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REVIEW

A phenomenological approach to spastic movement disorders: an international expert panel consensus

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Abstract

Background and objectives Hyperkinetic movement disorders, characterized by complex and overlapping motor patterns, present significant challenges in classification and treatment. The inconsistency in definitions and descriptors complicates both research and clinical communication.

This study aims to provide an overview of current terminology and definitions related to spasticity and other hyperkinetic movement disorders associated with central nervous system lesions. We propose a unified terminology and classification system for spastic movement disorders (SMD).

Methods In this scoping review, definitions of spasticity, dystonia, tremor, chorea, hemiballismus, athetosis, myoclonus, and dyssynergia were reviewed, with emphasis on overlaps and diagnostic challenges among these disorders. The goal was to develop a consensus expert opinion on a phenomenological approach to SMD.

Results The proposed classification system for spasticity includes clinical characteristics (Axis 1) and etiology (Axis 2). Axis 1 includes: age at onset, body distribution, disease course, phenomenological description, and impact on body function, activity, and participation.

The phenomenological description allows sub-classification of SMD into: phasic reflex overactivity, stimulus-induced muscle overactivity, constant muscle overactivity, and lack of muscle selectivity. These categories clarify disabling phenotypes such as clonus, dysregulated co-contraction, muscle spasms, activity-induced muscle hypertonia, associated reactions, and persistent muscle hypertonia.

Discussion This framework for classification of SMD aims to establish a common language for describing clinical phenotypes. By adopting a phenomenological approach, we underscore the importance of consistent descriptors and propose a systematic classification method for movement disorders, particularly spasticity. We hope this unified terminology will enhance clinical practice, research, and ultimately, patient care.

Keywords Phenomenological approach · Unified terminology · Hyperkinetic movement disorders · Spasticity · Classification system · Spastic movement disorder

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Introduction

Hyperkinetic movement disorders are often difficult to classify as the movement patterns are complex and may have an obvious overlap between different disorders with hyperkinesias. This is highlighted by the fact that there are multiple evolving definitions and that different specialties and professions (i.e., such as neurologists, physical and rehabilitation medicine specialists, neuro-pediatricians and physio- and occupational therapists) are treating hyperkinetic movement disorders and they tend to differ in their view of these specific disorders.

This confusion is particularly true when talking about spasticity, where there is neither a single accepted definition for the disorder, nor a consensus on the movement patterns to be included [1]. An example is the term spastic dystonia, where the term dystonia is used in conjunction with 'spastic' to describe one common expression of the spastic movement disorder (SMD) after acquired CNS lesion.

Spasticity syndrome has been proposed as an umbrella term including both neurological and non-neural components as well as patients' subjective experience [2]. Wherefore, spastic paresis characterized by hypokinetic and hyperkinetic movement abnormalities, arising from both muscular and neural causes, constitute the SMD [3].

When comparing definitions of different hyperkinetic movement disorders, there seems to be an overlap between descriptions of movements observed and the differentiation of these clusters of movements into diagnostic categories. To this end, often a proposed etiology of the movement disorder is applied to define the category of movement disorder (spasticity, dystonia, tremor, chorea), more than the phenotypes being observed. A more wanting definition and descriptors of hyperkinetic movement disorders are a hurdle in both research and clinical communication, as accurate terminology is essential for unambiguous communication and knowledge translation.

This present work provides an overview of the current terms and definitions of spastic movement disorder in relation to other hyperkinetic movement disorders that are related to lesions in the CNS. While there are various etiologies related to different hyperkinetic movement disorders like drug-induced, toxic-metabolic, diffuse-hypoxic among others, our aim is to focus on those causes that are hinged on clear localized central nervous system (CNS) lesions. The object is also to highlight common terminologies that define description of phenotypes. Thus said, we accentuate the value of a systematic approach for classifying movement disorders, with a focus in spastic movement disorder, the goal of which is to have a common language when treating muscle overactivity in patients with CNS lesions.

Method

As a scoping review, the purpose was not to capture all publications on hyperkinetic movement disorders, but rather to identify official consensus documents regarding their definition and classification. The search followed a hierarchical approach: definitions formulated by the Movement Disorder Society (MDS) were prioritized, followed by those from the World Federation of Neurology. If neither organization had published a consensus classification for a specific disorder, textbook sources or individual publications were consulted. Definitions of spasticity, dystonia, tremor, chorea, hemiballismus, athetosis, myoclonus, and dyssynergia were reviewed. To work towards a unified view on spasticity, an expert group of neurological rehabilitation specialists representing neurologists, physiatrists, and physiotherapist representing North America, Europe. and Asia joined in this multidisciplinary and multinational effort. Our expert group comprised eight neurological rehabilitation specialists (from eight countries with a combined experience of more than 220 years (range of 18–36 years, mean 27.5 years)) in treating spastic movement disorders and involved in spasticity education nationally and internationally. The group undertook repeated discussions and critical assessment of the current literature on hyperkinetic movement disorders with most focus on spasticity and conducted interactive meetings to create the expert opinion consensus on the presented phenomenological approach to spastic movement disorders.

Definition of spasticity

Several definitions alluding to spasticity have been proposed over the years. The term spasticity is derived from the Greek "spasticos" and "spaon" and was already in use by Hippocrates to describe an epileptic fit. Spasticity was first introduced to the English language by Good in 1829. He described "spastic wryneck…. from excess of muscular action on the contracted side"[4]. In 1868, Charcot described the symptoms of multiple sclerosis and introduced the term "spastic paraplexie"[5]. However, the first accepted and clinically used definition of spasticity is by Lance in 1980, who defined spasticity as a *"motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex"*[6]. However, this definition only focuses on the exaggerated resistance of a muscle to passive stretch, and not the whole spectrum of muscle overactivity seen in patients with a CNS lesion and how spasticity is used in a clinical context.

In an attempt to address the issue, other definitions have been proposed. The SPASM consortium in 2005 defined

spasticity as "*a disordered sensorimotor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles"* [7]. This broad definition tries to include all types of muscle overactivity that might occur following an upper motor neuron (UMN) lesion. However, there are no descriptions of the phenotypes nor suggestion of a common language to describe these phenotypes. In 2018, the Interdisciplinary Working Group on Movement Disorders published a consensus document suggesting a "practical new definition based on its various forms of muscle hyperactivity as described in the current movement disorders terminology" [8]. They defined spasticity as *"involuntary muscle hyperactivity in the presence of central paresis."* The authors also described that involuntary muscle hyperactivity can consist of various forms:

1. *Spasticity* sensu stricto*, that* describes involuntary muscle hyperactivity triggered by rapid passive joint movements.
2. *Rigidity* involuntary muscle hyperactivity triggered by slow passive joint movements.
3. D*ystonia* as a spontaneous involuntary muscle hyperactivity and
4. S*pasms* described as complex involuntary movements usually triggered by sensory or acoustic stimuli.

The Spasticity Study Group of MDS published an article in 2025 introducing the term "*spastic paresis"*, characterized by both hypokinetic and hyperkinetic movement abnormalities arising from muscular and neural causes. Together, these components constitute the *SMD* [3].

More recently, one of us delivered a keynote lecture titled "Does Spasticity Need a New Name?" on the occasion of the 19th Congress of the International Society of Physical and Rehabilitation Medicine in Marrakesh, Morocco [2]. The aforementioned lecture proposed "*spasticity syndrome"* as an umbrella term encompassing both neurological and non-neural components, including patient's subjective experience. In parallel, *spasticity* sensu stricto was defined as "a disorder of sensorimotor control resulting from an upper motor neuron disease and characterized by velocity- and length-dependent involuntary muscle overactivity, which is intermittent or sustained during passive stretch."

Dovetailing the variations in spasticity definition, we saw the need for a more comprehensive strategy in describing hyperkinetic movement disorders. To this end, we sought to unify the phenotypic terms and language applied so as to reach a more common terminology for use among collaborating health care professionals treating patients with muscle overactivity after a CNS lesion. In this effort, we do not include secondary changes in muscle or skeleton, such as muscle shortening and contractures, given the differentiation between these changes and muscle hyperactivity due to a CNS lesion can be clinically challenging.

Definition of other hyperkinetic movement disorders

To understand why it can be difficult to differentiate between different hyperkinetic movement disorders solely on the definitions of the movement disorders, we will shortly give an overview of the current definitions of other hyperkinetic movement disorders that might occur because of a CNS lesion. The examples of etiologies that can elicit the hyperkinetic movement disorders below are the ones that are described in the different consensus publications, but not a complete list of potential etiologies. To notice, all these etiologies can also elicit spastic movement disorder.

Dystonia

According to the resent revision by the MDS, published May 2025 [9], dystonia is defined as *"a movement disorder characterized by sustained or intermittent abnormal movements, postures, or both. Dystonic movements and postures are typically patterned and repetitive and may be tremulous or jerky. They are often initiated or worsened by voluntary action, and frequently associated with overflow movements."* Dystonic movements and postures are a result of involuntary muscle contractions.

It is also recognized that *"Dystonia syndromes have a remarkable degree of phenotypic variability with frequent overlap among different syndromes"* [10].

The classification scheme also identifies two distinct axes: clinical characteristics and etiology, and pathogenesis [9], where the etiology is divided into genetic (dystonia of proven genetic origin) and acquired (dystonia due to a known specific cause). Idiopathic dystonia refers to cases with unknown genetic or acquired causes and no evidence of neuroanatomical lesions.

Examples of acquired etiologies of dystonias are [10] (i) vascular: ischemia, hemorrhage, arteriovenous malformation (including aneurysm); (ii) neoplastic: brain tumor, paraneoplastic encephalitis; (iii) acquired brain injury: brain trauma, brain surgery (including stereotactic ablations).

The classification of dystonia illustrates that there is an overlap in what is described in the definition of a spastic movement disorder after a CNS lesion.

Tremor

A consensus statement on the Classification of Tremors was published in 2018 by the Task Force on Tremor of the MDS [11]. Tremor is defined as *"an involuntary, rhythmic, oscillatory movement of a body part"*. The tremor classification

has two axes, as dystonia, that describes clinical features and etiology. The authors emphasize that a tremor syndrome may have many etiologies, and a particular etiology may produce *multiple clinical syndromes* [11].

Etiologies are divided into genetic, acquired, or idiopathic, but here we will only focus on the acquired etiologies.

Examples of acquired etiologies of tremors according to the consensus statement are vascular: ischemia, hemorrhage, arteriovenous malformation; brain neoplasms; brain injury as head trauma and brain surgery [11].

This classification of tremor clearly illustrates that tremor can occur in patients that have a CNS lesion. One example is rubral tremor (Holmes tremor) that can occur as a result of stroke or traumatic brain injury and is accompanied by dystonia in up to almost 25% of the cases [12]. The overlap with dystonia is seen in the description of the "*Dystonic tremor syndromes which are tremor syndromes combining tremor and dystonia as the leading neurological signs"* [11].

If strictly using the definition of tremor, one might even argue that a non-fatigable clonus, usually classified as a spastic movement disorder, also can be classified as a tremor or a "Spastic tremor".

Chorea

To date, MDS has yet to publish a consensus document on the definition and classification of chorea. According to the Committee on Classification of the World Federation of Neurology (WFN), chorea is defined as "*a state of excessive, spontaneous movements, irregularly timed, non-repetitive, randomly distributed and abrupt in character. These movements may vary in severity from restlessness with mild intermittent exaggeration of gesture and expression, fidgeting movements of the hands, unstable dance-like gait to a continuous flow of disabling, violent movements"*[13]. In milder cases, chorea may even appear purposeful. Chorea can affect various body parts, and can interfere with speech, swallowing, posture, and gait, and disappears in sleep [14].

Chorea refers to movements that resemble dancing and is often accompanied by athetosis and ballism. Athetosis is considered a slower form of chorea, while ballism can be seen as a severe form of chorea (see below).

Chorea is usually classified as being primary (idiopathic, hereditary) or secondary (acquired) [14].

Examples of acquired etiologies of chorea are vascular conditions such as Ischemic or hemorrhagic stroke and brain tumors [13].

Hemiballismus

Neither MDS nor WFN have published a consensus paper on the definition for Hemiballismus. Hemiballismus is typically described as a hyperkinetic movement disorder characterized by intermittent, sudden, often violent, involuntary, flinging, or ballistic movements of high amplitude involving the arm and leg on one side [15]. Hemiballismus is a severe form within the spectrum of chorea.

Examples of acquired etiologies of hemiballismus are intracranial hemorrhage, ischemic stroke (most commonly from the small perforating branches of the basilar artery), brain neoplasm and traumatic brain injury [15].

Athetosis

Neither MDS nor WFN have published a consensus paper on the definition for athetosis. Athetosis is typically described as a slow, continuous, involuntary writhing movement that prevents maintenance of a stable posture [16]. Athetosis is continuous slow smooth movements that appear random and are not composed of recognizable sub-movements or movement fragments. Athetosis is typically slower and less jerky than chorea and with lack of rhythmicity and repeatability that is seen in tremor. The term "*athetosis*" derives from the Greek meaning "without position or place," reflecting the difficulty to maintain a stable posture. Contrary to other forms of chorea, athetosis continuously involves the same regions of the body [16].

Athetosis may worsen with active movements, but can be present when posturing and at rest. Athetosis usually involves the distal part of the extremities (hands or feet) more than the proximal, but can also involve the face, neck, and trunk [16].

The combination of chorea and athetosis is called "choreoathetosis". Examples of acquired etiologies of athetosis are cerebral palsy, anoxic brain injury, stroke, and traumatic brain injury [17], [18].

Athetosis should not be confused with pseudo-athetosis, which is the inability of the fingers or toes to remain still related to the loss of proprioception.

Myoclonus

Myoclonus is defined as a sudden, brief, shock-like, involuntary movement caused by muscular contractions or inhibition [19]. MDS uses the description *"involuntary jerks of a muscle or group of muscles"* [20]. Muscle contractions produce positive myoclonus, while inhibition of ongoing muscle contractions produces negative myoclonus [21]. Myoclonus can occur at rest, during an action, or maintaining a posture and can be provoked by tactile, acoustic, or visual stimuli (reflex or stimulus sensitive myoclonus) [21].

According to the neurophysiological classification, myoclonus can be divided into cortical, subcortical (including brainstem), spinal, or peripheral [20], [21].

Examples of acquired CNS etiologies of myoclonus are anoxic, brainstem vascular lesions, traumatic brain injury, neoplasm, multiple sclerosis, spinal cord injury [21].

Abnormal functioning of synergias or dyssynergia

Neither MDS nor WFN have published a consensus or statement on the role of abnormal functioning of synergias or dyssynergias that occur during the attempt to produce an active movement of the affected limb. In other words, when the patient voluntarily tries to move the spastic limb, the execution of the movement triggers the execution of dyssynergias that may lead to involuntary movements that can resemble choreatic-like movements or even imitating cerebellar ataxia and differentiates from disinhibited co-contraction by seeming more chaotic [22]. These dyssynergias, or abnormal muscle synergies, are movement disorders characterized by involuntary abnormal extra movements (lack of muscle selectivity) that occur in conjunction with intended voluntary movements [23]. They can arise as a result of a CNS lesion [23] and are present in combination with intended voluntary movements in the affected limb in an UMN syndrome. Dyssynergia is most common in the proximal segments of the limbs and adjacent trunk segments [23].

Dyssynergia can be differentiated from chorea and hemiballismus in that it occurs exclusively during voluntary movements. It also differs from activity-induced myoclonic movements by being less abrupt or "jerky" in character. Likewise, it can be distinguished from cerebellar ataxia because it lacks the typical cerebellar intention tremor and other cerebellar signs.

It is important to note, however, that the term *dyssynergia* is also used in the literature to describe a component of cerebellar ataxia—namely impaired muscular coordination resulting in uncoordinated and abrupt movements [24].

In addition, dyssynergias differ from synkinesias, which are typically observed in facial muscles due to aberrant facial nerve reinnervation, post Bell's palsy.

Non-CNS-related movement disorders

There are many other movement disorders with psychiatric or unknown origin. Examples are stereotypies, tics, as well as neuropsychiatric movement disturbances, i.e., those seen as part of Pediatric Acute-Onset Neuropsychiatric Syndrome (PANS) and Pediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcal Infections (PANDAS). As these are outside the scope of this paper, they will not be discussed further.

Overlap of hyperkinetic movement disorders with CNS lesions

As hitherto mentioned, those hyperkinetic movement disorders can potentially result from a CNS lesion. This means that we often see overlapping phenotypes of muscle overactivity as illustrated by Fig. 1. The most frequent combination of phenotypes related to a CNS lesion, in clinical experience, are spasticity and dystonia. This overlap of

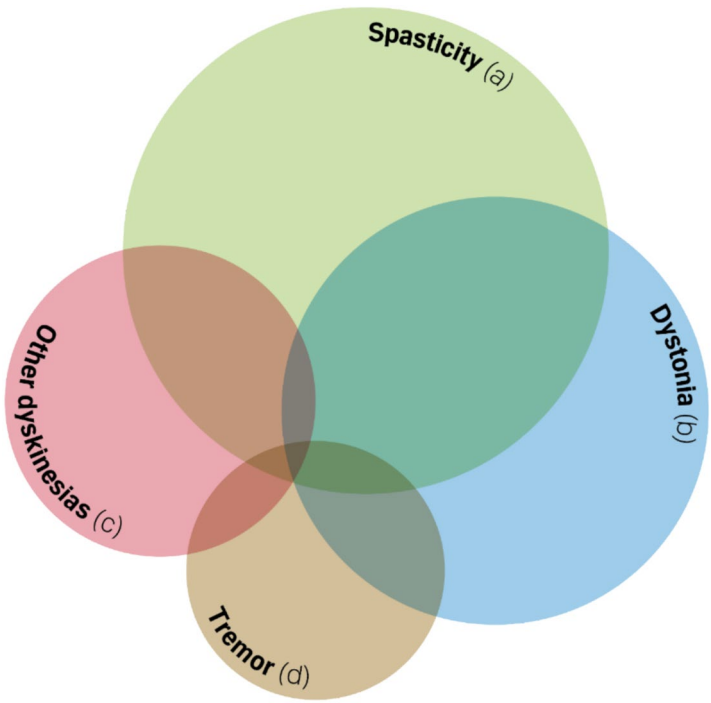

Fig. 1 Overlapping hyperkinetic movement disorders. The size of the circles relates to a crude approximation based on clinical experience of the frequency of the different types of movement disorders after a CNS lesion. [a]Proposed definition of spasticity "A movement disorder characterized by Involuntary muscle overactivity in the presence of a CNS lesion". [b]Dystonia is defined as a movement disorder characterized by sustained or intermittent abnormal movements, postures, or both. [c]Examples of other dyskinesias are chorea, hemiballismus, athetosis, myoclonus, dyssynergia. [d]Tremor is defined as an involuntary, rhythmic, oscillatory movement of a body part. In this context, excluding essential tremor and Parkinson disease.

different hyperkinetic movement disorders with overlapping phenomena can complicate definitions and descriptions of phenotypes.

Pathophysiology in hyperkinetic movement disorders

The scope of this present work is limited to a brief overview of the relevant supraspinal and spinal networks involved in the pathophysiology of hyperkinetic movement disorders. We acknowledge that pathophysiology is far more complex than this outline.

To understand the pathophysiology of hyperkinetic movement disorders, we need to understand the anatomy underlying the regulation of muscle tone. No matter the phenotype of the hyperkinetic movement disorder, muscle activation and tone generation in all phenotypes come from the same anatomical structures, although the primary dysregulation and the neurophysiological mechanism of the hyperkinetic movement disorder might vary.

Muscle tone is regulated and controlled by spinal and supraspinal mechanisms as illustrated by Fig. 2. Motor control involves the cortex (extensive processing capability), basal ganglia (learning and teaching of context dependent tasks), cerebellum (fine-tuning), brainstem reticular system (common pathway for ascending and descending tracts), spinal cord (the main pathway for ascending and descending tracts), and muscle spindle [25].

Normal muscle tone depends on central generators of tone, descending motor pathways, and the spinal-motor reflex circuit. Central generators of tone include cortico-striato-pallido-thalamo-cortical loops and cerebello-thalamo-cortical circuits [1]. These interacting systems modulate output through corticospinal pathways while other descending motor pathways are under no (or very limited) direct cortical control. These include the vestibulospinal tract, the rubrospinal tract, the tectospinal tract, the dorsal and

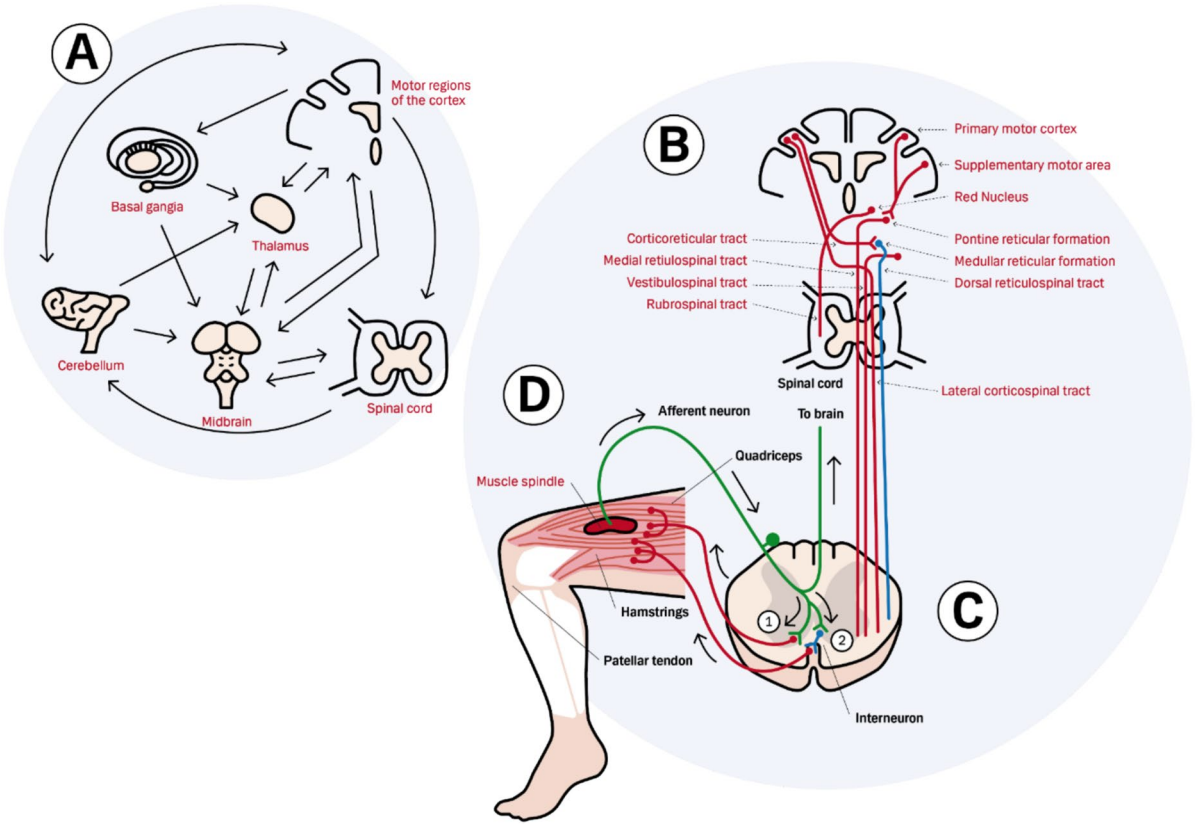

Fig. 2 Simplified anatomical model of regulation of muscle tone. **A**: Central generators of tone include cortico-striato-pallido-thalamo-cortical loops and cerebello-thalamo-cortical circuits. Modulates output through corticospinal pathways. **B** and **C**: The supraspinal control is regulated by excitatory and inhibitory extrapyramidal tracts, including input from basal ganglia and cerebellum. **D**: The spinal control depends on the interaction between muscle spindle and spinal cord along with the interneurons as well as the sensory input, e.g., from nociceptors. Red lines: excitatory neurons and tracts. Blue lines: inhibitory neurons and tracts. Will also include Renshaw cells. Green lines: afferents from the muscle spindle. Afferents also origin in the Golgi tendons as well as nociceptors. Afferents include Type 1a, Type 1b, and Type II

medial reticulospinal tracts. The spinal control depends on the interaction between muscle spindle and spinal cord along with the interneurons as well as the sensory input, e.g., from nociceptors. Supraspinal control is regulated by excitatory and inhibitory extrapyramidal tracts including input from the basal ganglia and cerebellum.

The pathophysiology of hyperkinetic movement disorders appears to include similar alterations in the physiological properties of neurons in these areas [26]. Phenotype differences in the hyperkinetic disorders illustrate the differing influence and degree of each of these changes and the various motor and, in some cases, non-motor pathways involved in mediating each disorder [26].

For example, dystonia is thought to have altered tone secondary to network disruption in the basal ganglia, the thalamo-cortical circuits, and their connections, including to the cerebellum and cortex. Abnormal function at any place of the network between the basal ganglia, thalamus, cerebellum, brainstem, and primary motor and sensory cortices (or pathway connecting them) may give rise to dystonia [1], [27].

Likewise, the pathophysiology of chorea involves a functional dysregulation of the basal ganglia motor circuit, where the final thalamic-cortical output is increased, resulting in increased movement and chorea [14], while the pathophysiology of Parkinson's tremor, essential tremor, dystonic tremor, and Holmes tremor generally involve the cerebello-thalamo-cortical pathway [28].

Proposed definition and classification of spasticity

MDS and WFN have no consensus on definition and classification of spasticity, and as described above, there is an overlap especially between the classification and definition of dystonia and spasticity. Sometimes these hyperkinetic movement disorders can be considered both as primarily spastic or primarily dystonic. Focal lesions involving cerebrum and spinal cord (e.g., stroke, MS, TBI, SCI) resulting in focal, segmental paresis tends to be considered as a spastic movement disorder, whereas diffuse lesions, especially involving basal ganglia, most often are considered as a dystonic movement disorder (e.g., encephalitis or toxic/metabolic encephalopathy).

We propose a simplified definition as: a spastic movement disorder is characterized by involuntary muscle overactivity in the presence of a CNS lesion, most often in conjunction with paresis. Non-neuronal components such as contractures and rheological changes should be assessed separately**.** When the spastic movement disorder hinders body function, activity, and/or participation, it is to be defined as disabling spasticity [29].

In our proposed classification of spasticity, the structure made by MDS for the axes used in dystonia and tremor is applied. Axis 1 in Table 1 describes the clinical characteristics of the spastic movement disorder.

The distribution of age of onset has been chosen from MDS definition and classification of dystonia [9]. The body distribution is from the European expert consensus paper on patient selection for the management of disabling spasticity. Here, definitions of focal, multi-focal, segmental, multi-segmental and generalized spasticity are described [29]. Disease course describes how the spastic movement disorder might progress, depending on the etiology described in Axis 2 (Table 2). In addition, the spastic movement disorder might change over time even if the etiology is considered a static disease as in children with cerebral palsy, and in these cases, “evolving disease course” should be used. The phenomenological description is differentiated into the observed phenomena when the patient is being examined both during active and passive movements. Last but maybe the most important in the treatment of spastic movement disorder is how the patient and/or caregiver experience the impact of the muscle overactivity on body function, activity, and participation.

To achieve a common language when describing the phenotypes observed in the clinic, Fig. 3 illustrates the

Table 1 Axis 1, clinical characteristics for spastic movement disorder

AXIS 1: clinical characteristics				
Age at onset	Body distribution	Disease course	Phenomenological description	Impact on body function, activity, and participation
Perinatal/infancy	Focal	Static	Phasic reflex overactivity	Is spasticity disabling?
Childhood	Multi-focal	Evolving	Stimuli-induced muscle overactivity	Patient-reported outcome measures (PROM)
Adolescence	Segmental	Progressive	Constant muscle overactivity	
Early adulthood	Multi-segmental		Lack of muscle selectivity	
Late adulthood	Generalized			

Table 2 Axis 2, examples of etiologies in spastic movement disorder

AXIS 2: etiology
Stroke
Central demyelinating disorders: MS, NMO and MOGAD
Traumatic brain injury
Spinal cord injury
Cerebral palsy
Anoxia or hypoxia
Encephalitis
Neoplasm
Hereditary spastic paraplegia
Myelopathies, inclusive of HTLV-1-associated myelopathy

phenomenological description from Axis 1. These are clonus as a phasic reflex overactivity that can be fatigable or non-fatigable, muscle spasms (extensor, flexor, adductor spasms), and activity-induced muscle hypertonia that are stimuli-induced muscle overactivity. Persistent muscle hypertonia is a constant muscle overactivity and associated reactions and dysregulated co-contraction that can be regarded as lack of muscle selectivity. Dysregulated co-contraction can also be elicited as a stimulus-induced muscle overactivity. The phasic and tonic stretch reflex have been highlighted in blue boxes and can be part of the different phenotypes of muscle overactivity. For definitions and description of phenotypes observed in a spastic movement disorder, see Table 3.

Discussion

Definitions and classifications will never be a true description of reality, but rather a practical simplification. As with maps of the world, they will never capture the real world lived by our patients but are essential for us to orient ourselves and for decisions on treatment.

The reason for changing the descriptors to pure phenomenological terms is that the old terms have a historical baggage where they sometimes are tentatively mechanistic or have different interpretations in different contexts.

With a phenomenological approach, we try to take into consideration that the hyperkinetic movement disorders of dystonia and spasticity often cannot be differentiated from each other by neither definition and description nor clinical observation. There is, therefore, a need to fuse how different descriptors of phenotypes are used in different clinical

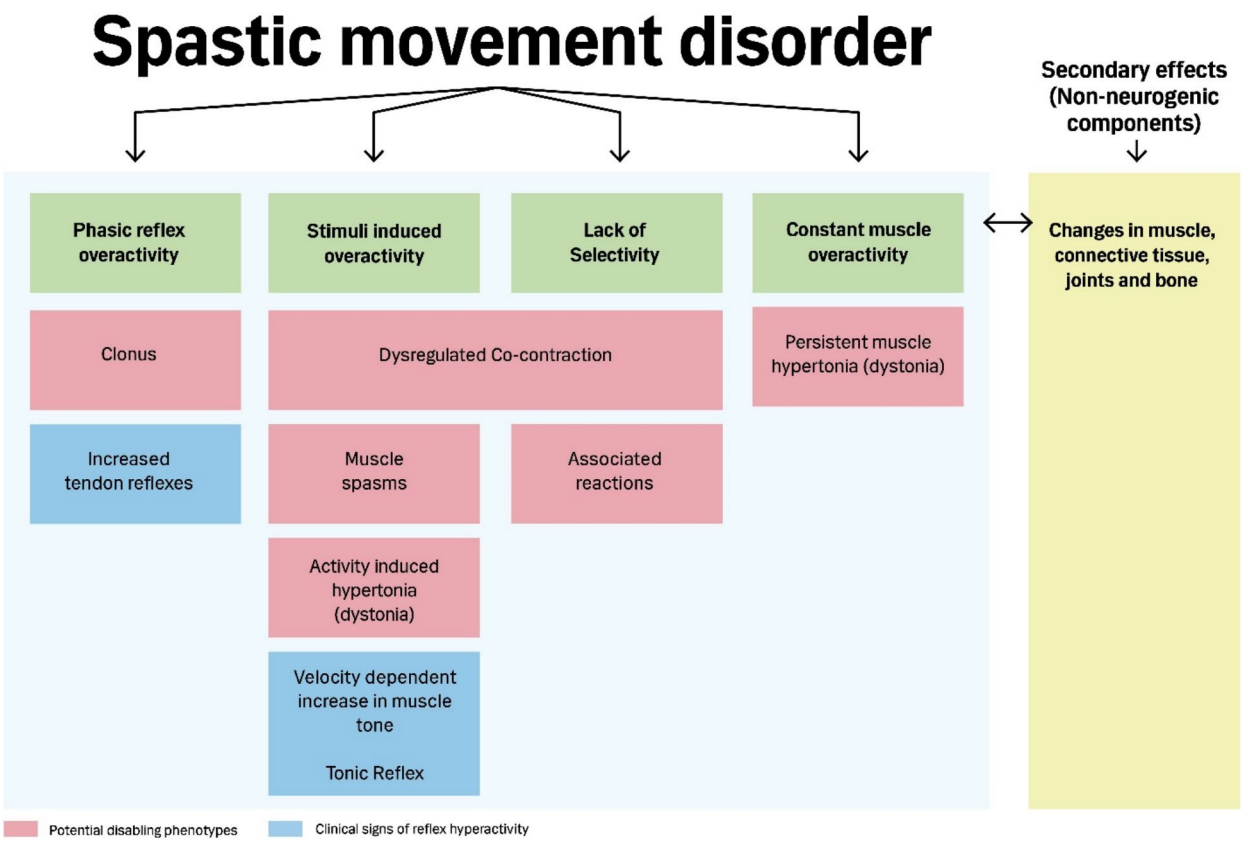

Fig. 3 Sub-classification of spastic movement disorder

Table 3 Definitions and description of potentially disabling phenotypes observed in a spastic movement disorder

Term	Description of phenotypes
Dysregulated co-contraction	A form of antagonistic muscle co-activation, refers to an abnormal redirection of voluntary supraspinal signals during an agonist muscle command (lack of selectivity and stimuli induced). This misdirection leads to the inappropriate activation of antagonist motor units [3]
Persistent muscle hypertonia (dystonia)	A constant, involuntary muscle activation occurring at rest without any stretch or voluntary movement. Its severity can be influenced by the degree of tension exerted on the affected muscle. Over time, spastic dystonia leads to abnormal postures and progressively worsening deformities [3]
Spasms	Characterized by rapid, short-lasting involuntary muscle contractions that occur while at rest, typically associated with heightened excitability of motor neurons [3]. Spasms are stimuli-induced overactivity and possible eliciting factors can be movement, pain, discomfort, posture, and even other medical problems like urinary tract infections and pressure sores can trigger spasms [30]
Clonus	A phasic reflex overactivity and presents as an involuntary, rhythmic muscle contraction and relaxation in response to sudden stretch and presents as a series of rapid, oscillating jerks (commonly seen at the ankle, knee, or wrist) with a frequency around 5–8 Hz [31]
Activity-induced hypertonia (dystonia)	An abnormal dystonic posturing that occurs during active movement of the affected limb (stimuli induced overactivity). In other words, when the patient voluntarily tries to move the spastic limb, the movement triggers an involuntary, often fixed, postural pattern because of overactivation of agonist muscles [22]
Associated reactions	Involuntary movements that occur in a paretic or affected limb during voluntary effort elsewhere or during involuntary activities like yawning, sneezing, or coughing (lack of selectivity). In a classic example, a hemiplegic stroke patient exhibits involuntary elbow flexion and upward arm movement on the weak side while walking or when exerting effort with the other limb [32]

contexts. One example of this is how the term "at rest and during action" as well as "fixed" (excluding psychogenic) [9] refers to the former term "persistent" muscle hypertonia [10]. The formerly used term to describe this phenotype in spastic movement disorders would be "static spastic dystonia" [1]. Persistent hypertonia, however, is more descriptive of the observed phenomena.

Likewise, in the nomenclature of dystonias, "action induced dystonia" [9] could as well be placed under the phenotype of "activity-induced muscle hypertonia", where hypertonia in spastic movement disorders is less likely to be task-specific. The formerly used term to describe this type of phenotype in spastic movement disorders would be "activity induced spastic dystonia" [1].

The phenomena of dyssynergias still need further exploration and clearer definition before the term can be included in Fig. 3. It may represent a lack of selectivity, activity-induced overactivity, or a combination of both, in analogy with co-contraction.

In both the scientific literature and clinical practice, different terms are used inconsistently, and are therefore often confusing. Beyond differentiating the various components of the spastic movement disorder, we would also like to advocate for the use of the term *spasticity syndrome* as an umbrella concept encompassing both neurological and non-neural components, including patient-reported symptoms. This allows for greater clarity regarding when non-neural factors and patients' subjective experiences are included.

The bodily distribution of muscle overactivity in Axis 1 is the one described in the "European expert consensus on improving patient selection for the management of disabling spasticity with intrathecal baclofen and/or botulinum toxin type A" [29]. In this manuscript, there is a clear description of the body distribution. This bodily distribution does not follow the same descriptions in the "definition and classification of dystonia" [9]. Here, e.g., the term "hemidystonia" being typically a patient who has an acquired brain lesion on the contralateral side. In the body distribution, in Axis 1, this would be called multi-segmental muscle overactivity, since two segments (one arm and one leg and the trunk) are affected. Further, it can be argued that a patient with a hemidystonia (according to the dystonia nomenclature) related to an acquired brain injury with paresis, rather than having dystonia, would be defined as having a spastic movement disorder.

Conclusion

This present work provides an overview of the current terms and definitions of hyperkinetic movement disorders that can be related to a CNS lesion and proposes suggestions on common terminology and definitions, including description of phenotypes observed in a spastic movement disorder. In addition, to provide a framework, we proposed using the classification axes previously utilized by the MDS in classifying dystonia and tremor. These proposed terms will hopefully address confusion and inaccuracy brought about by the frequent overlap in the pathophysiology and clinical presentation of the various movement disorders. In the near

future, we foresee these new terms being used commonly in communication between clinicians and researchers and in designing assessment tools that will guide clinical evaluation and choice of interventions, and measure outcomes.

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Declarations

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