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Biomechanical measurement of post-stroke spasticity

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Abstract

Background: spasticity following stroke is common, but clinical measurement is difficult and inaccurate. The most common measure is the modified Ashworth scale (MAS) which grades resistance to passive movement (RPM), but its validity is unclear.
Aim: to assess the validity of the MAS.
Methods: spasticity was clinically graded using MAS and RPM measured biomechanically in the impaired arm of 111 patients following stroke. The biomechanical device measured RPM, applied force, angular displacement, mean velocity, passive range of movement (PROM) and time required.
Results: the median age was 72 years, and 66 subjects were male. The clinical grading by MAS was '0' in 15, '1' in 15, '1+' in 14, '2' in 13, '3' in 43 and '4' in 11. There was no difference in RPM among '0', '1', '1+' and '2' ($P>0.1$). However, grade'4' was higher than '3' and below ($P<0.05$). The force required increased with the increasing MAS while velocity and PROM decreased ($P<0.01$). We regrouped the data using the algorithm: no stiffness = '0'; mild = '1' and '1+' and '2'; moderate = '3'; severe = '4'. There was no difference between 'no stiffness' and 'mild ' ($P>0.10$), but 'mild' and moderate' as well as 'moderate' and 'severe' were different ($P<0.01$).
Conclusion: the MAS is not a valid ordinal level measure of RPM or spasticity. Objective measurement of RPM is possible in the clinical setting. However, additional measurements of muscle activity (electromyography) will be required to quantify spasticity.

Introduction

Stroke is a major cause of disability in the UK [1]. The clinical features and subsequent disability following stroke occur secondary to ischaemia-induced neuronal loss. Damage to the corticofugal fibres (pyramidal and para-pyramidal) leads to motor deficits, which are present in >80% of stroke patients [2, 3]. These deficits, which are collectively described as the upper motor neurone (UMN) syndrome, are characterised by a combination of negative phenomena (e.g. motor weakness) and positive phenomena (e.g. spasticity) [3, 4]. Spasticity is a common impairment, which is one component of the UMN syndrome that could be present in more than a third of all patients at a year following stroke [5].

Spasticity is described as a velocity-dependent increase of tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the UMN syndrome [6]. Spasticity has been reported to cause stiffness, a reduced range of movement, painful spasms and contractures [7]. These can lead to problems with posture, transfers, physical therapy, nursing care and hygiene [7].

Treating and minimising the effects of spasticity has remained one of the major aims for physicians and therapists as part of rehabilitation following stroke [3, 7–10]. Current research trends would suggest that substantial effort and resources are still being invested in developing and studying the effectiveness of novel therapeutic technologies and strategies aimed at treating spasticity [e.g. 11–16]. Given this research focus, it is, therefore, important to be able to measure spasticity accurately and objectively in order to be able to plan, deliver and monitor various treatment strategies [17]. It is now widely acknowledged that establishing clearly defined goals and outcome measures are important strategies in the management of spasticity [18, 19].

Although there are many techniques available to measure spasticity [20], clinical rating scales such as the Ashworth scale [21] and modified Ashworth scale (MAS) [22] are the most commonly used [23]. When using clinical measures to assess spasticity, one assesses the resistance to imposed passive movement (RPM) when the limb is briskly stretched through the full range of available movement about a joint. Although the MAS has been shown to have varying degrees of inter- and intra-rater reliability for measuring RPM [24, 25], the validity of these scales has only been studied cursorily [e.g. 26–31]. Further, existing clinical scales of spasticity have been shown to correlate poorly with each other, and the lack of consistency in their reliability makes it difficult for them to be seen as anything more than subjective measures, and this limits their clinical relevance [26, 32]. Therefore, there is a need for a valid quantitative measurement of spasticity, which is easy to use in a clinical setting.

Aim

The aim of the study was to investigate whether a previously developed biomechanical measure of joint stiffness could be used in routine clinical practice as a measure of RPM and whether this had more resolution in the measurement of RPM at the elbow in a post-stroke population than the MAS.

Patients and methods

Study design and setting

This was a cross-sectional study with purposeful sampling to ensure an even representation of each level of spasticity. Stroke patients who had been admitted to the University Hospital with spasticity were recruited. An independent researcher selected the patients, and the clinical assessor took all study-related measurements. At the time of measurement, the clinical assessor was blinded to the instrumental (biomechanical) measures. The measurements were carried out at the bedside on the acute stroke ward, stroke rehabilitation unit, in the day hospital or at the patient's place of residence.

Inclusion and exclusion criteria

Patients with a diagnosis of ischaemic or haemorrhagic stroke were included. Those who had traumatic brain injury or subarachnoid haemorrhage were excluded. Receptive and expressive dysphasia were not used as exclusion criteria; patients were included if they were able to co-operate with the measurement and had sufficient basic communication to establish the pain-free range of movement in the arm.

Ethical issues

The Local Research Ethics Committee approved the study and measurement protocol. Informed written consent was obtained from the patient whenever possible, or written assent was obtained from the relative or carer. Patients and relatives were informed of the option to withdraw from the study of their own accord at any point.

Outcome measures

Demographic details including age, gender, affected side, stroke subtype and the time elapsed from the stroke to measurement were taken at recruitment.

Simultaneous clinical grading of spasticity and RPM measurement was carried out on the affected limb about the elbow. The clinical grading was done using the MAS [22]. For the purpose of RPM measurement, a biomechanical device consisting of a force transducer (measured the force used to stretch the forearm manually) and a flexible electrogoniometer (measured the resulting displacement) was used [29, 30]. The time taken for each measurement was also measured.

The measurement protocol was as follows: the device was attached to the affected arm. An initial slow stretch was first done to establish the pain-free range of movement and this was followed by the clinical test (involving a brisk stretch) as per the measurement protocol described by Bohannon and Smith [22]. The arm was extended from full flexion at the elbow through the range of pain-free passive movement for both the slow and brisk stretch. The device was removed after the brisk stretch was completed.

RPM was measured as the slope of the force angle plot, with data collected from the brisk stretch, within the range of pain-free movement using a linear regression technique. Goodness of fit was tested using the coefficient of determination (r^2)—a value of '1' suggests perfect fit and a value of '0' indicates no fit [33]. If the r^2 was >0.60, the slope was considered to have an

acceptable linear fit [34]. In addition to the above parameters, the average velocity of the fast movement was calculated as the ratio of passive range of movement (PROM) and time.

Data analysis

SPSS (Version 10.0) was used for statistical analysis.

In the primary analysis, validity and resolution was assessed between the MAS groups using analysis of variance (ANOVA). This was used to test for differences in force, PROM, time, velocity and RPM between the various MAS groups.

Previous studies have shown that the greatest problem with the MAS was to discriminate within the lower grades of stiffness (29). In view of this, for further analysis, we grouped the patients as normal (MAS 0), mild stiffness (MAS 1, 1+ and 2), moderate stiffness (MAS 3) and severe stiffness (MAS 4), and ANOVA was again used to compare the variation in mean RPM between these recoded groups.

Results

A total of 111 patients who had a previous stroke were tested. The median age was 72 years [interquartile range (IQR) 64–78]. The sample comprised 45 female and 66 male patients. The left arm was affected in 59 and the right arm in 52 patients. The median time post-stroke was 11 months (IQR 3–36). The measurements were carried out at the bedside and took an average of 5 min. The maximum time taken for measurement was 10 min, and all measurements were carried out by a single rater with the patient in a chair or bed.

The frequency distribution of the MAS is described in Table 1. It was not possible to demonstrate any difference in RPM among '0', '1', '1+' and '2' ($P>0.1$). MAS grade '4' was significantly different from individual MAS values of 3 and below ($P<0.05$).

The force required increased with the increasing MAS, and this was associated with a progressive decrease in velocity ($P<0.01$) (Table 2). The PROM decreased with increasing MAS ($P<0.01$). There were no significant differences in the time taken for passive movement of the arm across all grades of the MAS ($P>0.1$).

Further analysis based on the newly created groups shown in Table 3 showed no significant difference between 'no stiffness' and 'mild stiffness' ($P>0.10$). However, there were significant differences between 'mild' and moderate' as well as between 'moderate' and 'severe' ($P<0.01$).

Table 1. Biomechanically measured resistance to passive movement (RPM) for the clinically graded elbow stiffness according to the modified Ashworth scale (MAS)

MAS	Frequency	Mean RPM Newtons/° (SEM)
0	15	0.07 (0.03)
1	15	0.21 (0.05)
1+	14	0.31 (0.03)
2	13	0.37 (0.02)
3	43	0.72 (0.03)
4	11	2.21 (0.37)

Table 2. Biomechanically measured force required to achieve the maximum range of pain-free passive movement (PROM) and velocity of movement of the limb, for the clinically graded elbow stiffness according to the MAS

MAS	Maximum force (Newton)	PROM (°)	Velocity of movement (°/s)
0	8.9 (0.8)	79 (5)	65 (6)
1	16.7 (1.1)	82 (5)	55 (4)
1+	21.3 (1.8)	72 (4)	41 (4)
2	27.9 (1.5)	74 (5)	50 (7)
3	36.9 (1.5)	55 (2)	33 (2)
4	37.9 (3.1)	23 (2)	22 (4)

All values are given as mean (SEM).

Table 3. A four-point grouping of patients based on biomechanically measured RPM

Stiffness	Frequency	RPM Newton/° (SEM)
None	15	0.07 (0.01)
Mild	41	0.29 (0.03)
Moderate	45	0.71 (0.03)
Severe	10	2.21 (0.37)

Discussion

In this study, RPM was objectively measured according to current protocols of clinical measurement, using a previously developed biomechanical device [30]. We were able to carry out bedside measurements using the device in a short period of time in various clinical settings on the acute stroke unit, stroke rehabilitation ward, stroke review clinic and patients' place of residence (own home or institution). The measurement technique was quick, easy to administer and non-invasive. No adverse events were recorded throughout the study. The technique proved as easy to use in bed-bound patients, requiring high levels of nursing care, who had high grade stiffness of their arm, as in fully mobile patients with lower grades of stiffness. Because of this, we were able to include patients across the whole range of stiffness according to the MAS grading.

The protocol provided us with accurate measurements of the modalities of force, PROM, velocity and time using first principles. Subsequently, it was possible to quantify RPM objectively from the force angle plot using a linear regression technique. This provided us with a variety of biomechanically measurable components of a passive stretch at the elbow according to the definition of spasticity [6].

There was little difference in RPM at the lower grades of the MAS. This may have resulted from the inability of the MAS to discriminate among the lower grades of spasticity, and this observation is consistent with findings in other studies [29, 31, 32]. Although it was possible to identify accurately participants with higher grades of stiffness/RPM as per guidance in the MAS, it was not possible to confirm if the increase in stiffness exclusively represented changes in spasticity as defined by Lance [6]. Current evidence would suggest that the increase in RPM could result from the

biomechanical changes in the soft tissue and joints [27–30] or 'spastic dystonia', a phenomenon described by Sheean [3] as a state of continuous muscle activity which is not movement related, both of which can occur in people who have had strokes. The above two phenomena, if present, could be the main reasons for the reduction in the passive range of movement and the velocity of movement seen in MAS grades of '3 (4)' and '4 (5)' even though the time taken to complete the movement remained constant.

The secondary analysis showed that it may be more appropriate to use a four-point scale to measure RPM and that this may provide an alternative to the current clinical measures. The decreased sensitivity (obtained by merging the lower grades of the MAS) in clinical grading of RPM may have led to improved validity and could contribute to increased reliability. However, even after reducing the sensitivity, it was extremely difficult to establish clinically the difference between a normal tone and mild degree of limb stiffness. This would suggest that, for routine clinical practice, even the suggested four-point scale may lack sensitivity at the lower end of the scale.

In clinical practice and research trials [e.g. 13, 15, 16], antispasticity treatment is normally aimed at people who have moderate to severe levels of RPM as graded by the MAS. Therefore, there may be some advantage to using a three-point clinical scale (none/mild, moderate and severe) to measure stiffness. However, even such a scale will be inferior to objective measures of RPM, such as those described in this study. For example, the increased sensitivity of the biomechanical measure demonstrated that in people with an MAS grade of '4' described as having a 'rigid and fixed limb', angular displacement of ~20° was obtained.

It is clear from Lance's definition [6] that if one were to measure spasticity one needs to measure RPM. The implicit assumption is that the changes in RPM reflect changes in reflex-mediated neuronal activity. However, in clinical practice, the changes in RPM are often confounded by changes in biomechanical properties and neuronal activity, which is not exclusive to reflex hyperexcitability. Using first principles, we were able to use two primary measures, namely force and angular displacement, to quantify RPM objectively and accurately. This provides a valid measurement of RPM, which is an indicator of the stiffness of the limb and could represent spasticity. However, as muscle electrical activity was not measured (using electromyography), it was not possible to confirm whether the stiffness was purely biomechanical, purely neural or a combination of both.

MAS lacks the ability to discriminate small changes in RPM. This could be due to the fact that when using the MAS to quantify spasticity, one may be quantifying, in addition to RPM, other parameters (such as force applied, range of movement, time taken to move, momentum and catch), which then have the potential to confound the clinical grading [30].

It would be tempting to suggest the use of a three-point scale for the grading of arm stiffness in a clinical setting. However, the evidence from this study and others [e.g. 26–30] would suggest that any such scale would provide limited diagnostic information, i.e. the clinical scale would not be able to identify whether the stiffness is 'purely biomechanical', 'purely neural' or a combination of both. It is possible to measure stiffness objectively at the bedside, and this may provide valuable information in identifying patients for intervention and in measuring the effects of treatment. However, if one is interested in neural components of stiffness/RPM, then there is a need additionally to measure levels of muscle activity. Further work will be required to explore the feasibility of such a measurement system.

Key points
- The six-point clinical scale (MAS) is not a valid ordinal level measure of RPM or spasticity.
- Objective measurement of RPM is possible at the bedside.
- RPM is influenced by biomechanical factors, neural factors or a combination of both. Additional measurements of muscle activity will be required to obtain meaningful diagnostic information.

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