Introduction

Spasticity, as defined by Lance (1980), is one of many impairments that can develop following an injury to the central nervous system (Burke 1988). Although the disability and handicap resulting from the UMN lesion are not unequivocally associated with spasticity, its treatment represents a major challenge in neurological rehabilitation (Barnes 2001). A variety of treatment approaches are currently available to treat spasticity, however, the clinical efficacy of many of these techniques in reducing spasticity are unclear due to paucity in valid and reliable outcome measures.

The Ashworth scales (Ashworth 1964; Bohannon & Smith 1987) are used as principal measures of spasticity in routine clinical practice and research (e.g. Davies & Barnes 2001). A clinical rating of spasticity, on these scales, is made after an assessor tests the resistance to passive movement (RTPM) about a joint following a “brisk stretch”. However, a review of the properties of these scales suggests that these may have limited validity and reliability when used as measures of spasticity but may have sufficient validity and reliability as measures RTPM (Pandyan et al 1999).

Objective

The aim of this study was to investigate whether the modified Ashworth scale (MAS) provided a valid and sensitive measure of RTPM about the elbow. The elbow was selected as it is the joint in which the MAS has the highest reliability (Pandyan et al 1999) and, biomechanically, this is the best understood of the upper limb joints.

Methods

A system was developed to measure simultaneously the force applied by a clinician and the elbow angle while assessing RTPM (Figure 1). The measurement device is strapped on to the subject’s arm. The assessor abducted the humerus to 90 deg (or within a pain free range of abduction), fully flexed and then rapidly extended the forearm within a pain free range of movement. Data were collected by a laptop computer at 100 Hz and the RTPM was quantified as the slope of the force angle plot using linear regression. In order to study the performance of the system when measuring RTPM in patients with spasticity, a study was performed with 16 subjects (six female and ten male; mean age 67.3; range: 54 - 84) who had had a unilateral stroke one-week previously and no elbow contractures. RTPM, during elbow extension, was measured three times, first in the non-impaired and then in the impaired arm. The assessor simultaneously graded RTPM using the MAS. Velocity of movement was calculated from the displacement data. The assessor was blinded to the biomechanical measures.

Figure 1: The measuring device

Figure 2: A typical linear curve
Results

In the non-impaired arm the MAS was “0” in 43 measures and “1” in five measures. In the impaired arm, the MAS was “0” in 24 measures, “1” in 18 measures and “1+” in six measures. Although speed of passive movement was not significantly different, RTPM in the impaired arm was significantly higher (p < 0.01). In the impaired arm, RTPM was higher in subjects with MAS of “1+” (p < 0.01) and there were no significant differences between “0” and “1” (p > 0.1) (Table 1). The velocity of applied movement was significantly higher in subjects with a MAS of “0” (p < 0.01) and not significantly different between subjects with MAS “1” and “1+”.

<table>
<thead>
<tr>
<th>MAS (Frequency)</th>
<th>0 (24)</th>
<th>1 (18)</th>
<th>1+ (6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RTPM (N/deg)</td>
<td>0.17; 0.02</td>
<td>0.23; 0.02</td>
<td>0.46; 0.08</td>
</tr>
<tr>
<td>Speed (deg/s)</td>
<td>66; 5</td>
<td>44; 3</td>
<td>28; 5</td>
</tr>
</tbody>
</table>

Table 1. A summary of the results (mean; Standard Error) from the impaired arm for measures of resistance to passive movement (RTPM) and speed for each Ashworth group.

In both the impaired and non-impaired arm, there were no significant differences in RTPM between the three repeated measures (p > 0.20), however the velocity of passive movement was significantly lower for the first measure (p < 0.05).

<table>
<thead>
<tr>
<th>Non-impaired</th>
<th>Impaired</th>
</tr>
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<tbody>
<tr>
<td>RM1</td>
<td>RM2</td>
</tr>
<tr>
<td>RTPM (N/deg)</td>
<td>0.17; 0.02</td>
</tr>
<tr>
<td>Speed (deg/s)</td>
<td>45; 7</td>
</tr>
</tbody>
</table>

Table 2. A summary of the results (mean; Standard Error) showing the relationship in resistance to passive movement (RTPM) and speed between the three repeated measures (RMn = Repeated Measure n; n = 1, 2, or 3).

The force angle relationships were linear (R² > 0.6) in 90.6% of measurements (Figure 2). The non linear the curve shapes (9) were reclassified using breakpoint analysis (a technique to split a non-linear curve into two linear segments). All non linear curve shapes in the impaired arm were classified: yield (4) (Figure 3), initial catch (2), catch at end range of movement (1) (Figure 4). Such classification was not possible in the non-impaired arm (2).

Figure 3: A curve showing yield Figure 4: A curve showing a catch at end range of movement

Discussion

The prototype system developed in this study has been successfully used to quantify resistance to passive movement (RTPM) in a clinical environment under various patient testing conditions (e.g. in the acute care ward, in patients with poor sitting balance etc.). The data presented here show that the technique can provide reliable data that relate to clinically important variables when used with subjects one week after stroke.

Although all measurements were made one-week post stroke, the RTPM in the impaired arm was higher than that in the non-impaired arm and more than half the population had an MAS greater than “0”.
This could have resulted from the early development of spasticity, however, existing evidence would suggest that this does not occur (Speech & Dombovy 1994). Therefore it is likely that changes in viscoelastic properties associated with reduced use could have been the most likely cause of decreased compliance (Goldspink & William 1990).

Although there were no significant differences in RTPM between the three repeated measures, the first measured value was higher than the second and third, despite speed being significantly slower in the first. This could suggest that RTPM is influenced by the immediate past history of movement. Although reflex activity in the elbow flexors would have been triggered by the speeds measured in this study (Katz et al 1992) the changes in RTPM showed no velocity dependence. Together this would suggest that the increase in RTPM observed in the impaired arm might have been independent of spasticity (which is defined as a velocity dependent phenomenon - Lance 1980).

Poor subject compliance, i.e. subject actively assisting in the movement, could have resulted in non linear force angle plots in the non-impaired arm. Although the catch at end range of movement (Figure 4) was consistent with the biomechanical definition, a clinical catch was not identified. This raises doubts about the sensitivity of the MAS in identifying this phenomenon. The phenomena of yield and initial catch are both consistent with the description of the clasp-knife phenomenon (Sheean 2001). However, inertial effects encountered at the start of movement could also contribute to the initial catch.

**Conclusion**

The need to quantify neurological impairment is increasing rapidly with a perceived need to justify clinical procedures used routinely. The technical challenge of doing this relates to the need to provide reliable and sensitive measurement systems that can be used within the short time available in the clinic. A device to measure resistance to passive movement at the elbow, which is more sensitive and reliable than the MAS, has been presented in this paper. Evidence from this study supports previous findings suggesting that MAS may not have sufficient construct validity or reliability in assessing spasticity (Pandyan et al 1999). Further work is now required to study the relationship between MAS and RTPM in a general stroke population, to investigate the influence neuronal activity on RTPM and to validate the use of RTPM as a measure of spasticity.

**References**


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