Research report

Deficits in the coordination of agonist and antagonist muscles in stroke patients: implications for normal motor control

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Abstract

Movement impairments about a single joint in stroke patients may be related to deficits in the central regulation of stretch reflex (SR) thresholds of agonist and antagonist muscles. One boundary of the SR threshold range for elbow flexor and extensor muscles was measured in hemiparetic subjects by analysing electromyographic activity during stretching of relaxed muscles at seven different velocities. For each velocity, dynamic SR thresholds were measured as angles at which electromyographic activity appeared. These data were used to determine the sensitivity of the threshold to velocity and the static SR thresholds for flexors and extensors. In contrast to relaxed muscles in healthy subjects, static flexor and extensor thresholds lay within the physiological range in 11/12 and 4/12 subjects, respectively. This implies that, in the range between the static SR threshold and one of the physiological joint limits, relaxation of the muscle was impossible. Subjects then made slow movements against different loads to determine their ranges of active movement. Maximal flexor and extensor torques were lower in hemiparetic subjects throughout the angular range. In some subjects, ranges were found in which no active torque could be produced in either extensor or both muscle groups. These ranges were related to the boundary values of SR thresholds found during passive muscle stretch. The range in which reciprocally organized agonist and antagonist muscle activity could be generated was limited in all but one subject. When attempting to produce torque from positions outside their measured range of movement, excessive muscle coactivation occurred, typically producing no or paradoxical motion in the opposite direction. Results suggest a relationship between spasticity measured at rest and the movement deficit in stroke by demonstrating a link between motor deficits and control deficits in the central regulation of individual SR thresholds. © 2000 Elsevier Science B.V. All rights reserved.

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1. Introduction

Since the first descriptions of the ‘hemiplegic syndrome’ at the beginning of the century, the notion of altered tone or spasticity has been closely linked to that of disordered motor control seen after cerebro-vascular accidents (CVA [56]). In the first half of the century, spasticity and movement deficits were viewed as separate phenomena, focussing on the reflex nature of spasticity. Later studies acknowledged the influence of altered reflex behavior on voluntary motor control [11,25,51]. In 1980, Lance [39] proposed a new definition of spasticity emphasising that it is a motor disorder, thus recognising that spasticity is an independent phenomenon. Since then, it has been generally agreed that spasticity may be better assessed in the context of functional movements such as bicycling, walking and reaching, etc. [10,26,34,36,42,57]. To date, however, despite many experimental studies, the precise relationship between the clinical phenomenon of spasticity usually measured at rest and the active motor disability remains unclear. The goal of this study was to describe this relationship in the elbow joint of hemiparetic patients.

Following a central nervous system (CNS) lesion such as stroke, sensorimotor dysfunctions contralateral to the brain lesion occur in a large percentage of patients. Spasticity may appear predominantly in physiological flexor muscles while both flexors and extensors may be weak and manifest changes in motor unit properties [7,28,37,52]. Several types of motor impairments have been described in stroke patients. Movement times during goal directed arm
movements and gait can be prolonged up to four times that of healthy subjects [12,38,42]. In the upper limb, interjoint coordination during reaching movements is disrupted [42] and disruptions in the recruitment and derecruitment of agonist and antagonist muscles in the forearm [32] have been reported.

Although motor deficits have been detailed and well-described, the disturbances in control mechanisms underlying these changes have not been clearly elucidated. One reason for this may have been the lack of an integrated model of motor control combining reflex and central contributions to normal movement. Such a model would allow us to predict how changes following CNS damage may affect muscle tone and movement.

Previous studies have suggested that spasticity may be related to problems in regulating stretch reflex (SR) thresholds in specific muscles [35,41,44,50]. For example, at the elbow joint, stroke patients are unable to regulate SR thresholds of elbow flexors in the required range [44] and have a decreased ability to coordinate the regulation of agonist and antagonist SR thresholds [43] to stabilise the arm after rapid unloading.

The present study extends previous findings of decreased SR threshold regulation in a single muscle under passive conditions [44] to an agonist/antagonist muscle pair under passive and active conditions. We approached this problem by (1) measuring the range of regulation of the SR threshold in individual flexor and extensor muscles around the elbow joint in hemiparetic subjects; and (2) measuring the ability of hemiparetic subjects to produce coordinated changes in agonist and antagonist SR thresholds during voluntary elbow flexion and extension efforts. Some of the results have been presented in abstract form [46].

2. Materials and methods

2.1. Subjects

Twelve hemiparetic (mean age 42 years, range 24–65 years) and four healthy control subjects (age range 21–50 years) participated in the study after giving their informed consent. The study was approved by the hospital ethics committee. All testing was done using the subject’s dominant arm. The hemiparetic subjects were included if they (1) had sustained a unilateral stroke as documented by their medical history and appropriate medical tests (CAT scan, NMR) leading to motor deficits in their dominant arm; (2) were able to understand simple commands (no receptive aphasia); (3) had full passive range of motion in the elbow joint and (4) had no subluxation in the shoulder or pain in the affected arm. Healthy subjects were excluded if they had pain in the arm to be tested.

Prior to each session, spasticity and motor function in the affected arm of hemiparetic subjects were evaluated by two commonly used clinical scales fully described elsewhere [44,45]. Briefly, the spasticity scale includes measures of phasic reflex excitability (tendon jerk and clonus) and tonic muscular resistance to an imposed full-range stretch at a moderate speed (modified Ashworth scale; [2,6,49]. On this scale, composite scores ranging from 1–5, 6–9, 10–12 and 13–16 correspond to no, mild, moderate and severe spasticity, respectively. Previous studies have demonstrated that this composite spasticity score has a significant negative correlation with the threshold of the SR in the elbow flexor muscles in spastic hemiparetic subjects ($r = -0.65$, $p < 0.05$, [44]) and with clinically measured residual functional ability of the affected arm [42,43]. The residual functional ability of the arm was measured on a scale ranging from 0 (no function) to 66 (normal function) using the upper limb section of the Fugl-Meyer scale [24].

Table 1 describes the demographic characteristics as well as Fugl-Meyer and spasticity scores for the hemiparetic subjects. Eleven of the twelve subjects had sustained a left hemispheric stroke leading to a right-sided hemiparesis and one had sustained a right hemispheric stroke leading to left-sided hemiparesis. Lesion location studies revealed three subjects with cortical lesions only, three subjects with cortical/sub-cortical lesions and six subjects with only sub-cortical lesions. Levels of spasticity ranged from 2 (no spasticity) to 15 (severe spasticity). The Fugl-Meyer scores ranged from 12 (low motor function) to 65 (close to normal motor function).

2.2. Experimental protocol

Subjects were seated in an adjustable chair with the shoulder of the dominant or affected arm positioned midway between flexion and abduction at an angle of 70°. The flexion/extension axis of the elbow was vertically aligned with the rotational axis of a horizontal manipulandum (inertia about 0.03 kg m² which was coupled to a torque motor (Mavilor Motors, MT 2000). The forearm was held in the neutral position between pronation and supination by a padded polypropylene mold that encompassed the hand and forearm up to the head of the radius and was attached to the manipulandum. Full extension of the elbow was defined as 180° so that an increase in the joint angle was associated with stretching of the flexors and shortening of the extensors. The physiological range is the range of joint motion ($\theta_x, \theta_y$), the limits of which are defined by biomechanical constraints. The range of elbow movement is thus limited at one end by contact between the soft tissues of the arm and forearm ($\theta_x \approx 30^\circ$) and at the other end by bony contact between the olecranon of the ulna and the trochlea and olecranon fossa of the humerus ($\theta_x \approx 180^\circ$). A high precision digital resolver placed on the axis of the manipulandum was used to measure angular displacement and velocity of the forearm. The torque motor was controlled by both position and velocity feedback.
EMG signals were recorded from two elbow flexors (long head of biceps brachii, BB; and brachioradialis, BR) and from two elbow extensors (lateral head of triceps, TB; and anconeus, AN) using active bipolar surface electrodes (1 mm silver chloride strips, 1 cm long and 1 cm apart). After careful preparation of the skin, the EMG signals were recorded with a 40–500 Hz bandpass filter and amplified. All data were sampled at 1000 Hz.

### 2.3. Measurement at the muscle level: limits of regulation of the SR threshold in individual muscles

Velocity-dependent (dynamic) SR thresholds (LA<br>) were measured for each muscle group during passive displacement of the limb [44]. Since relaxed muscles may be activated by high velocity stretching (e.g., during a tendon jerk) and tonically active muscles may be deactivated by a sudden shortening (e.g., during an unloading reflex), the dynamic threshold is considered a decreasing function of velocity [19] (see Section 2.5).

Subjects were instructed to relax their arm completely and not to intervene while the arm in the manipulandum was being extended by the torque motor through an arc of 100°, starting from full flexion (approximately 30°). An oscilloscope was used to view the EMG activity of the muscle to be stretched (in this case the flexor EMG activity, see Fig. 1A) in order to ensure complete relaxation prior to each stretch. The passive joint was extended by the motor which produced near bell-shaped velocity profiles with mean velocities of: 8, 16, 32, 53, 80, 120 and 160°/s. Peak velocities however, were higher, ranging up to 360°/s. Eight trials were collected for each velocity. The velocity patterns were highly reproducible (see Fig. 2 in which all trials are superimposed) from trial to trial because of the high power of the torque motor (maximal torque about 60 N m) which allowed the programmed velocity profile to be generated regardless of the amount of muscular resistance. Correlations between velocity profiles from successive trials for each velocity ranged from 0.99 to 1.00. We chose to apply stretches with bell-shaped instead of step-like or ramp velocity profiles since the former better imitates the smooth changes in arm position during testing of muscle resistance by clinicians. In addition, step-like or ramp-shaped profiles may be associated with acceleration jerks at the onset and offset of the passive displacement leading to unwanted voluntary reactions by the subjects (see Fig. 4 in Ref. [19]). The order of presentation of stretches at each velocity was randomly distributed throughout the 56 trials. After each trial, the arm was passively returned to the initial, flexed position at approximately 50°/s. Between trials, a random rest period of 6–10 s was permitted in order to allow recovery of the muscle fibres and to minimise the influence of stretch history on the response to the subsequent stretch [31]. After testing passive stretch responses in the flexors, the same set-up was used to move the arm in the opposite direction for the measurement of dynamic SR responses in the extensors. The initial position for stretching of the extensors was approximately 170° and the EMG activity monitored prior to stretch was that of the elbow extensors.

### 2.4. Measurements at the joint level: the range of regulation of SR thresholds

The range in which subjects were able to actively generate net flexion and extension torques was then deter-

<table>
<thead>
<tr>
<th>S</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Side, type, site of lesion</th>
<th>Years since stroke</th>
<th>Motor score (66)</th>
<th>Spasticity score (16)</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>36</td>
<td>M</td>
<td>L, infarct, MCA, fronto-parietal lobe, para-central lobe, basal nuclei lesions</td>
<td>3.2</td>
<td>65</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>52</td>
<td>F</td>
<td>L, embolism, MCA, basal nuclei lesion</td>
<td>2.7</td>
<td>55</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>32</td>
<td>M</td>
<td>L, thrombosis, internal carotid artery, fronto-parietal lesion</td>
<td>2.5</td>
<td>61</td>
<td>8</td>
</tr>
<tr>
<td>4</td>
<td>35</td>
<td>M</td>
<td>L, haemorrhage, central infra-cerebral and posterior internal capsule lesions</td>
<td>5.0</td>
<td>49</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>F</td>
<td>L, embolism, MCA, corona radiata and internal capsule lesions</td>
<td>3.0</td>
<td>42</td>
<td>9</td>
</tr>
<tr>
<td>6</td>
<td>43</td>
<td>M</td>
<td>L, haemorrhage, thalamus, internal capsule and basal nuclei lesions</td>
<td>3.2</td>
<td>54</td>
<td>11</td>
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<tr>
<td>7</td>
<td>34</td>
<td>M</td>
<td>L, thrombosis, internal carotid artery, infarc sylvian artery, basal nuclei lesion</td>
<td>7.1</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>8</td>
<td>41</td>
<td>M</td>
<td>L, haemorrhage, temporal lobe and basal nuclei lesions</td>
<td>6.0</td>
<td>36</td>
<td>13</td>
</tr>
<tr>
<td>9</td>
<td>24</td>
<td>F</td>
<td>R, infarct, MCA, fronto-parietal lesion</td>
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<td>23</td>
<td>14</td>
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<tr>
<td>10</td>
<td>62</td>
<td>M</td>
<td>L, infarct, internal capsule and basal nuclei lesions</td>
<td>0.8</td>
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<td>14</td>
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<tr>
<td>11</td>
<td>30</td>
<td>F</td>
<td>L, aneurysm posterior communicating artery, cortical and subcortical lesions</td>
<td>6.5</td>
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<td>14</td>
</tr>
<tr>
<td>12</td>
<td>40</td>
<td>M</td>
<td>L, haemorrhage, fronto-parietal lobe lesion</td>
<td>4.3</td>
<td>15</td>
<td>15</td>
</tr>
</tbody>
</table>
Fig. 1. EMG and kinematic data from a subject (S9) with severe spasticity when the elbow flexors (A) and the extensors (B) were stretched at the mean velocities of 32 and 120°/s. BB, biceps brachii; BR, brachioradialis; TB, triceps brachii; AN, anconeus. The times of stretch onsets for each direction are indicated by thin arrows in the velocity traces. The onset of EMG activity threshold in the flexors in A and the extensors in B are shown by thick arrows. Note that, in both muscle groups, the tonic level of EMG at the final (longer) muscle length is higher than that before the onset of stretch (the tonic SR).

Maximal net joint torques and associated EMG activity were measured as a function of the elbow joint angle. Net flexor torques were considered as positive values and net extensor torques were considered as negative values. For the flexion direction, the elbow was placed in full extension (see Fig. 4, circle a) and subjects were instructed to slowly flex their elbows through the entire angular range. This was done initially with no load opposing the flexors (Fig. 4, circle a'). In subsequent trials, a constant load resisting elbow flexion was applied. The load was increased in steps of 2–4 N.m. For each load, the final elbow position was recorded at which the subject could not move their arm any further (Fig. 4, open circles) despite maximal verbal encouragement by the experimenters. The load was increased until the subject was unable to move their arm away from the initial position. When this occurred, the initial position was changed into one of more flexion in which the moment arm was increased, the muscle fibres were shortened and thus, active movement was once again possible (Fig. 4, line c). These experimenter-imposed changes in position varied between 10° and 30°. Three trials were collected at each load level and subjects were given enough rest time between trials to avoid muscle fatigue (e.g., a 5-s trial was followed by rest.

Fig. 2. Repeatability of the stretch input and the stretch responses. Velocity and angle traces recorded in subject 2 for eight trials are superimposed in each panel representing stretch of the elbow flexors at 53 (A) and 120°/s (B) and stretching of the elbow extensors at 120°/s (C). Also shown are flexor EMG from four trials for each velocity and direction of stretch (top four traces in each panel) and extensor EMG from the corresponding four trials (bottom four traces of each panel).
period of about 25 s). Limitations in the active range of motion were investigated throughout the entire range of loads. The same protocol as described for flexion was used for extension, except that movements started with the arm in full flexion instead of full extension.

In addition, we measured agonist and antagonist muscle activity when subjects were asked to actively generate flexion and extension movements from three different initial joint ranges: (1) mid-range (between 80° and 100°), (2) inner range for flexor muscles (near full flexion) and (3) inner range for extensor muscles (near full extension). For those subjects who could not attain the initial position voluntarily, the arm was placed in each position by the experimenter. Movements were unopposed except for minimal inertial and frictional torques (< 0.01 N m). For each movement, EMG, displacement and velocity data were recorded during three 2-s trials.

2.5. Data analysis

2.5.1. Muscle level: the limits of regulation of SR thresholds

To evaluate SR responses, EMG signals of individual trials were high-pass filtered at 40 Hz and rectified. For each velocity of stretch, the dynamic SR threshold was defined as the angle at which the EMG signal surpassed and remained above 2 S.D. of the pre-stretch baseline activity for at least 50 ms (see Fig. 1). The EMG onsets were measured in each of the stretched muscles (BB and BR when flexors were stretched; TB and AN when extensors were stretched) on a trial-by-trial basis using a cursor on an interactive video display. The threshold angle was defined as the angle of EMG onset of the first muscle that became active see Fig. 1. For each subject, the dynamic threshold angles were averaged for each velocity of stretch, the thresholds for flexors or extensors did not fall within the physiological range of the joint are not shown S1 for the flexors and S1±8 for the extensors.

Flexible SR thresholds for the flexors \( \lambda^f \) and extensors \( \lambda^e \) were averaged for each velocity of stretch (Table 2) and plotted on velocity/angle phase diagrams for each direction of passive stretch. Our data were consistent with the assumption that the dynamic threshold is a decreasing function of velocity [19]. Originally, it was assumed that the relationship is linear. We considered, however, a more general relationship between the threshold and velocity:

\[
\lambda^d = \lambda - f(\mu \omega) \tag{1}
\]

where \( \lambda \) is the static threshold, \( f(\mu \omega) \) is an increasing function of the angular velocity, \( \omega = \frac{d\theta}{dt} \); \( f(\mu \omega) = 0 \) if \( \omega = 0; \frac{df}{d\omega} = \mu \) at point \( \omega = 0; \mu \) is a coefficient characterising the velocity sensitivity of the threshold. Note that \( \mu \) is a time-dimensional parameter that represents the ratio of dynamic to static sensitivity of muscle spindle afferents [18]. In the case of a linear regression, function \( f(\mu \omega) \) can be replaced with \( \mu \omega \). In some cases, however, the experimental points suggested some small deflection from linearity such that the sensitivity of the threshold to velocity decreased with increasing velocity. To test this possibility, we used not only linear but also non-linear regression. In the latter case, the angular velocity was considered a second order polynomial function of threshold, which resembles \( f(\mu \omega) = a \sqrt{1 + 2 \mu \omega / a} - a \) where \( a \) is a positive parameter. The velocity-threshold relationship was considered linear if non-linear regression did not increase the correlation coefficient, \( r \). Otherwise the relationship was considered non-linear.

After the type of regression was chosen, the static thresholds for the flexors \( \lambda^f \) and extensors \( \lambda^e \) were estimated by extrapolating the regression curve to zero-velocity. In hemiparetic subjects, static thresholds \( \lambda \) determined in this way were usually found within the angular range.
For purposes of analysis, in those subjects with no SRs even at high velocities of stretch, \( \lambda_f \) for the flexors was set at the maximal physiological angle and \( \lambda_e \) for the extensors was set at the minimal angle. Since, in these subjects, the actual limits of \( \lambda \) were located beyond the physiological range, this approximation underestimated the actual threshold range.

### 2.5.2. Joint level: coordinated regulation of flexor and extensor SR thresholds

We classified the types of muscle activation patterns produced in different angular sub-ranges of the joint qualitatively. The sub-ranges were defined according to the experimentally measured boundary values (\( \lambda_f^1 \) and \( \lambda_e^1 \)) of the SR thresholds. For example, if these thresholds were both inside the angular physiological range (\( \theta_e, \theta_f \)) and \( \lambda_e^c < \lambda_f^1 \), then the sub-ranges were: \( \theta_e < \theta < \lambda_e^c ; \lambda_e^c < \theta < \lambda_f^1 \); and \( \lambda_f^1 < \theta < \theta_f \).

EMG signals were analysed both qualitatively and quantitatively. Qualitative analysis was done using an interactive video display to determine if reciprocal or coactivation patterns of agonist and antagonist muscle activity were present during attempts to actively flex or extend the elbow from the three different angular sub-ranges defined above. Similar to the analysis of threshold SR responses, a muscle was considered active when the level of muscle activation surpassed 2 S.D. of the activity recorded in the same muscles at rest.

EMG signals were then quantitatively analysed to determine the degree of agonist and antagonist coactivation during these movements. The EMG areas of flexors (BR) and extensors (AN) were calculated in the initial 250 ms window starting from the point at which the first EMG signal (BR or AN) surpassed 2 S.D. of its mean resting level. For each movement, a 4-s trial was divided into three consecutive 250 ms segments starting from the point at which the first EMG signal (BR or AN) surpassed 2 S.D. of its mean resting level. For each movement direction, two values were recorded: the initial and final combinations of position and torque. These were averaged for three consecutive trials and were plotted for each subject. A second-order polynomial curve was fit through these initial and final points describing the relationships between the maximal torque (\( T \)) and joint angle (\( \theta \)) for each movement direction. Maximal torque at 40°, 75°, 110°, 145° and 180° were derived from the polynomials and then averaged over subjects for each direction. These values were compared to those from healthy subjects.

### 2.6. Correlations

Spearman rank-order statistics were used to correlate clinical spasticity scores, motor function scores (Fugl-Meyer), \( \lambda \) and \( \mu \) for both flexors and extensors, torque, and the ranges of \( R \) and \( M \). In all tests, a significance level of \( p < 0.05 \) was used.

### 3. Results

#### 3.1. Stretching passive muscles: the limits of regulation of SR thresholds

In Fig. 1, examples of EMG and kinematic data from one subject are shown for single trials in which the flexors (A) and the extensors (B) were stretched at mean velocities of 32°/s and 120°/s, respectively. The data were recorded from subject 9 who had severe spasticity (spasticity score: 14/16) and moderately impaired motor function (Fugl-Meyer score: 23/66). The thin arrows on the velocity traces indicate the time of stretch onset and the thick arrows on the EMG traces show the onset of the SR response. The stretch response was characterised by gradually increasing activity in the stretched muscles followed by tonic EMG activity after the end of the stretch. In the initial position for stretching the extensors (Fig. 1B), the arm was held in a position near maximal extension that was beyond the threshold level of the flexors and thus...
Table 1. Velocity and angle curves for all eight stretches applied to the flexors at two velocities.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Flexors</th>
<th>Extensors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt;180</td>
<td>&lt;40</td>
</tr>
<tr>
<td>2</td>
<td>93.3 (3.4)</td>
<td>0.29 &lt;40</td>
</tr>
<tr>
<td>3</td>
<td>143.4 (8.8)</td>
<td>0.50 &lt;40</td>
</tr>
<tr>
<td>4</td>
<td>150.3 (2.9)</td>
<td>0.45 &lt;40</td>
</tr>
<tr>
<td>5</td>
<td>125.5 (3.0)</td>
<td>0.33 &lt;40</td>
</tr>
<tr>
<td>6</td>
<td>125.6 (6.1)</td>
<td>0.49 &lt;40</td>
</tr>
<tr>
<td>7</td>
<td>105.3 (2.2)</td>
<td>0.15 &lt;40</td>
</tr>
<tr>
<td>8</td>
<td>101.2 (2.0)</td>
<td>0.15 &lt;40</td>
</tr>
<tr>
<td>9</td>
<td>83.9 (4.4)</td>
<td>0.38 94.5 (1.3)</td>
</tr>
<tr>
<td>10</td>
<td>81.9 (2.7)</td>
<td>0.12 87.2 (3.4)</td>
</tr>
<tr>
<td>11</td>
<td>103.4 (2.3)</td>
<td>0.18 72.6 (1.9)</td>
</tr>
<tr>
<td>12</td>
<td>122.6 (3.1)</td>
<td>0.11 95.3 (6.2)</td>
</tr>
</tbody>
</table>

5.8°. For the extensors, the mean onset of EMG thresholds for the 120°/s stretch was 307.2 ms corresponding to an angle of 107.0 ± 2.0°. For this subject, the static flexor but not extensor SR threshold determined by non-linear regression lay within the physiological range of the joint. Dynamic SR thresholds for flexors and extensors for all subjects in whom calculated static SR thresholds lay within the physiological range of the joint are listed in Table 2. Some of the data points for stretches applied at the slower velocities were difficult to measure precisely due to a low signal-to-noise ratio. For these data points (missing data points at slower velocities in Table 2), we could not reliably determine the onset of the EMG response. Thus, these points were not estimated at all and static SR thresholds were determined by regression analysis through 4 or 5 instead of 7 points.

For the majority of cases the coefficients of variability of the angular component of the dynamic SR measure were lower than 10%. Coefficients reached between 10% and 20% in 20% of the cases (14 out of 70 cases). Velocities could be more variable however, with 10 out of 70 cases having coefficients of variability greater than 10% and 10 other cases with coefficients of variability greater than 20%.

The velocity and angle values corresponding to the mean EMG SR thresholds in each muscle group were determined for each subject and plotted together with the velocity/angle (phase) trajectories. Fig. 3 shows one such plot for subject S10 (see Tables 2 and 3). Positive and negative velocities represent stretching of the flexors and extensors, respectively. The averaged SR thresholds of the flexors and the extensors for each velocity are represented as filled circles in the phase diagram. Static SR thresholds

Movement kinematics and dynamic thresholds were highly reproducible for a given velocity of stretch in a given subject. This is illustrated in Fig. 2 for subject S2, who had a moderate motor deficit and mild spasticity (see Table 1). Velocity and angle curves for all eight stretches applied to the flexors at two velocities (53°/s in A and 120°/s in B) and to the extensors at 120°/s (Fig. 2C) are superposed and EMG traces for flexors (top) and for extensors (bottom) are illustrated for four trials. The correlations between velocity traces of successive stretches ranged from 0.99 to 1.00. In the example shown in Fig. 2, the mean onset of flexor EMG was 120.4 ms for the 53°/s stretch and 66.8 ms for the 120°/s stretch. These latency values corresponded to angles of 75.3 ± 9.0° and 58.5 ±
were estimated from the dynamic SR threshold values for each subject using both linear and non-linear regression analysis. The form of regression analysis in which most of the variance was explained by the equation and which had the best r value was used to estimate the static value of λ for an individual subject (see Section 2). Thus, non-linear equations were used for four (S2, S4, S6, S9) out of the 11 estimations for the flexor threshold (λf). In these subjects, r values ranged from 0.80 to 0.90 compared to 0.03 to 0.82 for the same subjects using linear estimates. For the remaining seven subjects, higher r values were obtained with linear approximations (range 0.65 to 0.93; mean 0.80 ± 0.9). Similarly, for estimates of extensor thresholds, λe, non-linear approximations were used in two subjects (S9, S12) and linear approximations were used in the two others. Non-linear approximations improved the correlation from 0.92 to 0.98 for S9 and from 0.72 to 0.94 for S12. R values from the linear estimates used for S10 and S11 were 0.92 and 0.98, respectively, and were not improved by the use of a non-linear estimation. The type of regression had little effect on the extrapolated value of the static threshold. The difference in the estimation of λ exceeded 2° in only two of the six cases in which non-linear instead of linear estimates were used. For these two cases, the difference in λ was 7.9° (S2) and 13.1° (S4).

For the subject shown in Fig. 3 (S10), the static SR thresholds (λf and λe) determined from linear regression analysis were 81.9° and 87.2°, respectively. The subscripts denote boundary values of the thresholds showing that flexors cannot be relaxed at joint angles where θ > λf = 81.9° and extensors cannot be relaxed where θ < λe = 87.2°. For these regressions, r values were 0.73 and 0.92 and the values of the time-dimensional parameter, μ were 0.12 and 0.19 s for flexors and extensors, respectively. In Fig. 3, the shaded areas to the right and left of the regression lines for the flexor and extensor muscles, respectively, represent the ranges in which the muscle groups were active. In other words, this subject could not relax his muscles when they were stretched beyond the threshold angle (into the shaded zone). Individual results for all subjects are listed in columns 2 and 4 of Table 3.

3.2. Active movement: range of regulation of torque and arm position

For zero load, all of the healthy subjects but only four of the hemiparetic subjects (Table 3, column ΔM = M+ − M−) were able to produce active movements in the whole physiological range of the joint. An example of the measurement of the range of active movement is shown in Fig. 4 for one hemiparetic subject (S6). This subject was able to actively flex his elbow from a starting position of full extension (180°, point a) to a final position of full flexion (30°, point a′) with no load. Further flexion was only limited by the juxtaposition of soft-tissue at the end of range. The range of active movement depended on the opposing load. Full active flexion was possible with loads of 0, 4, 7, 11 and 14 N m represented by five filled circles placed above the 180° position. When the load was increased to 16 N m (point not shown), S6 could not initiate flexion from the fully extended position of the elbow. The elbow was then passively flexed by the experimenter to 150° indicated by a vertical bar at point c. From this position, the subject was able once again to actively flex the elbow through the remaining range with loads up to 21 N m. When the load was further increased to 24 N m, flexion beyond 41° was not possible. With loads greater than 25 N m, the subject could not actively flex his elbow away from the starting position of 150°. Again, the arm was flexed by the experimenter to a position of approximately 120° from which loads of > 25 N m could be balanced. In this way, the borders of active torque generation were determined for the flexor and extensor muscles in each subject. The borders (solid curves) were calculated by fitting second order polynomials through the points corresponding to the torque limits at each angle investigated (only filled and open circles were used in this calculation). The area limited by the solid curves (Fig. 4) represents the attainable torque–angle combinations for this subject.
Fig. 5 compares the mean maximal torques throughout the angular ranges for the flexors (filled symbols) and the extensors (open symbols) in 12 hemiparetic (circles) and four healthy (squares) subjects. Average flexor and extensor torques in hemiparetic subjects were lower than those of healthy subjects, particularly net extension torque at more extended positions of the joint (Fig. 5, \( \approx 180^\circ \)). Since our sample of four healthy subjects is small and therefore limited, we contrasted maximal torques in the hemiparetic subjects with those of larger groups of healthy subjects obtained in other studies. For elbow flexors, maximal mid-range torques were 37.4% of that for healthy adult men (70.8 \pm 16.6 N m) and 48.2% of that for healthy adult women (39.3 \pm 4.4 N m; [9]). For elbow extensors, maximal values were 63.2% of those reported in healthy adult men (33.6 \pm 8.1 N m) and 57.5% of that for healthy adult women (19.3 \pm 5.6 N m; [9]).

For flexor muscles of most subjects (all but S3 and S4) the maximal torque peaked at mid-range (110°) and decreased substantially at each extreme of the joint range. The decline in maximal flexor torque in the inner range of flexion (i.e., angular range in which muscle fibres are shortest; \( \approx 40^\circ \)) ranged from 32% to 100%. In the outer range, this percentage was greater (56% to 100%). This is in contrast to the shape of the \( T/\theta \) relationship in healthy subjects in whom mean maximal torques developed at the extreme inner and outer joint ranges declined by 19% and 59%, respectively, of maximal torque. For the extensors, maximal torque peaked when muscles were at longer lengths (75°) and dropped off substantially when the extensor muscles were shortened. The decrease in maximal torque at the extremes of range was even more apparent when torque values were normalised by expressing them as a percentage of the maximal torque obtained at mid-range for the same muscle in each subject. Normalised torque/angle data for all subjects showed an asymmetrical distribution of maximal torque throughout the angular range that was more apparent for the extensors than the flexors (Fig. 5, lower panel).

3.3. Combining ranges of SR thresholds and active movement

Torque/angle combinations together with the range of active motion (\( M_{-}, M_{+} \)) when the load was zero are shown in Fig. 6 for three hemiparetic and in Fig. 7 for a healthy subject. For all subjects and for each muscle group, the ranges of active movement decreased with increasing load torque. For zero load, different ranges of flexor and extensor movement were recorded in the hemiparetic subjects. Five subjects (S1, S3, S4, S6 and S8) could produce active movements in the complete (\( n = 4 \)) or almost complete (\( n = 1 \)) joint range in both flexion and extension directions (Fig. 6, left panel). Five other subjects (S2, S5, S7, S9 and S12) could flex their elbows through the full angular range but could not fully extend the elbow (Fig. 6, middle panel). For these subjects, the maximal angle that they could reach by actively extending their elbow was 60–80° less than full extension (180°). We refer to this angle beyond which no further elbow extension movement was possible, as \( M_{+}. \) Starting from \( M_{+}, \) however, they could produce full flexion. One subject in this group (S12) was unable to produce any active extension at all. In two other subjects (S10 and S11), the range of active movement was limited in both flexion and extension directions (Fig. 6, right panel).

The values of static SR thresholds for flexors (\( \lambda_{f}^< \)) and extensors (\( \lambda_{e}^< \)) determined in the passive SR threshold task were plotted together with the active torque/angle diagrams (Fig. 6, filled and open arrow heads, respectively). The range (\( \lambda_{e}^<, \lambda_{f}^< \)) when \( \lambda_{e}^< < \lambda_{f}^< \) deserves special attention since, in this range, subjects could relax their muscles or produce active movement by appropriately activating their muscles (see below). To distinguish this range, we call it the range of \( R. \) Because of the limitation in SR regulation (\( \lambda_{e}^< \leq \lambda_{f}^< \)), all three subjects shown in Fig. 6 could not relax flexor muscles at any angle \( \theta > \lambda_{f}^+. \) This represents the upper limit (\( R_{+} \)) of \( R. \) There is no similar constraint for the lower limit of \( R \) (\( R_{-} \)) for S6. The fact that this subject could generate active flexor torque in a fully flexed position, \( \theta_{-} \), implies that \( R_{-} < \theta_{-} \).

The ranges of active movement (\( M_{-}, M_{+} \)) and of \( R \) (\( R_{-}, R_{+} \)) for subjects S6, S9 and S11 are compared at the bottom of Fig. 6 (light and dark horizontal bars, respec-
Note that the range of $M$ includes the range of $R$ and may extend beyond it. The open horizontal bars represent the zones of no movement or in which movement could be made in only one direction. For the healthy subject shown in Fig. 7, a similar representation of the zones is made showing that movement can be made in either direction throughout the range of joint angles. The data for hemiparetic subjects is summarised in Fig. 8. For all but one subject (S1), the upper value of the range of the $R$ command was limited. The lower value of $R$ was also limited in four subjects (S9, S10, S11, S12). In two subjects (S9 and S10), the SR of the flexors was less than that of the extensors, implying a zero range (fixed value) of $R$.

### 3.4. Muscle activation patterns

In all healthy subjects, slow movements were produced by reciprocal activation of muscles: agonists were active while antagonists were silent or did not change their activity. Coactivation ratios calculated in healthy subjects initiating movement from different segments of the angular range or making slow movements throughout the range never surpassed 20%. We tested whether or not hemiparetic subjects were able to produce such a reciprocal EMG pattern inside and outside of the range of $R$ defined for each subject. Different patterns of muscle activation were observed when subjects made movements or attempted to move from different initial positions of the joint range. These patterns consisted of either reciprocal activation or coactivation. Fig. 9 shows examples of extension efforts recorded in one hemiparetic (left) and one healthy subject (right) from a starting angle of 170°. The healthy subject produced extension by increasing extensor muscle activity without concomitantly increasing the activity of the flexors (right panel). For the hemiparetic subject (S12), the initial joint angle fell in a joint range beyond $R_\text{L}$. In this subject, the attempt to produce elbow extension resulted in strong agonist/antagonist coactivation (coactivation ratio ranged from 72% to 85%) with the net torque being in the flexion direction.

Thus, attempts to extend the joint outside of the range of $R$ were associated with agonist/antagonist coactivation. On the other hand, when the elbow was placed within the range of $R$, the movement could be accomplished in either direction by a reciprocal muscle activation pattern. This is illustrated for subject S11 in Fig. 10. Similar to S12, S11’s attempts to extend or flex the elbow outside of the $R$ range resulted in coactivation. When the arm was placed within the range of $R$ (i.e., 90° initial position; Fig. 6, right panel, between $R_\text{L}$ and $R_\text{R}$), movements were accomplished by a more reciprocal pattern of muscle activation (coactivation ratio ranged from 35% to 37%). This is illustrated for flexion and extension movements in
Fig. 7. The areas in which active torque was generated in flexor and extensor muscles in one healthy subject. Healthy subjects had full range of active movement ($M_{-}$, $M_{+}$) implying that they were able to regulate the $R$ command in a range larger than the physiological range of motion ($\theta_{-}$, $\theta_{+}$). The horizontal bar below the figure is produced as in Fig. 6.

Fig. 10A and B, respectively. However, when the arm was placed in a flexed position beyond the lower limit of $R$ (initial position of 40°, Fig. 6, right panel), extension could only be accomplished in association with a strong coactivation of the flexors (coactivation ratio = 50–60%). Attempts to extend in a range beyond the upper limit of $R$ (initial position of 170°, Fig. 6, right panel) resulted not in extension, but in slight flexion accompanied by a similarly high degree of coactivation (52–68%) of both muscle groups (Fig. 10D).

These behaviours and muscle activation patterns were typical findings in all subjects who had deficits in the ranges of regulation of $M$ or $R$. This is illustrated in Fig. 11 for four subjects (S2, S10, S11 and S12) making extension efforts from different initial angular ranges. For each subject shown, extension attempts initiated near the extremes of the angular range were characterised by high levels of coactivation. When movements were initiated from within the range of $R$, in general, more reciprocal patterns were observed and if the arm moved beyond the range of $R$ during the same attempt, coactivation patterns occurred. However, when movements were made from outside to within the reciprocal zone, the already established coactivation innervation pattern persisted throughout the movement.

3.5. Correlations with clinical measures

Threshold measures were correlated with clinical spasticity levels as well as with the deficits in voluntary muscle activation in the muscles about the elbow. The static thresholds of the flexors and the extensors defined by stretching passive muscles were significantly correlated with clinical spasticity ($r = -0.75$ and $-0.69$, respectively, Table 4). Thus, hemiparetic patients with more severe spasticity tended to have more severe limitations in the range in which SR thresholds for both flexors and extensors could be regulated. Not surprisingly, threshold disturbances were also correlated with deficits in the ability of hemiparetic subjects to regulate agonist and antagonist muscles during single joint elbow movements. This was reflected by significant correlations between the angu-
Fig. 9. Examples of the agonist and antagonist muscle activation patterns when a hemiparetic (left panel) subject was asked to produce extension movements of the elbow from a position beyond $R_1 (\approx 170^\circ)$. For comparison, kinematic and EMG patterns are shown in a healthy subject (right panel) asked to make the same movement from the same position. In the hemiparetic subject, the command to extend the elbow resulted in a strong coactivation of the flexor with the extensor muscles. Note that the resultant movement direction, in contrast to the healthy subject, was flexion (arrow) instead of extension.

lar locations of $\lambda^+$ and $\lambda^-$ and the range of $R (r = 0.90$ and 0.81, respectively). In addition, there was a stronger relationship between the more global measure of the voluntary motor deficit in the arm (Fugl-Meyer score) and the range of $M$ with the extensor rather than the flexor threshold. The clinical spasticity and Fugl-Meyer scores also showed significant relationships with the limitations in the ranges of reciprocal innervation ($R$) and active joint motion ($M$) but not with absolute muscle force in mid-range. Thus, the crucial impairment following CNS lesions leading to decreased SR thresholds and their ranges of regula-

Fig. 10. Reciprocal and coactivation patterns of muscle activation during attempts to make voluntary movement from different initial angles of the elbow in S11. Movements made into flexion (A) and into extension (B) from the reciprocal zone were accomplished by a reciprocal pattern of muscle activation. Outside of the reciprocal zone, attempts to extend the arm from an initial elbow angle of 40° (C) or 170° (D) were accompanied by strong flexor/extensor coactivation more commonly resulting in net flexion instead of net extension.
Fig. 11. Coactivation ratios for four subjects during attempts to actively extend the elbow from initial positions located in different parts of the angular range. Each circle represents the ratio of agonist and antagonist EMG areas calculated in a 250 ms window placed at the beginning of the EMG burst. The range of $R$ for each subject is indicated by the horizontal rectangle in each panel.

ion may be characterised by the inability to reach some combinations of net joint torque and joint angle. There was also a significant inverse correlation between the degree of spasticity and the value of $\mu$ for the elbow flexor muscles ($r = -0.70$). The parameter $\mu$ characterising the sensitivity of the threshold to velocity (Eq. (1)) contributes to the overall damping of the system which characterises the sensitivity of torque to velocity [19]. Damping affects the stability of the system. Thus, $\mu$ is an essential determinant of stability (see Section 4).

4. Discussion

4.1. Basic findings

In 11 of the 12 stroke patients studied, the range of regulation of the SR threshold of elbow flexors was limited because $\lambda^1_+$ was found to lie within the physiological range of the joint. A significantly smaller number of patients (4 out of 12) also had limitations in the range of regulation of the SR in the extensor muscles ($\lambda^e_+$) and this occurred only in those subjects with the highest levels of clinical disability (S9–S12; Fig. 8). All but one subject (S1) had limitations in the range in which flexor and extensor muscles could be activated reciprocally during active movement (range $R$). In spite of limitations in individual SR thresholds and ranges of $R$, 5 out of 12 subjects could still produce flexor and extensor torques in almost complete ranges of the elbow joint (Fig. 8). However, outside the range of $R$, this movement was associated with agonist/antagonist muscle coactivation (see Figs. 9–11). These findings support the hypothesis that motor deficits in hemiparetic patients may result from limitations in the central specification and regulation of SR thresholds at the muscle level and of central commands that produce coordinated changes in flexor and extensor thresholds at the joint level.

4.2. Flexor SR thresholds as a measure of spasticity

SR thresholds were significantly correlated with clinically measured spasticity in our study (Table 4). If limitations in SR thresholds can be considered as indicators of spasticity, then the finding that such limitations were predominantly found in arm flexor rather than in extensor muscles fits well with the observation that the typical pattern of spasticity following stroke is found in the physiological flexors [8]. Taken together, our data support the suggestion that the static flexor SR threshold may be an objective measure of spasticity in hemiplegic patients.

In the present study, we also measured the time-dimensional parameter, $\mu$, characterising the sensitivity of the threshold to velocity (see Eq. (1)). This parameter describes the time required to “drive” the changes in the muscle length at a given speed from the current (dynamic) to the static SR threshold. This parameter may be responsible for the velocity gain of the SR eventually also defining the damping properties of the system [43]. The data on $\mu$ obtained in the present study may be interpreted based on

Table 4

<table>
<thead>
<tr>
<th>Spasticity</th>
<th>Fugl-Meyer</th>
<th>Flexor threshold $\lambda^1_+$</th>
<th>Extensor threshold $\lambda^e_+$</th>
<th>Range of $R$</th>
<th>Range of $M$</th>
<th>Flexor $\mu$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spasticity</td>
<td>$1$</td>
<td>$0.86^{***}$</td>
<td>$0.65^*$</td>
<td>$0.71^{**}$</td>
<td>$0.51$</td>
<td>$0.67^{**}$</td>
</tr>
<tr>
<td>Fugl-Meyer</td>
<td>$0.75^{**}$</td>
<td>$0.65^*$</td>
<td>$1$</td>
<td>$0.86^{***}$</td>
<td>$0.78^{**}$</td>
<td>$0.21$</td>
</tr>
<tr>
<td>Flexor threshold $\lambda^1_+$</td>
<td>$0.69^*$</td>
<td>$0.71^{**}$</td>
<td>$0.51$</td>
<td>$1$</td>
<td>$0.81^{**}$</td>
<td>$1$</td>
</tr>
<tr>
<td>Extensor threshold $\lambda^e_+$</td>
<td>$0.69^*$</td>
<td>$0.71^{**}$</td>
<td>$0.51$</td>
<td>$1$</td>
<td>$0.81^{**}$</td>
<td>$1$</td>
</tr>
<tr>
<td>Range of $R$</td>
<td>$0.84^{**}$</td>
<td>$0.86^{***}$</td>
<td>$0.61^*$</td>
<td>$0.78^{**}$</td>
<td>$0.80^{**}$</td>
<td>$1$</td>
</tr>
<tr>
<td>Range of $M$</td>
<td>$0.67^*$</td>
<td>$0.86^{***}$</td>
<td>$0.61^*$</td>
<td>$0.78^{**}$</td>
<td>$0.80^{**}$</td>
<td>$1$</td>
</tr>
<tr>
<td>Flexor $\mu$</td>
<td>$0.45$</td>
<td>$0.67^{*}$</td>
<td>$0.21$</td>
<td>$0.55$</td>
<td>$0.34$</td>
<td>$1$</td>
</tr>
</tbody>
</table>

*** $p < 0.005$; ** $p < 0.01$; * $p < 0.05$. 

computer simulations made in the framework of the \( \lambda \) model [29,53]. These show that \( \mu \) describes the fundamentally non-linear behaviour of the system. Values of \( \mu \) in the range 0.04–0.1 s give an optimal movement stability (the arm reaches a final position with a small or without an overshoot). The system is unstable if \( \mu < 0.04 \) s (underdamping: the arm oscillates about the final position). The system is also unstable if \( \mu > 0.1 \) s (overdamping: the arm oscillates in an irregular fashion about the final position). Interestingly, the values of \( \mu \) measured in our group of patients (see also Ref. [44]) belong to the latter range associated with movement instability due to overdamping, suggesting deficits in important parameters determining the movement dynamics (see also Ref. [43]). It is also interesting that patients with high spasticity scores had lower \( \mu \) values, relatively close to the stability range, than patients with low scores, as follows from the finding of an inverse correlation between the degree of spasticity and the value of \( \mu \) for the elbow flexor muscles. This implies, in particular, that even patients with low spasticity scores may have hidden motor deficits that cannot be revealed during a regular clinical investigation.

4.3. The relationship between spasticity and the voluntary motor deficit

An interesting finding of this study was the relationship between the ranges of reciprocal (\( R \)) and of active (\( M \)) movement on the one hand, and the elbow extensor static \( SR \) threshold (Table 4) on the other. Ranges \( R \) and \( M \) were also significantly correlated with the degree of residual functional ability in the whole arm. This implies that the voluntary movement deficit in the arm is related to the presence of spasticity and weakness in the elbow extensor muscles and may not only depend on the degree of spasticity in the elbow flexors. The presence of spasticity in the elbow flexors correlated with the size of the range of \( R \) in which the patterns of muscle activation during active movement were well-organized. Indeed, these results, like those on \( \mu \) (see above), suggest that the relationship between spasticity and the voluntary motor deficit is complex.

4.4. Range of regulation of \( SR \) thresholds

The limitations in the range of \( SR \) regulation and the consequences for the control of muscle tone and movement in stroke patients may be discussed in the framework of the \( \lambda \) model. The limitation of the \( \lambda \) model in which motor control is based on the central modulation of \( SR \) thresholds [16,19]. The \( \lambda \) model is one of several versions of the equilibrium point hypothesis [4,17,40]. It integrates static and dynamic biomechanical properties of muscles (including torque/angle characteristics), reflex and central regulation of movement.

According to the \( \lambda \) model, the range in which muscle force and joint position are controlled is a consequence of the specification of \( SR \) thresholds at the muscle level and their coordinated regulation at the joint level. Expressed in length coordinates, EMG activity arises if the actual muscle length exceeds the threshold length set by the CNS [16,47]. The threshold can also be expressed in angular coordinates, as in the present study. In healthy subjects, the \( SR \) threshold can be specified at any point within the physiological range of the joint. The model describes at least four control variables at two different levels: the muscle level (\( SR \) threshold, \( \lambda \), and gain, \( \mu \)) and the joint level (reciprocal, \( R \), and coactivation, \( C \), commands). At the muscle level, within the physiological range, the \( SR \) threshold is defined as the joint angle at which EMG activity first appears. Normally, the threshold range of \( \lambda \) (\( \lambda_- \) to \( \lambda_+ \); Fig. 12A) should extend beyond the physiological limits of the joint. This is to allow complete relaxation when muscles are fully stretched (in which case, Fig. 12. The regulation of joint angle (horizontal axis) and muscle torque (vertical axis) based on the control of the \( SR \) threshold in the framework of the \( \lambda \) model. (A) In intact systems, the \( \lambda \) threshold, \( \lambda \), of each muscle group (e.g., flexors) can be regulated within a range (\( \lambda_- \) to \( \lambda_+ \)) which extends beyond the physiological range of angular motion (\( \theta_- \) to \( \theta_+ \)) so that muscles can develop force or relax at any angle inside the range. The shaded area shows attainable combinations of torque and angle. The left border of the activation area is defined by the lower physiological limit (\( \theta_- \)) of the joint angle (left vertical line) and by the torque/angle characteristic (left solid line) associated with the minimal value of the angular threshold, \( \lambda_- \). The right border is defined by the upper physiological limit of the joint (right vertical line). The upper border (dotted line) is defined by the force-generating capability of active motor units and by the moment arms of muscle action. (B) At the joint level, the reciprocal command (\( R \)) specifies movement by shifting \( \lambda \) of flexors (\( F \)) and extensors (\( E \)) in the same direction. This, in turn, causes a shift in the net joint torque/angle characteristic (solid line, dashed line) from one position (\( R_f \)) to another (\( R_e \)). (C) The coactivation (\( C \)) command normally does not elicit movement but shifts \( \lambda \) of flexors and extensors in opposite directions (dashed lines) leading (if \( C > 0 \)) to an increase in stiffness of the net joint characteristic (dotted line, compare with solid line as in B).
flexor $\lambda_e$ should be greater than $\theta_-$) and complete activation of the muscle when it is in the fully shortened position (in which case, flexor $\lambda_e$ should be less than $\theta_-$). At the joint level, two central commands regulate the thresholds of agonist and antagonist muscles for single-joint movement (Fig. 12B, C). The reciprocal ($R$) command specifies the threshold angle ($\theta_-$) at which the transition of agonist to antagonist activity or vice versa occurs (Fig. 12B). A coactivation ($C$) command may or may not occur with $R$, and specifies an angular range in which agonist and antagonist muscles may be simultaneously active (coactivation zone) if $C > 0$ (Fig. 12C) or not (no-coactivation zone) if $C < 0$ (not shown). The sign of the net joint torque produced by each muscle group to counteract external forces, changes at angle $R$ (Fig. 12B). In other words, changes in $\lambda^I$ and $\lambda^e$ elicited by the $C$ command, at least in healthy subjects, should produce equal flexion and extension torques and thus, no movement [20]. Thus, although the $R$ command when $C \neq 0$ no longer represents the threshold angle for the pure transition of activity from one group of muscles to the other, it still represents the referent angle which influences muscle recruitment and sets the location of the coactivation or no-coactivation zone in the physiological range. Control inputs to motoneurons may shift the point $R$ and/or change the width of zone $C$ (Fig. 12B, C).

4.5. Coordination of agonist/antagonist muscle activity

The range $(\lambda^e_-, \lambda^I_+)$ found in the present study may likely be associated with the range of the $R$ (reciprocal) command. This may be explained in the following way. According to the $\lambda$ model, slow active flexor movements against zero load can be produced by a decrease in the flexor SR thresholds. To prevent activation of extensor muscles due to stretching, extensor SR thresholds should be diminished in parallel. Such a reciprocal pattern of changes in thresholds is associated with an $R$ command (Fig. 12B; $R_2$ to $R_1$). Extension movements can be produced in a similar way but the direction of changes in thresholds would be reversed. Thus, reciprocal patterns of muscle activation imply the ability to move agonist and antagonist thresholds freely through and beyond the limits of the physiological range. Such a pattern seems possible for subject 6 (Fig. 6, left panel) with one constraint. Because of the limitation in the upper limit of the flexor threshold, the subject could not relax flexor muscles at any angle beyond the SR threshold of the flexors ($\theta > \lambda^e_+$). This may be an explanation for the development of reflexogenic torque in patients with spasticity (for review, see Ref. [55]). In this range, to make an extension movement, the subject could only increase extensor thresholds while attempts to increase flexor thresholds beyond $\lambda^e_+$ (i.e., to prevent flexors from resisting the movement) would be impossible. In other words, S6 could produce extension in the range $\theta > \lambda^I_+$ but only with coactivation of flexor and extensor muscles. Thus, in the group of subjects characterised by the first type of pattern, the upper limit of the reciprocal zone ($R_+$) is defined by the upper limit of the flexor threshold ($R_+ = \lambda^I_+$) while there is no similar constraint for the lower limit of the $R$ command.

In contrast, the control of not only flexor but also extensor muscles was limited in some subjects (Fig. 6, middle panel). At extremes of the physiological range (where muscle fibres were shortened), seven subjects (S2, S5, S7, S9–S12) could not extend the elbow beyond a certain joint angle and three (S10–S12) subjects had limitations in both active extension and flexion ranges (see Figs. 6 and 8). One explanation is that these subjects were unable to generate enough torque in the weak extensors to overcome the resistance of the reflexly coactivated flexors and thus movement beyond a certain angle ($M_+$) of extension was not possible. Note that the border, $\lambda^e_-$, does not necessarily coincide with $M_+$, since EMG activity in the extensor muscles could be observed in subjects’ ineffective attempts to extend their elbows beyond $M_+$. Similarly, notwithstanding the fact that subjects were unable to actively develop a net flexor torque in the extreme inner range of flexion, flexor EMG was always found. This implies that subjects were able to specify flexor thresholds $\lambda^e_+ < \theta_-$ and extensor thresholds $\lambda^I_+ > \theta_-$. This is an important conclusion because it indicates that the inability to fully flex or extend a joint may not be caused solely by muscle weakness but also by an inability to appropriately regulate and coordinate both agonist and antagonist thresholds in all joint ranges.

Limitations in the specification of SR thresholds (Fig. 8) may help to explain the presence of abnormal temporal and spatial patterns of agonist/antagonist muscle activation and inappropriate or maladaptive muscle coactivation which has been reported in stroke patients [10,13,22,38,32]. Abnormal coactivation has been related to isolated factors such as diminished agonist motor unit activation [54], impaired antagonist inhibition [32] or both [30]. Others have suggested that abnormal coactivation may be a consequence of a decrease in the number of possible muscle synergies that the CNS is able to produce because of damage to descending pathways [13]. The possibility that the CNS may be unable to regulate and coordinate SR thresholds in different muscle groups is not inconsistent with the latter interpretation. Our data suggest that altered descending commands may be reflected in changes in the regulation of SR thresholds leading to abnormal muscle synergies. For example, altered descending commands may be responsible for limitations in the range of $\lambda^I_*$. At angles greater than $\lambda^I_*$, flexor activation occurred involuntarily, creating a certain amount of torque. In order to stabilise the limb in this range, the excessive flexor torque was compensated by activation of the extensors creating an excessive amount of coactivation. The resultant coactivation may be due to local, segmental or central factors specifying $R$ and $C$ commands.
The finding that reciprocal patterns of muscle activation were possible within specific angular ranges indicates that there may be some preservation of pathways specifying the R command in some patients. Indeed, previous studies in which stroke patients show an ability to perform continuous tracking [33] or point-to-point movements [21] suggest that they may retain some ability to specify movements in terms of R commands. However, results of the present study suggest that the ability to fully regulate the R command may actually be deficient in most patients.

Deficiencies in the R command may be partly compensated by application of the C command. Theoretically, application of the C command in healthy subjects should produce equal flexion and extension torques without any additional movement. However, in patients with CNS lesions, the balance may be disrupted and application of a C command may abnormally lead to additional movement due to unequal torques generated in agonists and antagonists. This suggestion is consistent with our observation that movements made outside of the range of R were characterised by substantial agonist/antagonist coactivation, not typically seen in healthy subjects. The C command may be used in the disordered CNS to partially compensate the loss in the range of active motion. On the other hand, in some subjects the C command may have been applied inappropriately with the paradoxical result of movement being produced in a direction opposite to the desired one. This finding suggests that there may be deficits in the ability to regulate the C command. Additional support for this conclusion derives from a recent study in which hemiparetic subjects showed an inability to appropriately use muscle coactivation (C command) to limit joint oscillations around a final position after sudden unloading of the elbow [43].

4.6. Muscle weakness

Reductions in maximal voluntary strength of arm muscles in patients with unilateral stroke have been reported previously (e.g., [1,6,9]) and are consistent with the present data. The sources of muscle weakness are diverse. Evidence has been provided for the following factors contributing to muscle weakness in the hemiparetic limb: (1) lack of excitation arising in ascending pathways responsible for voluntary movement [8]; (2) muscle fibre atrophy and contracture [14]; (3) changes in the spatial and temporal patterns of muscle activation causing an inefficient EMG–torque relationship [32,54] and (4) loss of functioning motor units and changes in the properties of the remaining units [15,28,52,58]. Bohannon [5] explicitly investigated the relationship between maximal muscle force and deficits in the voluntary range of motion. The experimental set-up did not however eliminate possible compensatory movements in the shoulder and forearm, and only force at an elbow angle of 90° was directly measured. Nevertheless, a correlation was found between the maximal force in the elbow flexors of stroke patients and the deficit in the voluntary range of motion. Bohannon [5] argued that due to the force–length relationships in the muscles combined with an overall decrease of maximal force, motion is impaired at angles at which the agonists are more shortened or lengthened.

We directly measured active torque production in each subject throughout the physiological range of elbow angles. While some subjects had relatively normal torque–angle ranges (Fig. 6, left panel), in other subjects, the torque–angle ranges in the extensors only (middle panel) or in both the flexors and extensors (right panel) were impaired. Notably, for those subjects with flexor and extensor impairment, muscle strength was reduced asymmetrically over the physiological range. In contrast to the patterns of the distribution of maximal torque in healthy subjects, in the hemiparetic subjects, maximal torque in each muscle group occurred at a more extended muscle length (> 90° for the flexors and < 90° for the extensors; Fig. 5, lower panel). For the subjects with severe clinical muscle weakness, the area in which no torque was generated covered up to half of the physiological range.

The suggestion that there is an equal decrease in muscle force throughout the angular range of the elbow [5], cannot fully account for the changes in the general shapes of the torque–angle relationships observed in the stroke subjects in our study. In addition, Bohannon’s approach does not address the control mechanism by which motor function may be altered in stroke patients. The explanation of deficits in the regulations of SR thresholds may more adequately account for these alterations. Limitations in SR threshold regulation result in specific angular ranges in which agonist or antagonist muscle groups cannot relax. In Fig. 8, extensors are unable to relax in hatched and black zones to the left of R⊥ and flexors cannot relax in the light grey and white zones to the right of R⊥. In these zones, due to the presence of hypertonus in the antagonist muscle group, joint torque in the opposite direction may be actively generated but not without concomitant antagonist coactivation. In the white zones, movement could occur in only one direction (extension in far left zone and flexion in the far right zone). Muscles may be reciprocally activated and movement produced in both directions only in the black zones (range of R) defined by the SR thresholds for the flexors and extensors. Thus, in terms of limitations in the range of regulation of the control parameter, λ, patients with CNS lesions may have a more narrow range of attainable torque/angle combinations. This in turn may restrict the repertoire of limb configurations available to these patients when making multi-joint movements.

4.7. Motor deficits and the lesion location

Our subjects showed very similar limitations in the ranges of R and M in spite of differences in side, type and site of the lesion (Table 1). The lack of correlation be-
between the location of the brain lesion and the motor deficit is not surprising given that the majority of our subjects (9/12) had lesions involving sub-cortical structures (basal nuclei, thalamus and internal capsule). Although it has been shown that circumscribed capsular lesions may disrupt the output of well-defined anatomical motor areas (e.g., Ref. [3]), brain lesions following stroke are rarely distinct and isolated [23]. Indeed, in the clinical literature, there is no clear relationship between the anatomical site of the lesion and specific motor deficits [27]. This may be partially explained by the fact that lesions in the extremely compact internal capsule produce more widespread disability than lesions of comparable size in other regions of the brain [48]. Correlating specific motor deficits with precise lesions in man may only be possible if studies were done on groups of patients with small discrete lesions. However, the possibility that control of motor function is likely distributed throughout cortical and sub-cortical brain areas (see Ref. [42]) may well account for the similarity in motor deficits observed in our group of patients.

5. Conclusion

We have demonstrated that spasticity and motor function deficits in the arm of stroke patients may both be characterised by a similar mechanism: the limitation in the ability of the CNS to regulate the range of SR thresholds in flexor and extensor muscles. In healthy subjects, the range of regulation of \( \lambda \) is larger than the physiological range of joint angles. The full repertoire of available movement includes full muscle relaxation to full muscle activation in all parts of the physiological range. This ability to regulate muscle force in all parts of the physiological range may be lost in patients with CNS lesions because of a narrowing of the limits of regulation of SR thresholds.

The present results extend the applicability of the \( \lambda \) model to neurological deficits [44]. The model can account for: (1) the correlation between the static SR threshold and the level of clinically measured spasticity and residual motor function; (2) abnormal coactivation patterns observed in specific angular ranges and (3) the occurrence of ‘paradoxical’ movements (i.e., extension while attempting to flex or vice versa) near the extremes of the physiological range.

Our findings support the view that it may be inappropriate to consider spasticity separately from the motor control deficit. The correlations between the clinical spasticity and motor assessments with the measures of SR thresholds and ranges of active movement suggest an interdependency between these phenomena. Our data also support the suggestion that measures based on SR thresholds may be valid and reliable, objective measures of the degree of clinical impairment in stroke patients [35,41,50].

The explanation of the motor deficits in stroke patients using the \( \lambda \) model was based on the assumption that in healthy subjects, the regulation of SR thresholds may be a major mechanism of the control of movement. Our findings emphasise the significance of the individual and coordinated regulation of SR thresholds in producing basic motor patterns.

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