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Theoretical and methodological considerations in the measurement of spasticity

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Abstract

Purpose: To discuss the measurement of spasticity in the clinical and research environments, make recommendations based on the SPASM reviews of biomechanical, neurophysiological and clinical methods of measuring spasticity and indicate future developments of measurement tools.

Method: Using the results of the systematic reviews of the biomechanical, neurophysiological and clinical approaches, methods were evaluated across three dimensions: (1) validity, reliability and sensitivity to change; (2) practical quality such as ease of use and (3) qualities specific to the measurement of spasticity, for example ability to be applied to different muscle groups. Methods were considered in terms of applicability to research and clinical applications.

Results: A hierarchy of measurement approaches was identified from highly controlled and more objective (but unrelated to function) to ecologically valid, but less objective and subject to contamination from other variables. The lack of a precise definition of spasticity may account for the problem of developing a valid, reliable and sensitive method of measurement. The reviews have identified that some tests measure spasticity per se, some phenomena associated with spasticity or consequential to it and others the effect of spasticity on activity and participation and independence.

Conclusions: Methods appropriate for use in research, particularly into the mechanism of spasticity did not satisfy the needs of the clinician and the need for an objective but clinically applicable tool was identified. A clinical assessment may need to generate more than one 'value' and should include evaluation of other components of the upper motor neurone syndrome. There is therefore a need for standardized protocols for 'best practice' in application of spasticity measurement tools and scales.

Keywords: *Spasticity, assessment, measurement*

1. Introduction

1.1 Defining spasticity

Spasticity is a common problem associated with the upper motor neurone (UMN) syndrome. There are not many reports on the incidence or prevalence of this phenomenon, but Sommerfeld *et al.* [1] estimated that 19% of stroke patients developed spasticity during the first 3 months following stroke and Watkins *et al.* [2] reported that 38% developed spasticity in the first year. A panel of clinicians [3] estimated that the majority of patients with severe Traumatic Brain Injury (possibly 75%), 20% of patients with stroke and 60% of patients with moderate to severe Multiple Sclerosis require treatment for spasticity itself, its associated problems, or both. These figures are not, however, based on epidemiological evidence.

Most clinicians specialising in the treatment of people with UMN lesions would have little difficulty in recognizing spasticity, yet, perhaps partly because of the complexity of the underlying pathophysiology, we continue to debate 'what is spasticity'. This lack of a precise definition poses problems for developing

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valid, reliable and sensitive methods of measurement that are essential not only for determining and evaluating treatment but also for improving our understanding of the condition. Without a better understanding and robust measurement, progress towards improved methods of treatment and management of spasticity and associated phenomena are hampered. Lance's [4] definition of spasticity acknowledges that it is one component of the UMN syndrome. Lesions of the UMN pathways result in a complex pattern of impaired motor control [5,6], weakness, inappropriate reflex activity – including altered modulation of the stretch reflex [7] loss of normal reciprocal inhibition [8,9] and abnormal co-activation between agonist and antagonist muscles [10]. The extent and location of the lesion are important factors in determining the type and level of impairment.

Some neurogenic abnormalities may lead to biomechanical changes in the muscle, for example changes in mechanical properties, such as increased atrophia of type II muscle fibres, the presence of target fibres [11,12], structural changes mainly in type I fibres [13]. Probably most important and confused with the concept of spasticity is a decreased number of sarcomeres resulting in shorter muscles, a subsequently increased resistance to stretch, and contractures [14]. These non-reflex components influence the spastic muscle tone, particularly in the leg extensors and elbow flexors [13] and can therefore contribute to impaired movement, abnormal postures, pain and deformity [15,16]. These changes confound the measurement of spasticity, but should not be confused with spasticity per se. Methods of spasticity assessment that are particularly prone to this confusion are clinical and biomechanical approaches, where resistance to passive movement may be measured without distinction between the neurogenic and non-neurogenic components of the phenomenon.

In the introductory paper in this issue, we proposed that the term spasticity should be used to describe the entire range of signs and symptoms collectively described as the positive features of the UMN syndrome and that tests used to measure individual aspects of spasticity should be validated. A reliable and valid measure enables the clinician to diagnose spasticity and to direct and evaluate the effect of treatment. With this information, the clinician may want to conduct further tests to establish whether, in each case, spasticity interferes with function. Clinical assessment may therefore include measurement of other components of the UMN syndrome, phenomena associated with spasticity or consequential to it, and the effect of spasticity on activity and participation, all of which may change in response to treatment.

It is commonly assumed that there is a causal relationship between spasticity, activity limitation, participation and independence. In some cases however spasticity may not only *not* interfere with function, it may actually enhance it; in others, although it may not interfere with function, if left untreated, complications may later arise. In these cases the decision about whether to treat must be based on a balance of risk and may inevitably be partially subjective. If spasticity is not functionally disturbing, and risk of secondary complications minimal, then no specific treatment may be required. When loss or disturbance of motor function due to interruption of the cortico-spinal tracts (causing paralysis, muscle weakness and loss of dexterity – negative signs of the upper motor neurone syndrome) is functionally more disturbing than spasticity (caused by a lesion of the parapyramidal fibres – positive signs of the upper motor neurone syndrome) intervention to address these problems may take priority. For clinical effectiveness therefore, measurement of spasticity may need to be combined with assessment of muscle weakness, impaired function and the effect this has on activities of daily living (ADL) to define the most disturbing components of the upper motor neurone syndrome in each patient.

1.2 Rationale for the support programme for assembly of a database for spasticity measurement (SPASM) project

Three factors have provided the rationale for this research: firstly the importance of spasticity in the rehabilitation and long-term management of people with UMN lesions, secondly the inadequacy and controversy surrounding the current definition of spasticity, and thirdly, and probably related to this, the inadequacy of current measurement tools. To address these issues it was vital to review the current literature on the measurement of spasticity and this was the primary objective of the Support Programme for Assembly of a database for Spasticity Measurement (SPASM) project. The programme also sought to identify best practice and direction for future developments and, as a thematic network had a wider aim which was to foster European collaboration to facilitate future research into the understanding and measurement of spasticity.

The need to measure spasticity and the inadequacy of current measurement tools reflect the problem of measuring a collection of interrelated and changing phenomena. Our reasons for wanting to measure it are:

- To characterise it and thus improve our understanding of the nature of the phenomenon.

- To direct and evolve treatment – particularly lacking is a valid and reliable quantitative measure suitable for large sample clinical research and clinical practice.

The systematic review of the literature on measurement of spasticity was conducted by three specialist groups under the categories of Neurophysiological, and Biomechanical approaches and clinical scales. Details of the search terms used can be found in the review papers in this issue [17–19].

Neurophysiological approaches to assess spasticity investigate mainly the electrical responses of the motor control system upon a variety of stimuli and conditions. These include electrical stimulation of the peripheral nerves, mechanical stimulation via the muscle tendons and well defined passive and active movements. A common aspect of most of the methods applied is that the response of the neuromuscular system is measured at the level of the muscles with surface electrodes placed on the skin over them.

Biomechanical approaches are those that observe the behaviour of muscles, joints and limb segments in response to movement. For the purposes of the review, approaches were subdivided in to upper and lower limb applications and categorised into those that observed behaviour during passive or voluntary activity, methods that controlled the torque and displacement of the joint and those that observed behaviour during functional activity. In most cases stretch was applied to the muscle either whilst the spastic muscle or its antagonist was contracted or when both muscles were relaxed. In some methods a mechanical external force was applied to the muscle and the response measured, a mechanised tendon hammer was an example of this.

The clinical review was concerned with the psychometric properties of clinical scales that assess spasticity. While many scales that are intended to assess spasticity concentrate on resistance to passive movement as the main construct, spasticity might also lead to other clinically observable phenomena. Therefore, scales that measure associated clinical phenomena in the context of spasticity, i.e. passive range of motion, limb position at rest including postural alignment, tendon reflexes, clonus, spasms, or associated reactions were also included in the review. It was further sought to identify clinical scales for function that have an association with spasticity as assessed by clinical scales.

2. Method

In this paper the results of each group's review have been combined to present a broad overview of current measurement tools and, from the evidence found in the literature, recommendations for measuring spasticity have been made and ideas for the development of better methods have been proposed. The paper will not present results of each group as these are reported in the other papers in this issue [17–19].

Methods and scales were evaluated across three dimensions:

1. General quality as a measurement tool: validity and reliability, including construct validity, ecological validity, intra and inter-rater reliability and sensitivity to change.
2. Practical quality in terms of: interpretability of the data, correlation with existing tools, ease of application, cost and the skill or training required to administer the test.
3. Quality specific to the measurement of spasticity: whether tests were applicable to different ability levels, different muscle groups, whether they attempted to distinguish spasticity from other components of UMN syndrome, whether they measured spasticity during active or functional movement and whether they would be useful to measure changes in response to a variety of interventions – for example focal or systemic treatment.

Finally we asked the question: is the test more appropriate for use in the research or clinical environment?

3. Results

The findings of the complete review are described in previous papers in this issue [17–19].

For the purposes of this paper, attention has been focused on the relevance of the results of the review to clinical and research applications, and from there, to formulate recommendations. Although the division of the review into clinical scales, biomechanical and neurophysiological approaches to measurement was probably the only practical way of conducting such an extensive review, it has become apparent that many methods cross these boundaries and that for the needs of either the clinician or the researcher wanting to chose an appropriate way of measuring spasticity they may not be relevant. We have therefore synthesised the three approaches and presented our results in terms of recommendations for clinical and research applications, recognising that scales and methods are not specifically or wholly applicable to either research or clinical application, but lie on a continuum between the two.

3.1 Redefinition of spasticity

It became apparent during the project that the measurement approaches under review did not correlate with Lance's definition of spasticity. This definition makes no reference to the way in which spasticity is expressed during active movement, whether this be stretching of the voluntarily contracted spastic muscle or the abnormal behaviour of a spastic muscle during voluntary contraction of its antagonist – sometimes presented as impaired reciprocal inhibition or inappropriate co-contraction. Both of these may be due to abnormal reflex responses, but may also be caused by abnormal neural connectivity within the CNS. Whichever mechanism is responsible, response to active movement maybe more relevant to the patient (and therefore the clinician who is interested in treating spasticity) than the response of the muscle to passive stretch.

The Group has therefore proposed the following new definition of spasticity that does not specify velocity dependence or tonic stretch reflexes, but focuses of the importance of disordered sensory-motor control causing involuntary, inappropriate activity of skeletal muscles.

Spasticity has been defined as: '*disordered sensory-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles'*.

3.2 Approaches to measurement

Methods of measuring spasticity were found to lie on a continuum with, at one extreme, measurement being highly controlled and more objective but at the expense of being unrelated to functional problems experienced by the patient and, at the other, very relevant to function but likely to be contaminated by other variables such as sensory-motor control problems and other characteristics of the UMN syndrome. This hierarchy of measures is summarised below in Table I. In general the neurophysiological measures fell into the former category. Clinical scales were found to quite separately assess either spasticity and its related phenomena, or how spasticity affected function, in which case they fell into the latter category.

The most common neurophysiological approach to measuring spasticity was found to measure various responses of the muscle to either an electrical or mechanical stimulus. One clear conclusion from the large amount of literature is that the responses, although many of them are basically monosynaptic, are very much influenced by supraspinal processes which may easily lead to a considerable variability in the characteristics of the outcome. If these methods are used in clinical practice, strict protocols are required to avoid variations in response and there is, as yet, no evidence that these have been established. The association between neurophysiological responses and other more clinical measures is low, suggesting that they reflect only a narrow aspect of the sensory-motor abnormalities associated with the UMN syndrome.

Table I. The hierarchy of approaches to the measurement of spasticity.

Hierarchy	Examples
Spasticity measured in response to electrical/mechanical stimulation	H-reflex, T-reflex and F-response
At rest or during passive movement	Scales that subjectively assess resistance to manual passive movement Posture or distance between joints Stretch reflex Mechanical stretching
During well defined active but non-functional movements	Stretch reflex during active muscle contraction Muscle activation patterns during cyclic movements at various speeds Co-activation during active elbow movement [25]
During unrestrained functional, movements or activities	Muscle activation patterns during walking and changes in modulation of the stretch reflex during walking [23] Disability rating scale or carer burden (handling the arm of a hemi patient) and Associated reactions [26]

Where there is a single specific study relating to an example the reference is given; in most cases references are too many to include and can be found in the approach specific papers.

The study of muscle activation patterns during passive or active movement combines neurophysiological and biomechanical approaches. The literature in this area is mainly limited to small laboratory studies, using methods that are inappropriate for routine clinical use, but if extended and adapted may show a better association with the clinical findings. During the last decade, the distinction between neural and non-neural components and its role in the clinical expression and experimental evaluation of spasticity has been acknowledged. The only way that this distinction can be made is by combining neurophysiological and biomechanical methods.

3.3 Examples of recommended approaches

Using the hierarchy described above we have selected a sample of methods and scales that are

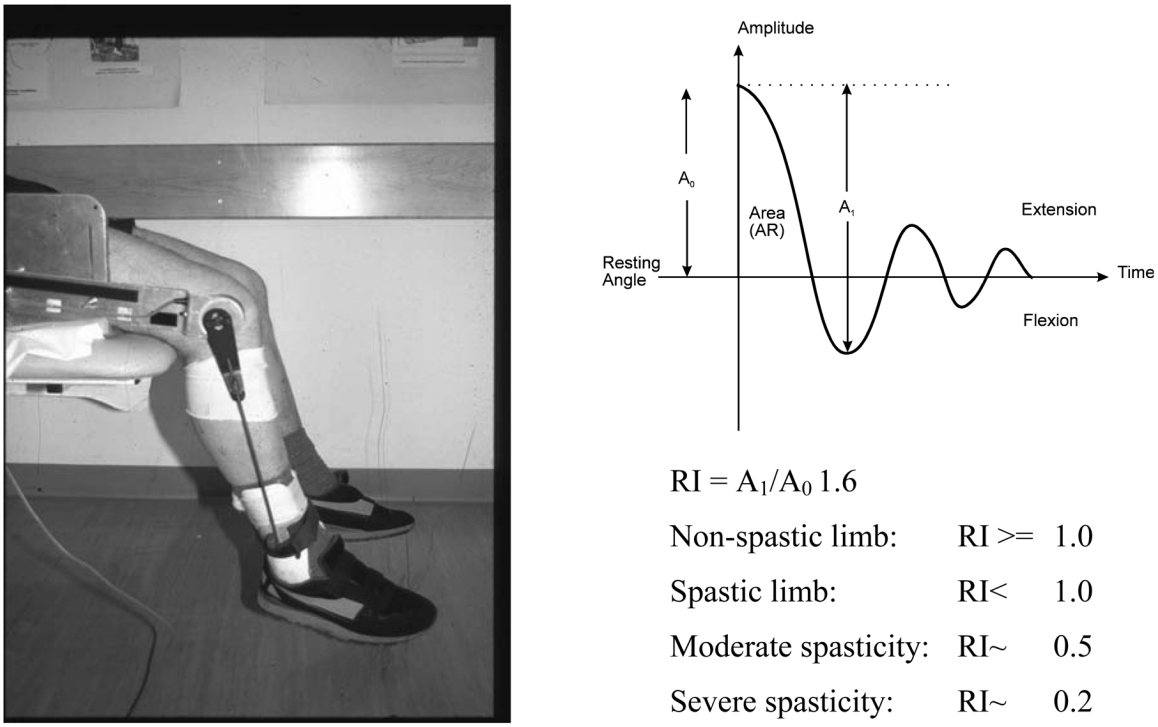

Figure 1. Shows a simple set-up for the Wartenburg pendulum test and illustrates how the relaxation index (RI) is derived. In this illustration EMG recording of activity in the hamstring and quadriceps muscles is not shown. The patient is positioned lying supine on a firm couch. The hips are in extension and the lower leg is allowed to hang freely over the edge of the couch. The assessor will hold the knee straight and asking the patient to relax will release the limb allowing it to swing freely. The excursion of the swing in illustrated and shows a normal damped simple harmonic motion. In spasticity, the limb will initially fall more slowly, describing a larger 'area under the curve' (AR), will have fewer cycles and the first swing will not extend so far (if at all) beyond the final resting angle.

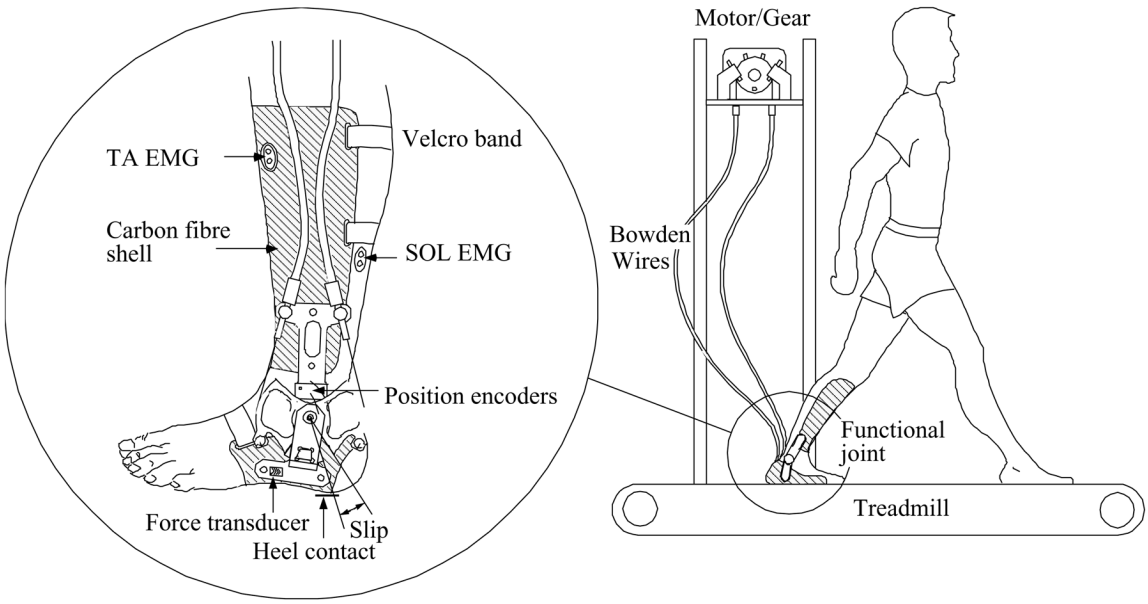

Figure 2. Shows a diagrammatic representation of the measurement of EMG in the soleus (SOL) muscle during treadmill walking. The experimental set-up allows a stretch to be imposed on the muscle at anytime during the gait cycle and the EMG response to be recorded. (*Note to reviewers: this figure is taken from Sinkjaer and permission will be sought for reproduction.)*

either widely used or have scored highly in our evaluation (Figures 1 – 3). Each is summarized in Table II in terms of the dimensions identified in the methods section.

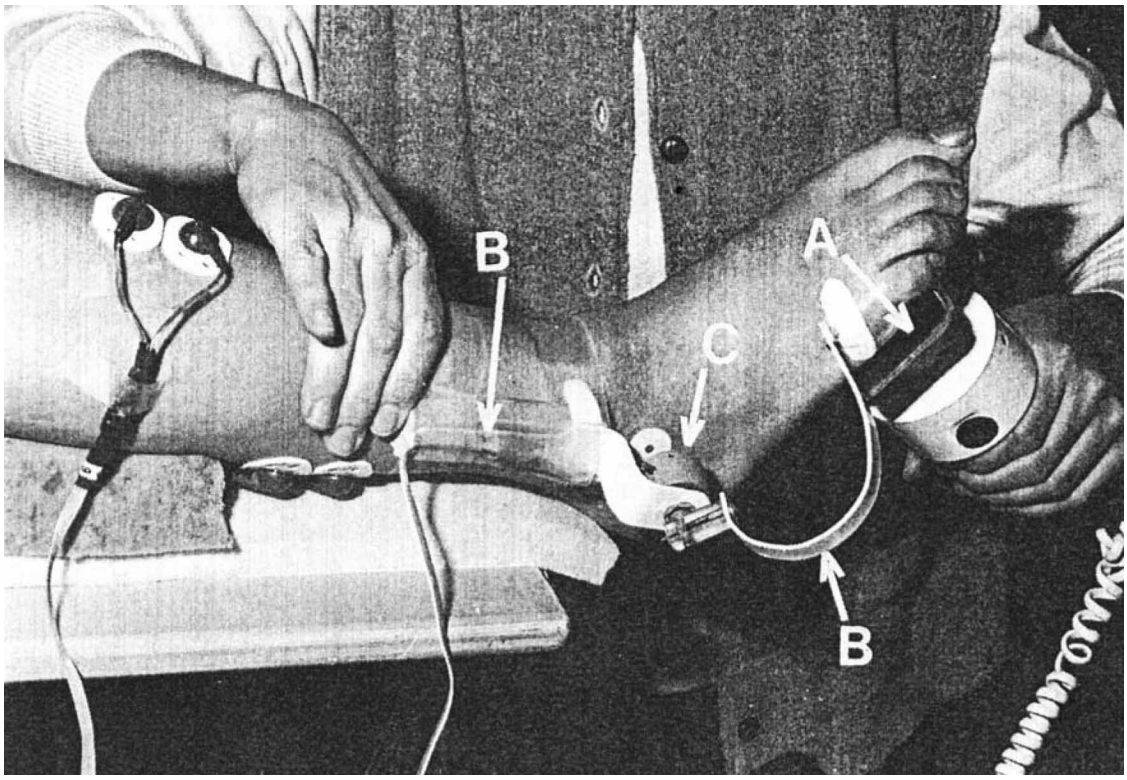

Figure 3. Shows the hand-held dynamometer used by Lamontagne to measure calf spasticity.

3.4 Clinical versus research applications

During the course of the review it became apparent that some measurement approaches were more appropriate to clinical use and others to research. Clinical scales clearly included most of the former, but we became aware of the problem of identifying an approach that satisfied our requirements in each dimension while remaining clinically appropriate. In an attempt to discover how the requirements of research differed from those of clinical practice we asked nine members of the research team involved in clinical measurement of spasticity for research purposes and eight clinicians specializing in the treatment of spasticity to rank the qualities of a measurement tool. Clinicians were asked to rank requirements in order of importance for a clinical tool and researchers in order of importance for a research tool. Three researchers and one clinician felt unable to rank the requirements and two clinicians did not respond. The replies were averaged and the results are presented in Table III.

The most obvious findings from this small informal study were that there was little correspondence between the requirements of research and clinical tools and, whereas researchers consider distinguishing spasticity from other components of the UMN syndrome as being of greatest importance, clinicians rate this lowest. Being simple and quick to use and having ecological validity were ranked highest for a clinical tool.

4. Discussion

Because spasticity is a complex phenomenon, exhibiting a range of clinical manifestations, and complicated by the accompanying disorders of the UMN syndrome, different methods of measurement may be required to evaluate different components. Data from measurement of resistance to passive movement cannot be related to spasticity, unless combined with data from neurophysiological measurement. Conversely, the isolated application of a neurophysiological method, without reference to effect of movement provides information only about the pathophysiological mechanisms underlying the different components of spasticity. Therefore we should not expect neurophysiological methods, when used in isolation, to provide accurate and clinically applicable measures of spasticity.

Many and varied clinical scales designed to measure spasticity, related clinical phenomena and associated changes in function were identified in the clinical review within this issue. Many are single item scales that assess muscle tone/resistance to passive movement or range of motion and are often more applicable to different muscles and ability levels than biomechanical approaches. Some scales are specific

to body parts, some measure muscle tone throughout the body and give summary information. Other phenomena related to spasticity such as spasms, tendon reflexes, clonus, and extensor toe signs can also be rated by clinical scales. In addition, scales that measure function associated with spasticity can be useful when used in conjunction with a combined biomechanical/neurophysiological measure to identify whether functional change is in fact due to change in spasticity.

During the literature review process, not only did we find a great variety of tests, but also a great variation in protocol and method of application of the tests. The variability of reliability estimates for the most frequently used scales (i.e. Ashworth and Modified Ashworth scale) may be due partly to a lack of standardisation of positioning and performance (as well as scoring).

In summary therefore, we recommend that a useful, clinically relevant assessment of spasticity as part of the UMN syndrome should include a range of different approaches. It may be useful to relate assessment to the WHO ICF as illustrated in Figure 4.

The literature review and informal ranking questionnaire also indicated that the perceived requirements of a clinical spasticity measurement tool are different from a research one. Our recommendations are however, that in both research and clinical practice, we should aim for certain standards of measurement. For example, both in clinical practice and in research, distinguishing neurogenic from mechanical changes is of primary importance and methods that measure response during active or functional movement as well as passive stretching should be considered. For both clinical and research applications, sensitivity to change and relevance to function are important and, dependent of the research question, ecological validity also. In clinical practice, ease of application and interpretability of results and cost are important factors, but it is important for clinicians to be aware that clinical scales are designed to measure associated clinical phenomena and function – although they may relate closely to spasticity they maybe contaminated by other variables associated with the UMN syndrome such as weakness and mechanical muscle and non-contractile tissue changes. Our recommendation is therefore that, because of the complexity of spasticity, it may be necessary to use more than one scale in a functional assessment so that impairments can be prioritized and any change in spasticity can be related to function.

Apart from clinical scales that addressed function related to spasticity, few methods attempted to measure spasticity of the contracted muscle or its antagonist [14,20–22,25] and even fewer the behaviour of the spastic muscle during a functional activity. The only example of this found in this review was during walking [23]. Measuring spasticity as neurogenic muscle response during active and preferably functional movement may be more informative, have greater ecological validity and relevance to function. It may also be more sensitive to relevant change.

The gulf that we have identified between measurements used clinically and those under laboratory conditions suggest that clinicians may be unwilling to use objective measures. It is not hard to see why. Most of the methods we have reviewed have been poorly tested in the clinical environment are expensive, time consuming and not commercially available. Yet there is clearly a need to measure spasticity more objectively to support and develop evidence based treatment. There is therefore a need for a quantitative clinical tool, that is also quick and easy to use and that generates information that can easily be interpreted by the clinician.

How might we design such a tool to do this and what should be the specific requirements? It must be able to distinguish neurogenic and non-neurogenic components of spasticity, be clinically applicable, therefore simple and easy to use and yield results that can be easily interpreted. It should incorporate:

- Variable velocities of displacement
- Simultaneous measurement of EMG and torque
- Be able to test response during passive and active conditions
- Include a clearly defined protocol.

Many experimental tools also feature methods to control displacement, but it is not yet entirely clear if this is essential for valid, reliable and clinically meaningful measurement[1] and this will need to be determined in future work.

Currently there is a range of tools available, which meet a number of the requirements listed above. At one end of the spectrum, we have complex testing equipment that measures a combination of neurogenic and biomechanical responses to carefully controlled perturbations under active or passive conditions (e.g. [7,22,23,25,33]). However, this category tends not to be practicable for routine clinical use. At the other end of the spectrum, we have uncontrolled manual techniques such as the Ashworth scale, which are easy to apply. In the middle, there is a variety of hybrid manual techniques in which the neurogenic and biomechanical responses have been measured using uncontrolled perturbation techniques (e.g. [24,27–32]), some of which simulate clinical routines.

Table II. A small sample of tests identified in the reviews.

Hierarchy		Dimension		
Assessment method	Examples	General quality as a measurement tool	Practical quality	Quality specific to measurement of spasticity
Response to electrical/ mechanical stimulation	H reflex	Extensively investigated, relevant factors well understood, broadly accepted protocols still missing.	Relatively simple technique, easy to use in neurology setting.	Low correlations with clinical scales.
During passive movement	Ashworth Scale	Clinical assessment of resistance to passive movement. Inter-rater reliability can be high, but this has not homogeneously been achieved. Can detect therapeutically induced changes of resistance to passive movement in the upper and lower limb. Few reports document an association between Ashworth scores and active motor control. Speed of movement is not standardized.	Simple clinical scale that can be used for all larger limb joints (passive movements). Standardized positioning and performance instructions for all joints would improve its practical applicability.	Can be applied to all ability levels. Does not distinguish between neurogenic and non-neurogenic components of resistance to passive movement. Does not evaluate spasticity in response to active or functional movement.
	Ashworth Scale – instrumented *[elbow]* [14,27–30]	Measures resistance to passive movement: (force during displacement) and EMG. Neither instrument nor method has been thoroughly tested for reliability (limited data in literature). Protocol e.g. speed of movement is not standardized. Preliminary tests show good sensitivity to change. Potential sources of error: • Variation in speed • Patient relaxation • EMG placement • Torque calculation	Requires computer and commercially available device. Some training required and as analysis is *post-hoc* it is only suitable at present for laboratory/research purposes. Execution of test is simple and simulates clinical test with some equipment that is easy to apply.	Applicable to elbow only, but currently being adapted for knee, ankle and wrist and to be used to measure force and EMG during isometric contractions. Can be used for any degree of spasticity at any stage following acute event. Does not measure response during functional activity.
	Wartenburg pendulum test (see Figure 2) [31–33]	Good inter and intra-rater reliability and sensitive to change. No ecological validity testing, but has shown correlation with clinical scales.	Simple but technical test requiring computer, some inexpensive equipment not commercially available and some training.	Can be applied to all ability levels, but only to the quadriceps muscles Does not distinguish components of the UMN syndrome unless used in conjunction with EMG recording. Does not evaluate spasticity in response to active or functional movement.

(*continued*)

Table II. (*continued*)

Hierarchy		Dimension		
Assessment method	Examples	General quality as a measurement tool	Practical quality	Quality specific to measurement of spasticity
During passive movement (cont'd)	Hand-held dynamometer (see Figure 4) [24]	Has shown correlation with commercially available laboratory equipment (Kin-Com) but not tested in a clinical environment. No data on sensitivity to change.	Simple but technical test requiring computer, hand-held commercially available dynamometer and electro-goniometer and some training. Requires signal processing – no commercial software available therefore only suitable at present for laboratory/research purposes.	Only reported use to test for calf spasticity. Distinguishes neurogenic and non-neurogenic components but not phasic from tonic stretch response. Has not been applied during voluntary muscle activity but potentially could be developed to do this.
During active muscle contraction	Response to calf stretch during walking (see Figure 3) [13]	Tested with small samples of normal and impaired subjects. No validity or reliability testing. Detected significant differences between normal and spastic muscle activity, but not tested in response to treatment.	For laboratory use only requires treadmill, motion analysis system, custom-made equipment and technical support.	A good example of an objective measure during an active functional movement. Only applicable during walking and only demonstrated on the calf
During undefined movements or functional activities	Disability Rating Scale [26]	Measures impact of severe upper limb spasticity on ('passive' arm) function. Sensitive to change.	Simple self-report scale that can be used for patients with severe arm spasticity. Reflects how difficult it is to handle the arm.	Can detect therapeutically induced changes when stroke patients with spasticity in a functionally useless arm received treatment for spasticity, e.g. after botulinum toxin treatment.

Tests are categorized by the assessment methods defined in the hierarchy in Table I and are critically summarized using the dimensions defined in the Methods section.

Therefore, in answer to the question above, there is probably no need to design an entirely new tool to measure the various aspects of spasticity, but there is a clear need to closely examine the systems already in existence, with the ideal of developing a valid and reliable tool that can be used by both researchers and clinicians. It is now possible to measure torque (moment), displacement (and derivatives) and EMG activity from a variety of muscles simultaneously (e.g. [7,22–25,27–33]). Most existing systems can also be adapted to measure neurogenic and biomechanical responses to externally imposed perturbations during active and passive conditions. Developmental work will be required to render these systems, especially the user-interface, suitable for routine clinical practice.

5. Conclusions

- If the purpose of the tool is to measure response to an intervention whether research or clinical it is important to distinguish the mechanical and neurogenic components of spasticity.
- Because of the complex nature of spasticity an assessment may need to generate more than one 'value' for spasticity.
- If a measure does not relate to clinical observations and function it is not useful for clinical practice, therefore the sole use of methods that only measure response to electrical stimulation should be avoided.
- While there is clearly a place for highly technical and sophisticated measures to improve understanding of the neurophysiology associated with UMN lesions or to measure a very specific response to an intervention in a research laboratory setting, such methods are unlikely to be clinically useful.
- The way in which any test is applied is as important as the test itself. There is therefore a need for standardized protocols for 'best prac-

Table III. The results of the informal questionnaire sent to researchers and clinicians.

Clinical applications	Median (SD) ranking of importance (9 = highest1 = lowest)		Research applications
	Clinical ($n = 5$)	Research ($n = 6$)	
Simple and quick to use	9(0.5)	9(0.5)	Distinguish spasticity from other components of the UMN syndrome
Ecologically valid	8(1.3)	8(1.2)	Inter and intra-rater reliability
Inexpensive	7 = (1.7)	7 = (1.4)	Sensitive to change
Sensitive to change	7 = (2.1)	7 = (2.4)	Ecologically valid
Easily interpreted results	5(1.2)	5(1.2)	Easily interpreted results
Inter and intra-rater reliability	4(1.2)	4 = (2.3)	Simple and quick to use
Correlate with existing tools	3 = (2.2)	4 = (1.0)	Correlate with existing tools
Applicable to different levels of ability	3 = (1.3)	2(2.1)	Applicable to different levels of ability
Distinguish spasticity from other components of the UMN syndrome	1(1.9)	1(0.6)	Inexpensive

Qualities of measurement tools have been ranked from 1 to 9 in order of importance for either clinical or research applications, where 9 = most important quality and 1 = the least important.

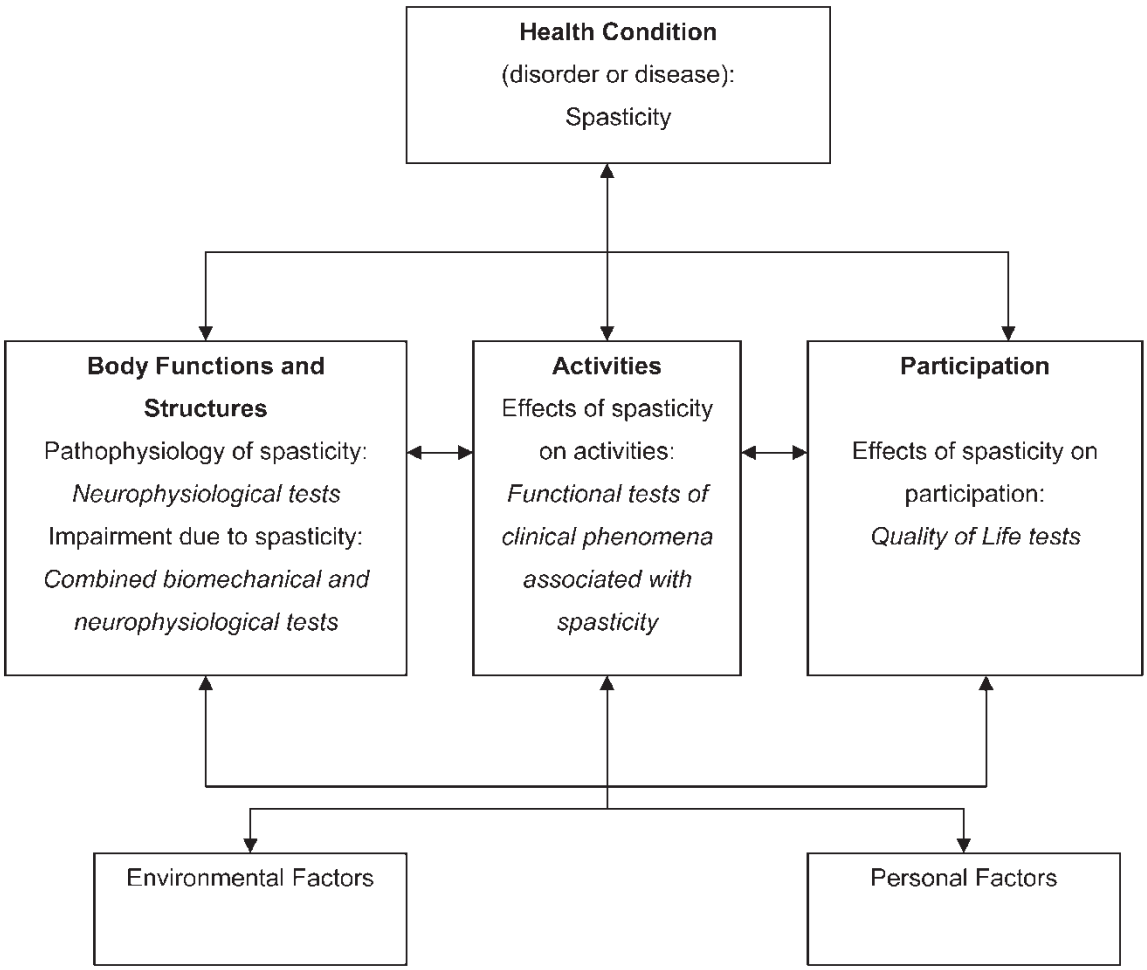

Figure 4. Presents a conceptual model of the assessment of spasticity within the WHO ICF framework. As in any clinical assessment levels ranging from pathology (impairment of body functions and structures) through activity to participation are all relevant to the patient. This figure shows how the spectrum of measurements of spasticity correspond to the ICF framework.

tice' in application of spasticity measurement tools and scales.
- There is a need for more in-depth evaluation of the (various) psychometric properties of tests when used with clinical populations.

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Notes

1. E.g. in most research systems, the angular velocity is controlled with an aim to standadise the perturbation. However, even if the angular velocity is controlled, the linear perturbation to the muscle spindle will still vary significantly between subjects and possibly even within subjects (i.e.) if there are changes in the biomechanical properties of the soft tissues).

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