

## COMMENTARY

### COMMENTARIES ON “SPASTICITY OR REVERSIBLE MUSCLE HYPERTONIA?”

#### *Spasticity and mechanical resistance after stroke should be differentiated*

Bakheit et al. (1) recently raised the question as to whether the term “spasticity” accurately reflects the clinical phenomenon of increased resistance to passive stretch in chronic stroke patients. The authors suggested that “reversible muscle hypertonia” may be a better alternative term when referring to the increased resistance to passive stretch. The authors reasoning was based on two main points: (i) there is often substantial remodelling of muscle and connective tissue in chronic stroke patients, which contributes to the increased resistance to passive stretch; and (ii) the stretch reflex does not contribute substantially to the increased resistance to passive stretch in chronic stroke patients. We agree with the authors on the first point, but not on the second.

The most-used definition of spasticity is the Lance definition, which defines “spasticity” as “a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex” (2).

Spasticity can coexist with other types of muscle overactivity in stroke patients (e.g. dystonia, co-contraction), and the term spasticity is often used clinically as an umbrella term to describe all these forms of muscle overactivity (3). Clearly, defining what is being described by the term spasticity is important both in the clinic and in research. Unclear terminology can cause confusion between clinicians and make it difficult to interpret scientific results. Given that the Lance definition is the most widely used in research studies (4), we propose that this definition should also be used consistently in the clinical context.

In contrast to Bakheit et al (1), we consider that the Lance definition is valid for the description of the clinical phenomenon of increased resistance to passive stretch in stroke since: (i) numerous stroke studies report the presence of abnormal electromyographic activity during passive stretch, a causal key component of spasticity according to the Lance definition (5–7); (ii) several studies provide evidence for a predominantly neural origin of the increased resistance to passive stretch (8, 9); and (iii) further studies show that the excitability in certain spinal cord circuits correlates with (the severity of) spasticity, which suggests a neuro-pathophysiological mechanism (10). We interpret this evidence as being consistent with a neural origin of spasticity, as defined by Lance.

Based on longitudinal studies showing a reduced post-stroke stretch reflex (11, 12), Bakheit et al. (1) argue that the significance of the stretch reflex decreases over time. However, there is no consensus: another study found no change with time (13), and a recent study suggests that the reflex activity may even increase in certain patients (14). Given these divergent findings, it seems likely that stroke patients, depending on several factors (such as degree of lesion, degree of paresis, degree of use, and

treatment type), evolve differently over time. This is the case for both spasticity and mechanical resistance (14). The systematic use of the term “reversible”, as suggested by the authors (1), therefore seems unwarranted.

A consistent application of the Lance definition implies a neural cause. Other factors that contribute to the increased resistance to passive stretch, such as increased mechanical resistance, need to be defined and measured separately. Separate measurement of the spastic and mechanical components would be in line with evidence showing that these mechanisms contribute differently to increased resistance to passive stretch in chronic stroke patients (8). Clearly, the commonly used Ashworth or modified Ashworth scales do not differentiate between these components, and this probably explains why these scales have not been found to relate to neurophysiological measurements of spasticity (15, 16). As a consequence of excluding the mechanical contribution to spasticity it should not be claimed that the Ashworth scale measures spasticity. Rather, the Ashworth scale measures a combination of spasticity and mechanical resistance. The unique contribution of each to the total resistance cannot be obtained, for example a high Ashworth score may be predominantly mechanical. This undermines the clinical value of the Ashworth scale, since patients should be treated differently depending on the relative contribution of these components to the increased resistance to passive stretch.

In conclusion, we propose that the Lance definition be used for spasticity, and that the mechanical resistance present in muscles and connective tissue be defined and measured separately. How this is to be achieved in clinical routine remains a challenge to medical research; however, a promising method has recently been developed (8).

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The article by Bakheit et al. (1) on the terminology of spasticity is very useful at this time, particularly in attempting to capture this aspect within the domains of the International Classification of Functioning, Disability and Health (ICF) (2). There has been a lot of work over many years in trying to clarify what we mean by the term spasticity, and I agree with the authors that using the nomenclature of spasticity to describe muscle hypertonia is inaccurate, and not always helpful in daily clinical practice and research. For instance, post-stroke spasticity shows considerable variability and often does not conform to any of the standard definitions, particularly with regard to the role of muscle tone in producing loss of activity. Malhotra et al. (3) found that a substantial proportion of post-stroke patients exhibiting involuntary muscle activity consistent with spasticity, as measured by biomechanical and neurophysiological measures, did not exhibit scores on the Modified Ashworth Scale (which measures muscle tone) that were diagnostic for spasticity. Eighty-seven out of 100 patients were diagnosed with abnormal muscle activity using biomechanical and neurophysiological measures. Spasticity was then measured in the same set of patients using the 6-point Modified Ashworth Scale, and 56 out of the 87 previously identified patients scored a 0, indicating no detectable spasticity (3). The Ashworth and Modified Ashworth Scales are frequently-used clinical methods for evaluating muscle tone. At best, such incongruity between common measures of spasticity and standard definitions of the condition (4) complicate the diagnosis, and, at worst, result in sub-optimal treatment of the patient with spasticity.

The question now is whether the alternative term “reversible hypertonia” proposed by Bakheit et al. (1) is more valuable. They make a good case for it, but again their emphasis on hypertonia may be relevant for what one is treating in some patients, but not in all. Young people with relatively mild spasticity due to

cerebral palsy have problems with motor control rather than the phasic and tonic aspects of hypertonia (5). The attempt to separate the very different presentations of the upper motor neurone syndrome is important, but will it make a difference to the treatments currently employed? The reason for asking this question is that most people demonstrate a multiplicity of features and no single term will capture all of them. Will changing the terminology change the treatment approach? All manner of manifestations of upper motor neurone syndrome are addressed in clinical practice, and these have been included in the term, “spasticity” because it is easy to do so. The product licences for the pharmaceutical interventions are for spasticity and the clinician does not then have to make a separate justification for treating associated reactions, reciprocal inhibition, mass synergy effects, etc. It is thus important to keep a watchful eye on practical issues, such as this, but my experience from examining data from post-stroke patients, who had rehabilitation goals to improve function, indicates that, where they were not achieved for walking, it may have been because they did not have true spasticity. The outcomes were thus diminished in terms of the goal of treatment, but the patients nonetheless improved to a degree. However, Bakheit et al. (1) suggest the terminology change on the basis that anti-spastic treatment is given for a range of findings in upper motor neurone syndrome. This is true, but there does not appear to be an advantage in dropping the term “spasticity” in favour of “reversible hypertonia”, which does not describe the impairments any better.

On the other hand, the operational definition of spasticity is focused on increased resistance of joints to passive rotation and the possible origin of this increased resistance in the induced tonic stretch reflex (6, 7). This term is applied in the context of both cerebral and spinal injury, implying that a similar reflex mechanism underlies the two disorders. Tonic stretch reflex does not

always contribute to clinical hypertonia in spinal cord dysfunction. Other reflex mechanisms must contribute to hypertonia, as assessed clinically and contrasts with similar studies of cerebral spasticity after stroke. Comparable low-frequency stretch perturbation produced clear evidence of a gain in increased tonic stretch reflex that was correlated with the hypertonia at rest, and low-frequency stretch perturbation clearly distinguished between spasticity after stroke and spinal cord injury. The conclusion was that spasticity in the two conditions was not equivalent, and care should be taken in generalizing results between them.

The authors make the point that the impairments change over time after an insult to the upper motor neurone. This is true, but changing the terminology may not change our thinking about the recognition of these changes or their treatment. Being more specific in determining the patient's problem may help, and identifying ways of teasing out the therapeutic challenges will clarify our thinking. Quantification has focused mainly on hypertonia, i.e. increased resistance at rest to passive movement. This could be caused by a combination of spasticity, spastic dystonia, soft tissue stiffness and thixotropy (8). Most attempts at quantification (measures, such as the Ashworth/Modified Ashworth, and Tardieu Scales) are very poor at distinguishing between spasticity and soft tissue stiffness, and all that can be said is that quantification of the spasticity portion of hypertonia remains difficult, at least in a clinical setting (9).

The aim of the article is to distinguish the neurogenic component of upper motor neurone syndrome from its mechanical consequence, i.e. fixed contracture. These are really two different things, and the treatment required for the neurogenic component of the clinical picture is different from that for the biomechanical consequences. While I accept the shortcomings of the term "spasticity", I do not think that changing the terminology will really help this distinction. I will keep an open mind about this, but I wonder whether a name change will really catch on. The article does not offer new treatment strategies for treating patients with "reversible hypertonia" as opposed to "spasticity". Similar pharmacological agents are used to treat

a variety of physiological features in "spasticity management" and it probably makes sense not to change a simple term, like spasticity, just yet until we propose different treatments for those different features.

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## RESPONSE TO LINDBERG ET AL.'S AND WARD'S COMMENTARIES

We thank Lindberg et al. and Ward for their comments on our paper "Spasticity or reversible hypertonia". In response to Lindberg et al's remarks, we wish to reiterate that the validity of Lance's definition (1) and the neural origin of spasticity are not disputed by us, nor does our proposed definition exclude the co-existence of spasticity with the other factors that contribute to the clinically demonstrable hypertonia. The fundamental point in our discourse is that "pure" spasticity, as defined by Lance, i.e. hyperexcitability of the spinal alpha motor neurones in the absence of changes in the viscoelastic properties and structure of muscle, is a transient phenomenon, at least in most patients with an upper motor neurone lesion. Furthermore, as Lindberg

et al. have conceded, the frequency of occurrence, the trajectory of evolution and the relevance of the H-reflex changes over time do not follow a consistent pattern. Therefore, given the transient nature of spasticity and its doubtful contribution to the long-term motor disability in these patients, we would argue that the use of the term hypertonia is a more appropriate and pragmatic description of the clinical situation, as it includes all the factors that are known to cause the resistance to muscle stretch. The addition of "reversible" in our proposed definition is meant to indicate that the hypertonia is amenable to conservative medical interventions. In other words, it means that a significant fixed shortening of the muscle-tendon unit (that requires surgery) has

not set in. The use of “reversible” in this context is not intended to suggest that the changes in the H-reflex reduce over time, as implied in Lindberg et al.’s commentary.

Lindberg et al. rightly state that the term spasticity is “used clinically as an umbrella term to describe all these [dystonia, co-contraction, etc.] forms of muscle overactivity.” We also agree with them that “unclear terminology can cause confusion between clinicians and make it difficult to interpret scientific results”. However, we believe their proposal that the term “spasticity” should be used routinely in clinical practice contradicts the latter statement. They also suggest that Lance’s definition (1) should be used for spasticity, and that “the mechanical resistance in muscle” is defined separately. As the increased excitability of the spinal alpha motor neurones and the structural muscle changes exist simultaneously it is difficult to understand how the two separate definitions for the same medical impairment enable a coherent approach to diagnosis and treatment.

The purpose of our proposed definition is to emphasise the component of muscle hypertonia, that is likely to respond to

conservative medical “antispasticity” treatment. We believe that the main value of this definition is that it does not capture all presentations of the upper motor neurone syndrome. Indeed, as Ward stated it is not desirable to use terminology that describes all manifestations of an upper motor neurone lesion because the disability resulting from it can be due to poor motor control rather than spasticity. The term “spasticity” does not allow clinicians to make this distinction.

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