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ORIGINAL COMMUNICATION

Defining spasticity: a new approach considering current movement disorders terminology and botulinum toxin therapy

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Abstract
Spasticity is a symptom occurring in many neurological conditions including stroke, multiple sclerosis, hypoxic brain damage, traumatic brain injury, tumours and heredodegenerative diseases. It affects large numbers of patients and may cause major disability. So far, spasticity has merely been described as part of the upper motor neurone syndrome or defined in a narrowed neurophysiological sense. This consensus organised by IAB—Interdisciplinary Working Group Movement Disorders wants to provide a brief and practical new definition of spasticity—for the first time—based on its various forms of muscle hyperactivity as described in the current movement disorders terminology. We propose the following new definition system: Spasticity describes involuntary muscle hyperactivity in the presence of central paresis. The involuntary muscle hyperactivity can consist of various forms of muscle hyperactivity: *spasticity sensu strictu* describes involuntary muscle hyperactivity triggered by rapid passive joint movements, *rigidity* involuntary muscle hyperactivity triggered by slow passive joint movements, *dystonia* spontaneous involuntary muscle hyperactivity and *spasms* complex involuntary movements usually triggered by sensory or acoustic stimuli. Spasticity can be described by a documentation system grouped along *clinical picture* (axis 1), *aetiology* (axis 2), *localisation* (axis 3) and *additional central nervous system deficits* (axis 4). Our new definition allows distinction of spasticity components accessible to BT therapy and those inaccessible. The documentation sheet presented provides essential information for planning of BT therapy.

Keywords Spasticity · Rigidity · Dystonia · Spasms · Contractures · Treatment · Botulinum toxin therapy

Introduction

Spasticity is a symptom occurring in many neurological conditions including stroke, multiple sclerosis, hypoxic brain damage, traumatic brain injury, tumours and heredodegenerative diseases. It affects large numbers of patients and may be the cause of major disability [13]. The need for its treatment is obvious [2]. However, as indicated by a high percentage of untreated patients, it does not seem to receive appropriate medical attention [8] although botulinum toxin (BT) therapy is now offering a highly effective and well-tolerated treatment option [4, 19].

So far, spasticity has merely been described as part of the upper motor neurone (UMN) syndrome or defined in a narrowed neurophysiological sense [12]. Referring to a previous definition of spasticity [3] this consensus wants to provide a brief and practical new definition of spasticity—for the first time—based on its various forms of muscle hyperactivity as described in the current movement disorders terminology. This new definition also wants to facilitate the use of BT therapy for treatment of spasticity.

This paper is based on a consensus of international spasticity experts. It was organised by IAB—Interdisciplinary Working Group for Movement Disorders, a multidisciplinary special interest group promoting multimodal and interdisciplinary therapies in movement disorders including spasticity.

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Clinical description

Spasticity describes the co-occurrence of involuntary muscle hyperactivity and central paresis. Spasticity is one of many signs of the UMN syndrome, but it is not identical with it. The UMN syndrome is not precisely defined. Table 1 gives an overview about its *primary signs* as featured by Barnes [1]. The signs of the UMN syndrome may be divided into positive and negative phenomena which both may induce various secondary functional deficits including deficits on walking, static or dynamic postural stability, hand use, swallowing and dysarthria. After some time, complications may occur. The most important one are contractures describing mechanical alterations manifesting as reduced range of passive joint motion. They may be caused by intramuscular (increased muscle viscosity) and extramuscular (shortening of tendons and ligaments) processes and by arthritic alterations. When and why contractures develop, is not fully understood. Another major complication of the UMN syndrome is pain. Despite its massive effects on the patient's quality of life, its role in the UMN syndrome is still not yet appropriately acknowledged. Other complications include decubitus, entrapment of nerves, veins and arteries and lymphedema. All of those complications may increase pain and, thus, increase muscle hyperactivity in return. Depending on the underlying aetiology, patients can also suffer from additional central nervous system deficits of the motor system, such as ataxia, apraxia, bradykinesia and of other systems including dementia, aphasia and incontinence. They frequently limit therapeutic strategies.

Aetiology

Spasticity can be caused by large number of aetiologies. Table 2 gives an overview about the different specific aetiologies. Stroke, multiple sclerosis and cerebral palsy are frequent causes of spasticity, hypoxic brain damage and traumatic brain or spinal cord damage are less frequent, but may produce particularly severe spasticity.

Table 2 Aetiology of spasticity classified by the localisation of their underlying pathological alterations

Supraspinal
Stroke
Multiple sclerosis
Cerebral palsy
Hypoxic brain damage
Traumatic brain injury
Mass lesions: tumours, vascular malformations
Inflammation
Spinal
Cervical myelopathy
Mass lesions: tumours, vascular malformations
Inflammation
Stroke
Traumatic spinal cord lesion
Hereditary spastic paraplegia
Spina bifida
Myelomeningocele
Tethered cord
Mixed
Multiple sclerosis
Motoneuron disease, primary lateral sclerosis
Inflammation

Classification

As shown in Table 2 spasticity may be classified according to its pathological origin within the central nervous system and its aetiology. Most frequently, however, it is classified according to its localisation within the body as arm (upper limb) spasticity, leg (lower limb) spasticity, hemispasticity, paraspasticity and tetraspasticity.

Table 1 Signs of the Upper Motor Neurone syndrome according to Barnes [1]

Positive phenomena	Negative phenomena
Increased tendon reflexes with radiation of effect	Weakness
Clonus	Reduced dexterity
Positive Babinski sign	Fatigue
Spasticity	
Extensor spasms	
Flexor spasms	
Mass reflex	
Dyssynergic patterns of co-contraction during movement	
Associated reactions and other dyssynergic and stereotypical spastic dystonias	

Pathophysiology

UMN's project to spinal motor neurons and spinal reflex circuits through various descending pathways exerting excitation or inhibition. These descending pathways include the excitatory monosynaptic pyramidal tract (lateral corticospinal tract) originating from pyramidal cells in precentral cortical areas 4 and 6. Isolated pyramidal tract lesions produce only little paresis with predominant impairment of fine finger movements, little hyperreflexia and a positive Babinski phenomenon [20]. They do not produce relevant muscle hyperactivity [20]. Their lesion, therefore, produces the extraordinary situation of a flaccid central paresis. Other descending pathways consist of several multisynaptic 'parapyramidal' tracts [19] including the excitatory medial reticulospinal tract, the excitatory vestibulospinal tract and the inhibitory dorsal reticulospinal tract. Lesions of the parapyramidal tracts produce most of the paresis and most of the muscle hyperactivity seen in UMN syndromes. The lesioning patterns of excitatory and inhibitory pathways at different levels within the motor tracts determine the degree of paresis and muscle hyperactivity and their localisation within the body. The duration of the lesion also seems to be important as spastic muscle hyperactivity develops with delay indicating the activation of plasticity processes.

Reduced spinal inhibition produces increased activity of the phasic stretch reflex (deep tendon reflex, tendon jerks) with cloni as its typical clinical manifestation. It also produces increased activity of nociceptive reflexes (flexor withdrawal reflex) manifesting as flexor spasms. It unmasks the Babinski phenomenon, a phylogenetically and ontogenetically suppressed ancient withdrawal reflex.

Spastic muscle hyperactivity can be explained by lesions affecting the complex system of central excitation and inhibition. In spasticity sensu strictu and clonus the phasic stretch reflex is disinhibited, when fast passive joint movements with strong afferent inflow are exerted. In rigidity the phasic stretch reflex is disinhibited already by slow passive joint movements with weak afferent inflow, whereas in dystonia muscle hyperactivity occurs spontaneously. In spasms nociceptive reflexes are released by external triggers.

Therapies

Table 3 gives an overview about current therapies of spasticity. Probably, the most effective treatment of spasticity is BT therapy [9, 14, 18, 21]. It produces robust effects and its direct intramuscular applications avoid typical adverse effects of oral drugs caused by irritation of absorption or excretion organs [5]. When spasticity is particularly widespread and severe, the total BT doses available may become exhausted. The introduction of the BT high dose therapy [6, 22]. Local perineural injections of phenol may produce definite and lasting nerve blockade whereas local intramuscular injections of phenol only produce temporary paresis [10, 15]. Continuous intrathecal baclofen application through an implanted pump system [7] is especially effective in paraspasticity. Additionally, tetraspasticity may be targeted with increased baclofen doses and catheter tip positions in the midthoracic level. Its use in hemispasticity is controversial as paretic adverse effects on the contralateral limb may occur. Oral spasmolytics such as baclofen, tizanidine, tetrazepam, tolperisone, dantrolene and clonazepam are less effective and frequently produce adverse effects especially in the altered brain. Cannabinoids have spasmolytic and analgesic effects. A fixed ready-made tetrahydrocannabinol/cannabidiol mixture for oromucosal absorption is registered only for spasticity due to multiple sclerosis [11]. It is generally well tolerated, but its efficacy, however, seems limited. Surgical interventions including selective dorsal rhizotomy, longitudinal myelotomy are controversial. Selective fasciculotomies seem to work in focal spasticity [16]. Orthopaedic surgery offers an option for complications of spasticity.

Definitions

With the aforedescribed features we propose the following new definition system. It is based on the current movement disorders terminology and summarised in Table 4.

Spasticity

Spasticity describes involuntary muscle hyperactivity in the presence of central paresis. The involuntary muscle

Table 3 Therapeutic options for treatment of spasticity

Botulinum toxin therapy, phenol
Intrathecal baclofen
Oral spasmolytics: baclofen, tizanidine, tetrazepam, tolperison, dantrolene, clonazepam, cannabinoids, tetrahydrocannabinol/cannabidiol
Surgical interventions: selective dorsal rhizotomy, longitudinal myelotomy, orthopaedic surgery
Adjuvant therapies: physiotherapy, occupational therapy, relaxation techniques, aquatics
Orthotics: splints, casting, taping wheelchairs, standers

Table 4 New definition of spasticity

Central paresis
Involuntary muscle hyperactivity
Spasticity sensu strictu
Dystonia
Rigidity
Spasms
Complications
Contractures
Pain

hyperactivity can consist of spasticity sensu strictu, of rigidity, of dystonia and of spasms or a mixture of those elements. Complications in the form of pain and contractures may occur.

Spasticity sensu strictu

Spasticity sensu strictu describes involuntary muscle hyperactivity triggered by rapid passive joint movements. The clasp knife phenomenon may occur.

Rigidity

Rigidity describes involuntary muscle hyperactivity triggered by slow passive joint movements. Rigidity is caused by neuronal hyperactivity. Additional mechanical factors, especially muscular viscosity, may contribute.

Dystonia

Dystonia describes spontaneous involuntary muscle hyperactivity. Dystonia frequently features co-contractions in antagonist muscle groups. It often becomes worst during voluntary activation of the dystonic muscle groups or of other non-dystonic muscle groups. This phenomenon may be called action-induced or dynamic dystonia.

Spasms

Spasms are complex involuntary movements usually triggered by sensory or acoustic stimuli. They may be painful because of the intensity of the muscle contractions, but also because of additional central sensory tract affection.

Description and documentation

For description and documentation of spasticity we propose the documentation sheet shown in Table 5. It is based on the new definition system of spasticity and it is grouped along 4 axes. Axis 1 describes the clinical picture. It reviews the primary signs of spasticity based on the new definitions introduced as well as secondary functional deficits and complications. Axis 2 addresses the specific aetiology, axis 3 the localisation and axis 4 additional clinical deficits. Severity of the muscle hyperactivity may be described by the well-established scale including the Modified Ashworth Scale, the Tardieu Scale and the Frequency of Spasms Score.

Consequences for BT therapy

Our definition allows distinction of spasticity components accessible to BT therapy and spasticity components inaccessible to BT therapy. Rigidity, dystonia, spasms and associated pain are directly accessible to BT therapy. Spasticity sensu strictu may be targeted, but given its low functional significance, BT therapy may not produce relevant functional improvement. Contractures are inaccessible by BT therapy as they are based on mechanical alterations unresponsive to BT's paretic effects. However, as contractures are often difficult to distinguish from spastic muscle hyperactivity probatory BT applications seem justified. Early BT therapy may prevent the formation of contractures [17]. The documentation sheet presented provides essential information for planning of BT therapy. The degree of central paresis is an important modifier of BT dosing: if it is severe, BT may be applied generously as no further functional impairment can occur. If central paresis is mild, muscle function may improve due to reduction of the spastic antagonist muscle hyperactivity. In patients with mild central paresis BT should be dosed carefully in order not to reduce residual target muscle functioning. Spastic posturing as described separately for all relevant joints is the key to identify target muscles for BT therapy. Information about secondary functional deficits and complications can help to focus BT therapy to the most relevant areas when maximal total BT doses are reached. Information on the aetiology is relevant for BT therapy as usually only spasticity due to stroke is a registered indication in most countries. Additional central nervous system deficits may limit BT-induced improvement as they may limit the outcome of any other therapy.

Table 5 Documentation sheet. Sheet to document spasticity according to 4 different axes. Individual patients can be described by circling each relevant item

Axis 1 : Clinical description			
Primary signs	Central paresis	Mild	
		Severe	
	Muscle hyperactivity	Spasticity sensu strictu	
		Dystonia	
		Rigidity	
		Spasms	
	Posture	Jaw	Closure
		Shoulder	Abduction
			Adduction
		Elbow	Flexion
			Extension
		Wrist	Flexion
			Extension
		Fingers	Flexion
			Extension
		Thumb	Flexion
			Adduction
		Thigh	Adduction
			Flexion
		Knee	Flexion
			Extension
		Ankle	Flexion
			Extension
			Equinovarus
		Toes	Flexion
			Extension
Secondary functional deficits	Reduced walking		
	Reduced postural stability		
	Reduced dexterity		
	Reduced swallowing		
	Dysarthria		
Complications	Pain		
	Contractures		
	Decubitus		
	Entrapment of nerves, veins and arteries		
	Lymphedema		
Axis 2 : Aetiology			
Stroke			
Multiple Sclerosis			
Traumatic brain injury			
Hypoxic brain injury			
Cerebral palsy			
Tumour			
Degenerative CNS disease			

Table 5 (continued)

Axis 3 : Localisation
Arm spasticity
Leg spasticity
Hemispasticity
Paraspasticity
Tetraspasticity
Axis 4 : Additional deficits
Dementia
Depression
Apraxia
Others

Compliance with ethical standards

Conflicts of interest Dressler D: DD received honoraria for services provided to Allergan, Ipsen, Merz, Desitin, Syntaxin, Abbvie, Medtronic, St Jude, Boston Scientific, Almirall, Bayer, Sun, Teva, UCB, IAB-Interdisciplinary Working Group for Movement Disorders. He is shareholder of Allergan and holds patents on botulinum toxin and botulinum toxin therapy. Bhidayasiri R: No conflict of interest to report. Stock Ownership in medically related fields: None. Consultancies: Ipsen Pharmaceuticals. Advisory Boards: Britannia Pharmaceuticals. Honoraria to speak: Novartis, BL Hua, Abbott. Partnerships: None. Grants: Newton Fund UK, Thailand research fund, Ratchadapisek sompot faculty grant and CU-CLUSTER fund of Chulalongkorn University. Intellectual Property Rights: Parkinson's disease laser cane, Tremor analysis algorithm, Nocturnal monitoring device (NIGHT-Recorder). Expert Testimony: None. Employment: Chulalongkorn University. Royalties: Blackwell-Wiley, Humana Publications. Others: None. Bohlega S: Nothing to declare. Chana P: Nothing to declare. Chien S: Nothing to declare. Chung TM: Honoraria from Ipsen, Allergan and Merz to lecture in Symposium, Training Couses and Advisory Board participation. Participation in clinical research from Ipsen. No research funding and no financial interest in Botulinum Toxin. Colosimo C: Consultancies with Zambon, Sunovion, Ipsen and Bial. Royalties from Cambridge University Press and Oxford University Press. Ebke M: Nothing to declare. Fedoroff K: Nothing to declare. Frank B: Nothing to declare. Kaji R: Nothing to declare. Kanovsky P: Nothing to declare. Kocer S: Nothing to declare. Micheli F: Nothing to declare. Orlova O: OO is scientific consultant for Ipsen, Allergan, Merz and Microgen. PausS: SP received research grants and lecturer fees from Ipsen Pharma and Merz Pharmaceuticals. Member of the Advisory Boards of Ipsen Pharma and Merz Pharmaceuticals. Pirtosek Z: Nothing to declare. Relja M: Northing to declare. Rosales RL: RR received advisory, research and travel grants from Ipsen Pharma and lecture and travel grants from Allergan and Merz Pharma. Sagastequi A: AS received honoraria for lecturing from Allergan and Ipsen. Schoenle PW: Nothing to declare. Shahidi G: Nothing to declare. Timerbaeva S: Nothing to declare. Walter U: UW received speaker honoraria and travel reimbursement from Merz Pharma, Ipsen Pharma, Allergan, Bristol-Myers Squibb, Daiichi Sankyo, Bayer Vital and Pfizer, and a research grant from Merz Pharma. Adib Saberi F: FAS received reimbursement from Abbot, Abbvie, Almirall, Allergan, Bayer, Desitin, Dynamed, Hempel GesundheitsPartner, Ipsen, Johnson & Johnson, Licher, Meda, Medtronic, Merz, Orion, PTZ Nawrath, Sensomotorik & Rehabilitation Hellmuth & Thiel, Sintetica, Sporlastic, Sun, Teva, Tricumed, TRS Med, UCB.

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