Spasticity, an impairment that is poorly defined and poorly measured

S Malhotra, AD Pandyan School of Health & Rehabilitation and Research Institute for Life Course Studies, University of Keele, CR Day Research Institute for Environment, Physical Sciences & Applied Mathematics, University of Keele, PW Jones Research Institute for Science and Technology in Medicine, University of Keele, UK and H Hermens Roessingh Research and Development BV, Enschede, The Netherlands

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Objective: To explore, following a literature review, whether there is a consistent definition and a unified assessment framework for the term 'spasticity'. The congruence between the definitions of spasticity and the corresponding methods of measurement were also explored.

Data sources: The search was performed on the electronic databases Web of Science, Science Direct and MEDLINE.

Review methods: A systematic literature search of publications written in English between the years 1980 and 2006 was performed with the following keywords: spasticity and tone. The search was limited to the following keywords: stroke, hemiplegia, upper, hand and arm.

Results: Two hundred and fifty references contributed to this review (190 clinical trials, 46 literature reviews, and 14 case reports). Seventy-eight used the Lance definition; 88 equated spasticity with increased muscle tone; 78 provided no definition; and six others used their own definitions for spasticity. Most papers used a single measure and some used more than one. Forty-seven papers used neurophysiological methods of testing, 228 used biomechanical methods of measurement or assessment, 25 used miscellaneous clinical measures (e.g. spasm frequency scales) and 19 did not explicitly describe a measure.

Conclusion: The term spasticity is inconsistently defined and this inconsistency will need to be resolved. Often, the measures used did not correspond to the clinical features of spasticity that were defined within a paper (i.e. internal validity was compromised). There is need to ensure that this lack of congruence is addressed in future research.

Introduction

Address for correspondence: AD Pandyan, 0.01 Mackay Building, School of Health and Rehabilitation, Keele ST5 5BG, UK. e-mail: a.d.pandyan@shar.keele.ac.uk

clonus and dyssynergic movement patterns) and 'negative features' (i.e. muscle weakness, loss of dexterity and fatigability). Although both positive and negative features contribute to the resulting functional loss in patients with an upper motor neuron lesion, there is a substantial focus on one particular positive feature: 'spasticity'. The focus on spasticity results from the premise that spasticity interferes with functional recovery and leads to secondary complications such as contractures, weakness and pain.^{1,2}

Spasticity was originally associated with a soft yielding resistance that appeared only towards the end of a passive stretch and an increased amplitude stretch reflex.³ Two decades later, during a post-conference discussion, it was suggested that spasticity could be defined as 'a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) and increased tendon jerks resulting from disinhibition of the stretch reflex, as one component of an upper motor neurone lesion'.⁴⁻⁶ The North American Task Force for Childhood Motor Disorders, attempting to improve the precision of the above definition, have suggested that spasticity should be redefined as 'a velocity dependent increase in hypertonia with a catch when a threshold is exceeded'.⁷ More recently, the members of the SPASM consortium, putting forward the argument that the existing definition were to narrow for clinical purposes, suggested that the definition be widened to 'disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles'.⁸ This latter definition purports to shift the focus of the definition to encompass current understanding of pathophysiology and clinical practice.

For research into spasticity to be valid it is important that the measures or outcome measures of spasticity are also valid and reliable. A prerequisite for identifying valid and reliable measurement(s) is either precise definition(s) or an unambiguous description(s). The aims of this work were to explore whether such a definition existed and, if one did, were the measures used congruent to the same definition. As the literature relating to the measurement and treatment of spasticity in the upper motor neuron syndrome is vast and all measurements developed for the lower limb have also been adapted for use in the upper limb, the search to support this review was limited to articles related to upper limb spasticity post stroke from (Web of Science, Science Direct and MEDLINE) between the years 1980 and 2006.

Methods

A search was performed by a single reviewer on published articles between 1980 (following the first formal definition by Lance) and 2006 on the following three electronic databases: Web of Science, Science Direct and MEDLINE, with keywords:

- (1) spasticity
- (2) tone
- (3) stroke
- (4) hemiplegia
- (5) upper
- (6) hand
- (7) arm

Search combinations were:

- (8) 1 or 2
- (9) 3 or 4
- (10) 5 or 6 or 7
- (11) 8 and 9 and 10.

Exclusion criteria

Animal studies, duplicates and references that were written in languages other than English were excluded from this review.

Inclusion criteria

Published references were fully reviewed if they fell into one of the following categories:

- characterization of spasticity
- measurement of spasticity
- treatment of spasticity
- modelling any association between spasticity and function, and
- literature reviews on any of the above.

Subsequent to having identified a suitable article from the title and abstract, the whole paper was

read and scanned to extract the necessary data for the paper. These were definition and outcome measures used to assess spasticity. All the data, including author details, year of publication, title of article, the definition of spasticity and the measures used, were stored on an Excel spreadsheet.

Results

The searches identified 272 papers from MEDLINE, 53 from Science Direct and 279 from Web of Science. After excluding duplicates and applying the inclusion criteria, 250 references contributed to the review. There were 190 clinical trials, 46 literature reviews and 14 case reports. (The list of references not cited in this paper can be found at: ftp://ftp.keele.ac.uk/pub/pta38/ Clinical_Rehabilitation.)

Results for definition of spasticity

Much of the research has not worked to a common definition (Table 1). Thirty-one per cent of the articles did not define spasticity; 31% of the articles cited the definition proposed by Lance in 1980; and 35% of the articles equated spasticity with increased muscle tone but no specific definition of altered muscle tone was provided. Other terms that were used within this context were 'abnormal tone', 'hypertonia' and 'hyperreflexia', however these terms were also not defined explicitly.

Table 1Definitions of the term 'spasticity' used in theliterature

Measures used:	Definitions used:				
	Lance	Muscle tone	None	Others	
Clinical trials Literature reviews Case reports Total	59 16 3 78 (31%)	69 13 6 88 (35%)	58 15 5 78 (31%)	4 2 0 6 (3%)	

This table demonstrates that the most common definition for spasticity equates the phenomenon with 'muscle tone' and that a significant number of articles have not provided an explicit definition for the phenomenon. Two examples to illustrate the variability of definitions are cited below:

A condition of paralysis or muscular weakness associated with hyperreflexia, the symptoms of which include increased resistance to manipulation, exaggeration of the deep reflexes, and clonus.⁹

An exaggerated activity of the stretch reflex loop with a length-dependent increase in tonic reflexes and a velocity-dependent increase in phasic reflexes.¹⁰

Three per cent of the articles equated spasticity with abnormal and involuntary muscle activity.⁸

Results for measurement of spasticity

Although most papers subscribed to a single definition (the others did not cite any specific definition), 314 different outcome measures were identified from the 250 papers (some articles used more than one outcome measure for spasticity). These measures could be clustered as described below:

- 15% (47 articles) attempted to measure aspects of spasticity directly, i.e. neurophysiological testing methods were used (37 used surface electromyographic (EMG) activity to quantify the muscle response to stretch, 9 either used the H-reflex response or the H-reflex standardized to the M-wave max, 1 used F-wave response).
- 71% used biomechanical measures/assessment (228 articles) to quantify spasticity indirectly. The perturbations and measurement methods varied:
- (a) instrumented measurement of stiffness during a controlled motorized perturbation (controlled velocity, controlled torque)
- (b) instrumented measurement of stiffness during a manual perturbation (uncontrolled velocity)
- (c) assessment of stiffness using clinical scales following manual perturbation (Ashworth Scale, Modified Ashworth Scale, Tardieu Scale, Clinical Score for Tone, Tone Assessment Scale, or Global Assessment Scale).
- 8% (25 articles) used miscellaneous methods consisting of a combination of clinical scales¹¹

and routine clinical tests (spasm frequency score, biceps tendon reflex, postural changes, passive range of movement or drawing test).

• 6% (19 articles) did not use/describe the outcome measure (neurological consultation or none).

Results for congruence between definition and measurement of spasticity (Table 2)

Congruence between definition and measurement was explored using the data from case reports and controlled clinical trials. Of the 204 such articles, 63 could not be used, as these did not define spasticity.

Among the 75 articles that defined spasticity as increased muscle tone, 60 used clinical scales to quantify stiffness, three used biomechanical measures of stiffness, four used neurophysiological measures, three used a combination of both biomechanical and electrophysiological measures, three used clinical measures of posture/range of movement and two did not describe the measure.

Among the 62 articles that cited Lance's definition, 33 used clinical scales to quantify aspects of stiffness, seven used instrumented biomechanical methods to quantify stiffness, eight used neurophysiological measures and 13 used a combination of both a biomechanical and electrophysiological measures and one measured resting posture.

Among the four articles that defined spasticity as muscle overactivity, one used muscle activity response to an external perturbation, two the Modified Ashworth Scale/Ashworth Scale and one did not describe a measure.

Discussion

The key findings from this review are that (a) the term spasticity is inconsistently defined and (b) the (outcome) measures often did not correspond to the definition (or the description of the key clinical features). Incongruence between definition(s) and measurement(s) can significantly compromise the internal validity of research and will need to be robustly addressed. This discussion will consist of two major sections: the first will critically evaluate the validity of existing definitions and the second will make recommendations on how to select an appropriate measure from the 'basket of measures' identified. While the focus of this paper is on spasticity it is important to note other such anomalies can be found throughout the rehabilitation literature a typical example being 'core stability'.

A critical evaluation of existing definitions

There are two broad approaches taken with respect to definitions of spasticity. The majority attempt at providing a narrow and precise description of spasticity. While this approach is probably the most valid it has not worked as well as it should have as these narrow definitions often do not conform to common clinical presentations.^{1,12}

The second type of definition takes the diametrically opposite approach, that is the definitions attempts to provide an umbrella statement to catch all possible variable interpretations of the phenomenon (the spasm definition is the only one in this category).⁸ While the latter type of definition is scientifically weaker it does provide

 Table 2
 Observed congruence between the definition of spasticity and methods of measurement

Measures used:	Definitions used:		
	Lance	Muscle tone	Others (spasm)
Clinical scales using an externally imposed stretch	33	60	2
Instrumented biomechanical measures	7	3	0
Neurophysiological	8	4	1
Hybrid (a combination of neurophysiological and biomechanical)	13	3	0
Posture	1	3	0
No measure described	0	2	1
Total	62	75	4

a framework from which narrow and precise definitions can be further developed. With respect to spasticity a decision has to be made as to whether the scientific community continues subscribing to traditional narrow definitions or takes a step backwards to using broader definitions. Based on this review it would appear that the time has come to move away from the existing narrow definitions as our current understanding does seem to challenge the validity of most of these definitions as discussed below.

The first formal definition for the term spasticity was proposed by Lance^{4–6} and there is one important assumption being made, that is the increase in stretch reflex-mediated muscle activity could be reliably measured by quantifying/assessing muscle tone (i.e. the stiffness) encountered when stretching a relaxed muscle during an externally imposed perturbation. Since the publication of this definition, our understanding of the pathophysiology associated with spasticity has progressed significantly and some of the early assumptions made in the original definitions will need to be reconsidered.

In addition to increased stretch reflex activity, the abnormal muscle activity may result from changes in the membrane properties of the alpha-motor neuron and/or changes in the threshold of activation of the alpha-motor neuron.¹³ The latter is influenced by a variety of pathways: group Ia presynaptic inhibition, group Ia reciprocal inhibition (from antagonist), recurrent Ib inhibition, group II afferents, group III and IV cutaneous afferents, and decreased recurrent Renshaw inhibition.^{13–15}

Both Denny-Brown and Lance seem to suggest that hyperexcitable deep tendon reflexes are a discerning feature of spasticity.^{3–7} Current evidence suggests that this may not be the case and that the variability of the reflex response in people with spasticity is high^{15,16} and may not be dissimilar to that of a population with no spasticity.

Indirectly measuring muscle activation by quantifying/assessing resistance to an externally imposed movement is fundamentally flawed as this is a confounded measure. The factors that can confound measurement of stiffness are the mechanical properties of the musculoskeletal structures being stretched, the compliance of the patient (i.e. the ability to relax) and muscle activity at rest. These confounding factors can contribute to substantial inter- and intra-subject variations. A further confounder of modelling the impact of muscle activity on stiffness is related to modelling the force generation during an eccentric contraction.⁸

To exclusively attribute a velocity-dependent increase in resistance to an externally imposed movement to spasticity may also be inaccurate. The muscle–tendon complex behaves as a viscoelastic material and will inherently demonstrate the same velocity-dependent behaviour in the absence of any muscle activation.¹⁷

A substantial proportion of the literature, ignoring the Lance definition,⁴ defines spasticity as an increase in muscle tone (i.e. an increase in the resistance to an externally imposed passive movement). Although it would appear to be a pretty straightforward definition, there is also a potential source of ambiguity in this definition. The word 'tone' can also be defined as state of readiness to act/ contract (i.e. innervation status) (e.g. ref. 18). Inferring which of these two definitions are being used is normally easy in papers discussing adult spasticity. However, this may not necessarily be the case in papers discussing spasticity in cerebral palsy. Using the same logic as discussed previously, the validity of using increased stiffness as an indicator of spasticity is flawed.

The North American Task for Childhood Motor Disorders attempts to make the Lance definition⁴ more precise by adding additional details.⁷ This modification has further confounded the original definition by introducing a new term (described as a 'catch') and one precondition (the catch occurs when a threshold has been exceeded). The key differentiating feature of spasticity, as per this definition, is the occurrence of a catch when some arbitrary (velocity) threshold is exceeded. Therefore, one has to conclude that the modifications do not provide any additional benefit to the original Lance definition.

The SPASM consortium attempted to widen the definition of spasticity in order to be able to reflect the vagaries in both research and clinical practice. This definition shifts the focus away from measurement of stiffness to the measurement of the 'abnormal' muscle activity. By doing this the term 'spasticity' can now be used to described most of the 'positive features' associated with the

upper motor neuron syndrome. However, this definition may exclude abnormal movement patterns triggered during voluntary movement, and will exclude all the negative features associated with the upper motor neuron syndrome. (Note: The phenomenon of associated reactions can also be observed in neurologically intact subjects when attempting tasks involving maximal voluntary contractions. Therefore, it is not clear if one should treat the resulting muscle activity as unwanted and involuntary.) While such a definition may be clinically relevant, the term can lose usefulness if researchers fail to identify which particular aspect of spasticity is being measured or studied.

In summary, it is reasonable to conclude that there is no adequate definition of the phenomenon of spasticity. Of the definitions currently available the broader definition proposed by the SPASM consortium provides a starting point for the development of future clinically usable definition.

Recommendations for measurement

To add to this problem of variable definitions, the frameworks used to underpin the measurement of spasticity is also substantially variable. Based on International Classification the of Functioning, Disability and Health (ICF) framework,¹⁹ spasticity can be classified as an impairment. So any attempt at using indirect measures of activity (e.g. measures of function) or participation (i.e. quality of life) is flawed. The main reason for this is that there is as yet insufficient evidence of a causal relationship between the impairment (i.e. spasticity) and the various measures of activity limitation and/or participation restrictions. The currently available measures of impairment can be classified as neurophysiological or biomechanical measures. These methods have been extensively reviewed in the literature^{8,16,20-22} and will only be described in brief to set the scene for identifying optimal measurement.

Neurophysiological measures provide the most direct way of studying (i.e. quantifying and classifying) spasticity. Most existing measures (i.e. the H-reflex, F-wave, response of a muscle (measured using electromyography) to an externally imposed perturbation) only measure aspects of spasticity.

The H-reflex bypasses the spindle and measures excitability in the reflex arc. The F-wave is primarily a measure of excitability of the alpha-motor neuron. Studying the muscle response to a tendon tap (or vibration) will provide a measure of excitability in the stretch reflex pathway. Studying the muscle response to an externally imposed passive stretch of the joint also provides information on the excitability of the stretch reflex pathways especially. Ideal measures when studying the muscle response to an externally imposed perturbation are threshold angles and patterns of muscle activation. All of the above measures can be confounded by the resting levels of muscle activity (which is commonly described as 'spastic dystonia'),²³ the ability to relax, pain, temperature and other environmental conditions, and cognitive capabilities.²⁴ Not surprisingly, most of these measures demonstrate a high degree of variability.⁸

Biomechanical measures can at best only provide an indirect method of measuring spasticity. Depending on the primary assumptions made one can measure aspects of spasticity by quantifying stiffness, posture at rest and range of movement. The one common assumption in all these cases is that biomechanical measures provide a valid reflection of the underpinning neurophysiological phenomenon (abnormal muscle activation to the externally imposed perturbation). Biomechanical measures can be administered in a variety of ways and these have also been extensively reviewed in the literature.²⁰ If instrumented methods are used, either interval level (instrumented hand held measures) or ratio level (e.g. threshold angle measures using controlled displacement methods) measurement of spasticity is possible. If clinical scales are used, either ordinal level (e.g. Ashworth Scale) or nominal level (e.g. Tardieu method of measurement) measurement of spasticity is possible. It is crucial to recognize that changes in the biomechanical properties of the musculo-tendenous and joint structures can significantly confound all biomechanical measurement and therefore significantly compromise validity of these measurements.²⁵

The key problem in the current literature is the lack of congruence between definition and measurement and this can lead to a compromise of internal validity (e.g. ref. 26). The solution to this problem is fairly simple: both researchers and clinicians will need to ensure that any outcome measures used in spasticity-related research is congruent valid and to the definition. Furthermore, when measurements are selected it is essential to minimize the effect of confounding factors not related to the definition in use. This would mean that wherever possible the aim should be to standardize to neurophysiological measures (as described above) or valid clinical scales (e.g. Spasm Frequency Scale, Myotatic Reflex Scale, original Tardieu Scale) to classify spasticity. As most biomechanical measures are confounded, using them in isolation is not advisable or recommended. However, using biomechanical measures in conjunction with simultaneous measurement of muscle activity (using surface or needle electromyography) may be recommended. In addition to control of the environmental conditions and time of testing, if the methods of measurement are dependent on an externally imposed biomechanical perturbation the following will also need to be considered.

Controlling the velocity of the externally imposed perturbation is not equivalent to controlling the stimulus to the afferent system. The main reasons for this are the polyaxial nature of human joints, the variations in the radius of rotation of the muscle-tendon units about a variable centre of rotation and the variability in the orientation of the ensemble of stretch receptors.

The efferent response to any externally imposed perturbation will be influenced by the resting length of the muscle, the range of movement employed during the test, the acceleration and the amount of support provided to the limb segment under test.

There were a few limitations to this narrative review paper. First, our search terms and database were narrow. Although unlikely, it is also possible that the spasticity-related literature within the field of stroke rehabilitation may not be representative of the spasticity-related literature in other conditions. In spite of these limitations we are of the view that the literature sampled for this review reflects the current state of the art with respect to spasticity-related research in all neurological conditions. There is also a potential bias in this paper: two of the authors involved in this paper (ADP and HH) played a key role within the SPASM consortium.

Clinical messages

- Define the term 'spasticity' precisely (even if this does not conform to any published definition).
- Select a valid measure/outcome measure that is congruent with the cited definition.
- Internal validity of research can be significantly compromised if measures are not congruent to definition.

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