noted that the inclusion of non-neural peripheral changes is grounded in well-established mechanistic evidence linking UMN-mediated paresis to obligate peripheral tissue changes via a bidirectional, mutually aggravating relationship with neural overactivity. Moreover, they considered that while morphologically similar changes occur with aging and disuse, the pathophysiology and clinical trajectories in UMN pathology are distinct. This thinking mirrors the recent contributions to movement disorders research[24,26], which advocates for incorporating both neuronal and non-neuronal components, alongside patient-reported symptoms, into the umbrella terminology used in this field.

At the conclusion of the meeting, the Toxnet group agreed that spasticity syndrome should be defined as "a constellation of neurological signs and symptoms secondary to upper motor neuron pathology, accompanied by non-neural peripheral soft tissue changes," and recommend that clinicians use it as an umbrella term, in conjunction with identifying the individual components that present in the patient's unique pattern. Notably, the IPMDS Spasticity Study group[24] used a specific movement disorder, namely spastic paresis, as a catch-all term to describe the signs and symptoms resulting from a CNS lesion. In contrast, spasticity syndrome is not a movement disorder; rather, it was introduced as a broader, inclusive label to encompass post-CNS lesion signs and symptoms.

Spasticity syndrome

Spasticity syndrome may encompass one or more of the components outlined below, which are also summarized in **Figure 1**.

Neural changes

Signs

- **spasticity *sensu stricto*** (spasticity [Lance[1]]): a velocity-dependent increase in muscle tone measured by passive assessment[1,18,20,23]
- **clonus**: characterized by rhythmic, alternating activation of antagonist muscles at frequencies around 3–8 Hz, triggered by muscle stretch[26]
- **exaggerated reflexes**: tendon reflexes that can be elicited beyond the usual reflexogenic zones and are larger in amplitude
- **associated reactions:** consistent movement resembling flexion or extension synergy patterns in the paretic limb during maximal efforts with the non-paretic limb[28]
- **dyssynergias:** abnormal synergistic movements triggered when a patient voluntarily attempts to move a spastic limb[25]
- **pathological reflexes**: reflexes that only occur after lesions in the sensorimotor system in the CNS. These aberrant motor responses are elicited during reflex testing, such as the Babinski sign and primitive reflexes, including the palmomental and grasp reflexes. These reflexes are typically suppressed after infancy but may reemerge in the context of neurological pathology

- **sustained involuntary muscle overactivity**, including **spastic dystonia**, a spontaneous (non-triggered) co-contraction of antagonistic muscles that leads to spastic-dystonic postures, believed to originate from lesions at the cortical level following Denny-Brown's experiments in 1966[29]

- **spastic paresis** (neither isolated nor pure motor weakness): hypokinetic and hyperkinetic movement abnormalities caused by both muscular and neural factors, which constitute spastic movement disorder[22,24]

- **decreased motor control during voluntary movement**

- **spastic co-contraction**: inappropriate co-contraction of agonist and antagonist muscles during active movement

- **spasms**: sudden involuntary muscle contractions that are usually triggered by sensory events that last from seconds to minutes[27]

Symptoms

- **stretch-induced pain** (passive evaluation)

- "**tightness**" as a manifestation of spasticity and sustained involuntary muscle activity as described above

- "**weakness**" as related to spastic muscle paresis as described above

Non-neural, peripheral changes

- connective tissue pathology

- muscle fibrosis
- muscle contracture
- joint contracture

Summary/Conclusion

Spasticity syndrome encompasses sensorimotor impairments resulting from UMN pathology, accompanied by non-neural peripheral changes and patient-reported symptoms. This new terminology serves as a reminder to clinicians that issues related to the UMNS extend beyond spasticity *sensu stricto* and that the complexity of the clinical presentation necessitates a comprehensive and holistic management approach. The Toxnet group strongly encourages clinicians to regard this phenomenon as a syndrome, an established concept denoting a specific constellation of signs and symptoms that reliably occur together. We endorse using "spasticity syndrome" as an umbrella term, with its individual components clearly delineated to enhance assessment precision and guide effective treatment choices.

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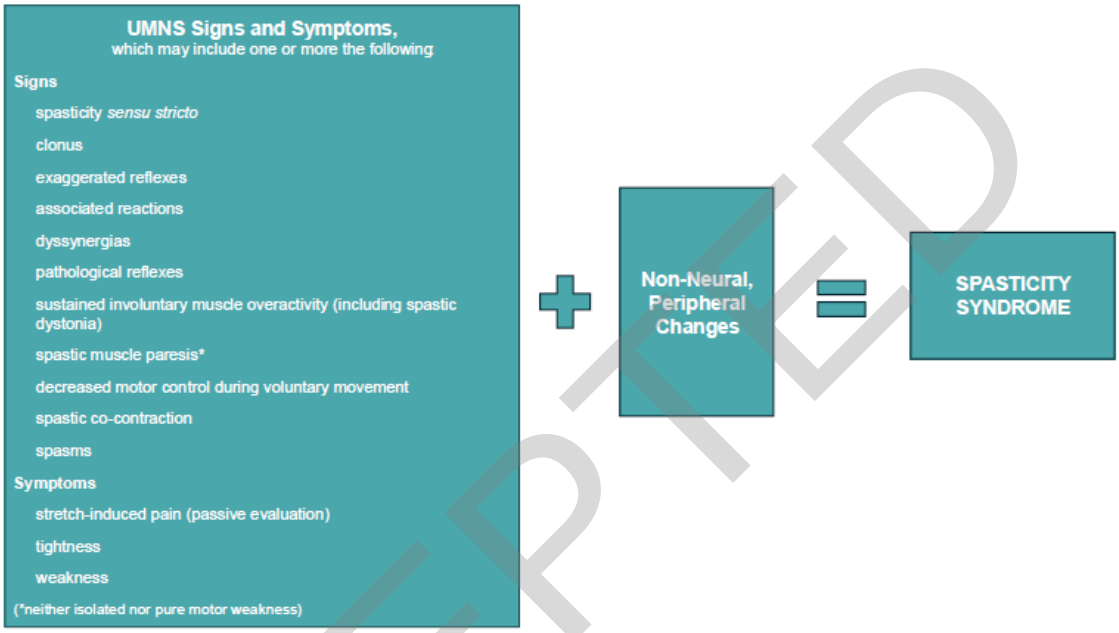
Figure 1. Spasticity syndrome overview



UMNS = upper motor neuron syndrome

Table 1. Comparison of well-known definitions and description of spasticity

Reference	One-line definition	Neural contributors					Non-neural contributors	Spasticity trigger or underlying condition
		Muscle tone	Stretch reflex	Type of stretch reflex	Other muscle "behaviors"	Velocity-dependence	Muscle change	
Little 1843[13]	Describes spastic rigidity of muscle and deformity: perfect paraplegia or hemiplegia (gradual/sudden) are frequent precursors	Spastic rigidity of muscles	ND	ND	Contracture	ND	ND	ND

	of universal paralytic contracture							
Burke & Ashby1972[14]	Describes how the absence or suppression of presynaptic inhibitory mechanisms may play a significant role in the increased muscle tone (or hyper-reflexive state) of spasticity	Increased muscle tone	Hyper-reflexive state	ND	ND	ND	ND	Absence or suppression of presynaptic inhibitory mechanisms

Landau 1974[15]	$Spasticity_{PRR}$ (proprioceptive reflex release)	ND	Increased proprioceptive reflexes	Phasic tendon jerk reflex, tonic stretch reflex	Tonic clasp-knife reaction	ND	ND	ND
	$Spasticity_{GRR}$ (generalized reflex release)	ND	Reflex release	Proprioceptive and polysynaptic flexion reflexes	ND	ND	ND	ND
	$Spasticity_{UMNs}$ (upper motor neuron syndrome)	ND	Reflex release (e.g., spastic paraparesis)	ND	ND	ND	ND	ND
	$Spasticity_{DR}$ (dystonic-rigid state)	ND	ND	ND	Involuntary continuous muscle contractions	ND	ND	ND

Lance 1980[1]	Spasticity is 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	Increased muscle tone	Hyperexcitability of the stretch reflex	ND	(Exaggerated tendon jerks)	Velocity-dependent increase in tonic stretch reflexes	ND	ND

O'Dwyer et al. 1996[19]	Describes how the shortening of muscle and its spindles due to contracture may increase tonic stretch reflex/spasticity in some patients (link between hypertonia following CNS lesions and contracture	Hypertonia	Increased tonic stretch reflex	Tonic	Contracture	ND	Shortening of muscle and spindles	CNS lesion

	s)							
Pandyan et al. 2005[3]	Spasticity is a disordered sensorimotor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles	ND	ND	ND	Intermittent or sustained involuntary activation of muscles	ND	ND	Disordered sensorimotor control following an UMN lesion
Gracies 2005[16]	Spasticity is an	ND	Increased phasic	Phasic	ND	Increased velocity-	ND	ND

	increase in the velocity-dependent reflexes to phasic stretch, detected & measured at rest		stretch reflex			dependent reflexes to phasic stretch		
van den Noort 2017[23]*	"Hyper-resistance" describes the phenomenon of impaired neuromuscular response during passive stretch	NA	NA	NA	NA	NA	NA	NA

Dressler et al. 2018[2]	Spasticity describes involuntary muscle hyperactivity in the presence of central paresis	ND	ND	ND	Involuntary muscle hyperactivity	ND	ND	Central paresis
Li et al. 2021[18]	Spasticity is manifested as velocity- and muscle length-dependent increase in resistance to externally imposed muscle stretch (it	ND	Exaggerated stretch reflex	ND	Increase in resistance to externally imposed muscle stretch	Velocity- and muscle length-dependent increase in resistance	ND	Hyperexcitable descending excitatory brainstem pathways

	results from hyperexcitable descending excitatory brainstem pathways and from the resultant exaggerated stretch reflex responses)							
Gracies 2025[24†]	Spasticity is but the symptomatic hallmark of the syndrome of spastic paresis	ND	ND	ND	ND	ND	Plastic changes	A gradual, plastic phenomenon of sprouting and synaptic formation

	following lesions that involve pyramidal pathways. Spastic paresis comprises hypo- and hyperkinetic movement abnormalities from both muscular and neural causes, which constitute the spastic movement disorder*							*

*Specifically refers to the van de Noort et al. 2017[23] description of "hyper-resistance" as a term to describe the phenomenon of impaired neuromuscular response during passive stretch, rather than terms like spasticity or hypertonia.
†Specifically refers to the Gracies 2025[24] depiction of spasticity as a symptomatic hallmark of spastic paresis.
CNS = central nervous system; NA = not applicable; ND = not described; UMN = upper motor neuron.

Table 2. Spasticity syndrome questionnaire results

Component	Number of votes	Percentage of respondents (%)
Exaggerated reflexes	21	91
Spasticity *sensu stricto* (spasticity [Lance 1980[1]])	20	87
Spastic co-contraction	19	83
Associated reactions and dyssynergias	19	83
Clonus	18	78
Spastic dystonia	18	78
Spasm	17	74
Spastic myopathy	15	65
Muscle shortening	13	57
Weakness	11	48
Stretch-induced pain (passive evaluation)	9	39
Babinski	8	35
Muscle fiber loss	7	30
Fatigability	6	26
Fatty degeneration	4	17
Altered sensation	3	13

n = 23 questionnaire respondents