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Spasticity: Clinical perceptions, neurological realities and meaningful measurement

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> When you can measure what you are speaking about and express it in numbers, you know something about it – but when you cannot measure it in numbers your knowledge is of a meagre and unsatisfactory kind – it may be the beginning of knowledge but you have scarcely, in your thought, advanced to the stage of science whatever the matter may be. (Lord Kelvin)

Spasticity, a neurological impairment,[1] is a common, but not an inevitable, consequence of an upper motor neurone (UMN) syndrome [1,2]. It is one of many sensory-motor signs and symptoms that may be present following an UMN lesion (Table I). Spasticity is usually associated with a lesion (or lesions) involving both the ''pyramidal'' and ''parapyramidal'' systems (the cortico-reticular pathways at the level of the cortex or internal capsule, and the reticulospinal and vestibulospinal tracts at the level of the spinal cord) [1,2]. Although no precise definition of this phenomenon exists, its clinical characteristics have been described for operational purposes by Lance (1980) [3] as ... *motor disorder characterized by a velocity dependent increase in the tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex, as one component of the upper motor neurone syndrome*. Although this is quoted as the most common definition of spasticity, it is essential to recognize that other researchers, notably Denny-Brown [4] and Tardieu [5], have also provided similar descriptions.

There is an implicit, and as yet unproven, assumption that a causal relationship exists between spasticity, and activity limitations,[1] participation restrictions,[1] including independence [6]. Further it is also claimed that spasticity leads to contractures, pain and weakness [6]. Therefore, the treatment of spasticity has been central to the clinical management of patients with injuries to the UMN pathways [6]. Current trends in research and clinical practice suggest that this focus has not changed substantially and that considerable resources are still being invested in both developing and optimising anti-spasticity treatment protocols.

Clinicians are now expected to implement evidence based practice and optimise health care interventions routinely. There are also pressures to rationalise the cost of treatment. With respect to spasticity, in particular, we need to establish why, in spite of the current focus on its management, the impact of treatment on activity, participation and independence is limited [7]. It is reasonable to suggest that a key obstacle to progress has been our limited understanding of this, reportedly common, phenomenon. The aim of this paper is to review briefly our understanding of the phenomenon of spasticity based in current evidence.

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What is spasticity?

Although Lance provided a precise definition for spasticity in 1980 [3] the use of this term in the clinical literature would suggest there is considerable confusion regarding the exact nature of the phenomenon. Clinicians view Lance's definition as narrow and limiting (e.g. [6,7]) and researchers use the term to describe the variety of pathophysiological phenomena, observed following an UMN lesion, that are not related to the features described in the original definition (e.g. [8–10]). This is not particularly surprising as the Lance definition [3], although commonly used, has never been fully validated. Given the significant advances in spasticity management and research since the publication of the Lance definition, it is essential that its validity and scientific underpinnings are re-examined. This will be done in the form of a brief literature review.

A review of spasticity

The phenomenon described by Lance [3] exists, i.e. if a joint of a person with an UMN lesion is flexed or extended passively at two different velocities, greater muscle electrical activity is associated with the higher velocity stretch (Figure 1). This observation is not unique to our group and there are others who have demonstrated this phenomenon (e.g. [11,12]). The commonality between many studies that have demonstrated this phenomenon is that stretch related muscle activity can be elicited with relatively low levels of stretch velocities.

If Lance's definition of the phenomenon is valid then it should be possible to demonstrate that

1. The increased muscle activity, during the imposed stretching phase, results exclusively from increased activity in the stretch reflex pathways.

Table I. Following a UMN lesion a person will present with a combination sensori-motor signs and symptoms that are broadly classified as *negative phenomena* (which are normally characterised by a reduction in voluntary motor activity) and *positive phenomena* (which are normally characterized by increased levels of involuntary motor activity) [1,2,6].

Positive features	Negative features
Increased tendon reflexes with radiation	Muscle weakness
Clonus	Loss of dexterity
Positive Babinski sign	Fatigability
Spasticity (a velocity dependent increase in resistance to passive movement)	
Flexor spasm	
Extensor spasm	
Mass reflex	
Dyssynergic patterns of co-contraction during movement	
Associated reactions and other dyssynergic stereotypical spastic dystonias	

Spasticity, as defined by Lance, is only one of the positive phenomena that may occur following an UMN lesion. Table reproduced from Barnes (2001) [6].

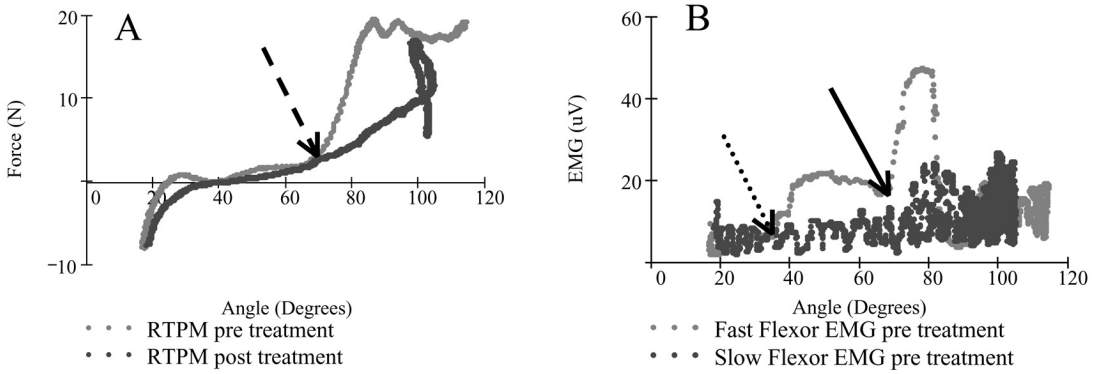

Figure 1. Graphs showing the resistance to passive motion (Force) (A) and the EMG (B) as functions of elbow angle recorded from the elbow flexors of a stroke patient during an imposed manual extension of the elbow at a low velocity (black trace – 8°/s) and a high velocity (grey trace – 74°/s). The muscle activity in the flexors during the fast stretch was higher than that observed during the slow stretch. (For further explanations see the text.)

2. The increased muscle activity, during imposed stretching, will contribute to an increase in resistance to passive movement.

Further, if the definition is to be accepted as valid, one should also be able to demonstrate that

1. Velocity dependent increase in the resistance to passive movement is exclusive to spasticity
2. Spasticity is a pure 'motor disorder'

Does the increased muscle activity result from increased stretch reflex activity?

Spasticity is reported to result from an increase in stretch reflex activity[2] (this is often described using the terms "hyper-excitable" or "exaggerated"). This can result from either or from a combination of

(a) Increased gain (amplification) in the stretch reflex networks, i.e. for a given afferent input (Ia and II) the response (output) from the respective efferent (α-motor neurone) is greater. Possible mechanisms that could contribute to an increased gain are increased α-motor neurone excitability, changes in the α-motor neurone properties, decreased Ia presynaptic inhibition, altered inhibition from the efferent pathway (in particular the group II fibres), altered reciprocal inhibition, decreased recurrent inhibition, increased excitability in the flexor reflex afferents (Group III and IV pathways) and altered force feedback (e.g. [1,2,10,13–20]).
(b) Decreased threshold in the stretch receptors, i.e. the reflex response in people with spasticity can be triggered with a much smaller stimulus than that used in people with no spasticity. Possible mechanisms that could contribute to a decreased threshold are increased receptor sensitivity and an increased excitatory drive to the muscle spindle efferents (e.g. [1,13]).

There is some evidence to suggest that increased gain in the reflex pathways, not just stretch reflex pathways, contributes to a variety of signs and symptoms associated with the positive features of the UMN syndrome (Table I and section above). The general assumption would appear to be that the increased excitability of the reflex responses would manifest as an increase in the amplitude response. However, this may not always be the case. Recent evidence would suggest that the amplitude response of the stretch reflex, to a controlled step perturbation at the elbow, is lower than that seen in people with no spasticity but the latency is significantly shorter in people with post stroke spasticity [21]. Further, there is evidence that spasticity (as defined by Lance, 1980) and stretch reflex hyperexcitability are not mutually exclusive [22]. With respect to the reduced threshold, although difficult to quantify, the evidence currently available suggests that spindle afferent activity is not necessarily abnormal in hemiparetic stroke [1,2,23,24].

In summary, there is insufficient evidence in the literature to support the hypothesis that the abnormal muscle activity observed in spasticity results exclusively from stretch reflex hyperexcitability. It would appear that activity in other afferent pathways (e.g. cutaneous), supraspinal control pathways (or systems) and even changes in the α-motor neurone may also contribute to the signs and symptoms associated with spasticity and other positive features of the UMN syndrome.

Does increased muscle activity contribute to increased resistance to imposed passive movement?

Based on the evidence available (e.g. [11,12,21, 24,25]), it is not possible to answer this question unambiguously. Muscle activity will normally contribute to force production; however, whether such a force can contribute proportionally to the resistance that opposes imposed passive stretching movement at all times is a moot point. For example, in Figure 1 it is possible to see that although there was an initial increase in the muscle activity (arrow with dotted line in Figure 1B) the resistance to the imposed passive movement only increased (arrow with dashed line in Figure 1A) after a further increase in muscle activity occurred (arrow with unbroken line Figure 1B).

Even with advanced instrumentation, the contribution from phasic stretch reflex activity (probably involving from monosynaptic or oligosynaptic feedback pathways) to stiffness has not been reliably measured. Although one would expect to be able to measure reliably the muscle activity contributions to stiffness from tonic stretch reflex activity (this will probably involve long latency polysynaptic pathways), it is surprising to note that it has also not been possible. The key confounding factors are likely to be inertial components from the limb segments, changes in the visco-elastic properties of soft tissues and joints, abnormal voluntary muscle activity, abnormal involuntary muscle activity resulting from phenomena other than stretch reflex hyperexcitability and the patient's cognitive and/or perceptuo-motor abilities (i.e. the ability to understand instructions to comply with testing).

In summary, spasticity related muscle activity may contribute to increased joint stiffness. However, under routine clinical or research conditions, the

exact relationship between spasticity related muscle activation and increased stiffness is yet to be modelled reliably (Figure 1A,B).

Is velocity dependent increase in stiffness exclusive to spasticity?

If we are to operationalize the phenomenon of spasticity according to Lance (1980), it is essential that the velocity dependent increase in resistance to imposed passive movement is exclusive to spasticity.[3] However, there is irrefutable evidence that this is not the case and that this velocity dependent change in stiffness is a characteristic response of the soft-tissue structures (e.g. muscles, tendons, ligaments, etc.) which normally demonstrate viscoelastic properties (e.g. [11,12,25]).

Is spasticity a ''motor disorder''?

Spasticity is without doubt an abnormal motor phenomenon, but, based on the current evidence, it would be wrong to treat it as a pure motor disorder. Stretch reflex activity is influenced by activity in other afferent (e.g. cutaneous and proprioceptive pathways) [26] and modulated by the higher centres in the nervous system [26]. Activity in other afferent pathways may also contribute to spasticity, e.g. benefits associated with antispasticity treatment involving electrical stimulation [11], and lycra garments [27] would suggest that cutaneous pathways have a role to play in spasticity. It is also possible that disordered feed-forward modulation of reflex activity, under both active and passive conditions, may also contribute to spasticity [21,26,28]. New evidence from studies of people with spinal cord injuries (and other animal models) suggest that an additional mechanism, i.e. a non-classical behaviour of motor neurons/inter-neurons described as plateau potentials[4] may have a role to play in spasticity [10,17,18,29,30]. However, as there are relatively few studies in this area it is difficult to draw any specific conclusion on the relationship between spasticity and the ''voltage-dependent persistent inward currents'' which lead to the production of ''plateau potentials'' [10,17,18,29,30].

In summary, it would appear that spasticity is not a pure motor disorder as specified in the definition, but instead it may be considered to be disordered motor control which appears to present the signs and symptoms associated with the positive features of the UMN syndrome.

Update on the Lance definition of spasticity

The evidence generated since the publication of the Lance definition suggests that spasticity is *not a pure motor disorder* and that *it does not exclusively result from hyper excitability of the stretch reflex*. The changes in resistance to imposed passive movement *cannot be uniquely related to muscle activity* and the phenomenon of *velocity dependence is not exclusive to stretch reflex hyperexcitability*. Therefore, it must be concluded that there is a need to update the definition of spasticity to reflect accurately the recent research findings and current clinical interpretations. Based on the evidence available, spasticity could now be redefined as ''*disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles*''.[5] Such a definition would imply that the term spasticity can now be used as a generic term to describe the entire range of signs and symptoms that are collectively described as the positive features of the UMN syndrome but narrows the term sufficiently to exclude the negative features of this syndrome and the pure biomechanical changes in the soft tissue and joints. This also opens up Lance's original definition to an interpretation that is clinically relevant. This definition also allows for the measurement/description of spasticity under both active and passive conditions. Although the definition will allow for a variety of signs and symptoms to be included under the umbrella of ''spasticity'', it does not imply or explicitly confirm a causal link between spasticity and other impairments (e.g. contractures), activity limitations, participation restrictions, and pain. If any such links exist they need to be demonstrated independently. It is now essential that the proposed definition, *whether it is adopted or not*, should be validated using appropriate and reliable measurement techniques that have clinical relevance. Furthermore, when using the term spasticity it is important that clinicians or researchers define precisely which particular aspect is being treated or studied and then ensure that valid measures are used for assessment.

During this review period the Consortium has also extensively reviewed the various approaches, viz neurophysiological, biomechanical and clinical, that could be used to measure individual aspects of spasticity. The outcomes of these reviews are contained in the next three papers. While accepting that not all aspects of spasticity can be (or need to be) measured at all times, the conclusions in this special issue will provide key recommendations with respect to clinically relevant measurement based on the reviews.

Notes

1. ICF classification: url http://www.who.int/classification/icf/intros/ICF-Eng-Intro.pdf (accessed 24th Aug 2004). *Impairments* are problems in body function or structure such as a significant deviation or loss. *Activity* is the execution of a task or action by an individual. *Activity limitations* are difficulties an individual may have in executing activities. *Participation* is involvement

in life situations. *Participation restrictions* are problems an individual ay experience in involvement in life situations.
2. The term ''tendon reflex'' is often used to describe a phasic stretch reflex. This anomaly probably reflects the clinical testing procedure used, i.e. the stimulus to elicit a stretch reflex is often obtained by using a hammer to tap on the tendon.
3. NB: The assumption one has to make here is that the muscle activity will proportionally contribute to increased stiffness.
4. Description of a plateau potential from Kiehn and Eken [30, p 746]: A plateau potential is a stable membrane potential that is more depolarized than the resting membrane potential. When a plateau potential is initiated, a cell can fire action potentials in the absence of continuous synaptic excitation [30].
5. For the purposes of this research project the SPASM consortium has reviewed the literature related to the measurement of spasticity using the definition described above. The Lance definition was too restricitive.

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