

# Muscle activation patterns of knee flexors and extensors during passive and active movement of the spastic lower limb in chronic stroke patients

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## Abstract

The aim of this study was to describe the characteristics of spasticity, quantified as muscle activity during stretch, during passive and active movement. For this cross sectional study 19 stroke patients with spasticity in the lower limb were recruited. Reflex activity was studied with surface electromyography of knee flexor and extensor muscles during passive and active movement of the lower leg.

On both the affected and unaffected side, root mean square values of the knee extensor muscles, while stretched, were higher during active than during passive movement ( $p < 0.05$ ). For the vastus lateralis (VL) the correlation was moderate ( $\rho = 0.54$ ,  $p = 0.022$ ), for the rectus femoris (RF) high ( $\rho = 0.83$ ,  $p < 0.001$ ). For the semitendinosus (ST) the correlation was low ( $\rho = 0.27$ ) and not significant.

During active movement the correlation between VL activity and activity of the antagonist ST, as an indicator for co-contraction of the affected muscles, was marked ( $\rho = 0.73$ ,  $p = 0.001$ ). A moderate negative correlation was found between reflex activity of RF during passive stretch and the active range of motion ( $\rho = -0.51$ ,  $p = 0.027$ ).

The results show that a passive stretch test alone is insufficient either as assessment method for spasticity during active motor tasks or as a measure for motor control.

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## 1. Introduction

Spasticity is a disorder which often develops after an upper motor neuron (UMN) lesion. Although prevalence figures vary between studies, it is estimated that 38–60% of patients surviving 12 months after stroke have spasticity (O'Dwyer et al., 1996; Watkins et al., 2002; Sommerfeld et al., 2004).

Spasticity is commonly described as a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes, resulting from hyperexcitability of the

stretch reflex (Lance, 1980 (1)). In this definition, the tonic stretch reflex is described as a response to an externally imposed passive stretch of relaxed muscle (Burke et al., 1970, 1971; Lance, 1980 (2)). Since the different positive signs after UMN lesions are often hard to discriminate in clinical practice, another definition was adopted for this study, as described by the support programme for assembly of database for spasticity measurement (SPASM) group. They defined spasticity as 'disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles' (Burrige et al., 2005; Pandyan et al., 2005). This definition includes all positive features of the UMN syndrome, like enhanced stretch reflexes, flexor and extensor spasms and clonus, all characterized by muscle overactivity. Pathological co-contraction, spastic dystonia and

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associated reactions (Sheean, 2002) can be added to this list as well. Whether the involuntary muscle activation is present during passive stretch or during active rotation about a joint, is left unspecified in this definition.

It is increasingly acknowledged that physical signs of spasticity, obtained during clinical examination, do not necessarily correspond with the functional impairment due to spasticity (Ibrahim et al., 1993; Dietz, 2003; Burne et al., 2005). The idea that spastic hemiparesis causes a movement disorder as a result of both the paretic and the spastic component is generally accepted (Levin et al., 2000). There are indications that stroke patients with spasticity are functionally more impaired than patients without spasticity (Kamper et al., 2001; Watkins et al., 2002; Francis et al., 2004). However, the exact relationship between the clinical phenomenon of spasticity, which is usually measured at rest, and the active motor disability remains unclear.

Knutsson et al. (1997) described that the weakness of voluntary knee movements in spastic paresis can be caused by different mechanisms. Besides the direct results of the paresis, spastic antagonistic muscles can produce exaggerated activity due to lack of reciprocal inhibition, resulting in dysfunctional co-contraction. Furthermore, diminished selectivity and the resulting activation of inappropriate muscles can disturb motor control. Secondary changes in biomechanical conditions of muscles and surrounding soft tissues will attribute to the movement limitation as well, in both passive and active muscles (Knutsson et al., 1997; Sinkjaer, 1997; Dietz, 2000).

Until recently, the majority of studies investigated electrical muscle activity during reflexes or during passive joint rotation (Voerman et al., 2005) rather than during more functional, active movements. In general, one of the difficulties of studying reflex activity during active movement is to differentiate it from voluntary muscle contraction. In a limited number of studies a comparison of polysynaptic stretch reflex activity between passive and active movement is presented, for both upper (Sahrmann and Norton, 1977; Ibrahim et al., 1993; Dietz, 2000; Burne et al., 2005) and lower limbs (Berger et al., 1984; Dietz et al., 1990, 1992, 2000, 2003; Ibrahim et al., 1993; Sinkjaer et al., 1993, 1999; Burne et al., 2005). Sahrmann and Norton (1977) found a moderate to marked correlation between stretch reflex activity of elbow flexors during passive stretch and the duration of an active motor task. Other authors indicated that EMG activity developing during passive stretch is responsible for the increased tone in spastic muscles, but that the development of spastic muscle tone during active contraction was influenced more by non-reflex stiffness (Ibrahim et al., 1993; Sinkjaer et al., 1993).

Findings of Dietz (2000) support that stretch reflex excitability and muscle tone are basically different in the passive compared to an active motor condition in both upper and lower extremities. It has been suggested that the modulation of reflex activity in the spastic limb becomes restricted to a smaller range (Berger et al., 1984;

Dietz, 1992, 2003; Sinkjaer et al., 1999) with a poor ability to switch off under passive conditions (Ibrahim et al., 1993; Burne et al., 2005).

In summary, the literature is still incoherent concerning differences in reflex activity between passive and active movements. It therefore remains uncertain what happens to reflex activity during simple motor tasks, when compared to reflex activity during similar but passive movements.

In this explorative study we compared muscle activity of spastic muscles during cyclic passive movement with comparable active movement of the lower leg, in order to assess the value of passive stretch tests in the measurement of spasticity. For this purpose we aimed: (1) to provide a qualitative and quantitative description of movement patterns and reflex activity of knee flexors and extensors during passive and active movement, and (2) to study the relationship between reflex activity during passively imposed movement and quality of the active movement.

## 2. Methods

This explorative study has a cross sectional design. The study received approval from the local medical ethics committee. Each subject signed an informed consent before participation.

### 2.1. Study population

Patients with spasticity in the lower limb following a unilateral cerebrovascular accident (CVA) were included if they were at least 6 months poststroke. Before inclusion, spasticity was assessed with the Ashworth scale (Ashworth, 1964) and scores of knee extensors and/or knee flexors should be  $\geq 1$ . In addition, patients had to be able to move the lower leg against gravity (Medical Research Council (MRC)  $\geq 3$ ) and understand simple commands. They were excluded if full hip or knee extension was not possible or if they had pain or other complaints in lower limbs.

Before testing, the passive range of motion of both hips and knees was assessed, as well as muscle length (slow Duncan–Ely test for the rectus femoris, popliteal angle for the hamstrings), to ensure that no structural contractures would interfere with the test results.

### 2.2. Procedure

Muscle activity was studied with surface electromyography (sEMG) of knee flexor and extensor muscles during passive and active movement of the lower leg. Movement patterns were assessed by goniometry of the knee joint.

Measurements were always performed by the same examiner. After placement of the sEMG sensors and the goniometer on both legs, the tests started on the unaffected side with passively imposed movement, followed by active movement. Subsequently, the same procedure was followed on the affected side. Before performing the tests each test was explained and the subject was allowed one practice session.

The subjects were in a comfortable sitting position with support for the back and lumbar region. During the passive movement test the lower leg of the subject was moved 10 times by the investigator, alternating from maximum extension to 90° flexion

of the knee. In order to approach the clinical setting as much as possible, it was chosen not to use an instrumented method to force the frequency of the movement. The frequency of the movement was standardized by moving the lower leg in a steady regular way at a pace that was least laborious for the investigator, which is similar to pendulum frequency. The subject had been instructed to relax and not to oppose or facilitate the movement of the swinging leg during these measurements. For the active movement test the subject was instructed to alternate 10 times between flexion and extension in a steady, regular manner in the same pace and over the same range of motion as the passive movement. All tests were performed three times with at least 10 min rest between sessions.

### 2.3. Instrumentation

The knee joint angle was measured with an electric goniometer (Biometrics Electro Goniometers, bi-axial), placed on the lateral sides of the knees. Surface EMG signals were obtained from the rectus femoris (RF), vastus lateralis (VL) and semitendinosus (ST) muscles, using electrode placement procedures according to the surface EMG for Non-invasive assessment of muscles – based protocol (Hermens et al., 2000). Bipolar, pre-gelled circular (diameter = 10 mm) electrodes (ARBO H93, solid gel) were used with an inter-electrode distance of 24 mm. A reference electrode was placed around the wrist.

EMG data were amplified (KL-100, Kinesiology Laboratories) and band pass filtered (third-order Butterworth; cut-off frequencies 20 Hz and 500 Hz) and sampled at 1000 Hz (12 bit analog to digital). The goniometer signal was low pass filtered with a cut-off frequency of 10 Hz. Software specifically developed for the analysis of muscle activation patterns during cyclic movements was used. Knee angle and sEMG signals were synchronized. Raw EMG-data were transformed to values of root mean square (RMS), related to the different phases (knee flexion and knee extension) of each cycle. The reverse points of the movement direction were set at an angle velocity of zero.

### 2.4. Outcome parameters

Two sets of parameters were used: one to describe the movement and the other to describe muscle activation patterns.

In order to describe movement characteristics the parameters duration and range of motion of the movement cycle were used. The cycle was divided into knee flexion and extension phases. The time taken for each phase was described in milliseconds, which is the duration of the flexion phase ( $D_{flex}$ ) or extension phase ( $D_{ext}$ ). The range of motion (ROM) represented the average knee angle range (in degrees) during the tests.

RMS values, calculated from EMG signals, were used to quantify the muscle activation patterns. It is a measure for the average amount of muscle activity during a period of time, in this case during the knee flexion or knee extension phase.

The interpretation of RMS values during the different phases depends on whether the muscle is stretched or shortened during a particular phase. For example, during knee flexion RF and VL are stretched and might show reflex activity, but no voluntary activity, when the subject is relaxed. For ST the opposite can be assumed: this muscle shortens during knee flexion and is stretched during knee extension.

### 2.5. Statistical analysis

The data were analyzed using statistical package for social sciences (SPSS, version 11.5) for Windows. For each subject the means of three measurements of both passive and active movement were used.

We compared muscle activation patterns during passive and active movements using the Wilcoxon signed ranks test, with a significance level of 0.05. RMS values during the stretch phase of each muscle are defined as stretch reflex activity. For comparison of the movement parameters we used the same non-parametric test.

Correlations were calculated between different parameters during passive and active movement using the Spearman's correlation coefficient.

For the relation between stretch reflex activity of a muscle during passive versus active movement the correlation coefficient was calculated for the average RMS values during the stretch phase of this muscle in both conditions.

To study co-contraction during active movement we calculated the correlation coefficient of the RMS value of the actively contracting agonist and the RMS value of the simultaneously stretched antagonist (Dewald et al., 1995).

To get insight in the influence of spasticity on motor control correlations were calculated between RMS values of stretched muscles during passive movement and movement characteristics during the same phase while actively moving.

Finally, the influence of paresis on motor control was estimated by calculation of the correlation coefficient between the RMS value of the agonist and the movement characteristics during the active task.

## 3. Results

### 3.1. Population

Twenty patients were recruited from the outpatient department of a rehabilitation centre. The results of one subject were excluded for further analysis, because this subject was unable to relax during the measurements.

Table 1 summarizes the baseline characteristics of all participating subjects.

Table 1  
Group characteristics

<i>N</i>	19
Mean age ± SD (yrs)	57.7 ± 12.3
Women ( <i>n</i> )	4
Right hemiparesis ( <i>n</i> )	6
Non-hemorrhagic ( <i>n</i> )	15
Mean months poststroke ± SD	32.7 ± 36.1
Median Ashworth score flexors (range)	1 (0–2)
Median Ashworth score extensors (range)	1 (0–3)

Note: Mentioned Ashworth scores are of the affected side. On unaffected side all Ashworth scores were '0'.

Abbreviation: SD, standard deviation.

### 3.2. Movement patterns and muscle activity during passive and active movement

#### 3.2.1. Qualitative observation of the data

During the passive movement test muscle activity was generally seen during the stretch phase of a muscle on the affected side, which was usually absent on the unaffected side. For instance, the stretched RF showed RMS values higher than  $5 \mu\text{V}$  in 14 of 19 subjects (74%) on the affected side, compared to only 2 subjects (11%) on the unaffected side.

During active movement remarkable differences in EMG activity were observed between the affected and unaffected side. Although all subjects were able to move their affected lower leg against gravity for the whole range of motion, which was a criterion for inclusion, many of them appeared to have problems with performing the movement repetitively. In most patients, the EMG activity of RF on the affected side persisted throughout active knee extension and even during knee flexion. Most patients showed inability to cease activity in the extensors after termination of knee extension, so that the leg returned to flexion very slowly. ST activity began at the end of extension, while the short burst often ceased shortly after flexion started.

Fig. 1 shows a representative example of muscle activation patterns during passive and active movement on the affected side of one of the subjects.

#### 3.2.2. Quantitative analysis of the data

Fig. 2 shows boxplots of the duration and range of motion of the cycle during passive and active movement, for both the affected and the unaffected side. The limited dispersion of the duration of passive movement cycles on both sides shows that standardization of the movement frequency was satisfactory.

On the affected side, we found that the differences in duration of flexion and extension between passive and active movement were not statistically significant ( $p = 0.064$  and  $0.198$ , respectively), although the dispersion of active movement data was much higher, with some outliers with extremely long duration of flexion and/or extension. On unaffected side these differences were smaller and not significant either.

The difference in average range of motion, however, was high (mean passive ROM  $76.0^\circ$ ; mean active ROM  $49.3^\circ$ ; mean difference  $26.7^\circ$ ) and significant ( $p < 0.001$ ). On the unaffected side this difference was significant as well ( $p = 0.002$ ) but much smaller (mean passive ROM  $85.1^\circ$ ; mean active ROM  $80.4^\circ$ ; mean difference  $4.7^\circ$ ).

Subsequently, RMS values were compared between passive and active movement (Table 2). As expected, the activity of any muscle during shortening differed significantly between passive and active movement, as during active movement the muscle contracts actively, while during passive knee rotation it is supposed to relax.

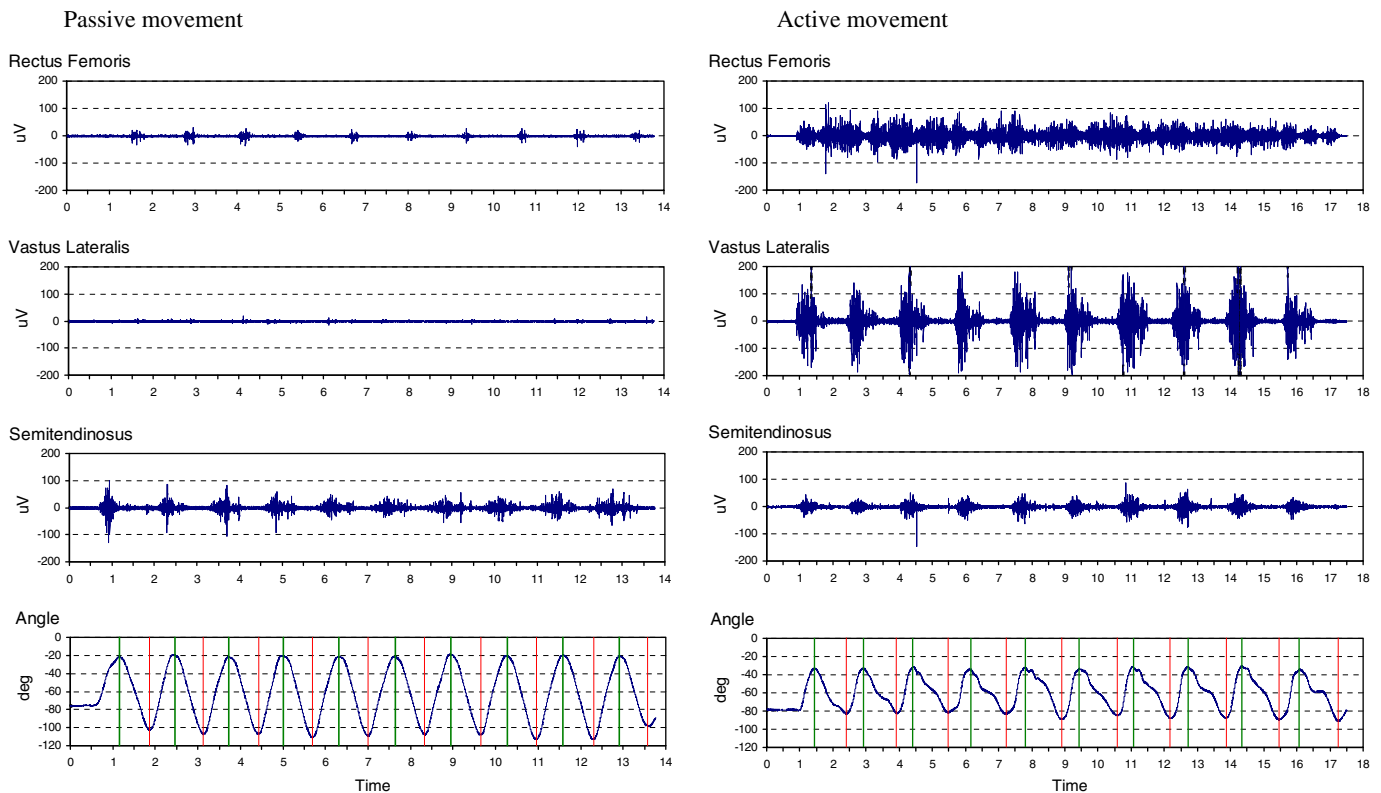


Fig. 1. Example of activation patterns during passive and active movement of a subjects' affected leg (muscle activity in  $\mu\text{V}$ , angle in degrees and time in seconds).

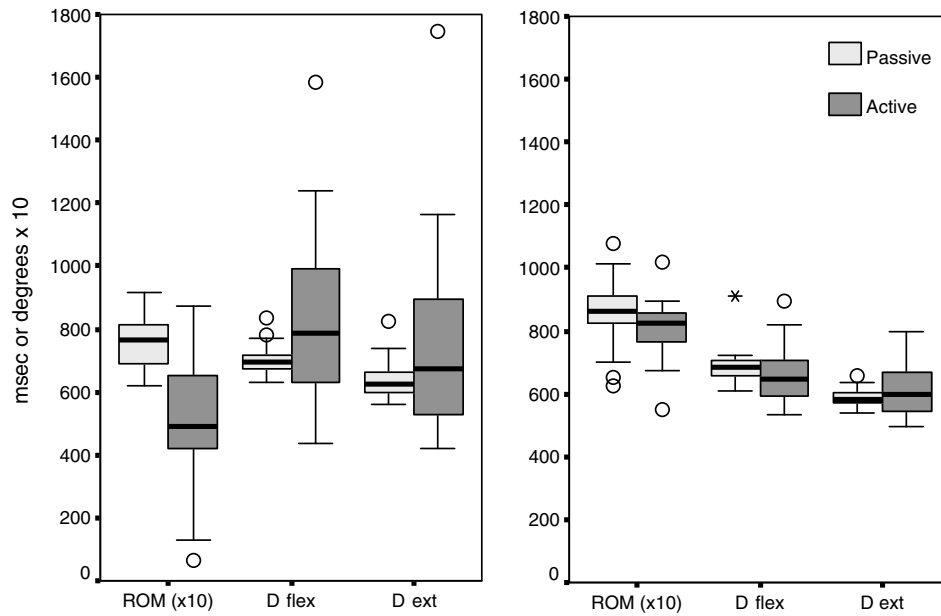


Fig. 2. Duration and range of motion of the movement cycle during passive and active movement on the affected (left) and unaffected side (right). Abbreviations: ROM, range of motion; D, duration; flex, during knee flexion phase; ext, during knee extension phase.

Table 2  
Comparison of muscle activity (in  $\mu\text{V}$ ) on the affected and unaffected side during passive versus active movement (Wilcoxon signed ranks test)

	Muscle	Passive, mean (SD)	Active, mean (SD)	<i>p</i>
<i>Affected side</i>				
During stretch	RF <sub>flex</sub>	12.8 (10.8)	24.0 (16.3)	0.001
	VL <sub>flex</sub>	5.5 (3.3)	20.8 (11.4)	<0.001
	ST <sub>ext</sub>	22.3 (14.5)	21.5 (16.1)	0.687
During shortening	RF <sub>ext</sub>	5.1 (4.5)	35.4 (21.5)	<0.001
	VL <sub>ext</sub>	3.6 (2.1)	45.5 (33.0)	<0.001
	ST <sub>flex</sub>	8.6 (4.9)	14.8 (14.5)	0.078
<i>Unaffected side</i>				
During stretch	RF <sub>flex</sub>	2.8 (1.9)	15.1 (15.0)	<0.001
	VL <sub>flex</sub>	3.7 (3.7)	20.6 (15.6)	<0.001
	ST <sub>ext</sub>	4.8 (2.8)	13.7 (7.1)	<0.001
During shortening	RF <sub>ext</sub>	3.9 (3.0)	42.4 (23.4)	<0.001
	VL <sub>ext</sub>	4.9 (4.0)	78.2 (35.6)	<0.001
	ST <sub>flex</sub>	6.4 (5.0)	12.8 (9.1)	0.001

Abbreviations: RF, rectus femoris; VL, vastus lateralis; ST, semitendinosus; flex, during knee flexion phase; ext, during knee extension phase; SD, standard deviation.

When comparing the stretch phases on the affected side, the mean RMS values of both knee extensors were higher during active movement than during passive movement ( $p \leq 0.001$ ). For ST no difference was found. On the unaffected side, muscle activity during stretch was found to be higher during active movement as well ( $p < 0.001$ ). RMS values during passive stretch were all below noise level.

Correlation coefficients between the muscle activity of a stretched muscle during active movement and the muscle activity of the same muscle during passive movement were calculated. The figures are presented in Table 3. For the

Table 3  
Correlation matrix of muscle activity in the stretch phase during passive versus active movement in the affected and unaffected limb (Spearman's rho)

		Passive		
		RF <sub>flex</sub>	VL <sub>flex</sub>	ST <sub>ext</sub>
<i>Affected side</i>				
Active	RF <sub>flex</sub>	0.825**		
	VL <sub>flex</sub>		0.536*	
	ST <sub>ext</sub>			0.267
<i>Unaffected side</i>				
Active	RF <sub>flex</sub>	-0.007		
	VL <sub>flex</sub>		0.068	
	ST <sub>ext</sub>			0.539*

Abbreviations: RF, rectus femoris; VL, vastus lateralis; ST, semitendinosus; flex, during knee flexion phase; ext, during knee extension phase.

\*  $p < 0.05$ .

\*\*  $p < 0.01$ .

knee extensors the correlation was moderate (VL  $\rho = 0.54$ ,  $p = 0.022$ ) to high (RF  $\rho = 0.83$ ,  $p < 0.001$ ). For ST the correlation was low ( $\rho = 0.27$ ) and not significant.

On the unaffected side, no relationship between reflex activity during passive and active movement was found for RF and VL. For ST the correlation was moderate ( $\rho = 0.54$ ,  $p = 0.017$ ).

### 3.2.3. Co-contraction during active movement of the lower leg

In Table 4 correlation coefficients are presented for agonist and antagonist activity during active movement. During active extension RF and VL are both contracting. The correlation between VL activity and activity of the antago-

Table 4  
Correlation matrix of muscle activity of agonists and antagonists during active movement on the affected side (Spearman's rho)

		Shortening phase (contraction)		
		RF <sub>ext</sub>	VL <sub>ext</sub>	ST <sub>flex</sub>
Stretch phase	RF <sub>flex</sub>			-0.39
	VL <sub>flex</sub>			0.21
	ST <sub>ext</sub>	0.33	0.73**	

Abbreviations: RF, rectus femoris; VL, vastus lateralis; ST, semitendinosus; flex, during knee flexion phase; ext, during knee extension phase.

\*\*  $p < 0.01$ .

nist ST was marked ( $\rho = 0.73$ ,  $p = 0.001$ ). Correlations of agonist activity of ST with the antagonists RF and VL during active knee flexion were low and not statistically significant. On the unaffected side, all correlation coefficients were neither relevant nor significant (not in the table).

### 3.2.4. Relation between stretch reflex activity and control of voluntary movement

To study the relationship between stretch reflex activity and the quality of voluntary movement, correlation coefficients were calculated between muscle activity during passive stretch and movement characteristics during active movement.

In Table 5 the correlation coefficients are presented. A moderate negative correlation ( $\rho = -0.51$ ,  $p = 0.027$ ) was found between stretch reflex activity of the RF during passive stretch and the active range of motion. The corresponding scatter plot is shown in Fig. 3. On the unaffected side no relevant or statistically significant correlations were found.

In order to get insight in the possible role of the paresis in control of the movement, we calculated correlations between RMS values during active contraction and active range of motion as well. For the duration of the flexion or extension movement, no relevant relationships were found. For the range of motion a moderate correlation was found with active contraction of VL ( $\rho = 0.57$ ;  $p = 0.012$ ) and ST ( $\rho = 0.47$ ;  $p = 0.047$ ), but not with RF activity ( $\rho = 0.22$ ;  $p = 0.371$ ). Again, no relevant or statistically significant correlations were found on the unaffected side.

Table 5  
Correlation matrix of stretch reflex activity during passive movement versus movement parameters during active movement (Spearman's rho)

		Passive		
		RF <sub>flex</sub>	VL <sub>flex</sub>	ST <sub>ext</sub>
Active	D <sub>flex</sub>	0.01	0.21	
	D <sub>ext</sub>			0.07
	ROM	-0.51*	-0.31	-0.30

Abbreviations: RF, rectus femoris; VL, vastus lateralis; ST, semitendinosus; flex, during knee flexion phase; ext, during knee extension phase; D, duration; ROM, range of motion.

\*  $p < 0.05$ .

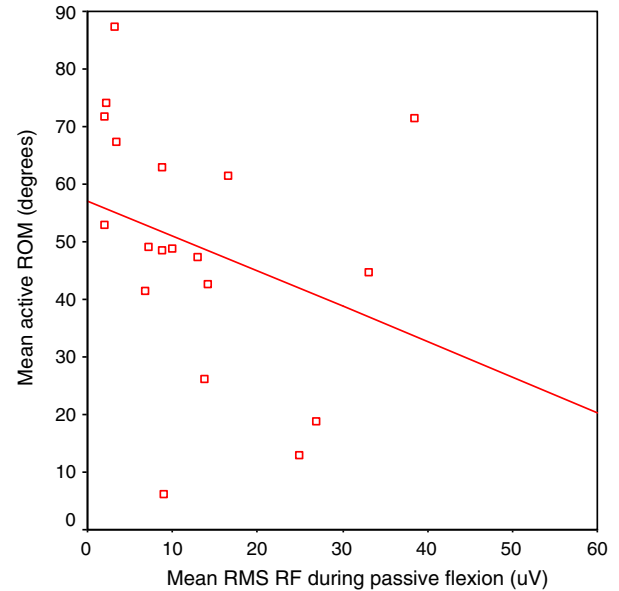


Fig. 3. Scatter plot of stretch reflex activity of RF during passive movement versus active range of motion of the knee on the affected side.

## 4. Discussion

The aim of this study was to get a better understanding of movement patterns and reflex activity of knee flexors and extensors during repetitive passive and active movements.

Our results support earlier studies, indicating that passive stretch and active movement elicit different manifestations of spasticity.

In the present study, we chose to use the pendulum or resonant frequency for passive movement of the lower leg, in order to approach the clinical setting in which the clinician moves the lower leg manually. This method appeared fairly easy to perform by the examiner and it resulted in consistent frequencies, as verified with the goniometric outcomes (see Fig. 2). Although patients were clearly instructed, active movement frequencies appeared considerably less controllable, as they were dependent on the physical abilities of each individual patient. To enable a valid comparison of muscle activity during passive and active movements, however, similar angular velocity is an important condition. Despite this, some interesting observations were made, particularly after including the movement characteristics as outcome values reflecting the quality of motor control.

A positive correlation was found between EMG activity of the stretched knee extensor muscles during passive and active movement, which indicates a parallel between the two conditions. Several reasons can be considered to explain this finding. Obviously, hyperexcitability of reflex activity, due to the cerebral lesion, is expected to result in relatively higher RMS values in both stretch conditions. Other individual factors affecting RMS values in general, like skin thickness, muscle cross-section etc., which are

constant in this intrasubject comparison, will contribute to a positive correlation as well.

In addition, we found some remarkable differences when we compared passive and active movements. We expected that, on average, the reflex activity would be reduced during active movement as a result of reciprocal inhibition of the antagonistic muscles. However, on the affected side the differences for RF and VL during the stretching phase appeared to be the opposite; these muscles showed higher muscle activity when stretched during active than during passive movement. At this point the lack of control of the time parameter must be considered. As the range of motion was smaller during active movement, without significant difference in cycle duration, consequently the velocity of stretch must have been lower. When we take this difference in velocity into account, less stretch reflex activity could have been expected during active movements. Yet the opposite was found, suggesting a considerably decreased reciprocal inhibition on the affected side. Sahrman and Norton (1977) encountered the same problem of variable range of motion during active elbow flexion. The authors solved the problem by normalizing the parameters of interest to a 120° range by a simple multiplication. A limitation of this method is the assumed linear relationship, which is probably not correct.

The relatively high electromyographic activity during the elongation phase in active movement was observed on both the affected and unaffected side. In this study we implicitly assumed the muscle activity during the elongation phase of a muscle to be stretch reflex activity. During active movement, however, it is likely to be contaminated by other factors. On the affected side, involuntary muscle activity during the stretch phase also involves muscle activity due to delayed relaxation of a contracted spastic muscle at the beginning of the stretch phase. In addition, early activity was seen at the end of the stretch phase, which seems to be anticipation on contraction. On the unaffected side, a similar overlap from contraction to relaxation phase was seen, but to a lesser extent (see Table 2). Since the RMS-values during passive stretch on this side were very low, the differences with active movement appeared relatively high.

Both phenomena are reflected in the electromyographic activity during stretch phase, but are not necessarily identical to stretch reflex activity. However, the delayed termination of the contraction can be regarded as one of the positive signs of the UMN syndrome, according to the definition of spasticity used for this study (Pandyan et al., 2005). A similar delay in termination of contraction was seen in the studies of Chae et al. (2002, 2006). The authors found a significantly prolonged delay in initiation and termination of voluntary muscle contraction in the paretic upper and lower limbs of chronic stroke subjects. In particular the delay in termination of the contraction correlated significantly with some functional tests. The authors brought up different

possible mechanisms, localized at different levels of the efferent pathways, varying from increased alpha motor neuron excitability to altered spinal and supraspinal mechanisms. Another explanation for this phenomenon might be the prolonged self-sustained firing in motor units, the so-called plateau potentials. Plateau potentials are sustained periods of depolarization that can amplify and prolong the effects of excitatory inputs, possibly due to changes in membrane properties of spinal motor neurons, as was studied in chronic SCI patients (Kiehn and Eken, 1997; Hornby et al., 2003). Anyhow, this delayed switching-off of the muscle underlines the complex relationship between spasticity and disordered motor control.

Additionally, pathological co-contraction might play a role during active movement as well. Chae et al. (2002) and Dewald et al. (1995) demonstrated a correlation between co-activation patterns of synergistic muscles in the paretic upper limbs, muscle weakness and functional outcome measures. In our study we found that on the affected side VL agonist activity correlated markedly with the antagonist activity of ST during active extension ( $\rho = 0.73$ ). An opposite association was not found, i.e. between ST agonist and VL antagonist activity. If this correlation represents a causal relationship, it might suggest that higher agonist activity elicited higher activity of the simultaneously stretched antagonist. On the unaffected side, no co-activation patterns were observed during this task. This finding fits into the concept of the extensor synergy pattern in the affected leg of stroke patients, with VL as one of the anti-gravity muscles (e.g. Berger et al., 1988; Ibrahim et al., 1993). An association was not found for RF, possibly because the biarticular RF does not have a prominent function during knee extension (Nene et al., 2004), but is active merely during the stance-to-swing transition in gait, acting as a hip flexor and on deceleration of excessive knee flexion (Perry, 1992; Nene et al., 2004). When observing the more or less continuous activity seen in the RF (see Fig. 1), which was often seen on the affected side, it can be considered dysfunctional eccentric contraction of this muscle during elongation. As it was not seen on the unaffected side, it may well be a result of disordered sensori-motor control.

Delayed termination of contraction, pathological co-contraction and the eccentric RF contraction, as described above, fit into the definition of spasticity that we used in this study, which encloses more than just stretch reflex activity.

For the assessment of the quality of the active movement the parameters cycle duration and range of motion were used. As all subjects were instructed to imitate the passively imposed movement in frequency and range of motion, deviations – in particular smaller range of motion and/or longer duration – were considered a consequence of poorer motor control. The fact that all subjects were able to perform the task properly on the unaffected side (see Fig. 2) confirms that they understood the task correctly.

We found a moderate negative correlation ( $\rho = -0.51$ ) between reflex activity of the RF during passive stretch and the active range of motion. On the unaffected side no relevant relationship was found. This finding shows that the amount of RF reflex activity during passive stretch is to some extent related to motor control.

However, presumably other factors intervene here as well. Although all subjects were able to move their lower leg against gravity over the whole range, the present paresis and other negative features will have contributed considerably to the poor motor control observed in this group. In particular increased fatigability might lead to deterioration during repeated performance. Provided that the subjects achieved maximal effort, the RMS values during active contraction of a muscle can be assumed a rough measure for paresis. A moderate positive association was found between RMS values during contraction of VL and ST and the active range of motion. Interestingly, no such relationship was found for RF, probably again due to its different function (Nene et al., 2004). It appears that this muscle is primarily impeding the movement by reflex activity rather than assisting in performing the requested task. The influence of the paresis was not taken into account in the study of Ibrahim et al. (1993), although conclusions were drawn concerning the actively contracting spastic muscle. The possible role of increased intrinsic muscle stiffness during active movement, however, cannot be addressed by our study.

Six of 19 subjects had right-sided hemiparesis (see Table 1). Fifteen subjects were diagnosed with an infarction, the others had cerebral hemorrhage. The extension of their lesion was not taken into account; both mild and severely affected patients were included. Nevertheless, all patients had spasticity and considerable paresis of the affected leg (MRC 3 or 4). It could be of interest to stratify data according to exact location or extension of the lesion, to investigate a possible relationship with different patterns of spasticity, equivalent to the distinction between cerebral and spinal spasticity. However, for this purpose our study population is too small.

Some limitations of this study should be considered. In the present study we chose a fixed order of tests to enable the patients to get used to the movements and the demanded tasks. Drawback of this procedure might be that the outcomes of the active movement test can be influenced by the earlier performed passive movement test (carry over effect). However, because the order of tests is relevant in this study to improve the comprehensibility of the active movement test, we accepted the possibility of systematic error, rather than introducing additional variability in the data.

Furthermore, we used the unaffected side as a reference to get insight in the clinical importance of the results found on the affected side. We assumed that changes on the unaffected side were not a result of spasticity. We realize that this assumption is not completely correct as pathological

changes on the ‘unaffected’ side can be found as well (Thilmann et al., 1990).

There are indications that reflex activity in spastic muscles in the lower extremities is comparable with that in upper limb muscles (e.g. Dietz, 2000). However, care needs to be taken with generalization of the described outcomes, as basic differences like synergic patterns and function might influence the general applicability.

## 5. Conclusion

Spastic upper leg muscles of stroke patients show remarkable differences in reflex behavior during passive movement compared with a similar active movement task. The amount of reflex activity in a muscle during passive stretch is related to the reflex activity during active movement. However, during voluntary movement other manifestations of spasticity are found to play a role as well. This study shows that the use of a passive stretch test alone is insufficient either as assessment method for spasticity during active motor tasks or as a measure for motor control.

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