# Assessment of spasticity

## From EMG to patients' perception

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# Contents

# 1

# General introduction

## Introduction

Spasticity is a common phenomenon which often develops after an upper motor neuron (UMN) lesion, such as stroke, multiple sclerosis or spinal cord injury. The prevalence of spasticity in the poststroke population is estimated at 38-60% of patients one year after stroke.[1,2,3] In a population with multiple sclerosis 84% reported spasticity.[4] Patients with spinal cord injury (SCI) also have a high probability to develop spasticity, up to 78% in a group with traumatic SCI.[5,6]

The clinical picture after an UMN lesion depends primarily upon its location and extent, and the time since it occurred, rather than on the pathogenesis of the lesion. In the acute phase after a lesion the so-called negative signs, such as paresis, fatigability and loss of dexterity, are usually most prominent. Muscle tone is initially flaccid with hyporeflexia. Spasticity is part of the positive phenomena, characterized by an exaggerated motor response, elicited for instance during physical examination. The interval between an acute lesion and the appearance of spasticity varies from days to months.[9]

In the field of Rehabilitation Medicine spasticity is an important topic. The decision whether or not to treat spasticity depends largely on its effect on the patient's functioning. Although some beneficial effects of spasticity have been reported,[5,8,10] it is more often associated with secondary negative consequences like pain, fatigue and deformities[3] and its overall impact on daily life seems to be negative.[11]

## Normal muscle tone

Early animal studies on the myotatic stretch reflex resulted in the model of an afferent-efferent neural circuit as the basis for understanding stretch reflex activity in humans.[12,13] Muscle spindles, small proprioceptive stretch receptors that lie in the muscle belly, have a key role in this process. They transmit information regarding muscle length and rate of change in muscle length. Depending on the velocity of

stretch, either dynamic (fast, powerful) or static (slower, longer) responses can be produced. When a muscle is stretched at high velocity, type 1a sensory fibres that surround specialized intrafusal muscle fibres within the muscle spindle are excited. The 1a fibres enter the cord via the posterior roots and make monosynaptic excitatory connections with alpha motor neurons of their muscle of origin. The 1a fibres also monosynaptically connect with inhibitory interneurons that project directly to the alpha motor neurons of antagonist muscles. Consequently, when the agonist muscle is excited antagonists are inhibited simultaneously; a mechanism which is called reciprocal inhibition (figure 1.1).

When the receptor portion of the spindle is stretched slowly, afferent terminals of type II fibres are stimulated. By changing their firing rate, they provide information on static length and position. Most type II afferents terminate on interneurons.

Two types of motor neurons originate from the anterior motor horn, alpha and gamma. A single alpha motor neuron innervates a varying number of muscle fibres; the whole entity is called motor unit. The smaller gamma motor neurons transmit impulses to intrafusal muscle fibres of the muscle spindle, thereby influencing the responsiveness of the spindle afferents by altering the continuous baseline discharge. This is referred to as the fusimotor system.[14]

Golgi tendon organs, located in the musculotendinous junction, detect changes in tension exerted by the muscle.[14,15] They supply feedback to the central nervous system via type 1b afferents. Together, muscle spindles and the Golgi tendon organs regulate muscle control and contraction, and therefore, muscle tone.

Interneurons are not simple relay stations in spinal reflex arcs, but receive a wide range of inputs from several different sources, both peripheral and supraspinal. As a consequence, spinal cord reflex responses are not stereotyped responses, but depend upon the ongoing activity in the surrounding interneurons.[12] Besides being involved in the mechanism of reciprocal inhibition, as described earlier, interneurons have a role in other types of signal processing as well. Specialized interneurons located in the anterior horns in close association with motor neurons, Renshaw cells, are excited by recurrent collateral branches of alpha motor neurons

before they exit from the spinal cord. Renshaw cells inhibit the alpha motor neuron and its synergists in order to limit and stabilize the discharge frequency (recurrent inhibition).

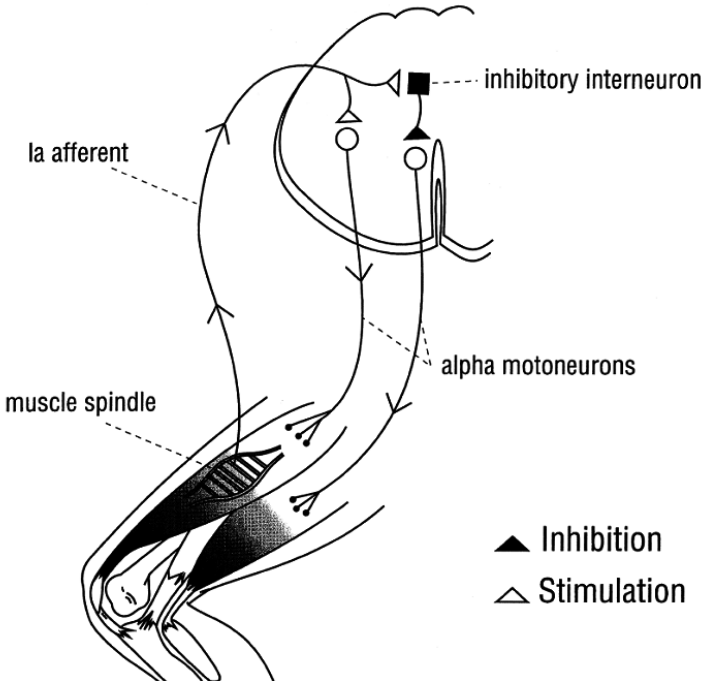


**Fig 1.1:** Monosynaptic stretch reflex arc and reciprocal inhibition (Mayer 1997)

Furthermore, inhibitory interneurons have presynaptic connections with 1a terminals and are under facilitatory supraspinal influences. Excitation of these interneurons reduces neurotransmitter release by 1a terminals on the alpha motor neurons, thereby maintaining a tonic inhibitory influence on the monosynaptic reflex arc, called presynaptic 1a inhibition. The 1b fibres, originating from Golgi tendon organs, also end on inhibitory interneurons. These in turn project to homonymous alpha motor neurons (nonreciprocal 1b inhibition). Reality is more complex, as the interneurons integrate afferent information of both 1a and 1b

afferents from a variety of muscles and each interneuron forms widespread inhibitory synapses with both homonymous and heteronymous alpha motor neurons.[12]

## Pathophysiology of spasticity

***Central neural changes***

The pathophysiological basis of spasticity is not completely understood. The changes in muscle tone probably result from an imbalance of inputs from central motor pathways, such as the cortico-reticulospinal and other descending pathways, to the interneuronal circuits of the spinal cord. The main tract that inhibits spinal reflex activity is the dorsal reticulospinal tract, which runs very close to the lateral corticospinal (pyramidal) tract.[16] It arises from the ventromedial reticular formation, which is under facilitatory control of cortical motor areas, thereby augmenting the inhibitory drive. The main excitatory pathway, also arising in the brainstem, is the medial reticulospinal tract.

Damage to these tracts gives rise to a net loss of inhibitory control, leading to increased alpha motor neuron excitability at the segmental cord level and subsequent increase in muscle tone.

***Peripheral neural changes***

Several studies claim that peripheral neural changes contribute to the increased muscle tone.[17] Direct changes in excitability of alpha motor neurons have not been demonstrated. However, denervation hypersensitivity of alpha motor neurons and collateral sprouting of excitatory afferents or interneuronal endings onto motor neuron membranes may be observed.[9,17] Another potential mechanism for alpha motor neuron hyperexcitability might be the self-sustained firing in motor units, the so-called plateau potentials. Plateau potentials are sustained periods of depolarization that can amplify and prolong motor output despite relatively short

or weak excitatory input.[18,19]

The theory that the fusimotor drive on muscle spindle afferents is increased, thereby increasing the muscle spindle sensitivity, has not been supported with direct evidence.[13,16] The role of fusimotor activity of gamma motor neurons in spastic muscle overactivity is still unclear. Enhanced spindle responses on a given amount of stretching force have been demonstrated, but primarily as a result of reduced compliance in stiffer muscles.

***Non-neural factors***

Early after UMN lesion, changes in mechanical, visco-elastic properties of muscle fibre and other soft tissues occur as a result of paresis and immobilization. Histological transformations in the muscles, such as muscle fibre atrophy and loss of sarcomeres, have been shown to contribute to muscle stiffness, leading to increased tension development and altered reflex sensitivity.[9,20,21] Accumulation of intramuscular connective tissue, increased fat content and degenerative changes at the musculotendinous junction cause reduced muscle compliance as well. Structural alterations in other soft tissues, including joint, ligaments, vessels and skin, also contribute to reduced range of motion.

In summary, spasticity is caused by net loss of supraspinal inhibition, i.e. decreased presynaptic inhibition on 1a afferents, decreased recurrent Renshaw cell inhibition, decreased Ib inhibition, and decreased reciprocal inhibition. In addition, peripheral mechanisms that have been shown to contribute to muscle overactivity include increased spindle stimulation by stiffer muscles and changes in contractile muscle properties. There is no direct evidence for alpha or gamma motor neuron hyperactivity, but evidence on the existence of plateau potentials in alpha motor neurons of spastic patients is growing.

## Definition of spasticity

The term spasticity is inconsistently defined in present medical literature.[22] Traditionally, spasticity was defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ('muscle tone') with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome", according to Lance (1980).[23] It thus focuses merely on enhanced stretch reflex activity, resulting from abnormal spinal processing of proprioceptive input.

In the clinical setting, the term 'spasticity' is frequently used in a broader sense. In addition to increased excitability of proprioceptive reflexes, several other reflex circuits, such as cutaneous and nociceptive reflexes, can also be affected by the disrupted supraspinal control.[16] Exaggerated responses originating from these afferents lead to distinct signs, which are generally included into the concept of spasticity as well. Because the various positive signs of the UMN syndrome are sometimes hard to differentiate in clinical practice, Lance's definition is often considered too narrow.[5,8,24-26] The SPASM (Support Programme for Assembly of database for Spasticity Measurement) consortium recently introduced an umbrella definition, which is increasingly being used. Spasticity was redefined as "disordered sensori-motor control, resulting from an UMN lesion, presenting as intermittent or sustained involuntary activation of muscles",[25] thereby including all afferent-mediated positive features of UMN syndrome.

## Measurement of spasticity

In patients with an UMN lesion, clinical problems of movement dysfunction arise from a complex interaction between positive features, negative features, and changes in the mechanical properties of muscles and other tissues. Therefore, careful assessment of all signs and symptoms that might contribute to impaired

motor function in the individual patient is essential in selecting the appropriate treatment.

Quantification of spasticity, in terms of 'Body Functions and Structures' within the framework of the International Classification of Functioning, Disability and Health (ICF),[27,28] requires reliable and valid measurement methods. Objective measurement of spasticity has therefore been a major goal for clinical researchers for many years.

To assess spasticity clinical, biomechanical and neurophysiological approaches have been used. Clinical scales for the assessment of spasticity mainly concentrate on resistance to passive movement.[29] Many of them are single item scales that can be used in different circumstances, that is, different joints and different underlying diseases. The Ashworth scale[30] or its' modified version[31] are the most commonly used clinical measurement methods for the assessment of tone. The Ashworth scale measures the resistance perceived by the rater when passively rotating a joint, which is scored on an ordinal scale from 0 to 4. The perceived resistance to passive movement is a sum total of neural stretch reflex activity and non-neural visco-elastic properties of joint structures and soft tissues.[32-34] The Spasm Frequency scale and Clonus score are examples of assessment methods for other manifestations of spasticity.[24] All these scales have in common that they are subjective, as they depend on the perception of the examiner or patient, that differentiation between neural and non-neural contributions is not possible and that the methodological qualities of the scales are doubtful.

In laboratory settings biomechanical or neurophysiological measurement methods can be used, assessing either the resistance to imposed passive movement or the electrical activity of the involved muscles. Use of the Hoffmann reflex, the Tendon reflex and the short latency Stretch reflex for the assessment of spasticity have been studied extensively,[35] but their clinical relevance seems limited. Using electromyography (EMG) with surface electrodes for assessment of (reflex) muscle activity during functional active or passive movements has shown to be a valuable method, when adequately standardized.[36] An obvious limitation of the single use

of a biomechanical approach, for example with a hand-held dynamometer, is the inability to distinguish between neural and non-neural components of spasticity.[37] Therefore a combination of the two is recommended.[38]

In current clinical practice several difficulties in spasticity assessment are encountered. First of all, it is increasingly acknowledged that the most commonly used clinical assessment methods, such as the Ashworth scale,[30] have considerable methodological limitations.[29,39,40]

Secondly, it becomes gradually more recognized that physical signs of spasticity, obtained during clinical examination, do not necessarily correspond with the functional impairment due to spasticity.[9,41,42] Although it is generally assumed that patients with spasticity are functionally more impaired than patients without spasticity,[2,44,45] there is inconsistency on this topic in medical literature (e.g. [38,46,47]). Hence the exact relationship between the clinical phenomenon of spasticity and the active motor disability remains unclear so far.

Furthermore, methods are needed that are closer to the patients' perception, because in decision making for optimal treatment the patients' perception plays an important role. Awareness of the patients' perception of spasticity and of treatment effect offers several advantages. It can help clinicians to better understand the patients' expectations and satisfaction of the received treatment. In addition, the opportunity for a patient to provide feedback about his perception of treatment success might enhance the patient's compliance with his treatment regimen.[28] In current practice, the patients' evaluation of spasticity is often an ad hoc report and is rarely documented by using measurement tools.[29,48] In addition, usually no explicit differentiation is made between the perceived degree of spasticity and the experienced spasticity-related discomfort, although the decision whether or not to treat spasticity depends mainly on its impact on a patient's daily functioning.

Finally, both objective and subjective assessments are commonly performed at one specific moment in time, thereby ignoring fluctuations of spasticity over the day due to personal and environmental factors.[28,35,37,38] Momentary assessment is thus

likely to be limitedly representative for spasticity experienced in normal daily life. An assessment method, coping with this shortcoming, can be useful, particularly in more complicated cases.

In summary, assessment of spasticity is complex due to its various manifestations, difficulties to distinguish between neural and non-neural components, and different characteristics during passive and active, more functional movements. Additionally, there can be a discrepancy between outcomes of objective tests and the patients' perception and, finally, a single momentary assessment may be erratic.
Consensus is growing that we need to measure spasticity at different levels,[38,49] covering the different manifestations of spasticity and representing spasticity at the different levels of the ICF framework.

## Objectives and outline of the thesis

The focus of this thesis was on the assessment of spasticity, with the aim to contribute to the development of a comprehensive set of clinically applicable measurement tools for spasticity, to support clinical decision making.

The first study, described in **chapter 2**, investigated the influence of posture and muscle length on clinical and neurophysiological measurement of spasticity in post-stroke patients. Stretch reflex activity was studied in stroke subjects with known spasticity, using the Ashworth scale, the pendulum test and passively imposed movement on the lower limbs in both sitting and supine position. Muscle activity was assessed non-invasively with surface EMG. Specific focus was on the quadriceps muscle, as in existing literature findings on length-dependency of spasticity in this muscle are contradictory.
**Chapter 3** focuses on the association between spasticity measured with passive

stretch tests and spasticity during active motor tasks. In poststroke patients, reflex activity of spastic upper leg muscles during cyclic passive movement was compared with reflex muscle activation during similar active movement of the lower limb.

The Ashworth scale is subject of investigation in **chapter 4**, in which the clinimetric properties of the scale for the measurement of spasticity are described. Although several studies about the methodological qualities of the (modified) Ashworth scale have been performed, this is the first study investigating both construct validity and inter-rater reliability of the Ashworth scale, using real-time sEMG and dynamometry recordings.

**Chapter 5** addresses the association between the subjectively perceived degree of spasticity and the experienced discomfort as a result of spasticity. It was studied in motor complete SCI patients by using a questionnaire that focused on the individual perception and description of spasticity in the lower limbs during daily life activities.

**Chapter 6** describes the relationship between patient ratings on the level of spasticity, measured with the Visual Analogue Scale, and objective spasticity measurement, using long-term sEMG recordings during daily activities, in motor complete SCI patients.

The aim of the study, described in **chapter 7**, was to quantify involuntary muscle activity patterns in the lower limbs of patients with motor complete SCI, using sEMG recordings during daily life activities. Analysis focused on the influence of daily activities on muscle activity and co-activation patterns.

The thesis is concluding with a general discussion in **chapter 8**, in which the findings of the different studies are discussed and integrated. Implications for clinical practice are presented and suggestions for further research are proposed.

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# 2

# INFLUENCE OF POSTURE AND MUSCLE LENGTH ON STRETCH REFLEX ACTIVITY IN POSTSTROKE PATIENTS WITH SPASTICITY

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*Arch Phys Med Rehabil. 2006 Jul;87(7):981-8*

## Abstract

The aim of this study was to investigate the influence of different positions on stretch reflex activity of knee flexors and extensors measured by surface electromyography in poststroke patients with spasticity and its expression in the Ashworth scale. Nineteen poststroke patients with lower-limb spasticity participated in this crossover trial, during which stretch reflex activity was assessed in both sitting and supine position, in randomized order. Main outcome measures were root mean square (RMS) values of muscle activity and goniometric parameters, obtained during the pendulum test and passive knee flexion and extension, and Ashworth scores.

Results showed that RMS values of bursts of rectus femoris activity were significantly higher in the supine compared with the sitting position ($p = 0.006$). The first burst of vastus lateralis activity during the pendulum test ($p = 0.049$) and semitendinosus activity during passive stretch ($p = 0.017$) were both significantly higher in the supine versus the sitting position. For both the pendulum test and passive movement test, the duration and amplitude of the cyclic movement of the lower leg changed significantly as well. In the supine position, we found significantly higher Ashworth scores for the extensors ($p = 0.001$) and lower scores for the flexors ($p = 0.002$).

It was concluded that the outcomes of both clinical and neurophysiological assessment of spasticity are influenced considerably by the positioning of the subject.

## Introduction

Spasticity occurs in 38% to 60% of patients surviving 12 months after stroke,[1] although prevalence figures vary between studies.[2,3] Functionally, patients with spasticity are significantly more impaired than patients without spasticity.[1]

Lance[4] defined spasticity as a motor disorder characterized by a velocity-dependent increase in muscle tone in response to stretching relaxed muscle. Recently, the Support Programme for Assembly of Database for Spasticity Measurement project redefined spasticity as "disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles".[5,6] This definition includes all the positive features of the upper motor neuron (UMN) syndrome, but excludes the negative features and the biomechanical changes in the joints and soft tissues.

Objective measurement is relevant for the indication for and evaluation of treatment of spasticity. In clinical situations, however, the assessment is very poorly standardized, and therefore its value for fine-tuning an intervention is limited. The Ashworth scale, in terms of assessment of resistance to passive movement, is the most common clinical measure for spasticity. The limited research concerning clinimetric properties of this scale shows that intra-rater and inter-rater reliability as well as test-retest reliability are moderate.[7,8] A lack of standardization during scoring might have contributed to these results. In the original description of the Ashworth scale,[9] instruction for positioning of the patient is not included. In practice, clinicians usually keep patients lying on a bed or sitting in a wheelchair for practical reasons.

Because spasticity is known to be length dependent, the positioning of subjects during testing is likely to influence the results of the spasticity assessment, particularly when bi-articular muscles are involved. Different researchers[10-17] have stated that in larger muscle groups increasing length of the muscle augments the stretch reflex activity. However, in the case of quadriceps muscle, a study by Burke et al.[13] showed that muscle lengthening seems to have an inhibitory effect.

In the trials reported in the literature,[13,14,16] subjects' positions vary greatly during the pendulum test and passive movement tests of the lower extremities, with some undertaking the tests with subjects supine and others having subjects sitting upright or in intermediate positions.

Only a few articles compare findings in two positions. Vodovnik et al[18] found that in hemiparetic patients a change in body position from sitting to supine increased the spastic state during the pendulum test, with more electromyographical activity in the quadriceps and changes in the goniogram. He[19] described similar findings in 59 patients with multiple sclerosis (MS). Kakebeeke et al[20] compared the elicited torques in the hamstrings and quadriceps muscles in the supine and sitting positions during passive movement in 20 patients with spinal cord injury with a complete motor lesion. For both knee flexors and extensors the torque was higher in the lengthened compared with the more shortened muscles.

Studies[21,22] involving the ankle and upper limb muscles have shown similar dependence of reflex response on joint position and muscle length. Even in people without neurological disorder, muscle lengthening has led to an increased reflex response in the preactivated gastrocnemius,[23-25] possibly because of changes in intrinsic muscle characteristics.

The contradicting findings in the literature about the influence of muscle length on stretch reflex activity, especially in the quadriceps muscle, raise two questions. The first is whether and how the stretch reflex in the quadriceps and hamstring muscles are influenced by the muscle length. Second, what is the consequence of positioning during clinical assessment of spasticity in patients with spastic hemiplegia?

We studied stretch reflex activity in stroke subjects with known spasticity, in both the sitting and supine positions, using the Ashworth scale, the pendulum test, and passively imposed movement on the lower extremities.

The aim of this study was to investigate the influence of the change in positioning on stretch reflex activity of the rectus femoris, vastus lateralis, and semitendinosus muscles on the affected and nonaffected sides as measured by surface

electromyography. A second aim was to assess whether the possible variability in stretch reflex activity in different positions is also expressed in a change in Ashworth score.

We hypothesized that the stretch reflex of the rectus femoris is elicited more strongly in the supine position when the muscle is elongated, compared with the sitting position. For the semitendinosus muscle, we expected the opposite: that is, more stretch reflex activity in the sitting position. The stretch reflex of the vastus lateralis was not expected to be influenced by changing the hip angle, because the length of this monoarticular muscle does not change. Finally, we expected that possible differences in electromyographical activity in the two positions during passive movement would not (or not to the same extent) be discriminated by the Ashworth scale.

## Methods

This explorative study was a crossover randomized trial in which the order of positioning was randomized for all patients. Randomization was performed mainly because of the occurrence of fatigue in repeated stretching of a spastic muscle.[26,27] The study received ethics approval from the medical ethics committee of Rehabilitation Centre Het Roessingh, in Enschede, The Netherlands.

### *Study population*

Patients with spasticity in the lower limb after a unilateral cerebrovascular accident were included if they were at least 6 months poststroke. In addition, they had to be able to move the lower leg against gravity and understand simple commands. Patients were excluded if full hip or knee extension was not possible, if they had pain or other complaints in the lower limbs or a history of (soft tissue) surgery on the lower limbs.

***Procedure***

Stretch reflex activity was studied clinically by the Ashworth scale and neurophysiologically during the pendulum test and passive movement of the lower leg. All 3 tests were performed in the supine and sitting positions, in random order. We divided the study population into two groups (A, supine-sitting; B, sitting-supine). Block randomization was performed by tossing a coin.

We chose for a fixed order of tests, starting on the unaffected side, to enable the patients to get used to the movements and the demanded tasks (appendix 1). Before performing the tests each test was explained and tried once.

Measurements were always performed by the same examiners. Initially the passive range of motion (ROM) of both hips and knees was assessed, as was 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.

In the supine position, each subject laid on the bed with a small pillow under the head and, if necessary, support under the back. The lower legs were hanging over the edge and could move freely. In the sitting position, each subject was in a comfortable upright position with hips ±90° flexed and with support for the back and lumbar region.

The Ashworth score was assessed by an experienced physiotherapist, blinded to the objective of the study or test results. The score was assessed for both knee flexors and extensors in the 2 described subject positions. No other instructions were given so as not to influence the therapist and thereby to approximate a typical clinical situation as much as possible.

Neurophysiological measurements consisted of the pendulum test and the passive movement test. For the pendulum test, the lower leg of each subject was held in full knee extension and released. During the passive movement test the lower leg of each subject was moved 10 times by the investigator, alternating from full extension to 90° of knee flexion. The lower leg was rotated in a steady regular way at a pace that was least laborious for the investigator, which is similar to pendulum or resonant frequency. Each subject had been instructed to relax his/her leg

and not to oppose or facilitate the movement of the swinging leg during these measurements. The pendulum and passive movement tests all were performed 3 times.



***Instrumentation***

The knee joint angle was measured with a biaxial electric goniometer (Biometrics Electro Goniometers; Biometrics Ltd, Gwent, United Kingdom), placed on the lateral side of the knee. Surface electromyographical signals were obtained from the rectus femoris, vastus lateralis, and semitendinosus muscles, using electrode placement procedures according to the Surface EMG for Non-Invasive Assessment of Muscles–based protocol.[28] Bipolar, pregelled circular (diameter, 10 mm; solid gel) electrodes (ARBO H93; Tyco healthcare, Zaltbommel, The Netherlands) were used with an interelectrode distance of 24 mm. A reference electrode was placed around the wrist.

Electromyographical data were amplified (KL-100; Kinesiologic Laboratories, Haarlem, The Netherlands), band-pass filtered (third-order Butterworth; cutoff frequencies, 20 Hz, 500 Hz) and sampled at 1000 Hz (12-bit analog to digital). The goniometer signal was low-pass filtered with a cutoff frequency of 10 Hz. We used software specifically developed for analysis of muscle activation patterns during the pendulum test and passive movement. Knee angle and surface electromyographical signals were synchronized. Raw electromyographical data were transformed to values of root mean square (RMS), related to the different phases (knee flexion, knee extension) of each cycle. In addition, an algorithm (the approximated generalized likelihood ratio) was used to determine the start and end of bursts in the electromyographical signals.[29]

***Outcome parameters***

We used two groups of parameters to get insight in the movement and muscle activation patterns.

The parameters describing the movement were derived from the goniometric signal

and divided the cycle into a flexion and extension phase. The duration reflects the time necessary for knee flexion (first half of the cycle) and knee extension (second half of the cycle). The amplitude of the cycle represents the ROM during the tests. These parameters are primarily relevant for the pendulum test, because changes in these parameters indicate a different degree of resistance against movement. For the passive movement test they are merely a verification of how accurately the test has been performed.

In the pendulum test the duration and amplitude of the first flexion phase decrease when more spasticity in the knee extensors is present.[30] The relaxation index (RI) is a frequently used ratio for the pendulum test, derived from the knee angle. It is defined as the ratio between the angle of the first drop and the initial angle (with the resting angle as 0°).[30] In healthy subjects, the relaxation index is found to be 1.6 or more. Lower scores represent spasticity.

We used RMS values derived from electromyographical signals to describe muscle activation patterns. This is a measure of the amount of muscle activity during a period of time (e.g. flexion phase, extension phase, during a burst of muscle activity).

The parameters for the pendulum test all were based on the first cycle (figs 2.1, 2.2): the duration of the first knee flexion ($D_{flex}$) and extension movement ($D_{ext}$), cycle amplitude of flexion ($A_{flex}$) and extension ($A_{ext}$) and the relaxation index. Furthermore, for each muscle RMS during flexion ($RMS_{flex}$) and extension ($RMS_{ext}$) were assessed, as was RMS of the first burst, if present ($RMS_{burst}$).

For the passive movement test, similar parameters were used as for the pendulum test, but averages of 10 cycles were calculated: Average duration of knee flexion and extension, average cycle amplitude of flexion and extension, average RMS during knee flexion and extension, and average RMS during burst activity, if present, for each muscle.

The parameters for muscle activity during knee flexion and knee extension have different significance for the antagonizing muscles: during knee flexion, the rectus femoris elongates and might show stretch reflex activity, but no voluntary activity

(besides co-contraction or when a subject is unable to relax). During knee extension, the rectus femoris shortens; we do not expect stretch reflex activity here, so the muscle activity we find in this phase is defined as active muscle contraction. For the semitendinosus muscle, the opposite is assumed.

The Ashworth scale was scored according to the original scale (range 0 – 4).[9]

***Statistical analysis***

The data were analyzed using Statistical Package for Social Sciences Version 11.5 (SPSS Inc, Chicago, United States) for Windows. We compared data from the sitting position with that from the supine position using the paired *t* test or Wilcoxon signed-rank test (depending on the distribution of the differences), with a significance level of .05. For the pendulum and passive movement test the means of 3 measurements were used for each subject.

To provide criteria for what might be normative changes not directly related to pathologic muscle activation, we also measured the unaffected side. To investigate the importance of the differences found on the affected leg, we compared these outcomes with the results on unaffected side. We used a linear mixed model with 2 factors (position, affected and nonaffected side), to compare the effect of changing position for the affected and unaffected sides. To determine whether an interaction between position and order of positioning (carryover effect) was present, a 3-factor analysis was performed with the group (A, B) as the third factor.

Furthermore, we calculated correlations between the Ashworth score and electromyographical parameters of knee flexor and extensor muscles with the Spearman correlation coefficient. We compared the Ashworth scores for flexors and extensors with RMS values of these muscles during stretching and during a burst of activity, in both positions.

# Results

Twenty patients were recruited from the outpatient Department of Rehabilitation Medicine. All patients were informed about the purpose of the study and gave informed consent. The results of one subject in group A were excluded for further analysis, because the subject appeared unable to relax during all the measurements.

Table 2.1 summarizes the baseline characteristics of groups A and B. The difference in mean age between the two groups was significant (Mann-Whitney *U* test, $p = 0.04$).

**Table 2.1:** Group characteristics

| ***Characteristics*** | ***Group A*** (supine - sitting) | ***Group B*** (sitting - supine) |
|---|---|---|
| n | 9 | 10 |
| Mean age ± SD (y) | 51.4 ± 12.4 | 63.4 ± 9.6 |
| Women (%) | 33.3 | 10.0 |
| Right hemiparesis (%) | 33.3 | 30.0 |
| Nonhemorrhagic (%) | 77.8 | 80.0 |
| Mean months poststroke ± SD | 38.9 ± 46.7 | 27.1 ± 24.5 |

*Abbreviation: SD, standard deviation.*

### *Pendulum test*

Figures 2.1 and 2.2 show the results of the pendulum test of one subject in the two positions. The differences in stretch reflex activity and the goniometric pattern can be observed of the affected leg. In this typical example, one can observe considerable stretch reflex activity in the rectus femoris and little continuous activity in the semitendinosus muscle.

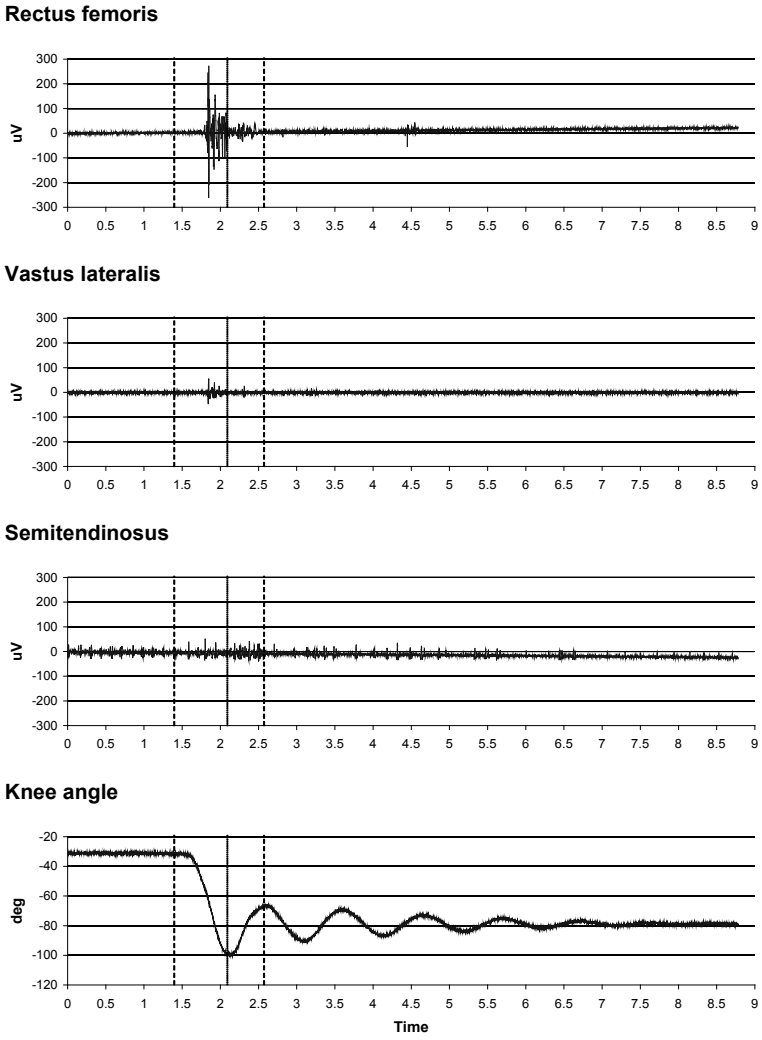


**Fig 2.1:** Example of the pendulum test on the affected side, in the sitting position. The Ashworth score was 1 for the extensors and 2 for the flexors

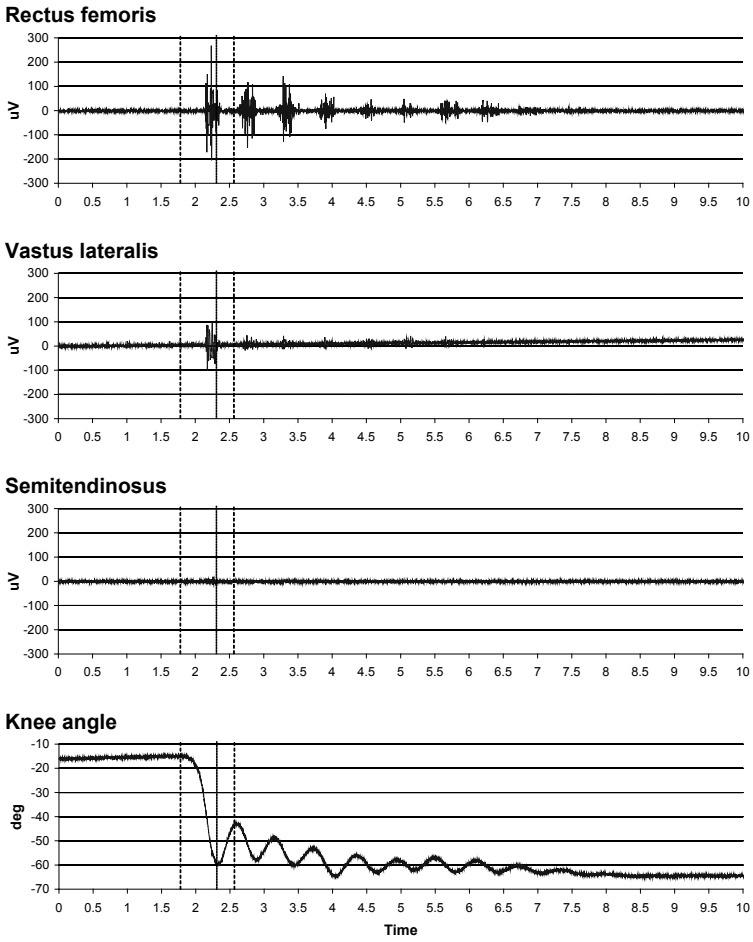


**Fig 2.2:** The pendulum test on the affected side, in the supine position (same subject as in fig 2.1). The Ashworth score was 3 for the extensors and 1 for the flexors

Table 2.2 summarizes the results of the pendulum test. Durations of the first knee flexion and extension were lower in the supine position (mean difference for flexion, 125.6 ms; $p < 0.001$; mean difference for extension, 65.7 ms; $p = 0.004$). The amplitude of the both halves of the cycle decreased as well (12.1°, $p < 0.001$; 7.1°, $p = 0.026$). The relaxation index was also lower in the supine position ($p = 0.001$).

The changes in RMS of the rectus femoris during knee flexion in the first cycle

(mean difference, –1.7 μV) were not statistically significant ($p = 0.145$). The same accounts for the RMS of the semitendinosus during extension ($p = 0.296$). However, the RMS values of the first burst in the rectus femoris and vastus lateralis were both significantly higher in the supine position (rectus femoris, $p = 0.006$; vastus lateralis, $p = 0.049$). Although the RMS of the burst in the semitendinosus was higher in the sitting position compared with the supine, this difference was not statistically significant ($p = 0.670$).

**Table 2.2:** Means of the parameters of the pendulum test on the affected side in two positions

| ***Parameter*** | ***Sitting*** | ***Supine*** | ***Mean Difference*** (95% CI) | ***p*** |
|---|---|---|---|---|
| $D_{flex}$ (ms) | 737.4 | 611.8 | 125.6 (69.9–181.3) | < 0.001 |
| $D_{ext}$ (ms) | 453.4 | 387.7 | 65.7 (24.3–107.1) | 0.004 |
| $A_{flex}$ (deg) | 69.8 | 57.7 | 12.1 (6.9–17.2) | < 0.001 |
| $A_{ext}$ (deg) | 38.0 | 31.0 | 7.1 (0.9–13.2) | 0.026 |
| Relaxation index | 1.7 | 1.4 | 0.3 (0.1–0.4) | 0.001 |
| $RMS(RF)_{flex}$ (μV) | 16.7 | 18.3 | –1.7 (–4.0 to 0.6) | 0.145 |
| $RMS(RF)_{ext}$ (μV) | 3.9 | 5.9 | –1.9 (–4.5 to 0.6) | 0.094† |
| $RMS(RF)_{burst}$ (μV) | 25.8 | 30.6 | –4.8 (–7.9 5 to –1.6) | 0.006 |
| $RMS(VL)_{flex}$ (μV) | 9.3 | 11.2 | –1.8 (–4.9 to 1.3) | 0.229 |
| $RMS(VL)_{ext}$ (μV) | 2.9 | 4.0 | –1.1 (–3.1 to 0.9) | 0.252 |
| $RMS(VL)_{burst}$ (μV) | 15.8 | 21.6 | –5.8 (–11.6 to –0.04) | 0.049 |
| $RMS(ST)_{flex}$ (μV) | 6.3 | 4.9 | 1.3 (–0.7 to 3.4) | 0.189 |
| $RMS(ST)_{ext}$ (μV) | 7.1 | 5.1 | 2.0 (–2.0 to 6.0) | 0.296 |
| $RMS(ST)_{burst}$ (μV) | 16.1 | 13.8 | 2.2 (–10.0 to 14.5) | 0.670 |

*Abbreviations: A, amplitude of movement; burst, during burst activity; CI, confidence interval; D, duration; ext, extension; flex, flexion; RF, rectus femoris; ST, semitendinosus; VL, vastus lateralis.*
*NOTE. p values are tested parametrically, unless mentioned.*
*† Wilcoxon signed-ranks test.*

Table 2.3 summarizes comparisons of the affected with the unaffected side. The differences in the parameters derived from the pendulum test, due to change of position, are presented for both the affected and unaffected sides. The *p* values indicate whether the effect of changing position differs for the affected compared with the unaffected side. Only parameters that show statistically significant

differences between the sitting and supine positions on the affected side (see table 2.2) are presented. It is necessary to mention that we found an interaction between the order of positioning and the effect of position (carryover effect) for the parameters cycle amplitude $A_{flex}$ ($p = 0.016$) and $A_{ext}$ ($p = 0.010$). When we analyzed the groups separately for these two parameters, the effect of changing position on cycle amplitude was stronger in group A (supine-sitting) than in group B (sitting-supine). For clarity of the presentation we have used the combined figures.

**Table 2.3:** Means of differences of the pendulum test on affected and unaffected side, compared by a 2-factor analysis of variance

| ***Parameter*** | $\Delta_A$ (95% CI) | $\Delta_{NA}$ (95% CI) | ***Interaction* (p)*** |
|---|---|---|---|
| $D_{flex}$ (ms) | 125.6 (69.9 - 181.3) | 19.7 (–12.3 - 51.8) | 0.002 |
| $D_{ext}$ (ms) | 65.7 (24.3 - 107.1) | 10.9 (–15.3 - 37.1) | 0.056 |
| $A_{flex}$ (deg) | 12.1 (6.9 - 17.2) | 2.0 (–4.5 - 8.6) | 0.042 |
| $A_{ext}$ (deg) | 7.1 (0.9 - 13.2) | –0.5 (–7.7 - 6.7) | 0.187 |
| Relaxation index | 0.3 (0.1 - 0.4) | –0.2 (–0.5 - 0.01) | 0.005 |
| $RMS(RF)_{burst}$ (μV) | –4.8 (–7.9 - –1.6) | –2.2 (–9.9 - 5.4) | 0.201† |
| $RMS(VL)_{burst}$ (μV) | –5.8 (–11.6 - –0.04) | –2.0 (–7.2 - 3.2) | 0.909† |

*Abbreviations: $\Delta_A$, mean difference (sitting - supine) on affected side; $\Delta_{NA}$, mean difference (sitting - supine) on nonaffected side; A, amplitude of movement; burst, during burst activity; CI, confidence interval; D, duration; ext, extension; flex, flexion; RF, rectus femoris; ST, semitendinosus; VL, vastus lateralis.*
** Interaction between position and side (affected or nonaffected), expresses whether the effect of changing position differs for the affected compared with the unaffected side.*
*† After log transformation of the data (the mean values presented are observed means).*

From table 2.3, it can be derived that the change of the duration of the first knee flexion movement ($D_{flex}$), due to changing position, was significantly larger on the affected side ($p = 0.002$) compared with the unaffected side. The change of duration of extension ($D_{ext}$) did not differ significantly, although the observed mean difference was larger on the affected side (65.7 ms) than the unaffected side (10.9 ms) ($p = 0.056$).

The amplitude of the movement differed more on the affected side for knee flexion ($p = 0.042$) but not for extension ($p = 0.187$). The changes in the relaxation index differed significantly between the affected and unaffected sides ($p = 0.005$).

On the affected side, the RMS values of the first burst in the rectus femoris and vastus

lateralis increased significantly in the supine position (see table 2.2). Compared with the unaffected side; however, these changes do not seem important.

***Passive movement test***

When comparing the parameters of the passive movement test between the two positions, we found that the duration of extension and amplitude of the movement changed significantly (table 2.4). The difference between duration of flexion in the sitting versus the supine position was not significant at the 5% level.

Rectus femoris activity was generally higher in the supine position, during knee flexion as well as during extension, although these differences were not statistically significant. The burst activity was significantly higher though in the supine position (mean difference RMS –4.0 μV; $p = 0.007$). For the vastus lateralis, all observed values were higher in the supine compared with the sitting position, but the differences were not statistically significant. The RMS of the semitendinosus during extension was higher in the sitting position (mean difference 6.6 μV; $p = 0.017$).

**Table 2.4:** Parameters of the passive movement test on the affected side in two positions

| ***Parameter*** | ***Sitting*** | ***Supine*** | ***Mean Difference*** (95% CI) | ***p*** |
|---|---|---|---|---|
| $D_{flex}$ (ms) | 703.6 | 760.3 | –56.7 (–109.7 - –3.6) | 0.059† |
| $D_{ext}$ (ms) | 640.5 | 608.7 | 31.8 (3.3 – 60.3) | 0.044† |
| $A_{flex}$ (deg) | 76.0 | 70.1 | 5.9 (1.7 – 10.1) | 0.008 |
| $A_{ext}$ (deg) | 76.2 | 70.2 | 6.0 (1.8 – 10.2) | 0.008 |
| $RMS(RF)_{flex}$ (μV) | 12.8 | 15.9 | –3.1 (–7.3 - 1.1) | 0.243† |
| $RMS(RF)_{ext}$ (μV) | 5.1 | 5.5 | –0.4 (–2.5 - 1.7) | 0.689 |
| $RMS(RF)_{burst}$ (μV) | 16.3 | 21.6 | –5.3 (–9.6 - –1.1) | 0.007† |
| $RMS(VL)_{flex}$ (μV) | 5.5 | 7.7 | –2.2 (–5.1 - 0.7) | 0.472† |
| $RMS(VL)_{ext}$ (μV) | 3.6 | 3.7 | –0.2 (–1.5 - 1.2) | 0.616† |
| $RMS(VL)_{burst}$ (μV) | 10.3 | 14.3 | –4.0 (–8.6 - 0.6) | 0.149† |
| $RMS(ST)_{flex}$ (μV) | 7.8 | 9.6 | –1.7 (–4.6 - 1.2) | 0.222 |
| $RMS(ST)_{ext}$ (μV) | 22.3 | 15.6 | 6.6 (1.5 - 11.7) | 0.017† |
| $RMS(ST)_{burst}$ (μV) | 26.5 | 21.3 | 5.2 (–1.8 - 12.1) | 0.135 |

*Abbreviations: A, amplitude of movement; burst, during burst activity; CI, confidence interval; D, duration; ext, extension; flex, flexion; RF, rectus femoris; ST, semitendinosus; VL, vastus lateralis.*
*NOTE. p values are tested parametrically, unless mentioned.*
*† Wilcoxon signed-rank test.*

For the passive movement test, the same type of comparison between affected and unaffected sides was performed. Table 2.5 shows the results of this analysis. On the affected side, the duration of knee extension and the amplitude of the movement changed significantly with changing position on the affected side. On unaffected side, however, these parameters changed as well ($p$ = 0.008 for duration; $p$ = 0.017 for amplitude of flexion; $p$ = 0.006 for amplitude of extension). The changes were comparable on both sides (all $p > 0.05$). Similarly, the changes in RMS of rectus femoris burst activity and the change of RMS of the semitendinosus during extension could not be discriminated.

**Table 2.5:** Means of differences of the passive movement test on affected and unaffected side, compared by a 2-factor analysis of variance

| ***Parameter*** | $\Delta_A$ (95% CI) | $\Delta_{NA}$ (95% CI) | ***Interaction* (p)*** |
|---|---|---|---|
| $D_{ext}$ (ms) | 31.8 (3.3 - 60.3) | 19.1 (4.0 - 34.2) | 0.542† |
| $A_{flex}$ (deg) | 5.9 (1.7 - 10.1) | 5.5 (1.1 - 9.8) | 0.906 |
| $A_{ext}$ (deg) | 6.0 (1.8 - 10.2) | 5.6 (1.2 - 10.0) | 0.911 |
| $RMS(RF)_{burst}$ (μV) | –5.3 (–9.6 - –1.1) | –2.9 (–6.5 - 0.6) | 0.688† |
| $RMS(ST)_{ext}$ (μV) | 6.6 (1.5 - 11.7) | –0.9 (-2.7 - 1.0) | 0.065† |

*Abbreviations: $\Delta_A$, mean difference (sitting - supine) on affected side; $\Delta_{NA}$, mean difference (sitting - supine) on nonaffected side, A, amplitude of movement; burst, during burst activity; CI, confidence interval; D, duration; ext, extension; flex, flexion; RF, rectus femoris; ST, semitendinosus.*
** Interaction between position and side (affected or nonaffected), expresses whether the effect of changing position differs for the affected compared to the unaffected side.*
*† After log transformation of the data (the mean values presented are observed means).*

### *Ashworth scale*

In the supine position, we found significantly higher Ashworth scores for the knee extensors (Wilcoxon signed-ranks test, $p$ = 0.001) and lower scores for the knee flexors ($p$ = 0.002). Table 2.6 shows the shift to lower scores for the extensors in the sitting position and for the flexors in the supine position. On the unaffected side, all scores for flexors and extensors were zero (no increase in tone) in both positions.
The correlation coefficients between the Ashworth scores for the extensors and the RMS values of the rectus femoris during stretch while performing the pendulum test were moderate in both the sitting and supine positions (table 2.7). All values were

significant at the 5% level. For the passive movement test, however, the correlation coefficients were low, particularly in the sitting position, and most of them did not reach a level of significance. For the knee flexors, correlation coefficients were low and nonsignificant in both the sitting and supine positions.

**Table 2.6:** Ashworth scores for knee flexors and extensors on the affected side in two positions

| | ***Extensors*** (N=19) | | ***Flexors*** (N=19) | |
|---|---|---|---|---|
| ***Ashworth scale*** | Supine | Sitting | Supine | Sitting |
| 0 = no increase | 2 | 8 | 9 | 4 |
| 1 = slight increase | 11 | 9 | 10 | 9 |
| 2 = more marked increase | 3 | 1 | 0 | 6 |
| 3 = considerable increase | 3 | 1 | 0 | 0 |
| 4 = passive movement impossible | 0 | 0 | 0 | 0 |

*NOTE. Data express the number of times a value is scored.*

**Table 2.7:** Spearman's correlation coefficients of Ashworth scores and RMS values of the knee extensors and flexors in two positions

| | ***Ashworth score extensors*** | |
|---|---|---|
| RMS Values (RF) | Sitting | Supine |
| ***Pendulum test*** | | |
| $RMS(RF)_{flex}$ | 0.55* | 0.51* |
| $RMS(RF)_{burst}$ | 0.51* | 0.48* |
| ***Passive movement*** | | |
| $RMS(RF)_{flex}$ | 0.31 | 0.51* |
| $RMS(RF)_{burst}$ | 0.35 | 0.45 |
| | ***Ashworth score flexors*** | |
| RMS Values (ST) | Sitting | Supine |
| ***Pendulum test*** | | |
| $RMS(ST)_{ext}$ | 0.37 | –0.14 |
| $RMS(ST)_{burst}$ | –0.22 | 0.00 |
| ***Passive movement*** | | |
| $RMS(ST)_{ext}$ | 0.38 | 0.27 |
| $RMS(ST)_{burst}$ | 0.24 | 0.35 |

*$* p < 0.05$*

## Discussion

The aim of this study was to investigate the influence of position on stretch reflex activity of knee flexor and extensor muscles in stroke subjects with known spasticity in the affected leg. In addition to what was done in earlier studies,[18-20] we performed the Ashworth scale in two positions and recorded surface electromyography during the pendulum test and passive movement of the limb.

The results of the neurophysiological tests in this study confirm our hypothesis that a muscle in an elongated state shows more stretch reflex activity compared with a muscle in a shortened state. The findings of Burke et al.[13] about the inhibitory effect of quadriceps lengthening are therefore contradicted by the results of our study. The graphically presented results of Burke et al.[13] show that, for a constant velocity of knee flexion, the stretch reflex of the quadriceps muscle diminishes in amplitude when the passively imposed stretching movement is started with the knee joint more flexed. Because the starting angle was not randomized in Burke's[13] experiment, fatigue might play a role in the extinguishing stretch reflex. Another explanation could be that a nonoptimal placement of the electrodes on the quadriceps muscle caused a high sensitivity of observed electromyographical amplitude on change of knee angle.[28]

In our study, the significant increase in burst activity of the vastus lateralis in the supine position during the pendulum test is noteworthy, because we did not expect to find any relevant change in this monoarticular muscle. Crosstalk is not expected to play a role here, because crosstalk from the rectus femoris in surface electromyography of the vastii is usually not seen, but rather the reverse. It might be a result of co-activation of the quadriceps muscle group, due to common pathways in the reflex arc, although the rectus femoris has been shown to function independently from the vastii during gait.[31] In addition, myofascial force transmission may contribute to this phenomenon. As shown by Huijing and Baan,[32] part of the total muscle force is transmitted to extramuscular connective tissue of a compartment and to adjacent muscles, rather than being transmitted to the

insertion of a muscle tendon. Related to this, it was shown that the relative position of a muscle, with respect to its surrounding structures, influences the proximodistal force distribution within the muscle itself.[33]

He[19] also performed the pendulum test under different postural conditions in 59 MS patients. He described that changes both in the rectus femoris and vastii (medial and lateral heads) are seen in some patients with moderate or severe spasticity but not in patients with very mild spasticity, as assessed with the Ashworth score. This difference between mildly and more severely affected patients is not observed in our data, possibly due to our limited sample size.

The changes in goniometric parameters of the pendulum test in the two positions are large and significantly higher compared with the unaffected side. The mean value of the relaxation index on the affected leg in the sitting position could even be considered as normal;[30] the mean relaxation index in the supine position, however, represents spasticity. These changes in goniometric parameters could be a result of both change in stretch reflex activity and changes in biomechanical factors. These cannot accurately be differentiated in this study, although an attempt is made by comparing with the unaffected side. Fowler et al,[24] evaluating poststroke subjects and healthy people, concluded that soft tissue changes rather than hyperreflexia may explain the goniometric changes found in their study. From different studies it becomes clear that the role of changes in intrinsic muscle characteristics after an UMN lesion is very complex.[2,21,34,35] Many authors[24,36,37] are now focusing on the changes in sarcomere length as a result of the UMN syndrome, which implicate an indirect effect on stretch reflex activity. The number of sarcomeres decreases[2,36] and sarcomere length increases in spastic muscles. Spastic muscle cells appear to be significantly shorter and less elastic than normal muscle cells,[37] implying an increased resistance to stretch.

In this study, stretch reflex activity has been shown to play a role in the changed goniogram after position change. Increased spindle sensitivity might be contributing as a direct result of muscle elongation or in combination with increased stiffness of the spastic muscle. A change in biomechanical properties of other soft tissues in

different positions probably is part of the cause as well.

These biomechanical changes well might explain the large differences in clinical assessment with the Ashworth scale between the two positions. These differences are remarkable, because we did not expect to find important changes measured by this rather crude scale. The low to moderate correlations between the Ashworth scores and the electromyographical parameters for muscle activity further emphasize the limited validity of the Ashworth scale as a measure for spasticity.

***Study limitations***

There are some limitations in this study that need to be mentioned. First, differences between the baseline characteristics of groups A and B were seen; of these, the difference in mean age was statistically significant. We do not expect, however, that these variables affect subjects' responses to the tests, because these variables do not seem to be related to the outcome variables.

Significantly larger differences in movement amplitudes were seen in the patients who were first measured in the supine and then in the sitting position, compared with the reverse order. This might indicate a carryover effect, but surprisingly no such difference was seen in the other pendulum test parameters, particularly not in the parameters describing the electromyographical activity of the knee extensors. Therefore, it might be a coincidental finding, not relevant for the interpretation of our results.

Furthermore, the influence of afferent stimuli was not included in this study. Sensation loss or the presence of neglect was not an exclusion criterion. Most subjects appeared to have at least some sensation on the affected side. Loss of sensation, particularly loss of proprioceptive input, however, might influence the results, especially when visual control of the movement is not possible (in the supine position). In addition, vestibular input probably has an important role in reflex modulation during stance and gait.[38] In this experiment, we considered the influence of vestibular input not relevant, because in both positions subjects were well supported and there was no floor contact. We standardized the position of the

head during testing, to eliminate the possible influence of head position on stretch reflex activity, similar to the H-reflex.[39,40]

Finally, in this study we used the unaffected side as a reference to investigate 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. However, this assumption is not completely correct as pathologic changes on the 'unaffected' side can be found as well, mainly because of damaged uncrossed fibers.[41]

## Conclusions

Clinical and neurophysiological assessment of spasticity is influenced considerably by change in posture and muscle length. For comparable assessment of spasticity exact documentation of patients' positions is essential. We strongly support the recommendation to standardize testing protocols.[7,8]

***Acknowledgements***

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# Appendix 1:

***Summary of the procedure in each position***

- Ashworth score of flexors and extensors on unaffected side
- Ashworth score of flexors and extensors on affected side

  *10-minute rest*
- Pendulum test (3 times) on unaffected side
- Passive movement test (3 times) on unaffected side

  *10-minute rest*
- Pendulum test (3 times) on affected side
- Passive movement test (3 times) on affected side

  *20-minute rest*

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# 3

# 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 = 0.536, $p = 0.022$), for the rectus femoris (RF) high (rho = 0.825, $p < 0.001$). For the semitendinosus (ST) the correlation was low (rho = 0.267) 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.

## 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.[1,2,3]

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.[4] In this definition, the tonic stretch reflex is described as a response to an externally imposed passive stretch of relaxed muscle.[5,6,7] 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".[8,9] 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 associated reactions[10] 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.[11-13] The idea that spastic hemiparesis causes a movement disorder as a result of both the paretic and the spastic component is generally accepted.[14] There are indications that stroke patients with spasticity are functionally more impaired than patients without spasticity.[2,15,16] 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.[17] 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.[17-19]

Until recently, the majority of studies investigated electrical muscle activity during reflexes or during passive joint rotation[20] 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[11,13,19,21] and lower limbs.[11-13,19,22-26] Sahrmann et al.[21] 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.[11,25]

Findings of Dietz[19] 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[12,22,24,26] with a poor ability to switch off under passive conditions.[11,13]

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 to: (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.



## 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.

***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[27] and scores of knee extensors and/or knee flexors should be ≥ 1. In addition, patients had to be able to move the lower leg against gravity (Medical Research Council (MRC) ≥ 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.

***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 minutes rest between sessions.

***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.[28] 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, Kinesiologic 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.



***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.

***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.[29]

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.

## Results

### *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 3.1 summarizes the baseline characteristics of all participating subjects.

**Table 3.1:** Group characteristics

| | |
|---|---|
| N | 19 |
| Mean age ± SD (yrs) | 57.7±12.3 |
| Women (n) | 4 |
| Right hemiparesis (n) | 6 |
| Nonhemorrhagic (n) | 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.*



### *Movement patterns and muscle activity during passive and active movement*

*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 μV 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.

Figure 3.1 shows a representative example of muscle activation patterns during passive and active movement on the affected side of one of the subjects.

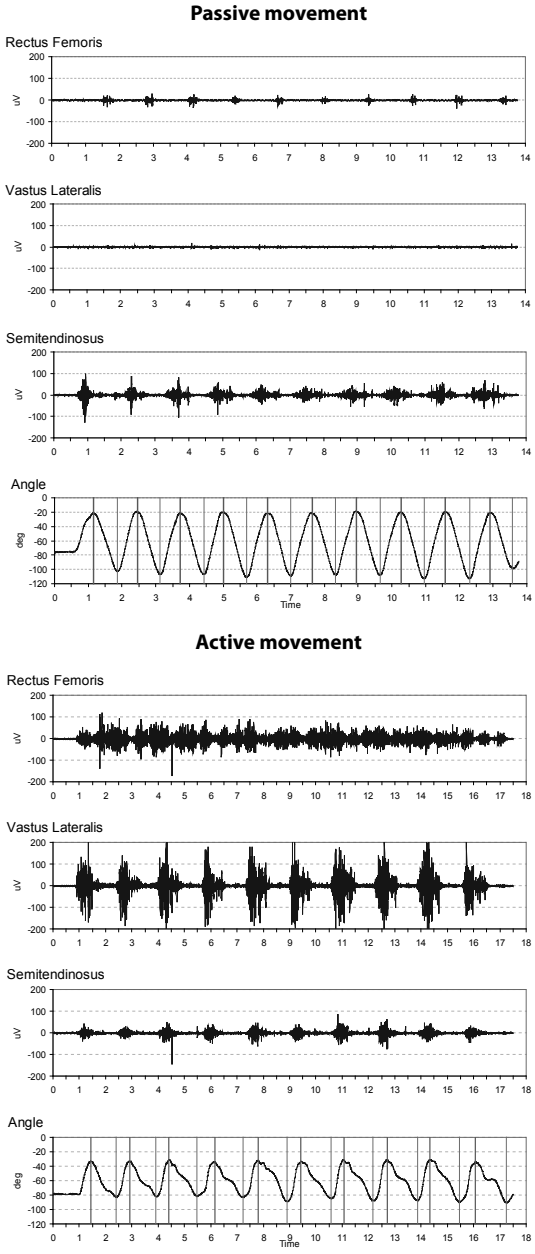


**Fig 3.1:** Example of activation patterns during passive and active movement of a subjects' affected leg (muscle activity in μV, angle in degrees and time in seconds)

*Quantitative analysis of the data*

Figure 3.2 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°; mean active ROM 49.3°; mean difference 26.7°) 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°; mean active ROM 80.4°; mean difference 4.7°).

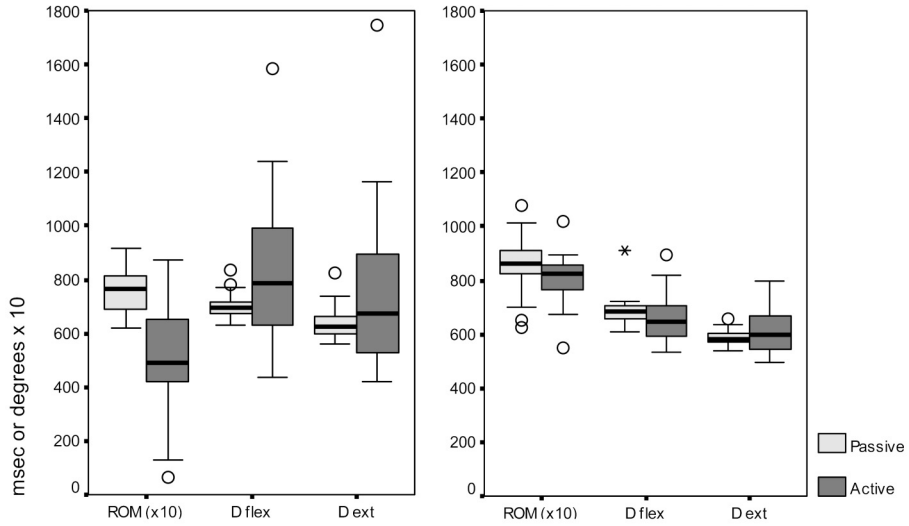


**Fig 3.2:** Duration and range of motion of the movement cycle during passive and active movement on the affected (left) and unaffected side (right)
Abbreviations: ROM, range of motion; D, duration; flex, during knee flexion phase; ext, during knee extension phase.

Subsequently, RMS values were compared between passive and active movement (table 3.2). 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.

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 3.3. For the knee extensors the correlation was moderate (VL rho=0.536, $p = 0.022$) to high (RF rho = 0.825, $p < 0.001$). For ST the correlation was low (rho = 0.267) 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 = 0.539, $p = 0.017$).

***Co-contraction during active movement of the lower leg***

In table 3.4 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 antagonist 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).

**Table 3.2:** Comparison of muscle activity (in μV) on the affected and unaffected side during passive versus active movement (Wilcoxon signed ranks test)

| **Affected side** | Muscle | **Passive**, mean *(SD)* | **Active**, mean *(SD)* | *p* |
|---|---|---|---|---|
| *During stretch* | $RF_{flex}$ | 12.8 *(10.8)* | 24.0 *(16.3)* | 0.001 |
| | $VL_{flex}$ | 5.5 *(3.3)* | 20.8 *(11.4)* | <0.001 |
| | $ST_{ext}$ | 22.3 *(14.5)* | 21.5 *(16.1)* | 0.687 |
| *During shortening* | $RF_{ext}$ | 5.1 *(4.5)* | 35.4 *(21.5)* | <0.001 |
| | $VL_{ext}$ | 3.6 *(2.1)* | 45.5 *(33.0)* | <0.001 |
| | $ST_{flex}$ | 8.6 *(4.9)* | 14.8 *(14.5)* | 0.078 |
| **Unaffected side** | Muscle | **Passive**, mean *(SD)* | **Active**, mean *(SD)* | *p* |
| *During stretch* | $RF_{flex}$ | 2.8 *(1.9)* | 15.1 *(15.0)* | <0.001 |
| | $VL_{flex}$ | 3.7 *(3.7)* | 20.6 *(15.6)* | <0.001 |
| | $ST_{ext}$ | 4.8 *(2.8)* | 13.7 *(7.1)* | <0.001 |
| *During shortening* | $RF_{ext}$ | 3.9 *(3.0)* | 42.4 *(23.4)* | <0.001 |
| | $VL_{ext}$ | 4.9 *(4.0)* | 78.2 *(35.6)* | <0.001 |
| | $ST_{flex}$ | 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*.

**Table 3.3:** Correlation matrix of muscle activity in the stretch phase during passive versus active movement in the affected and unaffected limb (Spearman's rho)

| **Affected side** | | **Passive** | | |
|---|---|---|---|---|
| | | $RF_{flex}$ | $VL_{flex}$ | $ST_{ext}$ |
| **Active** | $RF_{flex}$ | 0.825** | | |
| | $VL_{flex}$ | | 0.536* | |
| | $ST_{ext}$ | | | 0.267 |
| **Unaffected side** | | **Passive** | | |
| | | RFflex | VLflex | SText |
| **Active** | $RF_{flex}$ | -0.007 | | |
| | $VL_{flex}$ | | 0.068 | |
| | $ST_{ext}$ | | | 0.539* |

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

** $p < 0.05$*

*** $p < 0.01$*

**Table 3.4:** Correlation matrix of muscle activity of agonists and antagonists during active movement on the affected side (Spearman's rho)

| | | **Shortening phase** (contraction) | | |
|---|---|---|---|---|
| | | $RF_{ext}$ | $VL_{ext}$ | $ST_{flex}$ |
| **Stretch phase** | $RF_{flex}$ | | | -0.39 |
| | $VL_{flex}$ | | | 0.21 |
| | $ST_{ext}$ | 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*

### *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 figure 3.3. On the unaffected side no relevant or statistically significant correlations were found.

**Table 3.5:** Correlation matrix of stretch reflex activity during passive movement versus movement parameters during active movement (Spearman's rho)

| | | **Passive** | | |
|---|---|---|---|---|
| | | $RF_{flex}$ | $VL_{flex}$ | $ST_{ext}$ |
| **Active** | $D_{flex}$ | 0.01 | 0.21 | |
| | $D_{ext}$ | | | 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*

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.

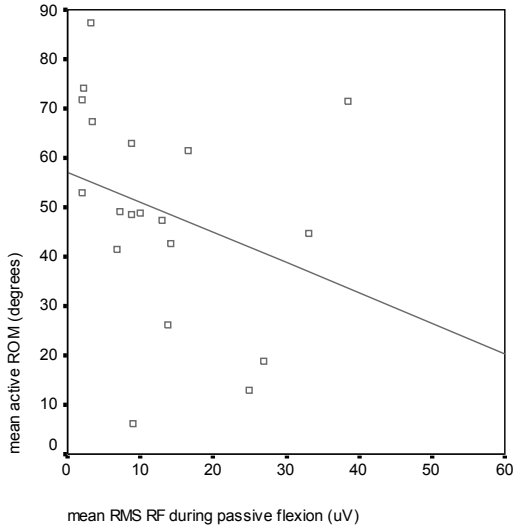


**Fig 3.3:** Scatter plot of stretch reflex activity of RF during passive movement versus active range of motion of the knee on the affected side

## 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 figure 3.2). 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. Sahrmann and Norton[21] 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 degree 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 3.2). 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.[9] A similar delay in termination of contraction was seen in the studies of Chae et al.[30,31] 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.[32,33] 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.[30] and Dewald et al.[29] 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. [11,34]). An association was not found for RF, possibly because the biarticular RF does not have a prominent function during knee extension,[35] but is active merely during the stance-to-swing transition in gait, acting as a hip flexor and on deceleration of excessive knee flexion.[35,36] When observing the more or less continuous activity seen in the RF (see figure 3.1), 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 figure 3.2) 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.[35] 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.,[11] 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 3.1). 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.[37]

There are indications that reflex activity in spastic muscles in the lower extremities is comparable with that in upper limb muscles (e.g. [19]). 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.

## 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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# 4

## STOP USING THE ASHWORTH SCALE

## FOR THE ASSESSMENT OF SPASTICITY

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## Abstract

Many studies have been performed upon the methodological qualities of the (modified) Ashworth scale, but overall these studies seem insufficiently conclusive. Aim of this study is to investigate the construct validity and inter-rater reliability of the Ashworth scale (AS) for the assessment of spasticity in upper and lower extremities.

A cross-sectional study on spasticity in the elbow flexors (part 1) and knee extensors (part 2) was carried out. In both parts AS was assessed, while muscle activity and resistance was recorded simultaneously, in patients with upper motor neuron syndrome. Each patient was measured by three raters.

Thirty patients participated, nineteen in each part of the study. For elbow flexor muscles, AS was not significantly associated with electromyographic parameters, except for rater 2 (rho = 0.66, $p < 0.01$). A moderate significant association was found with resistance ($0.54 \leq \text{rho} \leq 0.61$, $p < 0.05$). For knee extensors, AS scores were moderately associated with muscle activity ($0.56 \leq \text{rho} \leq 0.66$, $p < 0.05$) and also with resistance ($0.55 \leq \text{rho} \leq 0.87$, $p < 0.05$). Intraclass correlation coefficient for absolute agreement was 0.58 for elbow flexors and 0.63 for knee extensors. In linear mixed model analysis the factor Rater appeared to be highly associated with AS.

It was concluded that validity and reliability of the Ashworth scale is insufficient to be used as a measure for spasticity.

## Introduction

Spasticity is a common phenomenon in patients with upper motor neuron syndrome and is characterized by involuntary muscle activity.[1] Traditionally, spasticity is defined as a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes resulting from hyperexcitability of the stretch reflex[2] and is particularly present in the anti-gravity muscles, like the knee extensors and the elbow flexors.[3]

In clinical practice as well as in scientific research either the Ashworth scale (AS)[4] or its modified version (MAS)[5] is the most commonly used method for the measurement of spasticity.[6,7] In many neurology books this scale is still referred to as being the principal method for assessing spasticity (e.g. [8]). However, its methodological limitations are now increasingly being acknowledged.[6,7,9,10] While performing the test the joint under investigation is passively rotated and the examiner rates the perceived resistance during the movement. This resistance is scored on an ordinal scale from 0 to 4. To be a measure for spasticity, the (M)AS can only be valid when the increase in resistance to passive movement is exclusively associated with an increase in neural, stretch reflex activity.[11- 13] However, this is probably not the case as the resistance to passive movement is a sum total of reflex muscle activity and non-neural mechanical characteristics. It is influenced by changes in visco-elastic properties of joint structures and soft tissues after an upper motor neuron lesion.[13,14] In addition, changes in mechanical muscle-fibre properties might contribute to spastic muscle tone.[3] Biomechanical changes are hard to differentiate from reflexive muscle activity without the use of highly sophisticated instruments, although in clinical practice nerve blocks with local anaesthetics can be of assistance.[15]

Two comprehensive reviews have been published that address the validity of the (M)AS.[6,7] Both reviews found that associations of (M)AS with electromyographic parameters were moderate, and the association with objective measures of resistance was generally stronger.

Besides concerns with regard to validity, the reliability of the AS is questioned as well. Platz et al. concluded that the intra- and inter-rater reliability of the (M)AS appeared to vary highly between studies.[7] Generally somewhat higher levels of reliability were found in the upper extremity compared to the lower extremity,[7,16,17] which could be due to the larger mass of the legs. Another factor that might affect reliability is lack of standardization of the (M)AS.[6,18,19] Possibly, factors like the velocity and range of motion may affect the perceived resistance, but these have never been quantified for this scale so far.

In summary, several studies about the methodological qualities of the (M)AS have been performed, investigating either the validity or the reliability of the scale. Overall, these studies are not conclusive and that has led to the continued use of a measurement method with doubtful methodological qualities. Therefore, there is a need for a comprehensive study on the clinimetric properties of the AS, using a design that overcomes the major drawbacks of existing literature. The goal of present study was twofold: first, to investigate the construct validity of the AS for the measurement of spasticity, and second, to assess the inter-rater reliability and identify potential sources of variability between raters. For these purposes, surface electromyography (sEMG) recording was performed during AS scoring by different trained raters, in both elbow flexor and knee extensor muscle groups. Additionally, dynamometry recording was done simultaneously for objective assessment of resistance during passive movement.

## Methods

### *Study design and population*

This cross-sectional study consisted of two parts: the first part focused on spasticity in elbow flexor muscles and the second part on spasticity in knee extensor muscles. Patients with self-reported spasticity in the upper arm and/or upper leg following an upper motor neuron lesion were recruited from the in- and outpatient departments

of a local Rehabilitation Centre. To take part in the study patients had to be able to understand simple commands. Presence of pain or severe contractures of the elbow or knee were exclusion criteria.
The study was approved by the local medical ethics committee. Patients signed informed consent before participation in the study.

***Procedure***

Each patient was measured by three raters. Patients were measured in random order, with 30-45 minutes of rest in between two ratings. The raters, three physicians and a physiotherapist involved in daily spasticity care and experienced with using the AS, were instructed in detail about the measurement protocol a few weeks prior to assessment during a group session. Before the actual measurement the full passive range of motion was assessed by the rater, starting from the position with maximum shortened muscles and rotating to the position of full muscle stretch at a low velocity. Subsequently, raters performed the movement twice at fast velocity throughout the entire range of motion. Raters noted the AS score on a form. In case of doubt, the lower score obtained was documented.

For measurement of the elbow flexors (part 1), patients were comfortable in supine position with the affected arm on a supporting scale, with the shoulder abducted to about 20° (figure 4.1a). The rater rested one hand on the upper arm of the patient and the other on the palmar side of forearm just proximal to the wrist simultaneously holding the dynamometer. From the starting position of full elbow flexion the rater extended the elbow to maximum extension. For the measurement of the knee extensors (part 2) the patients were positioned lying on the non-tested side, with ± 45° of hip flexion on the tested side (figure 4.1b). The raters were instructed to hold the tested leg with one hand just above the knee and the other around the distal part of the lower leg holding the dynamometer that was fixed ventrally above the ankle. From the starting position of maximum knee extension the rater flexed the knee fully. The standardized positions were maintained without

forceful stretching or discomfort for the patient. Patients were explicitly instructed to relax fully and not to react to stretches. Raters and patients were blinded for the sEMG and dynamometry output and other raters' scores.

(a) (b)

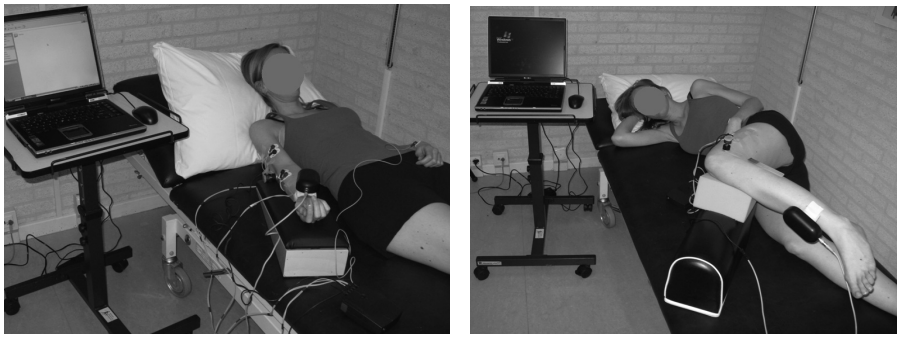

**Fig 4.1:** Demonstration of the patients' positioning for measurement of the elbow flexors (a) and knee extensors (b)

***Instrumentation***

Surface EMG signals were obtained using bipolar, pre-gelled circular (diameter = 10 mm) electrodes (ARBO H93, solid gel), with an inter-electrode distance of 24 mm. A reference electrode was placed around the wrist.

Electrodes were placed on the elbow flexor muscles biceps brachii (BB) and brachioradialis (BR), or on the knee extensor muscles rectus femoris (RF) and vastus lateralis (VL). Sensors on BB, RF and VL were placed according to the electrode placement recommendations of the SENIAM-based protocol.[20] BR electrodes were placed at 1/4$^{th}$ of the line between the distal radius and the fossa cubit. A reference electrode was placed at the medial aspect of the contralateral wrist.

For force measurements a handheld dynamometer (Biometrics M500) was used, which was positioned at the palmar aspect of the wrist or anterior aspect of the lower leg, such that the rater could easily hold it during AS scoring. In addition, the elbow or knee angle was registered using a mono-axial electronic goniometer, placed on the lateral side of the joint, to enable the characterization of the movement performed during measurement by determining start and end of the

movement. All sensors were connected with the Mobi measurement device (Mobi, TMSi, The Netherlands), a blue tooth recording and processing system that allowed temporal synchronization of the sEMG signals with analogue data from the angle and force sensors. Sample frequency was 512 Hz and data were bandpass filtered at 15 - 256 Hz using 2nd order Butterworth filter.

***Data analysis and outcome parameters***

The AS was scored according to the original scale (score 0 – 4).[4]

Outcome parameters were selected in order to reflect (1) reflex muscle activity on stretch while rating the AS, using sEMG, and (2) the total resistance felt by the raters while rotating the limb, using a dynamometer.

From sEMG recordings, root mean square values (RMS; in μV) of each muscle during joint rotation were calculated, representing the average muscle activity during stretching of the muscle. Subsequently, the total amount of EMG activity was calculated by integrating the RMS during the whole joint rotation (area under the curve, $AUC_{muscle}$; in μV*sec). The latter was considered to give better representation of the intensity of muscle activity during the whole rotation.

Similarly, the area under the force curve was calculated from the dynamometer data, representing the intensity of applied force or the resistance during joint rotation (Resistance; in Newton*sec).

Goniometry recordings were used to determine the start and end points of the muscle stretching phase, from which movement characteristics during joint rotation, such as range of movement (ROM; in degrees), duration (Duration; in seconds) and mean angular velocity (Velocity; in deg/sec) were derived.

The parameters AS score and $AUC_{muscle}$ were used as primary outcome measures.

***Statistical analysis***

Descriptive statistics were used for sociodemographic and outcome variables. Mean values of each instrumentally obtained parameter were computed out of the 2 performed elbow extension or knee flexion movements. In a few cases only

one cycle was used for analysis, due to failure to perform one of the movements properly, indicated by the rater, or for technical reasons (e.g. recording problems).
Construct validity of AS for each rater separately was investigated, by calculating the association between AS and $AUC_{muscle}$, using Spearman's correlation coefficient. Additionally, the correlation coefficient was calculated between AS, Resistance and Velocity.
Inter-rater reliability of the AS was evaluated first by calculating raw overall agreement between raters. Subsequently generalized kappa for three raters was calculated. In addition, the intraclass correlation coefficient (ICC) for absolute agreement was calculated.[21] A linear mixed model analysis was performed to get insight in the weight of each independent variable ($AUC_{muscle}$, Resistance and co-variate Velocity) in explaining the dependent variable AS. In order to explore the variability between raters, the factor Rater was added to the model as well. The 'eyeball test' was used to test the assumption of normally distributed residuals. The percentage of explained variance (1st level $R^2$) for the model was calculated according to the formula of Snijders and Bosker.[22]
For statistical analysis SPSS 11.5 was used. For calculation of the generalized kappa Excel software was used. Alpha was set at 0.05 for statistical significance.

## Results

Altogether thirty patients were included in this study, of which 19 patients participated in each part. The characteristics of both groups are summarized in table 4.1. In the first part of the study all 19 patients were measured by rater 1, 18 by rater 2 and 16 patients by rater 3. Dynamometry and sEMG data were partly missing in 3 patients. In part two all 19 patients were measured by raters 1, 3 and 4. Dynamometry and sEMG data were partly missing in one patient; dynamometry data were missing in another patient.

AS score '0' was rated in 22% of the measurements, '1' in 44%, '2' in 23%, '3' in 11% and '4' in 1% of the measurements, with comparable distributions in both parts of the study. During slow stretch no severe contractures were found.

Figure 4.2 shows an example of muscle activation patterns of knee extensors and resistance during AS scoring by rater 1. The medians and ranges of the movement characteristics during scoring for each rater are shown in table 4.2.

**Table 4.1:** Group characteristics

| **Characteristics** | **Part 1 (Elbow flexors)** N=19 | **Part 2 (Knee extensors)** N=19 |
|---|---|---|
| Age, in yrs (mean ± sd) | 57 (± 13) | 57 (± 16) |
| Male (n) | 16 | 15 |
| Diagnosis (n) | | |
| Stroke | 18 | 11 |
| Cerebral palsy | 1 | 2 |
| Neuromuscular disease | 0 | 4 |
| Spinal cord injury | 0 | 2 |

**Table 4.2:** Descriptive statistics (median, range) of the movement characteristics per rater

| | **Elbow flexors (part 1)** | | |
|---|---|---|---|
| **Parameter** | **Rater 1** | **Rater 2** | **Rater 3** |
| ROM | 97.3 (67.7 – 119.0) | 104.0 (85.4 – 131.7) | 90.9 (71.8 – 115.0) |
| Duration | 0.97 (0.70 – 1.56) | 1.54 (0.96 – 2.99) | 1.37 (0.72 – 2.70) |
| Velocity | 100.6 (57.9 – 156.0) | 62.7 (33.2 – 126.7) | 66.6 (34.0 – 123.3) |

| | **Knee extensors (part 2)** | | |
|---|---|---|---|
| **Parameter** | **Rater 1** | **Rater 3** | **Rater 4** |
| ROM | 84.7 (51.6 – 99.8) | 78.8 (47.7 – 97.2) | 101.9 (68.7 – 119.3) |
| Duration | 0.82 (0.46 – 2.08) | 0.87 (0.49 – 2.47) | 2.05 (0.57 – 4.02) |
| Velocity | 112.9 (49.6 – 208.2) | 91.7 (34.4 – 176.4) | 50.9 (21.2 – 177.4) |

*Abbreviations: ROM, range of motion.*

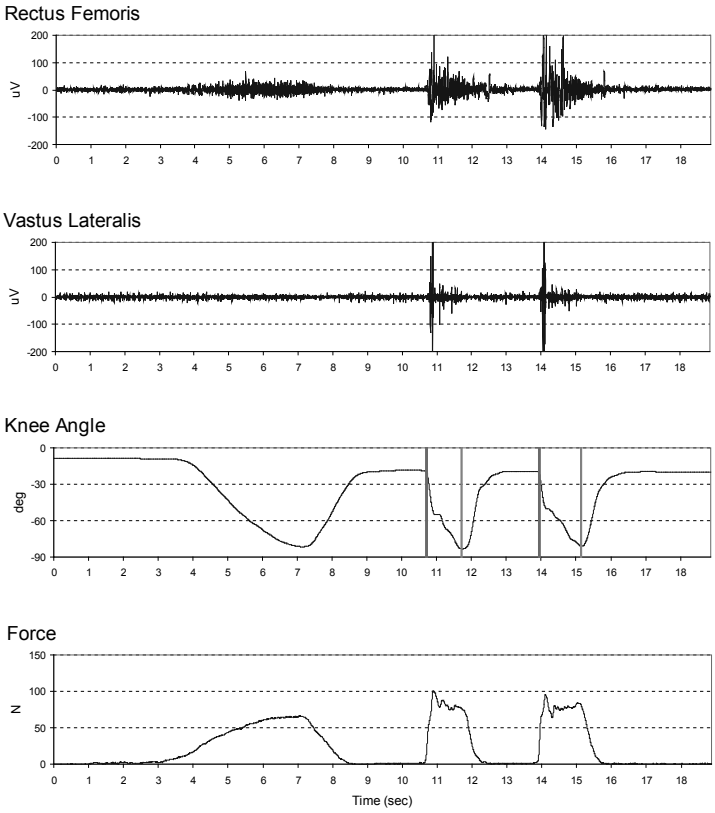


**Fig 4.2:** Example of sEMG, knee angle and dynamometry output during measurement of a patient with Hereditary Spastic Paraparesis, measured by rater 1. The goniometry signal between the vertical lines correspond to the two consecutive fast knee flexion movements and represent the stretch phase of the knee extensor muscles.

### *Construct validity*

*Part 1 (Elbow flexor muscles)*

The results for each rater are shown in table 4.3. For all raters AS was significantly moderately associated with Resistance. AS was not associated with sEMG parameters, except for rater 2, for whom a positive association was found with $AUC_{BB'}$ but not with $AUC_{BR}$.

**Table 4.3:** Spearman correlation coefficients for the association between AS and muscle activity, resistance and angular velocity

| | **AS score elbow flexors** (part 1) | | |
|---|---|---|---|
| **Parameter** | **Rater 1** | **Rater 2** | **Rater 3** |
| $AUC_{BB}$ | 0.05 | 0.66** | 0.07 |
| $AUC_{BR}$ | 0.13 | 0.12 | 0.31 |
| Resistance | 0.55* | 0.54* | 0.61* |
| Velocity | -0.44 | -0.73** | -0.65** |

| | **AS score knee extensors** (part 2) | | |
|---|---|---|---|
| **Parameter** | **Rater 1** | **Rater 3** | **Rater 4** |
| $AUC_{RF}$ | 0.34 | 0.56* | 0.54* |
| $AUC_{VL}$ | 0.57* | 0.65** | 0.66** |
| Resistance | 0.67** | 0.87** | 0.55* |
| Velocity | -0.71** | -0.79** | -0.77** |

** p < 0.05*
*** p < 0.01*
*Abbreviations: AS, Ashworth scale; AUC, area under the curve; BB, biceps brachii; BR, brachioradialis; RF, rectus femoris; VL, vastus lateralis.*

*Part 2 (Knee extensor muscles)*

Table 4.3 shows the results of the knee extensor measurements. A consistent finding was the marked positive association between AS and Resistance across the three raters. In addition, all three raters showed a moderate positive correlation between AS and $AUC_{VL}$, rater 3 and 4 also between AS and $AUC_{RF}$.

### *Reliability*

*Part 1 (Elbow flexor muscles)*

Overall agreement of AS scores between the raters 1 and 2 and between the raters 1 and 3 was 44%. Overall agreement between the raters 2 and 3 was 38%. The generalized kappa for the three raters (n = 16) was 0.20. The ICC for absolute

agreement concerning the AS scores of the three raters was 0.58 (95% confidence interval 0.30 – 0.81).

*Part 2 (Knee extensor muscles)*

Overall agreement of AS scores between raters 1 and 3 was 32%. Between raters 1 and 4 it was 53% and between raters 3 and 4 it was 42%. The generalized kappa for the three raters (n = 19) showed very low agreement: $\kappa = 0.16$. ICC for absolute agreement was 0.63 (95% confidence interval 0.39 – 0.82).

**Table 4.4:** Spearman correlation coefficients for the association between angular velocity, muscle activity and resistance per rater

| | **Elbow flexors** (part 1) | | |
|---|---|---|---|
| **Parameter** | **Rater 1** | **Rater 2** | **Rater 3** |
| | **Velocity elbow extension** | | |
| $AUC_{BB}$ | -0.18 | -0.68** | -0.50 |
| $AUC_{BR}$ | -0.09 | -0.59* | -0.56* |
| Resistance | -0.74** | -0.86** | -0.77** |
| | **Resistance elbow extension** | | |
| $AUC_{BB}$ | 0.11 | 0.66** | 0.62** |
| $AUC_{BR}$ | -0.18 | 0.59* | 0.45 |

| | **Knee extensors** (part 2) | | |
|---|---|---|---|
| **Parameter** | **Rater 1** | **Rater 3** | **Rater 4** |
| | **Velocity knee flexion** | | |
| $AUC_{RF}$ | -0.50* | -0.56* | -0.53* |
| $AUC_{VL}$ | -0.64** | -0.61** | -0.52* |
| Resistance | -0.75** | -0.79** | -0.41 |
| | **Resistance knee flexion** | | |
| $AUC_{RF}$ | 0.24 | 0.65** | 0.43 |
| $AUC_{VL}$ | 0.36 | 0.70** | 0.25 |

** $p < 0.05$*
*** $p < 0.01$*
*Abbreviations: AUC, area under the curve; BB, biceps brachii; BR, brachioradialis; RF, rectus femoris; VL, vastus lateralis.*

***The role of angular velocity in the variability between raters***

*Part 1 (Elbow flexor muscles)*

Table 3 shows negative associations between AS and Velocity in raters 2 and 3 ($p < 0.01$). The association between Velocity, sEMG parameters and Resistance was explored further (table 4.4): in all raters, Velocity was highly negatively associated with Resistance. In rater 2, Velocity was also moderately negatively associated with $AUC_{BB}$; in raters 2 and 3 with $AUC_{BR}$.

Table 4.5 shows the results of linear mixed model analysis. None of the parameters $AUC_{muscle}$, Resistance or Velocity appeared significantly associated with AS. However, the factor Rater significantly affected the AS outcome ($p < 0.05$). The model explained 34% of the variance in AS of the elbow flexor muscles.



**Table 4.5:** Linear mixed model with AS score of elbow flexors as dependent variable (part 1)

| Parameter | Estimate | Std. Error | df | t | Sig. | 95% CI |
|---|---|---|---|---|---|---|
| **Intercept** | 1.111 | 0.735 | 41.352 | 1.511 | 0.138 | -0.374 – 2.596 |
| **$AUC_{BB}$** | -0.003 | 0.008 | 18.822 | -0.397 | 0.696 | -0.019 – 0.013 |
| **$AUC_{BR}$** | -0.001 | 0.004 | 29.324 | -0.252 | 0.803 | -0.008 – 0.006 |
| **Resistance** | 0.011 | 0.006 | 38.591 | 1.924 | 0.062 | -0.001 – 0.022 |
| **Velocity** | -0.010 | 0.005 | 33.875 | -1.867 | 0.071 | -0.020 – 0.001 |
| **Rater = 1** | 0.706 | 0.348 | 38.895 | 2.028 | 0.049 | 0.002 – 1.410 |
| **Rater = 2** | 1.257 | 0.298 | 37.750 | 4.217 | 0.000 | 0.653 – 1.860 |
| **Rater = 3** | 0 | 0 | . | . | . | . |

*Abbreviations: AS, Ashworth scale; Std, standard; df, degrees of freedom; Sig, significance; CI, confidence interval; AUC, area under the curve; BB, biceps brachii; BR, brachioradialis.*

*Part 2 (Knee extensor muscles)*

The negative correlation between AS and Velocity was marked and statistically significant for each of the three raters (see table 4.3). Table 4.4 shows a marked and statistically significant negative association between Velocity and Resistance, except for rater 4. The negative association with muscle activity of the knee extensors was moderate and statistically significant in all cases.

Table 4.6 shows the results of linear mixed model analysis for the knee extensor measurements: Resistance and Velocity were significantly associated with AS, while $AUC_{RF}$ and $AUC_{VL}$ were not. However, the factor Rater showed a highly significant association as well ($p < 0.001$). This model explained 65% of the variance in AS of the knee extensors.

**Table 4.6:** Linear mixed model with AS score of knee extensors as dependent variable (part 2)

| Parameter | Estimate | Std. Error | df | t | Sig. | 95% CI |
|---|---|---|---|---|---|---|
| **Intercept** | 0.670 | 0.435 | 43.090 | 1.539 | 0.131 | -0.208 – 1.548 |
| **$AUC_{RF}$** | 0.007 | 0.007 | 42.198 | 1.024 | 0.312 | -0.007 – 0.021 |
| **$AUC_{VL}$** | 0.126 | 0.131 | 46.037 | 0.960 | 0.342 | -0.014 – 0.389 |
| **Resistance** | 0.010 | 0.002 | 25.623 | 4.136 | 0.000 | 0.005 – 0.014 |
| **Velocity** | -0.010 | 0.003 | 43.964 | -3.593 | 0.001 | -0.015 - -0.004 |
| **Rater = 1** | 1.042 | 0.227 | 41.740 | 4.581 | 0.000 | 0.583 – 1.501 |
| **Rater = 3** | 1.079 | 0.230 | 41.635 | 4.702 | 0.000 | 0.616 – 1.542 |
| **Rater = 4** | 0 | 0 | . | . | . | . |

*Abbreviations: AS, Ashworth scale; Std, standard; df, degrees of freedom; Sig, significance; CI, confidence interval; AUC, area under the curve; RF, rectus femoris; VL, vastus lateralis.*

# Discussion

This is the first study investigating both the construct validity and the inter-rater reliability of the Ashworth scale, using real-time sEMG and dynamometry recordings. The results of this study show that the methodological characteristics of AS are unsatisfactory for the assessment of spasticity.

### *Construct validity*

The contribution of muscle activity parameters, representing involuntary muscle activity when the muscle is stretched, on the variability in the AS score appeared to be low, in particular in the elbow flexor muscles. The association between the AS and Resistance was generally stronger than the association between AS and $AUC_{muscle}$. This finding is in agreement with most other studies,[7] however, a

recent study on the agreement between various measures of spasticity[23] found no consistent relationship of biomechanical measures with the MAS. In the same study, sensitivity of the MAS, when compared with sEMG outcomes, was low.

In present study, the association between Resistance and $AUC_{muscle}$ was not strong either, indicating that even objectively measured resistance is not a good representation of reflex muscle activity. Resistance was calculated as force-time integrals, instead of the more commonly used slope of the force-angle curve.[24] Force-time integrals are influenced by the duration of joint rotation, which was assumed to be one of the features that contributed to the eventual score for resistance perceived by the individual rater. Remarkable is the large inconsistency between raters. It appeared that the large variation in angular velocity within and between raters, which was associated highly with both AS and Resistance, acts as a confounder in AS assessment.

The results of our study differ significantly from the study of Sköld et al.,[25] who recorded sEMG while scoring the MAS of knee flexors and extensors in SCI patients. Although comparison with present study is problematic due to difference in measurement procedures, they found the majority of sEMG parameters to be strongly associated with the MAS. However, 50% of the measurements in the study were rated as MAS scores '0'. For calculation of the Spearman rank correlation coefficient, these scores all become assigned to the same rank, causing misleading results and overestimation of the association.

***Reliability***

There is little consensus in literature and among statisticians about what statistical methods are best to analyze rater agreement.[6] The overall agreement between raters gives a general idea about the agreement of the individual scores in the same patient group. In this study rather low values, up to maximum 53%, were found. The kappa coefficient was very low in both upper and lower limb measurements. However, the kappa coefficient can be considered less appropriate for this study, because it is very stringent for a scale with five categories.[21] A weighted kappa,

which takes partial agreement into account, could have been used. It is, however, exactly identical to the intraclass correlation coefficient, when the most commonly used weighing scheme is applied.[21] The ICC for absolute agreement between three raters was 0.58, for AS scoring of elbow flexors, and 0.63, for AS scoring of knee extensors. However, overestimation of the ICC is a major problem when the between-subjects variance is high,[26] which is likely in this study population. Another limitation of using ICC is that it assumes equal spacing between categories, which is probably not the case in the AS.

Several explanations can help to understand the findings in this study. Most importantly, raters may differ in defining the specific rating levels of the AS or in the definition of the measured construct itself (i.e. spasticity, or perceived resistance against passive movement). The actual rating levels of the AS (0 to 4) can be viewed as an arbitrary categorization of the underlying construct spasticity, which is a continuous trait. The perceived resistance to passive movement is an aggregate composed of various physical features and weights attached to each feature. Raters may vary in terms of which feature they notice and the weights they associate with each. This is well illustrated by the results of this study. As was shown in table 2, the movement characteristics of the rotations differed largely between and within raters. The differences between raters were shown to play a significant role in the eventual AS score, which gives ground for stratified presentation of the results per rater.

There is no standard on how much reliability is 'good enough'.[21] Anyhow, an ICC of 0.63 can be considered insufficient for use in scientific research, in particular when used as single outcome measure (e.g. [27]). When the test is used for individual judgement, even higher reliability is required.[21]

***Study limitations***

For the assessment of the contribution of each variable to the AS, we used a linear mixed model. This model assumes that the dependent variable is a continuous variable, which is not the case for AS. In addition, sample size for the study was

rather small. Nevertheless, for illustration purposes only we considered the model sufficiently robust.

Although there is some heterogeneity in our population, there is no reason to assume that the nature of the upper motor neuron lesion influences the results in this cross-sectional study, although it might have increased the between-subjects variance. Finally, in the upper limb study some additional error might have been introduced by the fact that gravitational force opposed elbow extension during the first part of the movement and assisted during the second part. In the lower limb study this effect was reduced due to limb rotation in the horizontal plane. In addition, the brachialis muscle, another important elbow flexor, was not measured with sEMG due to its deep position.



In summary, the results of this study show that the methodological characteristics of AS are unsatisfactory and that AS should not be used as single outcome measure for the assessment of spasticity. It is essential that both researchers and clinicians are very well aware of the limitations of this scale.

## Conclusion

Ashworth scale outcomes appeared to be poorly related to simultaneously measured reflex muscle activity. In its current form the Ashworth scale is therefore insufficiently valid and reliable as a measure for spasticity. We should therefore stop using it as single outcome measure and focus on newer and promising methods, preferably including sEMG application.

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# 5

# PERCEPTION OF LOWER LIMB SPASTICITY IN PATIENTS WITH SPINAL CORD INJURY

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## Abstract

Aim of this study is to investigate the manifestation of spasticity in daily life of spinal cord injured patients, their perception of spasticity and spasticity-related discomfort.

Twenty-six patients with motor complete spinal cord injury (SCI) and spasticity in the lower limbs completed a questionnaire. The following outcome measures were used: manifestation of spasticity, activities during which spasticity occurs, perceived degree of spasticity and resulting discomfort, measured with Visual Analogue Scale and Borg scale, respectively.

In general, spasticity manifested as extensor spasms (84.6%), flexor spasms and/or clonus (both 69.2%), and less often as continuous tension (57.7%). The registered activities were categorized into five main groups: 'changing position' was the largest group (22.0%) with a median VAS of 6.8 (range 2.5 – 9.5) and median Borg scale of 3.0 (range 1.0 – 7.0). Other groups of activities were 'making a transfer' (20.7%), 'activities of daily living' (17.1%), 'being active' (17.1%) and 'stable body position' (12.2%). The overall correlation between VAS and Borg was moderate (Spearman's rho = 0.53, $p = 0.005$).

In conclusion, patients with complete SCI experienced several manifestations of spasticity, with extensor spasms being the most common. Many daily life activities elicited different manifestations of spasticity. The experienced discomfort was only moderately related to the perceived degree of spasticity during an activity. Possibly, the discomfort is influenced by other factors than the perceived spasticity alone.

## Introduction

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.[1] For patients with spinal cord injury (SCI), however, this definition can be considered too narrow, as spasticity and other positive signs of the upper motor neuron syndrome (UMNS) are sometimes hard to differentiate in clinical practice. For that reason different broader definitions are used increasingly.[2-4] In this study, a definition introduced by the SPASM consortium[5] is used. The definition describes spasticity as disordered sensori-motor control, presenting as involuntary muscle activation following an upper motor neuron lesion, thus it includes all positive features of UMNS.

Patients with SCI have a high probability to develop spasticity.[3] Of all SCI patients, 25-43% reported problematic spasticity that hindered their daily activities.[3,4,6,7] Although some beneficial effects of spasticity have been reported,[3,4,8] it is more often associated with secondary negative consequences like pain, fatigue and deformities,[3] and its overall impact on daily life seems to be negative.[9] A decision to treat spasticity depends largely on whether or not it interferes with patient's daily life. In patients with SCI, it has not been identified when spasticity mostly presents. However, this information is essential to improve our understanding of the impact of spasticity on their daily life.

Treatment of spasticity requires reliable assessment methods, which allow therapy to be individually optimized. In order to be able to assess whether 'we are treating what we want to treat', we need objective measurement methods that could measure spasticity in terms of Body Functions and Structures within the framework of the International Classification of Functioning, Disability and Health (ICF).[10] On the other hand, methods are needed that are closer to the patients' perception, because in decision making for optimal treatment, patients' perception plays an important role. The patients' evaluation of spasticity is often an ad hoc report and is rarely documented by using measurement tools like the Spasm Frequency Scale

or a Visual Analogue Scale (VAS).[11-13] Usually no differentiation is made between the perceived degree of spasticity and the experienced spasticity-related level of discomfort.

Figure 5.1 shows a proposed basic model, showing the interrelationship between the different components of the ICF framework and the perception of the patient.

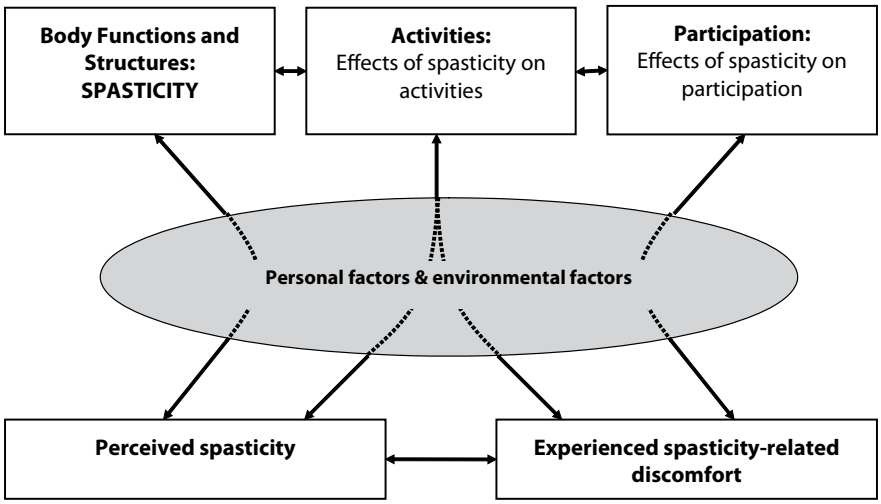


**Fig 5.1:** Conceptual model of spasticity (in terms of impairment of Body Functions and Structures within the WHO ICF framework) and the patients' perception

Both the perception of spasticity and the experienced discomfort are influenced by personal and environmental factors, maybe in different proportions. It has been suggested that patients might include other physical sensations, such as pain, in their perception of spasticity.[3,8] Psychological factors, such as personal interpretations and coping strategies, will probably play a role as well.[8] The experienced spasticity-related discomfort is expected to be influenced considerably by environmental factors, for example, the context in which spasticity occurs. It is recognized that a high degree of perceived spasticity does not necessarily imply high resulting

discomfort,[8,9] although the relation between perceived spasticity and spasticity-related discomfort has not yet been explored. This knowledge could, however, provide important information for the interpretation of spasticity assessment and will, therefore, be essential in evaluating the effect of treatment.

In summary, this study aims to answer two questions. First, how and when does spasticity manifest in patients with motor complete SCI? Second, what is the relationship between the perceived degree of spasticity and the level of spasticity-related discomfort?

## Methods



This explorative cross-sectional study was performed by using a questionnaire that was developed for this study. Patients with motor complete SCI (American Spinal Injury Association (ASIA) Impairment Scale[14] grade A or B) were recruited from in- and outpatient departments of a rehabilitation centre. The inclusion criteria were SCI at least six months old, stable medical condition and self-reported spasticity in the lower limbs. Patients signed informed consent before participation.

### *Questionnaire*

The questionnaire consisted of three parts. The first part was a general section concerning sociodemographics and information on the date and level of injury and use of medication.

The second part focused on the individual perception and description of spasticity in the lower limbs. Four manifestations of spasticity were predefined in the questionnaire, of which patients could choose one or more when applicable: (a) a sensation of continuous tension or stiffness in one or both legs, (b) sudden bending of one or both legs (flexor spasms), (c) sudden straightening of one or both legs (extensor spasms) and/or (d) 'shaking' of the leg or the ankle (clonus). If necessary, patients could provide a description in their own words as well.

Subsequently, questions were posed about whether spasticity was experienced as problematic, and if yes, how, and questions concerning the identification of factors influencing spasticity. These factors were prelisted, including the possibility to add others: (a) infection or inflammation, (b) (change of) posture, (c) full bladder or bowel, (d) skin problems, (e) time of the day, (f) emotions or mental stress, (g) tight clothing, and (h) other.

In the final section, patients were asked to list a maximum of five activities during which they experience high degree of spasticity in the lower limbs. For each activity, patients were asked to rate the degree of spasticity as well as the level of spasticity-related discomfort they experienced. The perceived degree of spasticity was assessed using the VAS, a 10 cm line with 'no spasticity' and 'most imaginable spasticity' at the extremes. The VAS scale is a valid and reliable measure in rating pain intensity and is presently being used more often for the assessment of spasticity.[11,13] The level of spasticity-related discomfort was scored using the Borg scale. The Borg scale is a widely used scale for perceived exertion in exercise,[15,16] but is new in this field. In this study a 12-point ratio scale was used with both numbers and verbal anchors,[16] varying from 'no discomfort at all' (0) to 'extremely much discomfort' (10).

The questionnaires were completed in the presence of the investigator (JF or GV), so that instructions on how to fill out the questionnaire could easily be provided. Subjects without sufficient hand function were assisted in writing down their verbally given responses. For completing the VAS score, the investigator slowly moved a pencil from the left to the right extremity of the line. The mark was placed at the position indicated by the patient.

The questionnaire, in Dutch language, can be provided separately on request.

***Analysis***

Descriptive statistics were used to analyze the general characteristics of perceived spasticity. The activities with the highest VAS score per patient were grouped for an overall estimation of the relationship between perceived spasticity and spasticity-

related discomfort. Subsequently, to facilitate interpretation each of the listed activities was allocated to one of six main groups. For each group, the reported characteristics of spasticity, as well as the medians and ranges of both VAS and Borg scores, were mapped.

To assess the strength of association between the self-rated scores of VAS and Borg the non-parametric Spearman Rank correlation coefficient, with a significance level of 0.05, was used. To avoid bias due to paired observations, in case patients had written down more than one activity assigned to the same group, only the activity with the highest VAS score per patient was used for all calculations.

## Results



### *Sociodemographics*

Twenty-six patients participated in the study. Each questionnaire took approximately 30-45 minutes to be completed.

Table 5.1 summarizes the baseline characteristics of the study population.

**Table 5.1:** Group characteristics (N = 26)

| Characteristics | Values |
|---|---|
| Age (in years) | 41.0 (± 10.6) |
| Female | 6 |
| Level of lesion | |
| C3 – C7 | 14 |
| T3 – T12 | 12 |
| ASIA Impairment Scale A/B | 22/4 |
| Duration since injury (in months) | 100.9 (± 76.5) |
| Duration of spasticity (in months) | 95.8 (± 75.0) |
| Use of spasmolytics | 17 |

*Abbreviation: ASIA, American Spinal Injury Association.*
*NOTE. Values are mean (± standard deviation) or number.*

***General characteristics of perceived spasticity***

Fifteen patients (57.7%) described the spasticity in the legs as continuous tension. Flexor and extensor spasms were perceived by 69.2% and 84.6% of the patients, respectively. Eighteen subjects (69.2%) had clonus. Additionally, two patients described spasticity in different ways (slow contraction of muscles in toes and burning sensation, and slowly increasing tension).

More than half of the patients (57.7%) found the perceived spasticity problematic, mostly due to decreasing function (73.3%) rather than pain (33.3%). Other problems, such as annoyance, contractures and wounds, as a result of spasticity, were mentioned by six patients (23.1%).

In 20 patients (76.9%) the manifestation of spasticity was affected by infection or inflammation. Change of posture (92.3%), time of the day (53.8%), full bladder or bowel (50%), skin problems (42.3%), tight clothing (26.9%) and emotions or mental stress (23.1%) were also mentioned. Fifteen patients added other factors, of which temperature/climate (both coldness and heat), external stimuli (like riding on rough surface) and fatigue were the most frequently mentioned.

***Activities with high degree of perceived spasticity***

Altogether 82 activities were included. The registered activities were divided into six main groups (table 5.2): 'Changing position' was the largest group (22.0%). Other groups of activities were 'Making a transfer' (20.7%), 'Activities of daily living' (17.1%), 'Being active' (17.1%) and 'Stable body position' (12.2%). The 'Stable body position' group included activities during which the patient remains in the same position for a long time. The remaining group consisted mainly of reactions on stimuli, and thus contained no specific activities (11.0%). This group was, therefore, left out of further analysis.

**Table 5.2:** Classification of activities (n=82) with a high degree of perceived spasticity

| Group | n | Examples |
|---|---|---|
| **1. Changing position** | 18 | Changing position from sitting to lying down<br>Changing position from lying down to sitting<br>Changing position of arms in wheelchair<br>Changing bed position<br>When the knees are extended |
| **2. Making a transfer** | 17 | Transfers (both lifted and sliding, or not specified)<br>Transfer into/out of the car<br>When muscles stretch during transfer |
| **3. Activities of daily living** | 14 | Washing and clothing<br>Taking a shower<br>Catheterization of bladder |
| **4. Being active** | 14 | During physiotherapy<br>Handbiking<br>Riding in wheelchair (outside, irregular road)<br>Starting to move |
| **5. Stable body position** | 10 | Lying in bed<br>Sitting in wheelchair for a long time |
| **6. Other** | 9 | When startling<br>When legs are touched<br>When breathing in deeply in bed<br>When yawning in the morning in bed<br>After activity |



In table 5.3, the types of manifestation per group of activities are presented. Extensor spasms were the most commonly occurring manifestation in all groups except in the 'Stable body position' group, where flexor spasms were the most common (87.5%).

**Table 5.3:** Type of spasticity per group of activities (%)

| Group | N | Tension | Flexor spasms | Extensor spasms | Clonus | Other |
|---|---|---|---|---|---|---|
| **1. Changing position** | 13 | 30.8 | 53.8 | 61.5 | 30.8 | 15.4 |
| **2. Making a transfer** | 14 | 50.0 | 42.9 | 78.6 | 35.7 | 0 |
| **3. Activities of daily living** | 8 | 41.7 | 66.7 | 66.7 | 41.7 | 8.3 |
| **4. Being active** | 12 | 45.5 | 45.5 | 54.5 | 36.4 | 9.1 |
| **5. Stable body position** | 11 | 37.5 | 87.5 | 62.5 | 37.5 | 0 |

***Relationship between perceived spasticity and spasticity-related discomfort***

The (mixed) group of activities with the highest VAS score per patient had a median VAS of 7.6 (range 2.5 – 9.5) and a median Borg of 6.0 (range 0.0 – 10.0). The correlation between VAS and Borg scale was moderate (Spearman's rho = 0.53, $p < 0.01$).

The association between VAS and Borg scores was also calculated for all five groups of activities (see table 5.4). For the groups 4 ('Being active') and 5 ('Stable body position') the correlation between the VAS and Borg score was marked (rho = 0.71 and 0.78, respectively) and statistically significant ($p = 0.01$ and 0.02). For group 3 ('Activities of daily living') the correlation was moderate (rho = 0.58) but statistically significant ($p = 0.046$). For the other two groups correlation was low and not significant.

**Table 5.4:** Median VAS and Borg scores (range) per group and correlations between VAS and Borg

| Group | N | VAS | Borg | Spearman's rho |
|---|---|---|---|---|
| **1. Changing position** | 13 | 6.8 (2.5-9.5) | 3.0 (1.0-7.0) | 0.34 |
| **2. Making a transfer** | 14 | 6.9 (2.1-9.0) | 3.5 (1.0-10.0) | 0.37 |
| **3. Activities of daily living** | 8 | 7.4 (4.6-8.9) | 6.5 (0.0-10.0) | 0.58* |
| **4. Being active** | 12 | 5.1 (1.2-8.9) | 3.0 (0.5-8.0) | 0.71* |
| **5. Stable body position** | 11 | 6.9 (3.5-8.2) | 4.0 (1.0-10.0) | 0.78* |

* $p < 0.05$

Five patients mentioned positive effects of spasticity. Some described it as practical, for example, while getting dressed, especially when putting on trousers, or when stretching the trunk. Another subject experienced it as a pleasant feeling because an outburst of spasms relaxes the muscles afterwards. Prevention of muscle atrophy and prevention of skin sores were mentioned as well.

Several patients indicated that they had experienced high discomfort at the onset of the symptoms, but later they had got used to the spasticity and it was no longer regarded as problematic. One patient said that it had taken her time to learn how to interpret the new body signals. Another patient used to be ashamed of the

spasticity shortly after his injury, but not anymore. Most patients had found ways to suppress spasms or clonus when they felt it coming up, for example, by pushing with hands or arms on the knees or by changing position.
Presence of high discomfort at the time of participation in this study was usually associated with the fear of falling (for example, out of the wheelchair, in the shower) or otherwise dangerous situations, such as driving.

## Discussion

Spasticity is a multidimensional phenomenon, which seems to be looked upon in different ways by patients and clinicians. It is important to have insight in these different perceptions, especially when considering treatment to reduce spasticity, to be able to meet the patients' needs.
The aim of this study was to provide epidemiological data on the manifestation of spasticity in patients with motor complete SCI and to investigate how the perceived degree of spasticity and spasticity-related discomfort are associated.
A high degree of spasticity was most frequently perceived during 'Changing position' and 'Making a transfer'. These two groups of activities partially overlap, as part of the perceived spasticity during transfers is possibly elicited by changing position. Other explanations for perceived spasticity during transfers are touching the skin, increasing abdominal pressure or great physical exertion. From clinical observations, it is known that extension of the hips can elicit spasms, particularly extensor spasms. Flexor spasms are usually seen after skin stimuli. Experimental observations have confirmed that proprioceptive stimuli from the hip are the most likely triggers for extensor spasms in patients with SCI.[17,18] The influence of knee joint rotation in provoking extensor spasms is also described.[18] In addition to skin stimuli, imposed movements of the ankle or knee joint can also elicit flexor reflexes in SCI.[19,20]
Perception of spasticity by ASIA-A subjects is hard to explain in neurological

context. Spasms and clonus, causing limb movement, can obviously be perceived indirectly, for example, by vision. A possible consequence of continuous tension might be the increased difficulty in performing the activities.

A highly interesting finding in this study is the modest correlation between perceived spasticity and spasticity-related discomfort during activities as measured with the VAS and Borg scale, respectively. Correlation between the VAS and Borg scale is higher during activities involving a 'stable body position' and while 'being active'. This finding confirms the idea that the impact of spasticity on daily life is related to the context in which it occurs. It would appear that in some situations, a high degree of spasticity is disturbing, while in other situations, the patient can adapt more easily. The experienced discomfort can be based on practical reasons, such as disturbance of sleep, compromised safety, and so on, or on psychological factors such as coping strategies or negative self-image. The latter is supported by the indication that overall negative impact of spasticity seemed to decrease with time since injury, as was brought up by a number of patients in this study. This finding agrees with other observations.[3,7,8] Perhaps the growing acceptance of their situation, better understanding of the altered body functions and/or finding ways how to deal with it, can explain the diminishing impact of spasticity on daily life of patients with SCI through the years. These observations imply that treatment of spasticity might serve different purposes for patients in different phases after injury, as 'confounding' of the patients' perception seems to change throughout time. This information is of high clinical interest as it might improve communication between patients and clinicians. However, further research is needed to clarify the complexity of this process.

## Conclusions

Patients with motor complete SCI experienced several manifestations of spasticity. Extensor spasms, mainly elicited by activities incorporating change of position,

were most common.

Self-ratings on the perceived degree of spasticity by the patients should be added to the set of assessment methods for the evaluation of spasticity. It is useful to distinguish between the perceived degree of spasticity and experienced spasticity-related discomfort, because they were only moderately associated. How these self-rating scores relate to spasticity in terms of involuntary muscle activity, assessed with more objective tools, will be an interesting area for further research.

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# 6

# Patient ratings of spasticity during daily activities are only marginally associated with long-term surface electromyography

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## Abstract

This study aimed to investigate the association between subjective spasticity ratings and objective spasticity measurement using a new tool for spasticity assessment, that is long-term surface electromyography (sEMG) recordings during daily activities. For monitoring, processing and analysis of this long-term sEMG data, a muscle activity detection algorithm was developed.

Surface EMG of the rectus femoris, vastus lateralis, adductor group, and semitendinosus of 14 complete spinal cord injured patients, in whom voluntary muscle contraction was absent, was recorded continuously during daily activities. Synchronously, subjects stored their activities in a diary and scored their experienced level of spasticity on a Visual Analogue Scale (VAS) for that particular activity. sEMG data were analyzed using a high quality burst detection algorithm that was developed and validated within this study. Derived sEMG parameters were clustered using Principal Component Analysis (PCA) and used in a linear mixed model analysis to study their association with VAS.

Results showed that VAS scores appeared significantly associated with the PCA components representing the number and the duration of bursts, but not burst amplitude. Furthermore, VAS scores were associated with the activity performed. The percentage explained variance was however low, that is 27 - 35%.

It was concluded that patient ratings of the level of spasticity appear poorly associated with spasticity in terms of involuntary muscle activity assessed with long-term sEMG recordings. It is likely that other factors such as pain and cognitions are also incorporated in these patient ratings. Clinicians are therefore strongly advised to perform complementary objective assessments using long-term sEMG recordings.

# Introduction

Spasticity affects about 12 million people all over the world.[1] Several definitions have been provided in the literature to describe this phenomenon. Although Lance's definition[2] of spasticity is the most cited definition, it has also been considered to be too narrow.[3] The umbrella definition of Pandyan and colleagues, "spasticity is a sensori-motor disorder resulting from upper motor neuron lesion (UMNL) presenting as intermittent or sustained involuntary activation of muscles",[3] was therefore recently introduced.

Spasticity is associated with impaired motor control, pain and joint deformity, and interferes with activities of daily living and quality of life.[4] As a result, its management is a major goal in rehabilitation.[5] Proper management requires sound assessment methods for spasticity, which can be classified into objective and subjective. Objective methods concern biomechanical and neurophysiological approaches. In particular, neurophysiological methods, using surface electromyography (sEMG) to quantify muscle activity, are close to the definition of Pandyan[3] and may thus be considered valid. A main disadvantage is that these methods are not suitable for clinical use. For this purpose, subjective methods are employed, which comprise besides ratings from clinicians, for example the Ashworth scale,[6] also patient ratings, whether or not using a 'standardized' measure such as the Visual Analogue Scale (VAS).[7] Subjective ratings commonly direct the decision on and evaluation of spasticity management strategies. A clear disadvantage of this approach is, however, that subjectivity inherently introduces measurement error.[8] Furthermore, the use of subjective ratings, for example from the patient, to evaluate spasticity management strategies directed at reducing muscle activation, implies an association between these subjective ratings and objective measurements of involuntary muscle activity (sEMG). Evidence on this relation is largely lacking,[5] however, but it is required because a dissociation might imply suboptimal management evaluations with all its associated consequences.

Both objective and subjective assessment approaches face problems with

ecological validity: observations are commonly performed at one specific moment in time, thereby ignoring fluctuations of spasticity over the day due to temporal and environmental factors.[5,9,10] Momentary assessment is thus likely to be limitedly representative for spasticity experienced in normal daily life. There is a clear need for a spasticity assessment method that incorporates the requirements of objectivity and usability outside the laboratory during normal daily life. Long-term sEMG monitoring fulfills these requirements. A few studies have reported on this method several decades ago.[11-13] sEMG recordings were performed in complete spinal cord injured (SCI) patients in whom periods of muscle activation can be considered spasticity as voluntary contractibility is lost. Due to technical limitations at that time, the method never matured: sEMG data were analyzed by visual inspection only,[14] rather than using objective criteria combined in an automated algorithm. Recent advances in technology enable the development of such algorithms and to ultimately use this for spasticity assessment. Herewith, new opportunities arise to further scrutinize the association between subjective (patient ratings) and objective measures of spasticity: instead of comparing both measures obtained non-simultaneously in the clinic and laboratory, it is now possible to study this association during daily life, obtained simultaneously. Because of the important role of subjective ratings in spasticity management evaluation, knowledge on this association is highly useful.

This study aimed at investigating the association between subjective patient ratings on the level of spasticity on one hand and objective spasticity measurement using long-term sEMG recordings during daily activities on the other hand. For proper monitoring, processing and analysis of this long-term data, a muscle activity detection algorithm was developed.

# Methods

### *Subjects*

Fourteen motor complete chronic SCI patients (lesion above Th12) were included. All patients reported to experience spasticity in the upper leg(s). Spasticity of the hip adductors, hip abductors, and hip and knee flexors and extensors was additionally assessed clinically using the Ashworth scale.[15] Severe contractures and pain that might interfere with the measurements were exclusion criteria. The study was approved by the Medical Ethics Committee of Roessingh, Enschede (NL), and subjects signed informed consent prior to participation. General demographic characteristics are presented in table 6.1.

**Table 6.1:** Sociodemographic characteristics

| **Subject** | **Gender** | **Age** (years) | **Time since lesion** (months) | **Level of impairment (motor)** | **Level of impairment (sensory)** | **ASIA** | **AS hip add** | **AS hip abd** | **AS hip ext** | **AS hip flex** | **AS knee ext** | **AS knee flex** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| M01 | M | 51 | 7 | C6 | C7 | B | 1 | 0 | 1 | 0 | 0 | 1 |
| M02 | M | 31 | 16 | C5 | C5 | A | 1 | 1 | 2 | 1 | 0 | 0 |
| M03 | F | 45 | 18 | C5 | C5 | A | 1 | 1 | 1 | 1 | 2 | 1 |
| M04 | M | 40 | 187 | C5 | C4 | B | 3 | 1 | 0 | 1 | 0 | 0 |
| M05 | M | 37 | 229 | C5 | C4 | A | 1 | 1 | 0 | 0 | 0 | 1 |
| M06 | M | 35 | 90 | Th5 | Th7 | A | 2 | 0 | 1 | 2 | 0 | 1 |
| M07 | M | 51 | 26 | Th3 | Th3 | A | 1 | 0 | 0 | 1 | 1 | 2 |
| M08 | M | 55 | 42 | Th8 | Th8 | A | 3 | 0 | 1 | 3 | 2 | 3 |
| M09 | M | 40 | 147 | Th4 | Th4 | A | 3 | 2 | 0 | 2 | 0 | 0 |
| M10 | F | 33 | 32 | Th7 | Th7 | A | 3 | 0 | 0 | 0 | 0 | 0 |
| M11 | F | 28 | 89 | C6 | Th6 | A | 0 | 2 | 0 | 0 | 0 | 0 |
| M12 | M | 46 | 30 | Th3 | Th3 | A | 0 | 0 | 0 | 0 | 0 | 0 |
| M13 | F | 25 | 138 | C6 | C5 | A | 2 | 0 | 0 | 0 | 0 | 0 |
| M14 | M | 31 | 163 | C7 | Th2 | B | 2 | 3 | 0 | 0 | 1 | 2 |

*Abbreviation: AS, Ashworth scale.*

***Measurement protocol***

Each subject was measured at two or three days, with a cumulative minimum of about 10 hours, during normal daily activities. sEMG was recorded continuously, and patients noted each activity in a diary along with a score on the subjectively experienced level of spasticity during that particular activity, using the VAS.

*sEMG recordings*

Skin preparation and electrode (bipolar, pre-gelled ARBO H93, interelectrode distance 24 mm) placement were performed according to international guidelines for sensor placement.[16] Activity of four muscles was recorded: the rectus femoris (RF), the vastus lateralis (VL), the adductor group (including gracilis and adductor magnus) (AD) and the semitendinosus (ST). The reference electrode was placed at the lateral malleolus. Electrodes were connected to a portable measurement and storage device (Mobi, sample freq 1024 Hz, manufactured by TMSi, Oldenzaal, The Netherlands) by means of cables taped to the skin (figure 6.1).

*Diary*

Subjects were instructed to note their activities including start and end times meticulously in a diary. Examples of activities were making transfers, reading, etc. For each activity the experienced *level* of spasticity assessed with VAS was noted in the diary as well: patients were explicitly instructed that this could be deviant from the experienced *hindrance* of spasticity. The VAS consisted of a 100 mm horizontal line, with 'no spasticity' and 'spasticity as bad as it can be' at the two extremes.[7] Patients with sufficient hand function marked the line at the position they felt corresponded best to their experienced level of spasticity. For subjects without sufficient hand function, the experimenter was continuously available for assistance. The experimenter slowly moved a pencil from the left to the right extremity of the VAS, and the mark was placed at the position verbally instructed by the patient. Indications for sufficient psychometric properties of the VAS for spasticity have been shown.[17]

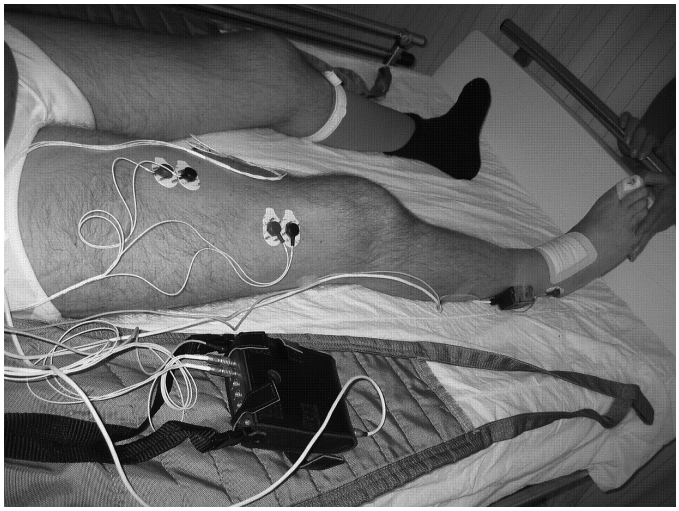

**Fig 6.1:** Electrodes connected to a portable measurement and storage device, with cables taped to the skin

***Data reduction***

sEMG was band-pass filtered at 30 – 500 Hz. This is a common filter setting for long-term sEMG monitoring during which movement artifacts are likely to occur.[18] Beginnings and endings of bursts of muscle activity were subsequently detected using custom-made software based on the Approximated Generalized Likelihood Ratio (AGLR) algorithm developed by Staude.[19] This algorithm detects time instances that correspond to sudden changes in the variance of the signal. A postprocessor was then developed to detect which changes in variance indeed corresponded to bursts in muscle activity. For this purpose, two experts (LK and GV) independently manually marked starts and endings of bursts in a random subset of data from seven patients. Data marking by experts has the advantage that the results of burst detection coincide with human intuitive judgment.[20] The marks corresponded to a subset of changes in variance that were detected with the AGLR algorithm. Postprocessor criteria defining when a detected change in variance corresponded to a start or end of a burst were agreed on by the experts, also based on existing literature:

1) The non-burst value of the sEMG was assessed by taking the minimum value of 100 randomly selected 1 second data samples across the signal;
2) The start of a burst was defined when the change in variance detected by the AGLR algorithm exceeded twice the RMS value of the non-burst RMS value;
3) The minimum burst duration was set to 100 ms, to prevent that activity of single motor units was considered a burst;
4) The minimum period between two bursts was set to 200 ms, since the electromechanical delay of a muscle is longer when muscle activity is ended than when it is started;
5) Bursts with an amplitude of >1000 μV were excluded, as these were considered to be artifacts.

These thresholds correspond quite well to what can be derived from physiological characteristics of motor control.[21,22] Using this algorithm, the mean and standard deviation of the RMS amplitude and duration across all bursts were calculated, as well as the number of bursts during an activity, resulting in five variables for the four muscles.

The 'quality' of the algorithm with regard to detecting bursts was evaluated using data from the second group of seven patients. Fourteen data samples of 2 minutes duration (two data samples per patient) were randomly selected, and the beginnings and endings of bursts were marked by the two experts independently. The percentage agreement on the number of bursts detected between experts and algorithm was considered indicative of the 'quality' of the burst detection algorithm and was calculated.

***Statistical analysis***

The 20 sEMG variables were calculated for each activity scored. The variables were anticipated to be interrelated, and so a Principal Component Analysis (PCA) was performed to reduce their number. Requirements for normality, linearity, singularity, and multicollinearity were explored, and (random) missing values were

replaced by the mean. The sEMG variables were generally not normally distributed, and so logarithmic transformations were performed, resulting in acceptable normality. There is no one singular approach for extracting the 'right' number of components in PCA, but one of the most often used methods is to plot the eigenvalues against components in descending order in a so-called scree-plot[23] and to extract components with eigenvalues over 1. Besides this, the 'optimum' number of components extracted also needs to comply with the requirements of interpretability.[24] Orthogonal, varimax rotation was used, and the Kaiser-Meyer-Olkin measure and Bartlett tests were evaluated for testing sampling adequacy and sphericity. Variables that loaded fairly ($> |0.4|$)[25] on more than one component were removed.

The components, representing objective quantifications of spasticity, were studied for their association with self-rated spasticity (VAS). A initial analysis contained only the principal components as fixed factors and provided an insight into the relative association between involuntary muscle activity and spasticity rated by the patient. To include context dependency, a second analysis was performed, containing, next to the components derived from the PCA, also the fixed factors 'part of the day' (dichotomized into morning and afternoon/evening) and 'activity' performed by the subject. Activities were classified into: 1. Transfers (including activities inducing an obvious change in body position (change in muscle length)); 2. Activities of daily living; 3. Being active; 4. Therapy; 5. Stable body position; and 0. Other.

For both models, the random factor included was 'subject'. The factors were entered in the model, and only significant factors remained ($p < 0.05$) after manual backward elimination. The percentages of explained variance (first level $R^2$) for the final models were calculated according to the formula of Snijders and Bosker[26]. Model fits were reflected in −2 log likelihood.

# Results

### *Quality of the algorithm*

The 'quality' of the algorithm was studied by comparing the number of bursts detected by the experts and the number of bursts detected by the algorithm. The algorithm detected slightly more bursts than defined by the experts together (161 versus 156; i.e. 3%). The percentage of agreement between experts and algorithm was thus high, that is 97%.

### *Description of data*

Figure 6.2 shows an example of RF activity during dressing (2-6 minutes), transferring (10-15) and quiet sitting (15-40). Fourteen subjects scored 263 activities (table 6.2).

**Table 6.2:** Number of activities, related Visual Analogue Scale (VAS) scores, and number of hours recorded per patient

| **Patient** | **Summed duration recordings** (hours) | **No of activities scored with VAS** | **Median and interquartile range VAS** |
|---|---|---|---|
| 1 | 12.4 | 17 | 5 (0 – 13.5) |
| 2 | 9 | 10 | 39.5 (7.5 – 68.5) |
| 3 | 3.5* | 17 | 33 (0 – 50) |
| 4 | 12.2 | 34 | 36.5 (8.75 – 73.3) |
| 5 | 11.2 | 22 | 1.5 (0 – 8.3) |
| 6 | 10.6 | 24 | 42.5 (27.5 – 61.5) |
| 7 | 10.1 | 20 | 65 (47 – 72.8) |
| 8 | 14.1 | 15 | 29 (10 – 40) |
| 9 | 18.6 | 7 | 27 (0 – 42) |
| 10 | 16.1 | 17 | 32 (7 – 57.5) |
| 11 | 16.9 | 15 | 18 (13 – 28) |
| 12 | 13.3 | 13 | 13 (8 – 22) |
| 13 | 12.2 | 26 | 19 (6.8 – 32) |
| 14 | 14.5 | 26 | 22.5 (13.8 – 32.5) |

** Because of technical errors, only data for one measurement were suitable for analysis*

'Transfers' were the activities scored most often (47%, including transfers from sitting to supine and vice versa as well as sitting sitting transfers), followed by 'Activities of daily living' (24%, e.g. getting dressed), 'Being active' (13%, e.g. performing sports), 'Stable body position' (7%, e.g. working behind computer), 'Therapy' (5%, e.g. occupational and physical therapy), and 'Other' (4%, e.g. emotional conversations, clinical evaluations). The sEMG burst data for each group of activities are provided in table 6.3.

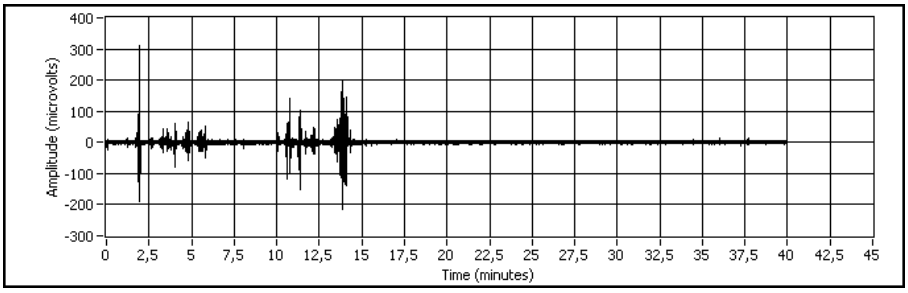


**Fig 6.2:** Muscle activity of the rectus femoris during activities of daily life



***sEMG components defined by PCA***

PCA results indicated the extraction of seven components, as for these components, eigenvalues were > 1 (figure 6.3). Inspection of the component loadings indicated that all variables were strongly loaded on one component only (see table 6.4). In addition, the residual correlation matrix indicated a good fit between observed and reproduced correlations, and the Kaiser-Meyer-Olkin measure was 0.62, which fulfills the minimum requirement for satisfactory PCA analysis. Finally, the interpretability of the factors was satisfactory. Therefore, the seven-component structure was maintained (see table 6.4).

**Table 6.3:** Median and interquartile range scores for the separate sEMG variables

| | **Activity 1** | **Activity 2** | **Activity 3** | **Activity 4** | **Activity 5** | **Activity 0** |
|---|---|---|---|---|---|---|
| Mean RMS RF | 9.3 | 9.5 | 6.6 | 9.6 | 9.7 | 10.5 |
| | (6.5 – 12.7) | (6.5 – 12.5) | (5.5 – 9.8) | (5.7 – 14.5) | (7.4 – 15.5) | (9.4 – 11.9) |
| Mean RMS VL | 9.9 | 8.4 | 6.2 | 12.8 | 9.2 | 8.8 |
| | (7.2 – 14.4) | (5.8 – 14.9) | (4.8 – 12.5) | (7.0 – 18.6) | (5.4 – 18.5) | (6.1 – 12.5) |
| Mean RMS AD | 7.2 | 8.2 | 7.6 | 6.9 | 7.8 | 7.2 |
| | (5.2 – 11.2) | (5.6 – 10.6) | (5.0 – 10.2) | (5.6 – 14.2) | (6.1 – 14.5) | (5.8 – 10.1) |
| Mean RMS ST | 8.4 | 9.2 | 6.8 | 5.6 | 7.4 | 6.6 |
| | (6.3 – 10.5) | (7.6 – 12.2) | (4.8 – 7.2) | (4.6 – 7.9) | (5.8 – 9.4) | (5.6 – 16.7) |
| Sd RMS RF | 5.9 | 5.7 | 4.8 | 6.2 | 8.8 | 6.5 |
| | (2.9 – 10.1) | (2.3 – 10.8) | (3.4 – 7.5) | (2.0 – 11.9) | (4.7 – 16.9) | (5.7 – 9.7) |
| Sd RMS VL | 6.7 | 7.3 | 5.0 | 6.9 | 5.0 | 5.8 |
| | (3.6 – 12.2) | (2.3 – 13.7) | (1.0 – 9.4) | (3.6 – 16.8) | (0.9 – 15.0) | (2.4 – 16.7) |
| Sd RMS AD | 4.0 | 4.4 | 3.7 | 4.6 | 5.8 | 3.5 |
| | (1.8 – 7.4) | (2.1 – 6.9) | (1.5 – 13.2) | (2.1 – 15.4) | (2.7 – 13.2) | (2.3 – 5.7) |
| Sd RMS ST | 4.4 | 6.7 | 2.6 | 2.8 | 3.3 | 3.6 |
| | (2.5 – 8.3) | (3.4 – 10.9) | (1.7 – 5.2) | (2.0 – 4.3) | (2.7 – 8.2) | (2.2 – 8.6) |
| | | | | | | |
| Number of bursts RF | 4 | 5 | 11 | 7 | 8 | 7 |
| | (4 – 13) | (2 – 11.5) | (2.5 – 46) | (1 – 21.5) | (3.3 – 33.8) | (4 – 9) |
| Number of bursts VL | 13 | 8 | 54 | 26 | 12.5 | 8 |
| | (4 – 21.3) | (3 – 27.5) | (8 – 85) | (6 – 69.5) | (1.8 – 25.6) | (6 – 16) |
| Number of bursts AD | 6 | 5.5 | 5 | 13 | 6 | 3 |
| | (3.3 – 11) | (2 – 13.3) | (2 – 35) | (2 – 29.5) | (1.8 – 48.5) | (2 – 7) |
| Number of bursts ST | 7 | 6 | 7.5 | 7.5 | 15.5 | 6.5 |
| | (4 – 13) | (2 – 12) | (4 – 16.5) | (4 – 16.5) | (2.8 – 27.0) | (3.0 – 18.8) |
| | | | | | | |
| Mean burst duration RF | 1.22 | 1.0 | 0.9 | 0.8 | 1.0 | 2.9 |
| | (0.5 – 2.7) | (0.4 – 2.9) | (0.3 – 1.7) | (0.5 – 31.5) | (0.5 – 1.8) | (2.0 – 4.3) |
| Mean burst duration VL | 0.7 | 2.3 | 0.4 | 0.7 | 0.6 | 1.8 |
| | (0.4 – 1.8) | (0.3 – 3.1) | (0.2 – 0.8) | (0.2 – 2.0) | (0.4 – 1.7) | (1.0 – 2.4) |
| Mean burst duration AD | 3.2 | 2.1 | 2.1 | 3.2 | 3.1 | 4.5 |
| | (1.4 – 6.4) | (0.7 – 4.6) | (0.5 – 3.2) | (1.7 – 4.4) | (2.2 – 6.5) | (3.1 – 5.2) |
| Mean burst duration ST | 5.3 | 3.1 | 2.5 | 3.4 | 3.0 | 4.6 |
| | (2.6 – 8.5) | (1.2 – 7.1) | (0.9 – 9.1) | (2.9 – 8.7) | (1.4 – 3.4) | (3.8 – 5.0) |
| Sd burst duration RF | 1.6 | 1.3 | 1.0 | 2.6 | 0.7 | 3.0 |
| | (0.6 – 2.7) | (0.4 – 2.7) | (0.3 – 2.0) | (1.1 – 175.4) | (0.2 – 2.9) | (2.2 – 4.9) |
| Sd burst duration VL | 1.2 | 1.1 | 0.7 | 3.1 | 0.5 | 2.3 |
| | (0.5 – 2.4) | (0.6 – 2.4) | (0.4 – 1.9) | (0.6 – 17.6) | (0.1 – 2.7) | (1.6 – 3.6) |
| Sd burst duration AD | 3.5 | 2.4 | 1.7 | 3.8 | 2.3 | 3.9 |
| | (1.7 – 5.7) | (1.3 - 3.8) | (0.5 – 3.8) | (2.1 – 5.5) | (1.1 – 4.1) | (1.5 – 5.0) |
| Sd burst duration ST | 5.1 | 3.5 | 39.5 | 4.8 | 4.1 | 3.7 |
| | (2.8 – 7.9) | (1.7 – 7.0) | (2.0 – 67.5) | (1.1 – 8.4) | (2.3 – 5.3) | (2.6 – 6.1) |

*Legend: Activity 1. Transfers (including activities inducing an obvious change in body position (change in muscle length)); 2. Activities of daily living; 3. Being active; 4.Therapy; 5. Stable body position; 0. Other. Abbreviations: AD, adductor group; RF, rectus femoris; RMS, Root Mean Square; sd, standard deviation; ST, semitendinosus; VL, vastus lateralis.*

**Table 6.4:** Loadings, percentage of variance for principal components extraction and varimax rotation on sEMG variables

| | Comp.1 | Comp.2 | Comp.3 | Comp.4 | Comp.5 | Comp.6 | Comp.7 |
|---|---|---|---|---|---|---|---|
| Mean burst duration AD | **,686** | -,080 | -,058 | -,046 | -,042 | -,042 | ,299 |
| Mean burst duration ST | **,832** | -,129 | -,004 | ,176 | ,082 | ,001 | -,030 |
| Sd burst druration AD | **,685** | ,006 | ,088 | ,020 | -,033 | ,024 | ,338 |
| Sd burst duration ST | **,791** | -,081 | ,069 | ,217 | ,166 | ,061 | -,093 |
| Mean RMS RF | ,055 | **,789** | ,077 | -,225 | ,076 | ,166 | ,055 |
| Mean RMS AD | -,256 | **,754** | ,129 | ,205 | ,084 | ,025 | -,042 |
| Sd RMS RF | ,082 | **,795** | ,147 | -,261 | ,070 | ,120 | ,120 |
| Sd RMS AD | -,293 | **,718** | ,200 | ,169 | ,130 | ,052 | -,042 |
| Number of bursts RF | ,127 | ,180 | **,769** | -,080 | ,123 | ,044 | ,138 |
| Number of bursts VL | ,214 | ,131 | **,693** | ,039 | -,161 | -,183 | ,010 |
| Number of bursts AD | -,071 | ,138 | **,760** | ,038 | ,039 | ,200 | ,032 |
| Number of bursts ST | -,138 | ,033 | **,760** | -,006 | -,027 | ,164 | -,034 |
| Mean burst duration RF | ,164 | -,047 | -,015 | **,918** | ,061 | ,022 | ,119 |
| Sd burst duration RF | ,139 | -,049 | ,006 | **,919** | ,078 | ,027 | ,130 |
| Mean RMS ST | ,091 | ,144 | -,103 | ,075 | **,914** | ,077 | ,015 |
| Sd RMS ST | ,044 | ,123 | ,085 | ,059 | **,924** | ,066 | -,015 |
| Mean RMS VL | ,018 | ,098 | ,046 | -,014 | ,044 | **,923** | -,070 |
| Sd RMS VL | ,022 | ,187 | ,190 | ,065 | ,104 | **,882** | ,034 |
| Mean burst duration VL | ,103 | -,008 | ,007 | ,090 | ,032 | -,002 | **,855** |
| Sd burst duration VL | ,174 | ,093 | ,105 | ,143 | -,031 | -,039 | **,839** |
| **Eigenvalues** | **3,770** | **3,359** | **2,096** | **1,683** | **1,620** | **1,334** | **1,026** |
| **Cumulative percentage of explained variance** | **18,848** | **35,645** | **46,126** | **54,543** | **62,641** | **69,312** | **74,443** |

*High component loadings on a variable are printed in bold.*
*Abbreviations: AD, adductor group; RF, rectus femoris; RMS, Root Mean Square; sd, standard deviation; ST, semitendinosus; VL, vastus lateralis.*

Components 1, 4, and 7 were composed by the mean and standard deviation of the burst duration of the AD and ST, RF and VL respectively. On components 2, 5, and 6, on the other hand, the mean and standard deviation of burst activity (RMS) of the RF and AD, ST and VL were loaded respectively. Finally, component 3 consisted of the number of bursts of each of the four muscles.

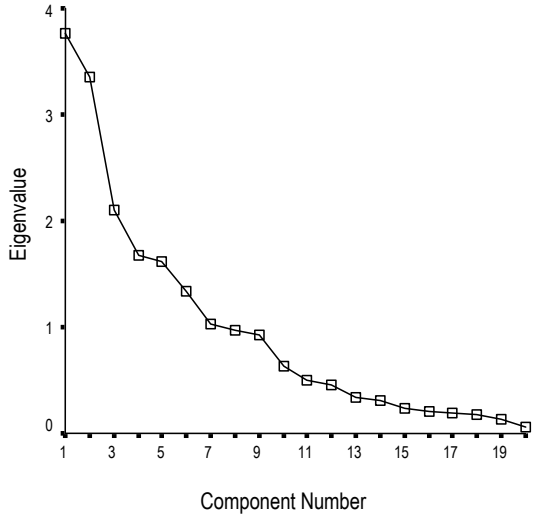


**Fig 6.3:** Scree plot

***Association between VAS, sEMG components, and context***

Seven fixed factors (the seven components) and one random factor (i.e. subject) were entered in the first mixed linear model, with VAS being the dependent measure. The first, third and seventh component were significantly associated with VAS (see table 6.5): patients reported higher levels of experienced spasticity with increasing duration of AD, ST and VL bursts, and a larger number of bursts. This model (-2 log likelihood = 2321.7 compared with 2377.5 for model without fixed factors) explained 27% of the variance in VAS.

The second analysis also integrated the context variables 'part of the day' and 'activity'. Again, components 1, 3, and 7 were significantly associated with VAS,

and the factor 'activity' showed a significant relation (see table 6.5). Higher levels of experienced spasticity were reported with increasing duration of AD, ST and VL burst duration, and a larger number of bursts. In addition, the level of spasticity experienced during activities depended on which activity was being performed. For activities classified as 'Transfers' (median VAS score 30.5; interquartile range 13 – 56.8) and 'Other' (39.0; 0 – 74) significantly higher VAS scores were reported compared with activities classified as 'Stable body position' (12.5; 1 – 41.5), while 'Activities of daily living' (16.5; 3 – 39.3), 'Being active' (16.0; 6 – 34), and 'Therapy' (18; 0 – 55) did not (see table 6.5). This model (-2 log likelihood = 2273.6, compared with 2377.5 for model without fixed factors) explained 35% of the variance in VAS.

**Table 6.5:** Multilevel models

| Parameter | Estimate | Std. Error | df | t | Sig. | 95% CI |
|---|---|---|---|---|---|---|
| *Model incorporating sEMG components only* | | | | | | |
| **Intercept** | 29.30 | 3.84 | 13.09 | 7.63 | 0.00 | 21.01 – 37.60 |
| **Component 1** | 6.43 | 1.35 | 257.88 | 4.76 | 0.00 | 3.77 – 9.09 |
| **Component 3** | 6.38 | 1.36 | 258.21 | 4.70 | 0.00 | 3.70 – 9.05 |
| **Component 7** | 4.16 | 1.27 | 252.53 | 3.27 | 0.00 | 1.66 – 6.67 |
| *Model incorporating sEMG components and context factors* | | | | | | |
| **Intercept** | 20.78 | 5.90 | 61.10 | 3.52 | 0.00 | 8.99 – 32.57 |
| **Activity= 0** | 18.31 | 7.44 | 244.91 | 2.46 | 0.02 | 3.65 – 32.96 |
| **Activity = 1** | 12.69 | 4.84 | 243.71 | 2.62 | 0.01 | 3.16 – 22.22 |
| **Activity = 2** | 4.49 | 5.12 | 243.87 | 0.88 | 0.38 | -5.59 – 14.58 |
| **Activity = 3** | -1.83 | 5.61 | 243.62 | -0.33 | 0.75 | -12.88 – 9.22 |
| **Activity = 4** | 13.51 | 7.04 | 246.01 | 1.92 | 0.06 | -0.35 – 27.37 |
| **Activity = 5** | 0(a) | 0.00 | . | . | . | |
| **Component 1** | 5.34 | 1.32 | 252.27 | 4.04 | 0.00 | 2.74 – 7.94 |
| **Component 3** | 6.90 | 1.34 | 252.35 | 5.16 | 0.00 | 4.27 – 9.53 |
| **Component 7** | 3.12 | 1.26 | 247.52 | 2.49 | 0.01 | 0.65 – 5.59 |

*Legend: Activity 1. Transfers (including activities inducing an obvious change in body position (change in muscle length)); 2. Activities of daily living; 3. Being active; 4. Therapy; 5. Stable body position; 0. Other. Dependent Variable: VAS*

## Discussion

The aim of this study was to investigate the association between subjective patient ratings on the level of spasticity, on the one hand, and objective spasticity measurement using a new tool, that is long-term sEMG recordings during daily activities, on the other hand. Fourteen motor complete SCI patients performed their normal daily activities and scored their experienced level of spasticity on a VAS, while sEMG of four upper leg muscles (RF, VL, AD, ST) was recorded synchronously. To enable processing and analysis of the sEMG data, an automated burst detection algorithm was developed which proved to be of high quality. The burst duration and number of bursts explained 27% of the variance of the self-rated level of upper leg spasticity, and when relevant context parameters were added the level of explained variance increased to 35%.

The self-rated level of spasticity appeared only marginally (27% to 35%) related to the synchronously recorded objective quantification in burst duration, number of bursts and activity performed. This finding is highly relevant, as it objectifies that opinions of the patient, indicating involuntary muscle activation in the evaluation of management strategies, should be interpreted with caution.

The duration and number of bursts were, though marginally, significantly related to higher levels of patient ratings of spasticity and more relevant than the amplitude of bursts. From a pathophysiological perspective, the occurrence of bursts is associated with the (hyper)excitability of neural pathways due to loss of supraspinal control: increased alpha motor neuron excitability, and decreased presynaptic and recurrent inhibition[5] have been reported in spasticity. As a result, involuntary muscle contractions are more easily evoked by any form of stimulation. Furthermore, it has been shown that the duration of reflexive muscle contraction increases in spasticity.[5] Bursts with longer duration are more likely to be noticed by the patient than shorter bursts, also because these may interfere more seriously with activities. Furthermore, lasting bursts may be associated with development of secondary spasticity symptoms such as contractures.

The weak association between VAS and sEMG may be explained by the fact that patients have difficulties with properly sensing muscle spasticity because the majority of patients had a sensory lesion as well (ASIA A, n = 11). One might hypothesize that the association would thus be different in patients with 'normal' sensibility (ASIA B). Visual inspection of scatters plotting VAS scores with the PCA components, stratified for ASIA A and B, did not provide preliminary evidence for this hypothesis, probably due to small sample, and further research is required. Another explanation is that the discrepancy may originate from the methods used for quantifying spasticity intensity. When considering the classification of these assessment methods according to ICF levels, sEMG assessments are at the level of 'Body Functions and Structures', while VAS ratings are at the level of activities or participation. This means that the VAS score for spasticity intensity is at risk of incorporating more factors than spasticity intensity alone, despite careful instructions to the patients.

But what are these other factors that potentially contribute to patients perceptions of spasticity? Lechner and colleagues[7] showed that complete SCI patients include sensations like pain into their spasticity rating, explaining the discrepancy between self- and clinically rated spasticity. A good example of this dissociation is provided by subject 12 of the current study: Ashworth scores were zero but self-evaluation indicated considerable spasticity. Furthermore, it may be valid to assume that other factors like cognitions, interpersonal and economic factors, and social considerations are integrated in the concept of spasticity by patients. Evidence for this was reported in a well-conducted ethnographic design study by Mahoney et al.[27] The relative contribution of these factors and considerations to the total concept of spasticity is however not clear and needs to be further explored. Furthermore, it would be very interesting to focus on the exploration of the uniformity of the concept of spasticity among patients: variability in this concept might as well have accounted for the low association observed.

Based on existing literature and clinical perceptions, it was hypothesized that spasticity would also be dependent on context variables like time of the day and the activity that was being performed. Sköld[17] showed, for instance, fluctuating hourly VAS ratings in cervical SCI patients. Results of the current study confirmed the relevance of the activity being performed for the level of spasticity experienced and that spasticity was significantly higher during transfers compared with when stable body position was kept. During transfers, knee (and hip) flexion and extension may occur, which causes muscle stretch. Within the light of changed neural pathways like increased alpha motor neuron excitability, this stretch evokes a reflexive muscle contraction that is measured with sEMG[5] and sensed by the subject. Finally, there appeared to be no (linear) association between VAS and time of the day. Subsequent inspection of scatter plots indicated that the patterns are characterized by high inter- and intrasubject variability. Further research should clarify this.

From a methodological perspective several comments have to be made. First of all the quality of the algorithm appeared to be good. Future efforts could be invested in cross-validation of burst detection and the exploration of other sEMG parameters. Second, the sample size was relatively small, and the number of observations available was marginal for what is generally considered justified for PCA. Although one could thus debate the justification of PCA and the validity of the results, it should be noted that sampling adequacy, sphericity, accumulated explained variance (i.e. 74%) and validity of the components in terms of interpretability were all satisfactory. Furthermore, despite the fact that several components consisted of only two variables, the individual loadings were high enough to be robust.[24,25] The small sample size was also accounted for during the multilevel approach: no more than four parameters were included in the models to ensure stability and are herewith stable and valid. However, interaction terms could not be investigated. Therefore, the results of this study need to be interpreted with caution and require validation with larger subject samples. These samples are preferably composed of patients with varying degrees of spasticity.

## Conclusions and clinical implications

Patient ratings on the level of spasticity should be interpreted with caution when evaluating spasticity management aiming at reducing involuntary muscle activity. To date, there has been no gold standard for spasticity assessment. However, monitoring muscle activity in motor complete SCI patients can be considered close to the umbrella definition of spasticity[3] and may therefore be seen as one of the most valid assessment methods. From this perspective, the results of this study strongly suggest that patient ratings are invalid for spasticity assessment. This stresses the need for clinically applicable, objective methods such as long-term (sEMG) monitoring for proper evaluation of spasticity management. The findings do however not imply that patients' perceptions are not useful in clinical practice: spasticity from a patient's perspective comprises more than muscle activity alone and is likely to be affected by psychological factors such as coping, and pain as well. Exactly which factors are involved needs to be further explored, as these may need to be dealt with as well for proper management. It should be considered whether the findings of the present study are generalizable to other patient groups with spasticity, such as patients with stroke. As, in this population, involuntary muscle activation interferes with voluntary contractions, future studies should aim first at distinguishing between these two components of muscle activity.



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# 7

# INVOLUNTARY MUSCLE ACTIVITY IN PATIENTS WITH MOTOR COMPLETE SPINAL CORD INJURY

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## Abstract

The aim of this study is to quantify involuntary muscle activity patterns in the lower limbs of patients with SCI during daily life. The study focused on the questions whether the individual muscles show different behaviour, in terms of duration of muscle activity and co-activation, and whether and how this was affected by the type of activity that was performed. Longterm monitoring of muscle activity was performed with sEMG of four upper leg muscles in motor complete SCI patients, in whom muscle activation can be considered spasticity as voluntary contractibility is lost.

The results indicate that the four recorded muscles show different muscle behaviour ($p < 0.0001$) and that the type of activity a patient performs influences the relative duration of both muscle activity ($p < 0.0001$) and co-activation ($p < 0.0001$). Generally, duration of muscle activity was relatively short and, when muscles were active, they were simultaneously active with at least one of the other recorded muscles most of the time. However, the level of co-activation differed among muscles and type of activities performed. In particular transfers appeared to elicit more muscle activity and more co-activation. These results may help clinicians in decision making concerning reducing involuntary muscle activity in SCI patients and should encourage the clinician to refine history taking concerning spasticity.

## Introduction

Spasticity is a common phenomenon in patients with an upper motor neuron disorder. It is associated with an imbalance of excitatory and inhibitory control from reticulospinal and other descending pathways to the interneuronal circuits of the spinal cord, usually resulting in a net loss of inhibitory control, such as decreased presynaptic or reciprocal inhibition.[1] Traditionally, spastic responses have been attributed to velocity-dependent reflexes upon passive stretch.[2] However, in particular for patients with spinal cord injury (SCI), this definition is increasingly considered too narrow, because input from other afferent systems can elicit increased activation of spinal reflex circuits as well.[3-5] As a consequence, motor responses are produced that are not necessarily stretch-induced, leading to different manifestations of 'hyperreflexia'.[1] As was proposed by the SPASM (Support Programme for Assembly of database for Spasticity Measurement) consortium, all these afferent-mediated manifestations can be referred to as spasticity.[4]

Currently, literature on the different manifestations of spasticity in SCI patients is emerging. It increases our insight in the different positive features related to the upper motor neuron syndrome, such as spasms and clonus. Spasms appear to be the most frequently reported manifestation of spasticity by motor complete SCI patients.[6] Extensor spasms are multijoint reflex responses, commonly seen in combination with hip movement, such as changing from sitting to supine position.[3,7] Clinically they are characterized by hip extension, knee extension and, most often, ankle plantar flexion. Typical muscle activation patterns that were observed on induced hip extension are activity of rectus femoris, medial vastus and soleus muscle.[3] On hip flexion movement, in phase muscle activity of medial hamstrings was observed. Adductor muscle activity did not show consistent patterns across subjects.

Flexor spasms are considered to be a result of hypersensitivity of the flexion reflex pathways.[8] They can be elicited via for instance stimulation of the skin, causing generalized flexion of the leg at multiple joints. Modulation of the flexion reflex is

seen by imposed hip[9] or knee rotation.[8] Clonus may result from recurrent activation of stretch reflexes, from the involvement of a central oscillator or from interaction of both mechanisms.[10]

Not clinically observed, but also afferent-mediated are the rhythmic, locomotor-like activation patterns in motor complete SCI patients that were shown to be initiated by load and hip joint-related afferent input.[11] Another symptom of muscle overactivity, which is often reported by SCI patients, is continuous tension or stiffness in the legs.[6] It might be a result of passive stretch and altered intrinsic motor neuron properties,[12] but this manifestation is not studied in more detail so far.

Hence, knowledge is increasingly built on how spastic muscle is reflexively responding to imposed stimuli in controlled laboratory settings. In daily life, however, many different and more subtle triggers can occur during the day that might elicit different manifestations of spasticity. Information on the actual muscle activity is essential when considering spasticity treatment aiming at reducing involuntary muscle activity. Up to now, literature on involuntary muscle activity during daily life is hardly available.[13] Patient ratings on the level of spasticity were shown to be inadequate for this purpose, as they were only poorly associated with spasticity in terms of involuntary muscle activity.[13] Therefore involuntary muscle activity needs to be quantified otherwise, for instance with surface electromyography (sEMG).

The aim of the present study is to quantify involuntary muscle activity patterns in the lower limbs of patients with SCI during daily life. Longterm monitoring of muscle activity was performed with sEMG of four upper leg muscles in motor complete SCI patients, in whom muscle activation can be considered spasticity as voluntary contractibility is lost. The following questions were investigated: (1) do the individual muscles show different behaviour, in terms of amount of involuntary muscle activity and co-activation, and (2) is the type of activity that is performed affecting the amount of involuntary muscle activity and co-activation patterns? If so, (3) which muscles are mostly (co-) active during the specific activities?

## Methods

### *Study population*

Patients with motor complete SCI (American Spinal Injury Association Impairment Scale[14] grade A or B) were recruited from in- and outpatient departments of a Dutch rehabilitation centre. Patients were included when they had SCI at least six months old, a lesion above Th12 level, stable medical condition and self-reported spasticity in the lower limbs. Presence of severe contractures or pain that might interfere with the measurements were exclusion criteria. The study was approved by the local medical ethics committee. Patients signed informed consent before participation in the study.

### *Procedure*

Each patient was measured at two or three days for several hours per day, during which surface EMG was recorded continuously. Patients were instructed to note their activities including start and end times in a diary. Examples of activities were making transfers, reading, clothing etc. For each activity, patients were asked to provide a description of the individually perceived manifestation of spasticity in the lower limbs during that particular activity. Four manifestations of spasticity were predefined, of which patients could choose one or more when applicable: (a) a sensation of continuous tension or stiffness in one or both legs, (b) sudden bending of one or both legs (flexor spasms), (c) sudden straightening of one or both legs (extensor spasms) and/or (d) 'shaking' of the leg or the ankle (clonus). If necessary, patients could provide a description in their own words as well.

### *Instrumentation*

Surface EMG signals were obtained using bipolar, pre-gelled circular electrodes (ARBO H93, solid gel), with an inter-electrode distance of 24 mm. Activity of four upper leg muscles was recorded: rectus femoris (RF), vastus lateralis (VL), the adductor group, including gracilis and adductor magnus muscles, (AD)

and semitendinosus (ST). Electrodes on RF, VL and ST were placed according to international guidelines for electrode placement.[15] AD electrodes were placed at 50% on the line between the pubic tubercle and the medial femur condyle in the direction of the line. A reference electrode was placed at the lateral ankle. Electrodes were connected to a portable measurement and storage device (Mobi, sample frequency 1024 Hz, manufactured by TMSi, Oldenzaal, The Netherlands) using cables taped to the skin.

Surface EMG was band-pass filtered 30 – 500 Hz, a common filter setting for long-term sEMG monitoring during which movement artefacts are likely to occur.[16] For detection of the start and end of bursts of muscle activity an algorithm, based on the Approximated Generalized Likelihood Ratio (AGLR) algorithm,[17] was used. Duration and root mean square (RMS) was calculated for the detected bursts.

***Outcome parameters***

The primary outcome parameters are expressed as time ratios. The first parameter is the relative duration of muscle activity per muscle (RelDur), reflecting the proportion of time that a muscle was active during the whole measurement (range 0 – 1). It is calculated as the sum of all burst durations (for each muscle) divided by the duration of the entire measurement.

The second outcome parameter is the level of co-activation (CoAct), which is the proportion of time during which two muscles were simultaneously active (range 0 – 1). It is calculated as the sum of time periods that a reference muscle was simultaneously active with one of the other muscles (referred to as 'couple'), divided by the duration of muscle activity of the reference muscle.

***Statistical analysis***

Descriptive statistics were used for sociodemographic and outcome variables. Each of the listed activities from the diaries was allocated to one of five main groups (Transfer, Activities of daily living, Being active, Stable body position and Other). For each group of activities, the reported manifestations of spasticity, as well as the

EMG parameters per muscle, were mapped. Due to its limited size the group 'Other' was left out of further analysis. Data from the periods of time during which no activity was registered, were clustered as reference group 'No activity registered'.

Statistical modelling was conducted to assess whether RelDur (dependent variable) was affected by the factors Muscle (RF, VL, AD or ST) and Activities. A second model investigated whether CoAct (dependent variable) was affected by the factors Couple and Activities. For this purpose, a beta regression model was used. The main assumptions in such model are that the dependent variable may be regarded as continuous and that it is bounded between two known endpoints, such as proportions.[18] Each sample in the database represented one of the time periods (performed activity or period during which no activity was registered) of a single patient.

In this study, both outcome parameters RelDur and CoAct have highly skewed and heteroscedastical distributions between 0 and 1. The beta regression approach models both location (means) and dispersion (variances) with its own set of shape parameters. Thereby, instead of assuming equal variances, like in more conventional models, it is modelling heteroscedasticity. Shape parameters are obtained with maximum likelihood estimation. Goodness of fit was assessed by plotting the predicted versus the observed values. In order to avoid zeros and ones in the data, transformation was done according to Smithson and Verkuilen.[18]

Outcome parameters RelDur and CoAct are presented graphically for each muscle or couple of muscles by kernel density estimates, based on the corresponding histograms of each distribution. By definition the area under the curve for each density function is equal to one. It implies that a small amount of (co-)activity is visualized by a high early peak and a relatively thin right hand tail; a higher amount is visualized by a shift to the right. As a result of normalization the units of the y-axis have no importance and are therefore removed from the graphs. The estimated density lines exceed the limits '0' and '1' in the figure, which is a result of the kernel density estimation of the true unknown density that is bounded by definition between '0' and '1'.

For statistical analysis Statistical Package for Social Sciences (version 11.5) and 'R'[19] was used. Alpha was set at 0.05 for statistical significance.

# Results

### *Sociodemographics*

Table 7.1 summarizes the baseline characteristics of the study population. Nineteen patients were included in this study. On average 10.7 hours (sd ± 3.4) were measured per patient.

**Table 7.1:** Group characteristics (N = 19)

| Characteristics | Values |
|---|---|
| Age in yrs (mean, sd) | 40.2 (9.1) |
| Male / female (n) | 13 / 6 |
| Level of lesion: cervical / thoracal (n) | 10 / 9 |
| ASIA Impairment Scale A / B (n) | 16 / 3 |
| Duration since injury in months (mean, sd) | 102.0 (87.4) |
| Use of spasmolytics (n) | 13 |

*Abbreviations: sd, standard deviation; n, number, ASIA, American Spinal Injury Association.*

Altogether, 374 activities were registered during monitoring, with a median of 17 activities per patient (range 9 - 32). All reported activities were assigned to one of five main groups. 'Transfer' was the largest group, which included activities incorporating change of position, mainly transfers. It was reported 151 times (40.4%). Second largest group was 'Activities of daily living' (26.7%). Examples were getting dressed, brushing teeth, drinking coffee and having lunch. 'Being active' was the next group (19.0%), comprising activities such as having therapy, exercising and riding in wheelchair, followed by 'Stable body position' (9.6%). This group included activities during which the patient remains in the same position for a long time. The remaining group 'Other' was small (4.3%) and included activities that could not be categorized otherwise.

During 85.0% of the activities spasticity was perceived (table 7.2). Overall, continuous tension was reported most often (42.0%), followed by extensor spasms (29.9%), flexor spasms (22.7%) and clonus (15.5%).

### *Relative duration of muscle activity*

First the entire measurements, irrespective of the performed activities, were analyzed to get insight in the overall muscle activity during the day. Muscle activity characteristics per muscle are shown in table 7.3. Each muscle appeared to be active during only a small proportion of the total duration of the measurement. The intensity of muscle activity per burst (RMS) is comparable for the four muscles.

**Table 7.2:** Percentage of each activity that a perceived manifestation of spasticity was reported (mixed data of all 19 patients). Per activity more than one manifestation could be reported.

| Activity (n) | No spasticity | Tension | Flexor spasms | Extensor spasms | Clonus | Other |
|---|---|---|---|---|---|---|
| **Transfer** (151) | 7.3 | 45.0 | 27.2 | 38.4 | 20.5 | 3.3 |
| **Activities of daily living** (100) | 19.0 | 36.0 | 24.0 | 27.0 | 8.0 | 3.0 |
| **Being active** (71) | 14.1 | 57.7 | 16.9 | 23.9 | 25.4 | 7.0 |
| **Stable body position** (36) | 27.8 | 25.0 | 22.2 | 19.4 | 2.8 | 16.7 |
| **Other** (16) | 37.5 | 18.8 | 0 | 18.8 | 0 | 6.3 |
| **All activities** (374) | 15.0 | 42.0 | 22.7 | 29.9 | 15.5 | 5.3 |

*Abbreviations: n, number of times an activity has been reported.*

**Table 7.3:** Relative duration of muscle activity per muscle (median, IQR) in percentage of total duration of the measurement

| | **RelDur** (%) | **Mean RMS bursts** (μV) |
|---|---|---|
| **RF** | 1.5 (0.9 – 4.4) | 9.0 (6.8 – 10.8) |
| **VL** | 1.9 (0.9 – 4.2) | 10.4 (8.0 – 13.7) |
| **AD** | 3.7 (1.3 – 6.9) | 6.4 (5.3 – 7.9) |
| **ST** | 5.1 (2.3 – 10.3) | 7.5 (5.7 – 8.7) |

*Abbreviations: IQR, interquartile range; RMS, root mean square; RF, rectus femoris; VL, vastus lateralis; AD, adductor group; ST, semitendinosus.*

In figure 7.1 the same data are visualized by statistically estimated density functions, representing the distribution of RelDur per muscle. The figure illustrates the highly skewed distributions for all muscles, with most observations approximating '0', indicating extremely short duration of muscle activity. Consistent with the median values in table 7.3, the means of RelDur appeared to differ between muscles, with RF having the lowest and ST the highest mean RelDur.

In addition, the figure shows that the variability, represented by the varying thickness of the right hand tails, differed between muscles as well. ST had the highest variability, indicating more inter- and/or intra-subject variation. Beta regression analysis demonstrated that the differences between the four muscles, in terms of both means and variances, were highly significant ($p < 0.0001$).

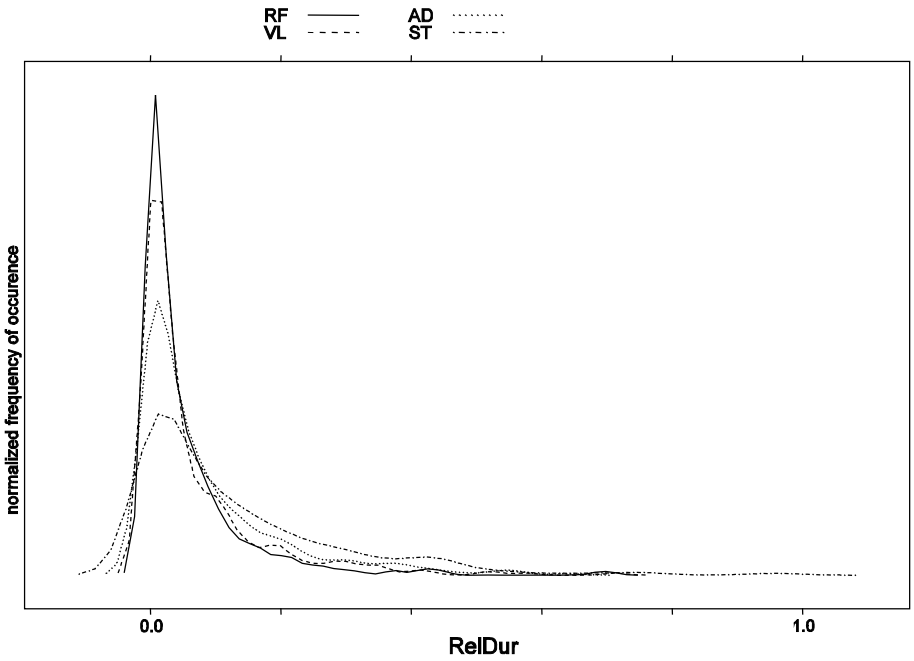


**Fig 7.1:** Estimated density of RelDur (range 0 – 1) for each muscle. Ratios are normalized to the total duration of the measurement. Legend: 0 = not active; 1 = continuously active.
Abbreviations: RF, rectus femoris; VL, vastus lateralis; AD, adductor group; ST, semitendinosus.

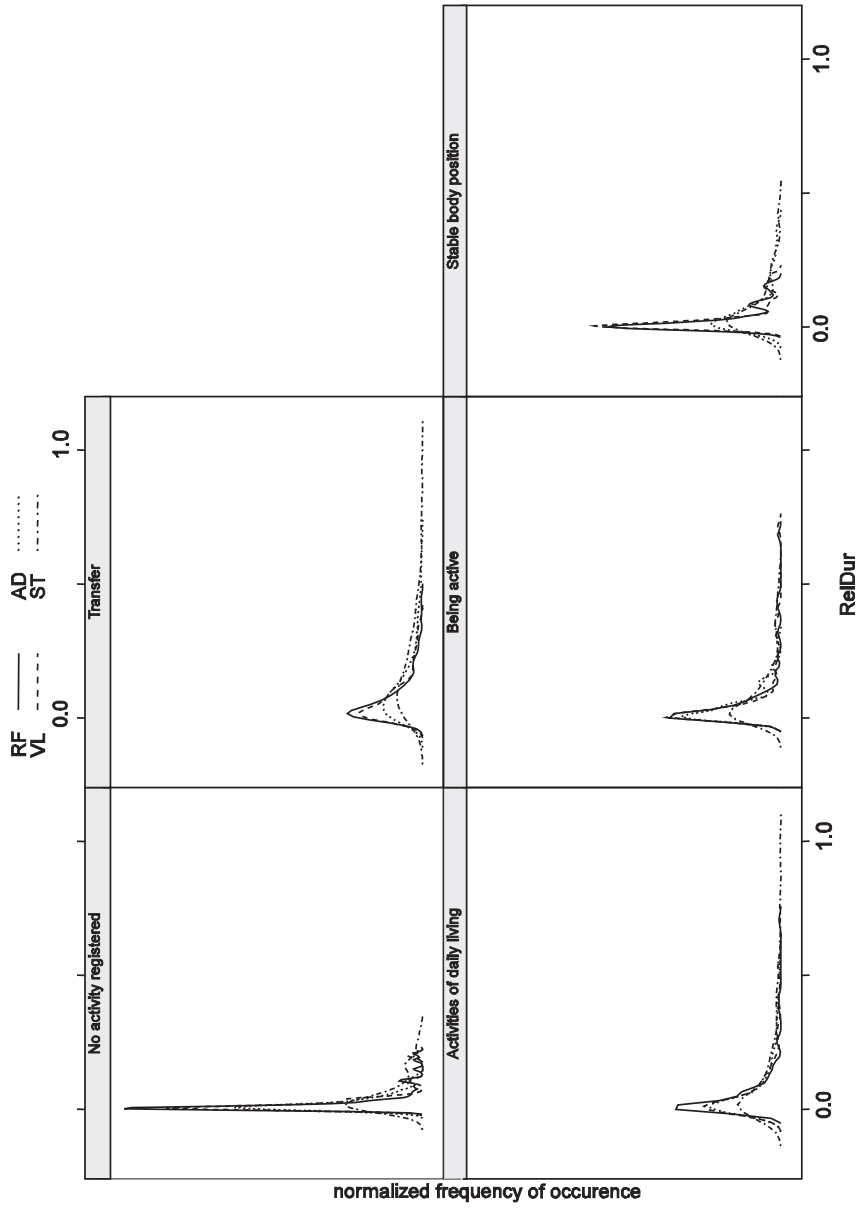


**Fig 7.2:** Estimated density of RelDur (range 0 – 1) for each muscle per activity. Ratios are normalized to the total duration of the measurement. Legend: 0 = not active; 1 = continuously active.
Abbreviations: RF, rectus femoris; VL, vastus lateralis; AD, adductor group; ST, semitendinosus.

***Duration of muscle activity and association with daily activities***

Figure 7.2 shows the density function of RelDur per muscle in five different conditions, i.e. one of four activities or while no activity was registered. Overall, the effect of performed activity on RelDur was significant ($p < 0.0001$). When comparing with 'No activity registered', the graphs of the four activity groups show a shift of distributions to the right side, illustrated by lower but broader curve peaks. It indicates that during all performed activities relative duration of muscle activity was higher compared to the periods that no activity was registered. The distribution patterns of RelDur during 'Transfer' deviate most from those while no activity was registered. The other activity groups show intermediate patterns.

***Quantification of co-activation patterns***

In tables 7.4 and 7.5 the co-activation patterns for the entire measurement are summarized. Table 7.4 shows the percentage of time each muscle was active solitarily or co-active. In table 7.5 it is specified with which muscles each recorded muscle was co-active mostly.

ST showed more single muscle activity, compared to the other muscles. When co-active, it was mostly with AD. In the other muscles, RF and VL, it appeared more equally distributed. These findings are confirmed by the estimated densities of CoAct, presented in figure 7.3. The figure shows evident variety in distribution patterns. Some couples show a homogeneous or almost bimodal pattern, in particular the couples with reference muscles RF and VL (the upper two graphs). In the lower two graphs of the reference muscles AD and ST, different distribution patterns are observed: Some couples approach '0' (e.g. ST – RF and ST – VL), representing hardly any co-activity. This is consistent with the relatively high single activity of ST. Other couples approach '1' (AD – ST), indicating that when the reference muscle (AD) is active, it is almost always co-active with the other muscle (ST). The observed differences between couples, in terms of both means and variances, were highly significant ($p < 0.0001$).

**Table 7.4:** Single muscle activation or co-activation in percentage of total duration of muscle activity of each reference muscle (median, IQR)

| | **Reference muscle** | | | |
|---|---|---|---|---|
| | **RF** | **VL** | **AD** | **ST** |
| **Single activation** | 22.4 (7.2 – 30.2) | 27.0 11.6 – 52.3) | 19.8 (11.5 – 35.9) | 43.3 (29.5 – 60.4) |
| **Co-activation with 1 other muscle** | 21.3 (10.2 – 25.5) | 18.1 (13.5 – 29.2) | 26.6 (23.6 – 41.3) | 23.9 (18.5 – 29.4) |
| **Co-activation with 2 other muscles** | 21.9 (17.4 – 34.6) | 19.2 (11.5 – 30.1) | 20.1 (16.1 – 26.3) | 12.6 (8.4 – 19.9) |
| **Co-activation with 3 other muscles** | 28.5 (14.4 – 44.1) | 22.1 9.2 – 57.7) | 14.0 8.8 – 34.6) | 12.5 (6.2 – 23.5) |

*Abbreviations: IQR, interquartile range; RF, rectus femoris; VL, vastus lateralis; AD, adductor group; ST, semitendinosus.*

**Table 7.5:** Identification of co-activating couples in percentage of total duration of muscle activity of each reference muscle (median, IQR)

| | **Reference muscle** | | | |
|---|---|---|---|---|
| Co-activity | **RF** | **VL** | **AD** | **ST** |
| **With RF** | - | 41.1 (28.6 – 67.5) | 35.3 (24.5 – 50.5) | 22.7 (14.0 – 35.8) |
| **With VL** | 48.9 (38.0 – 72.1) | - | 35.3 (21.5 – 61.7) | 30.3 (11.2 – 54.0) |
| **With AD** | 67.1 (50.3 – 80.0) | 53.4 (28.9 – 74.3) | - | 42.3 (34.4 – 60.2) |
| **With ST** | 53.2 (26.1 – 75.6) | 59.8 (27.9 – 75.6) | 64.7 (42.6 – 82.0) | - |

*Abbreviations: IQR, interquartile range; RF, rectus femoris; VL, vastus lateralis; AD, adductor group; ST, semitendinosus.*



### *Co-activation patterns and association with daily activities*

Figure 7.4 shows the densities of CoAct for each couple of muscles in five different conditions. Overall, the effect of performed activity on CoAct was significant ($p < 0.0001$). The figure shows that the columns of 'Transfer' and 'Stable body position' co-activation patterns deviate most from the pattern during 'No activity registered' (the first column). This is illustrated by evident shifts to the right side, indicating a proportional increase in duration of co-activation.

The most prominent differences can be observed during 'Stable body position' and 'Transfer', such as the increase in co-activity of RF and AD during 'Stable body position' and co-activity of AD and ST during 'Transfer'.

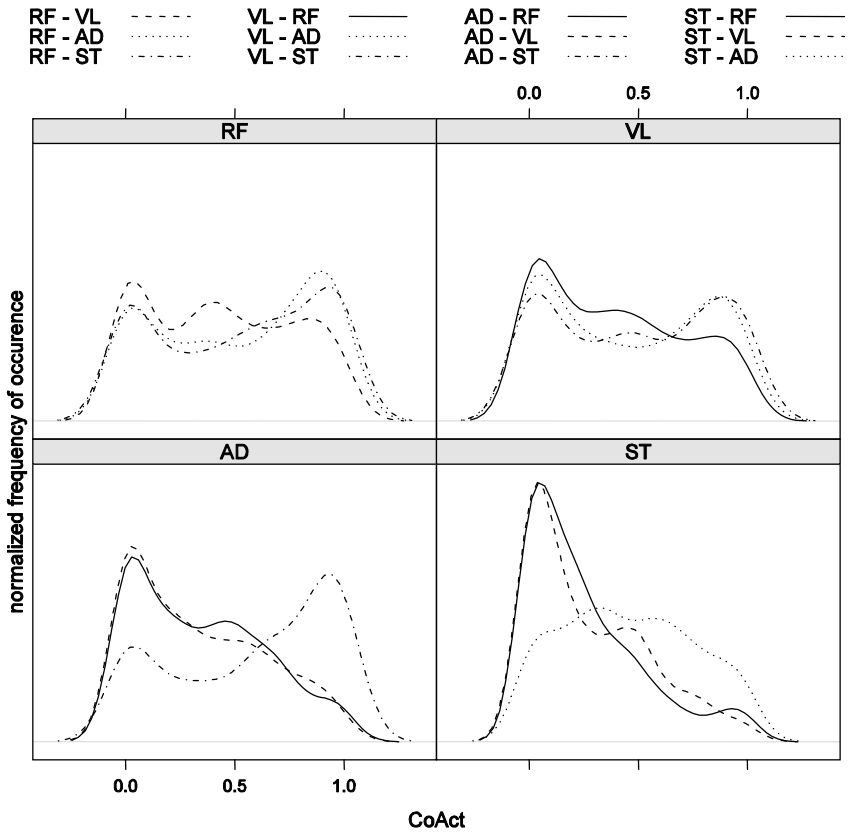


**Fig 7.3:** Estimated density of CoAct (range 0 – 1) for each couple. Each couple is indicated with reference muscle (first) and each of the other recorded muscles (second). Ratios are normalized to the total duration of activity of the reference muscle. Legend: 0 = not co-active; 1 = continuously co-active. Abbreviations: RF, rectus femoris; VL, vastus lateralis; AD, adductor group; ST, semitendinosus.

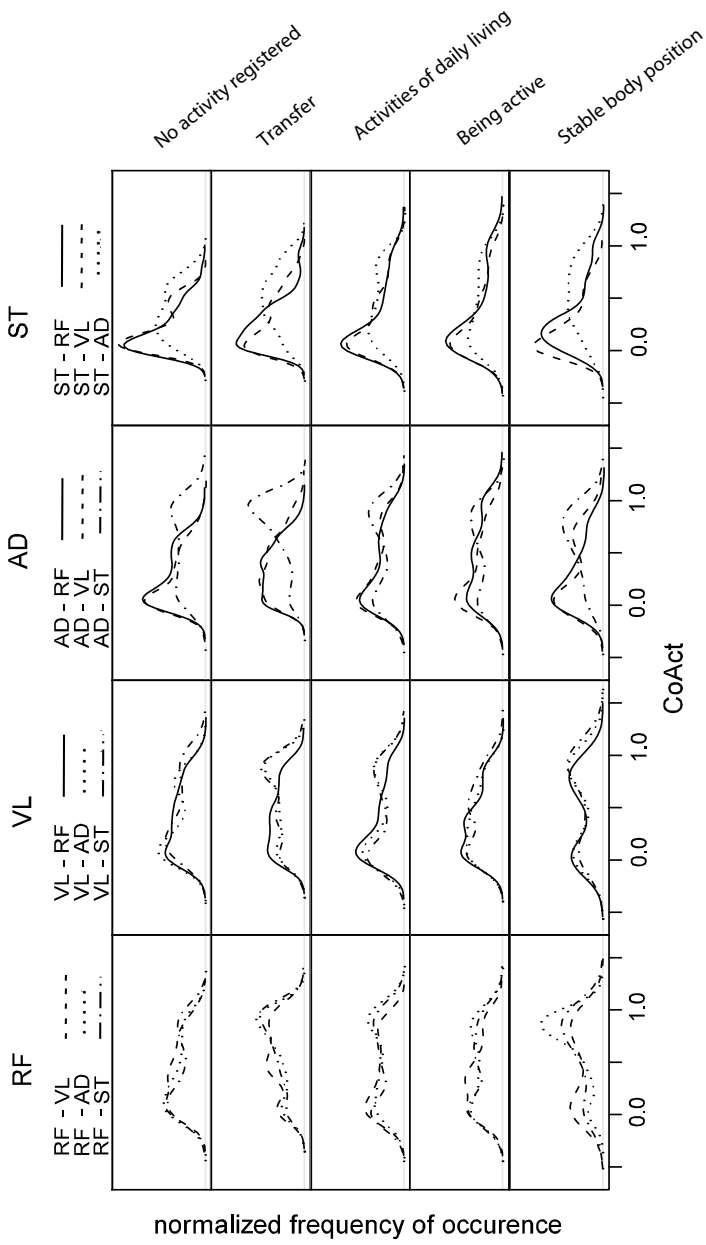


**Fig 7.4:** Estimated density of CoAct (range 0 – 1) for each couple per activity. Each couple is indicated with reference muscle (first) and each of the other recorded muscles (second). Ratios are normalized to the total duration of activity of the reference muscle. Legend: 0 = not co-active; 1 = continuously co-active.
Abbreviations: RF, rectus femoris; VL, vastus lateralis; AD, adductor group; ST, semitendinosus.

# Discussion

The aim of this study was to investigate involuntary muscle activity patterns in the lower limbs of patients with SCI during daily life. In particular answers were sought to the questions whether the individual muscles show different behaviour, in terms of duration of muscle activity and co-activation, and whether and how it was affected by the type of activity that was performed. The results indicate that the four recorded muscles indeed show different muscle behaviour and that both RelDur and CoAct are influenced by the type of activity a patient performs. Generally, duration of muscle activity was relatively short and, when muscles were active, they were simultaneously active with at least one of the other recorded muscles most of the time. However, the level of co-activation differed among muscles and type of activities performed.

***Individual muscle behaviour and co-activity***

Generally, patients reported having 'continuous tension' as the most commonly occurring manifestation during all activities. It was the most frequently reported manifestation during 'Being active' and during 'Transfer'. This finding was only partially in agreement with sEMG outcomes. The parameter RelDur was highest during 'Transfer' for all muscles, but not during 'Being active' (figure 7.3). ST appeared to be active most often. Both the commonly present hamstrings shortening and a sitting position in a wheelchair during the day may increase tension development and thereby lead to enhanced reflex sensitivity.

How 'continuous tension' is perceived by ASIA-A subjects is hard to explain in neurological context. Increased non-neural stiffness might have contributed as well. Possibly, it results in more difficulty to perform activities and may thus be noticed indirectly.

The frequencies of reported manifestations of spasticity in the present study differ slightly from an earlier study,[6] in which extensor spasms were the most common. In the former study patients were asked to report perceived manifestations

of spasticity during certain activities retrospectively, while in this study the perceived manifestations applied to the activity that was just performed. The latter observations may therefore be more reliable, as a less manifest symptom such as 'continuous tension' might not be remembered after some time.

The overall low proportion of muscle activity is remarkable, because all patients had self-reported spasticity in the legs, despite of the use of oral spasmolytic medication in 68% of the patients. Therefore more involuntary muscle activity might have been expected. A recent study[13] already demonstrated that self ratings of the level of spasticity by motor complete SCI patients are poorly associated with spasticity, in terms of involuntary muscle activity.

Co-activity patterns appeared to be highly complex, with some muscles showing very little co-activity and others being co-active most of their 'active time'. ST was mostly active solitarily and thus showed little co-activation. AD was the least solitarily active muscle and seemed to co-activate most often with ST. The apparently low selectivity of AD is consistent with other studies.[3]

***Influence of performed activities on individual muscle behaviour and co-activity***

In general, all activities appeared to enhance the relative duration of muscle activity and the amount of co-activation. The results support the clinical observation that muscle tone is built up when a patient comes to exertion. The effect was the highest during performing transfers, inducing the largest increase in involuntary muscle activity in all four recorded muscles and largest increase in co-activation in most couples. During a transfer an increase of afferent input is likely, such as proprioceptive and cutaneous stimuli, thereby activating the different disinhibited sensori-motor spinal reflex circuits. This is in agreement with the perception of patients themselves.[6]

Spasms were often reported during transfers. Some patients reported more extensor spasms and others more flexors spasms. Possibly the manifestation was depending on how a transfer was performed (with lift, sliding) but this information

was not registered. As the two manifestations involve opposite muscle actions, it might explain some of the bimodality in the distribution of the variable CoAct. For example, strong RF-VL coupling is observed during extensor spasms[3] (the right hand peak in figure 7.4), but is not expected during flexor spasms[8] (the left hand peak).

The distinct muscle couples appeared to behave differently, depending on the activity that was performed. The most prominent example of large activity-induced change is the couple RF-AD, which shifted towards more co-activity during 'Stable body position' and, to a lesser extent, during 'Transfer'. On the other hand, ST appeared to keep on acting merely solitarily, regardless of the activities that were performed. Its co-activation pattern appeared to be relatively insensitive for performed activities, although co-activation with AD increased slightly (figure 7.4). VL and AD showed intermediate patterns.

***Clinical implications and therapeutic consequences***

The first clinically relevant message is that the actual amount of muscle activity during the day, in this group of motor complete SCI patients, is rather low. Second clinically important finding is that duration of muscle activity and co-activation increases when daily activities are performed, in particular during transfers or during activities involving long-lasting stable body position. As concluded earlier[6] it appears rewarding to ask a patient with spasticity-related discomfort to specify when it is mostly experienced. Although still in an explorative stage, figure 7.4 gives some insight in what type of co-activation might be expected.

The results indicate that, when treatment of spasticity is considered, a systemic approach is probably first choice in most cases. In particular AD is commonly co-active with other muscles when it is active. Therefore, local management like obturator nerve blocks might deal with part of the problem, but might leave hamstring spasticity unresolved.

***Methodological considerations***

In this study timing parameters derived from sEMG of four muscles were used to quantify muscle (co-)activation. Amplitude parameters are known to be considerably more variable than timing parameters in the analysis of dynamic sEMG,[20] partly due to the large inter-subject variability.[21]

Due to uncorrectable skew and multimodality of the dependent variables and the residuals, linear modelling was not possible in this study. Beta regression modelling appeared very well applicable, not only because of the assumptions as mentioned earlier. It has been shown that in this study the location (mean) shifts are not the only measure of effect, as is routinely assumed. It is, however, also possible that a factor's influence manifests primarily through variation, which can only be revealed when heteroscedasticity is modelled as well.[18]

Unfortunately, correction for paired observations was not possible.[18] As a result, bias could have been introduced, because the number of reported activities differed significantly between participants. Although not evident during data checking, systematic errors due to observations that were not independent can not be ruled out.



## Conclusions

Upper leg muscles in motor complete SCI show relatively short duration of involuntary muscle activity. However, contextual factors were shown to play a prominent role: Both the duration of muscle activity and the degree of co-activation of muscles appear to be influenced significantly by the activity a patient is performing. In particular transfers appeared to elicit more muscle activity and more co-activation. The results give more insight in involuntary muscle activity patterns in SCI and should encourage the clinician to refine history taking concerning spasticity.

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# 8

# General discussion

# Introduction

In the field of Rehabilitation Medicine spasticity is an important topic. Spasticity is a common phenomenon in patients with an upper motor neuron (UMN) lesion. When a patient's functioning is negatively affected by the presence of spasticity, treatment to reduce spasticity is required. In that case, quantitative assessment of spasticity is essential to evaluate the effect.

In clinical practice several difficulties in spasticity assessment are encountered, as outlined in the first chapter. Measurement of spasticity appears to be complex due to its various manifestations and the difficulty to distinguish between neural and non-neural components. Furthermore, the value of passive stretch tests for the representation of spasticity during active, more functional movements is unclear. Additionally, there can be discrepancy between outcomes of objective tests and the patients' perception. Finally, a single momentary assessment may be insufficiently reliable, due to variability of spasticity during the day.

The objective of this thesis is to contribute to the development of a comprehensive set of clinically applicable measurement tools for spasticity, by concentrating on questions concerning the measurement of spasticity that were existing in clinical practice. In the previous chapters some parts of the puzzle have been found, but on the other hand new questions have arisen.

In this final chapter, the main findings are integrated and evaluated within the context of the existing literature. The main questions to be answered are: do the presented results offer new insights for clinicians and researchers and do they provide clear recommendations to improve spasticity assessment?

Before discussing measurement the construct 'spasticity' needs to be defined clearly, as consensus on its definition is still lacking.[1] Yet a shift seems to be taking place from the more traditional definition by Lance,[2] concentrating on the velocity-dependent increase in stretch reflex activity, to broader definitions that include other positive symptoms following an UMN lesion as well.[3-5] The definition introduced

by the SPASM (Support Programme for Assembly of database for Spasticity Measurement) group comprises any involuntary muscle activation resulting from disordered sensori-motor control,[4] thereby including all afferent-mediated positive features of UMN syndrome. It does, however, exclude some other positive symptoms, such as dystonia and associated reactions,[6] as these are probably mostly efferent-driven. Within this regard, the phenomenon of pathological co-contraction is debatable. It can be simply regarded as a lack of motor control due to reduced reciprocal inhibition and consequently being considered as an efferent-mediated phenomenon.[6] On the other hand, agonist contraction is prerequisite to attain pathological co-contraction. When contraction of the agonist is reflexive, an afferent stimulus is involved which also excites the antagonist alpha motor neuron via interneurons. In addition, stretch reflex activity of the antagonist can be involved as well, which cannot be discriminated from co-contraction.

In this thesis the latter, broader definition[4] is mostly cited. It is considered useful in both clinical and scientifical context, as long as it is identified which manifestation is studied. In particular in the clinical environment its use is considered pragmatic. In practice, both patients and clinicians often intermingle the different manifestations of spasticity, like for example spasms and stretch reflex activity. In addition, spasmolytic treatment generally acts upon all hyperreflexive signs. Drugs like baclofen have effect on several polysynaptic reflex arcs by decreasing alpha motor neuron sensitivity directly, mainly via enhancing presynaptic inhibition,[7,8] or indirectly, via reduction of spindle sensitivity.[9]



## Measurement of spasticity and the ICF

When discussing the methodological qualities of the described measurement methods it should be taken into account that each instrument might measure a different construct, being a part of or related to spasticity. Understanding can be improved by relating the different methods to the framework of the International

Classification of Functioning, Disability and Health (ICF) of the World Health Organisation.[10,11] The ICF classifies health and health-related states. The classification organizes information in two parts: part 1 deals with Functioning and Disability, while part 2 covers Contextual Factors. Each part has two components. The first part consists of domains, described from the perspective of the body (Body Functions and Structures) or the individual and society (Activities and Participation), as shown in figure 8.1. The second part consists of a list of Environmental and Personal Factors, representing the complete background of an individual's life and living, which may have an impact on the individual and that individual's health.

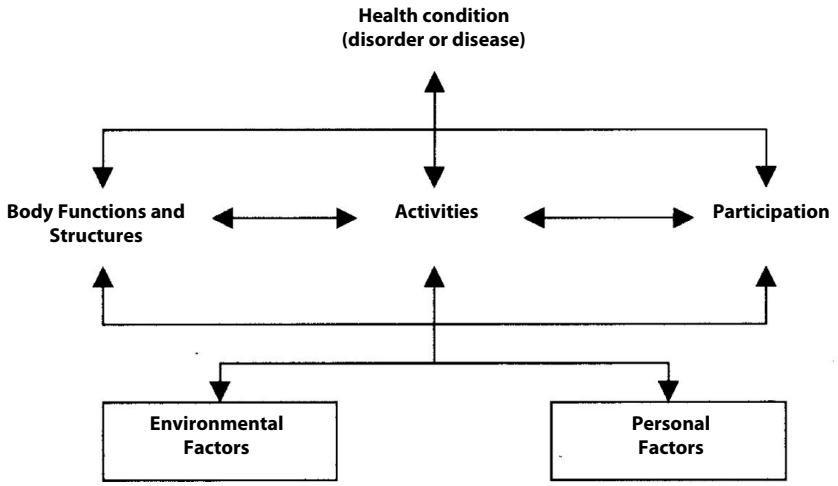


**Fig 8.1:** Interactions between the components of the ICF(Geneva World Health Organization 2001)

When spasticity is considered as a health condition, the measurement methods can roughly be ordered by relating them to the components of Functioning and Disability, as follows:

1. *Body Functions and Structures*
    - Measurement of reflex muscle activity with surface electromyography (sEMG) during standardized test situations (**chapters 2 and 4**), during active tasks (**chapter 3**) or during daily life activities (**chapters 6 and 7**);
    - Measurement of resistance against passive movement with dynamometry (**chapter 4**) or performed manually (e.g. Ashworth scale) (**chapters 2 and 4**);
    - Measurement of perceived degree of spasticity during a specific activity with Visual Analogue Scale (VAS) (**chapters 5 and 6**).
2. *Activities and Participation*
    - Measurement of perceived degree of spasticity during a specific activity with VAS (**chapters 5 and 6**);
    - Measurement of experienced spasticity-related discomfort during a specific activity with Borg scale (**chapter 5**).

***1. Body Functions and Structures***

Most conducted studies described in this thesis primarily focused on the ICF component Body Functions and Structures by the application of sEMG for quantification of (reflex) muscle activity. This neurophysiological method has potentially good criterion and construct validity for the assessment of spasticity, as it is close to its definition.[4,12] However, the measurement method appears to be rarely used in clinical practice. This can partly be explained by the fact that the instrument is not generally available, that its use is time-consuming and that a meticulous protocol is lacking.

Another major difficulty of studying reflex activity is to differentiate it from voluntary muscle contraction during active movement. Voluntary muscle contraction may interfere with involuntary reflex activity, depending on the chosen protocol and the context. In the presented studies the latter problem is avoided by focusing merely on the stretch phase of a muscle or to study patients with motor complete

spinal cord injury (SCI), hence without presence of voluntary muscle activity. The first approach is considered valid when only stretch reflex activity is subject of study. The second approach addresses all reflex-mediated positive symptoms. Measurement protocols are therefore crucial in determining how to interpret muscle activity measured with sEMG. As a consequence, cautiousness is needed when generalizing the results to other situations or other patient groups with spasticity.

An important conclusion from the conducted studies is that the currently widely used clinical method, the Ashworth scale, is neither sufficiently valid nor reliable for spasticity measurement.

Because in clinical practice standardization of the test is generally poor, the influence of positioning of the patient during testing was investigated (chapter 2). It was shown that both clinical measurement, by using the Ashworth scale, and neurophysiological measurement, with sEMG, were considerably affected by change in posture and muscle length. The results of the neurophysiological tests in this study confirmed our hypothesis that muscles, including the quadriceps muscle, in elongated state show more stretch reflex activity compared with muscles in shortened state. This can be understood from the neurophysiological background that increased muscle stiffness and increased tension development lead to altered reflex sensitivity.[13-15]

In spite of standardized positioning of the patient, as was done in the study described in chapter 4, clinimetric properties of the Ashworth scale for spasticity measurement were insufficient. Although the Ashworth scale was originally used for the assessment of stretch reflex muscle activity, it is now increasingly regarded as an instrument for perceived resistance during passive rotation of a joint.[16] However, our study showed that clinimetric properties of the scale to be a measure for resistance were also unsatisfactory. This finding is in line with other studies (e.g. [17]). In our study, correlations of Ashworth scores with dynamometry outcomes were stronger in comparison with the EMG outcomes, but inter-rater reliability figures were low.

***2. Activities and Participation***

Improvement of daily functioning is an important goal of spasticity treatment.[12] However, the correlation between observed reduction in spasticity and improvement in function was found to be poor.[18-20] Spasticity reduction will probably not improve all aspects of daily functioning, but only specific activities of an individual patient, such as fewer spasms disrupting sleep or the improved ability to use the affected arm during a specific task.[12] In addition to the severity and distribution of the spasticity itself, there are various factors that can influence whether a positive functional outcome occurs as a result of spasticity treatment, such as presence of muscle weakness, sensory loss, visual impairment, neglect or cognitive disability.

Chapter 3 addressed the ecological validity of passive stretch tests. The study aimed to clarify whether the result of a passive stretch test has some relevance for every day life situations and reflects not just a clinical phenomenon. In a group of poststroke patients, the outcomes of reflex muscle activity during passive tests appeared not to be related strongly with reflex muscle activity during an active task. Even though functional significance of the demanded active motor task in this study was limited, it was clearly shown that muscle activation during stretch showed different behaviour in the two conditions. During the active motor task other pathological mechanisms appeared to play a role, such as delay in start and termination of contraction and co-activation. This observation is strongly supported by other studies.[20] It has been demonstrated that during active movement several reflex systems are involved, which become modulated during its execution and control.

***The impact of Contextual Factors***

The effect of internal and external factors on spasticity is well known by clinicians. Several physical and psychological factors can increase spasticity, such as infection, tight clothing or emotional stress.[12,21] The influence of environmental factors, like for instance climate or riding on rough roads, is known as well.[22]

It is likely that the patients' perception of spasticity in itself is affected by contextual factors as well. From the clinical point of view, subjective ratings on spasticity may be regarded as important measurement tools for spasticity, in particular when spasmolytic treatment is considered or evaluated. Eventually, patients themselves must decide whether or not treatment is justified. For most clinicians, the patients' perception of their own health state seems an uncontrollable variable, in particular in the acute disease episode, during which their perception is thought to be highly confounded by emotional distress. In addition, other psychological and physical sensations, such as pain, might influence the patients' perception.[21-23]

It is subject to discussion how the subjectively perceived degree of spasticity should be positioned in the ICF framework. In chapter 5 a conceptual visualisation was presented of the position of the patients' perception in the ICF framework and its interrelationship with the different components (figure 8.2). First, the perceived degree of spasticity is thought to be derived from the actual amount of involuntary reflex muscle activity. Secondly, the patients' perception of spasticity is expected to be influenced by personal and environmental factors. Both the perception of spasticity and the experienced discomfort are likely to be influenced by these contextual factors, but probably each to a different extent.

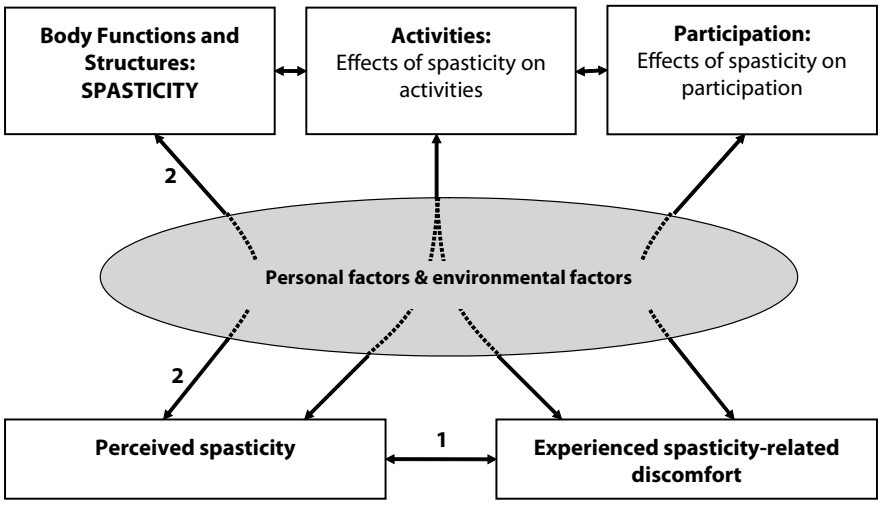


**Fig 8.2:** Conceptual model of spasticity (in terms of impairment of Body Functions and Structures within the WHO ICF framework) and the patients' perception

## Interrelationships between the different components in the model

The perceived degree of spasticity, measured with VAS, and the experienced spasticity-related discomfort, measured with Borg scale, appeared to be only moderately associated (arrow 1 in figure 8.2). It was shown that the impact of spasticity on daily life was related to the context in which it occurs. Correlation between the VAS and Borg scale was higher during activities involving a 'stable body position' and while 'being active'. Patients indicated that the experienced discomfort was often associated with practical concerns, such as disturbance of sleep, compromised safety etc. On the other hand psychological factors, like the degree of acceptance of their situation or negative self-image, were reported as well. The influence of contextual factors is strongly supported by the study described in chapter 6: Ratings of the level of spasticity by motor complete SCI patients appeared to be poorly associated with spasticity, in terms of involuntary muscle activity assessed with long-term sEMG recordings (arrow 2 in figure 8.2). The finding that muscles were active during only a fraction of the day in a similar population with self-reported spasticity was remarkable (chapter 7). Apparently the upper leg muscles showed relatively little involuntary muscle activity, yet it was once more demonstrated that contextual factors have prominent influence: Both the duration of muscle activity and the degree of co-activation of muscles became significantly higher during specific daily activities. In particular transfers and other activities involving change of position appeared to elicit more muscle activity.



## Implications for clinical practice

Based on the outcomes of the conducted studies some recommendations for clinical practice can be formulated. Beforehand, it is useful to identify the goal of spasticity measurement in a specific patient. Spasticity measurement is most often

associated with the selection for or evaluation of treatment. The goal of treatment can, however, be basically different in an immobile bed bound patient in a nursing home, compared with an active self-supporting patient. Primary treatment goals for the first - at low level functioning - patient might be improving daily nursing care, decrease of pain, prevention of contractures. Improvement of walking ability and increasing independency in self-care will probably be treatment goals for the second - at high level functioning - patient. For that reason, simple bed-side clinical tests might suffice in the first patient, whereas for the second patient a more functional evaluation of spasticity is required.

Clinicians still tend to rely on passive stretch tests, such as (modifications of) the Ashworth scale or Tardieu scale, probably because it provides useful information to perceive what happens while rotating the limb. However, from this thesis it has become clear that passive stretch tests for the assessment of spasticity have some serious shortcomings. The commonly used Ashworth scale appeared to be invalid and unreliable as a measure for spasticity. It is therefore strongly recommended to at least document the patients' position for comparable assessment or, better, to avoid use of the scale at all. Additionally, clinicians should realize that the outcome of a passive stretch test alone is not representative for spasticity during active motor tasks nor for motor control.

Most clinicians involve the patient's judgment into the decision making process. However, patient ratings on the degree of spasticity appear to be invalid for spasticity assessment, in terms of measuring involuntary muscle activity: Spasticity from a patient's perspective apparently comprises more than muscle activity alone. Yet it is generally acknowledged that it provides useful additional information from the patients' perspective. The described results can help clinicians to refine history taking concerning spasticity. First of all, asking a patient to specify during which activities spasticity is experienced mostly can provide valuable information. Secondly, it is useful to distinguish between the perceived degree of spasticity and experienced spasticity-related discomfort, as the two concepts were only

moderately associated. Therefore, standardized self-ratings on the perceived degree of spasticity should be added to the assessment set.
Surface EMG recording, under standardized test circumstances or during daily life activities, is a useful tool particularly in more complex cases. When there are serious doubts about the role of actual reflex muscle activity in the complaints or symptoms of an individual patient, sEMG can provide crucial information. When a high degree of perceived spasticity or discomfort can not be confirmed by sEMG findings, then other physical or psychological explanations must be searched for. A detailed measurement protocol is essential for adequate interpretation of the results.

In conclusion, different levels of spasticity measurement can currently be covered in daily practice, depending on the goals of assessment. Simple VAS and Borg scores can be used to incorporate the patients' perception into the assessment in more standardized manner. Secondly, passive stretch tests can be applied, but all pro's and con's that were outlined earlier must be taken into account. Thirdly, the use of surface EMG is highly recommended in more complex cases, either under standardized circumstances or during daily activities. Finally, although not subject of study in this thesis, functional tests are recommended in high level patients.

## Methodological considerations and topics for future research



In the conducted studies presented in this thesis, potential differences between spasticity of spinal and supraspinal origin have not been taken into consideration. However, evidence exists that reflex activity behaves differently in the two groups.[5,24] Clinical differences can be understood by the location of the UMN lesion, as described in chapter 1. In patients with a spinal lesion afferent activity can lead to multilevel reflex responses. Cerebral patients often demonstrate recognizable

antigravity postural patterns.[25] Therefore the reported results cannot be generalized to all patients with UMN syndrome.

The clinimetric value of the Borg scale for the assessment of the experienced spasticity-related discomfort is indefinite so far. In our study (chapter 5), the Borg scale was used to measure the impact of spasticity during the self-defined activities. It seems an appropriate tool for this purpose, although its qualities in this field must be proven yet. Patients appeared very well able to differentiate between the degree of spasticity as such and the discomfort related to it. A limitation of this rating scale is the fact that patients can only score a negative impact. Even though this was the case most of the time, possible positive consequences of spasticity, could not be specified with this scale. There are some other interesting alternatives, described in literature. The evaluation tool described by Adams et al[26] is an example of a bidirectional self-rating scale concerning the impact of spasticity in daily life situations. The Canadian Occupational Performance Measure (COPM)[12] might be a useful tool for tracking both functional changes and patient satisfaction following, for instance, spasticity treatment, by means of a semi-structured interview. It is, however, not diagnosis-specific and test scores cannot be norm-referenced.

New initiatives for improvement of the clinimetric properties of passive stretch tests have been described in literature recently, for example the Spasticity Test[27] and the REPAS.[28] The first results are promising, but it seems too early for widespread clinical application and, more importantly, insight is needed in the relationship between passive stretch tests and more functional outcomes.

Some literature exists on other factors that potentially contribute to the patients' perception of spasticity.[22,23] The relative contributions of these factors to the total concept of spasticity are, however, not clear and need to be further explored.

The results, presented in this thesis, stress the need for clinically applicable, objective methods for proper evaluation of spasticity management. Surface EMG, applied in standardized conditions or for long-term use, has potential as a valid instrument for objective quantification of involuntary reflex muscle activity. To

facilitate its clinical use, the choice of parameters representing involuntary reflex muscle activity must be elaborated. In addition, future studies should continue to focus at distinguishing between voluntary and involuntary components of muscle activity.

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# Summary

Spasticity is a common phenomenon which often develops after an upper motor neuron (UMN) lesion, such as stroke, multiple sclerosis or spinal cord injury (SCI).
In the acute phase after a lesion the so-called negative signs, such as paresis, fatigue and loss of dexterity, are usually most prominent. Spasticity is part of the positive phenomena, characterized by an exaggerated motor response. The umbrella definition of spasticity that was recently introduced, defining spasticity as "disordered sensori-motor control, resulting from an UMN lesion, presenting as intermittent or sustained involuntary activation of muscles", is increasingly being used. It includes all afferent-mediated positive features of UMN syndrome.

In patients with an UMN lesion, clinical problems of movement dysfunction arise from a complex interaction between positive features, negative features, and changes in the physical properties of muscles and other tissues. Careful assessment of all signs and symptoms that might contribute to impaired motor function in the individual patient is essential in selecting the appropriate treatment.
Quantification of spasticity requires reliable and valid assessment methods, measuring spasticity in terms of 'Body Functions and Structures' within the framework of the International Classification of Functioning, Disability and Health (ICF). Objective measurement of spasticity has therefore been a major goal for clinical researchers for many years.
In current clinical practice several difficulties in spasticity assessment are encountered. Assessment of spasticity appears to be complex due to its various manifestations, difficulties to distinguish between neural and non-neural components, and different characteristics during passive and active, more functional movements. Additionally, there can be a discrepancy between outcomes of objective tests and the patients' perception. Finally, a single momentary assessment may be insufficiently reliable, due to variability of spasticity during the day.
So far there is no agreement in literature with regard to what assessment method is best in which situation. Consensus is growing that we need to measure spasticity at

different levels, covering the different manifestations of spasticity and representing spasticity at the different levels of the ICF framework.

In **chapter 1** the (patho)physiology of normal and increased muscle tone is described. Secondly, difficulties in clinical assessment of spasticity are described and the aim of this thesis is formulated. The principal aim is to contribute to the development of a comprehensive set of clinically applicable measurement tools for spasticity, to support clinical decision making.

**Chapter 2** presents the results of a study investigating the influence of posture and muscle length on stretch reflex activity in post-stroke patients with spasticity. In this study, stretch reflex activity is studied in nineteen stroke subjects with known spasticity, using the Ashworth scale, the pendulum test and passively imposed movement on the lower limbs, in both sitting and supine position. Muscle activity was assessed non-invasively with surface electromyography (sEMG). The results show that both clinical and neurophysiological assessment of spasticity is influenced considerably by change in posture and muscle length. All recorded muscles showed more stretch reflex activity in elongated state in comparison with shortened state of the muscle. Exact documentation of the patients' position is therefore essential for reliable assessment of spasticity.

The study described in **chapter 3** aimed at assessing the value of passive stretch tests in the measurement of spasticity. Stretch reflex activity of spastic muscles during cyclic passive movement was compared with reflex muscle activity during similar active movement of the lower leg. The outcomes of reflex activity during passive and active tests in post stroke patients appeared not to be related strongly. It was shown that muscle activation during stretch shows different behaviour in the two conditions. During the active movement task other mechanisms, such as delay in start and termination of contraction and co-activation, appear to play a role. Therefore it is concluded that the outcome of a passive stretch test alone is not considered representative for spasticity during active motor tasks nor for motor control.

**Chapter 4** describes the clinimetric properties of the current widely used clinical measurement method, the Ashworth scale. In this study both the construct validity and the inter-rater reliability of the Ashworth scale were investigated, by using real-time sEMG and dynamometry recordings. In the first part of the study three experienced (para)medics rated spasticity in the elbow flexor muscles of nineteen patients with upper motor neuron lesion. The second part of the study focused on spasticity in the knee extensors. It became clear that the Ashworth scale is neither sufficiently valid nor reliable to serve as a measurement method for spasticity, in both the upper and lower extremity. It is therefore essential that clinicians are well aware of the limitations of this scale. It is recommended that its use is avoided in clinical practice and in research.

**Chapter 5** focuses on the association between the patients' perception of spasticity, measured with the Visual Analogue Scale (VAS), and the experienced discomfort as a result of spasticity, measured with the Borg scale. Twenty six motor complete SCI patients were asked to fill out a questionnaire. The study showed that the experienced discomfort during an activity was only moderately related to the perceived degree of spasticity and that the impact of spasticity on daily life was related to the context in which it occurs. Correlation between VAS and Borg scale was higher during activities involving a 'stable body position' and while 'being active'. Patients indicated that the experienced discomfort was often associated with practical concerns, such as disturbance of sleep, compromised safety etc. On the other hand psychological factors, like the degree of acceptance of their situation or negative self-image, were reported as well.

The influence of contextual factors is strongly supported by the study described in **chapter 6**. This chapter addresses the association between patient ratings on the level of spasticity, using the VAS, and objective spasticity measurement, using long-term sEMG recordings during daily activities, in fourteen motor complete SCI patients. Patient ratings of the level of spasticity appeared to be poorly associated with spasticity, in terms of involuntary muscle activity assessed with long-term sEMG recordings.

**Chapter 7** addresses the quantification of involuntary muscle activity patterns in the lower limbs of nineteen patients with motor complete SCI during daily life activities. In these patients, with self-reported spasticity, the recorded muscles appeared to be active during only a small proportion of the day. Contextual factors were shown to play a role here as well: both the duration of muscle activity and the degree of co-activation of muscles were higher during specific daily activities. In particular activities involving change of position, such as transfers, appeared to elicit more muscle activity.

The thesis is concluding with a general discussion in **chapter 8**, in which the findings of the different studies are discussed and integrated. Emphasis is on the implications for clinical practice.

# Samenvatting

Spasticiteit is een fenomeen dat vaak voorkomt bij patiënten die een centraal neurologisch letsel hebben doorgemaakt, zoals een beroerte, multiple sclerose of een dwarslaesie.
In de acute fase na een letsel van het centraal motorisch neuron ('*upper motor neuron*', UMN) staan de zogenaamde negatieve symptomen, zoals verlamming, verhoogde vermoeibaarheid en verlies van behendigheid, meestal op de voorgrond. Spasticiteit behoort tot de positieve symptomen, die gekenmerkt zijn door een versterkte motorische reactie. De overkoepelende definitie van spasticiteit, die recent werd geïntroduceerd, wordt in toenemende mate gebruikt. Deze definitie beschrijft spasticiteit als 'verstoorde sensomotore regulatie na een centraal neurologisch letsel, die zich presenteert als intermitterende of aanhoudende onwillekeurige spieractivatie'. Deze definitie omvat alle afferent-gemedieerde positieve kenmerken van het UMN syndroom.

Bij patiënten met een UMN letsel kunnen problemen in bewegingsaansturing ontstaan als gevolg van een complexe interactie tussen positieve symptomen, negatieve symptomen en veranderingen in fysieke eigenschappen van spieren en andere weefsels. Het nauwkeurig vastleggen van de kenmerken die kunnen bijdragen tot verstoorde motorische functie is belangrijk voor het selecteren van de juiste behandeling voor een individuele patiënt.
Voor het kwantificeren van spasticiteit zijn betrouwbare en valide meetmethoden nodig, die spasticiteit meten op het niveau van 'Functies en Anatomische eigenschappen' in het raamwerk van de *'International Classification of Functioning, Disability and Health'* (ICF). Voor klinisch onderzoekers is het objectief meten van spasticiteit daarom al jarenlang een belangrijke doelstelling.
In de huidige klinische praktijk komt men diverse problemen tegen tijdens het meten van spasticiteit. Spasticiteit meten blijkt complex door de diverse uitingsvormen en de moeilijkheid om neurale en niet-neurale componenten te onderscheiden. Daarnaast heeft spasticiteit andere kenmerken tijdens passief bewegen vergeleken met actieve, meer functionele bewegingen. Bovendien kan

er discrepantie bestaan tussen de uitkomst van objectieve testen en de ervaring van de patiënten zelf. Tenslotte kan een enkele meting onvoldoende betrouwbaar zijn, als gevolg van de variabiliteit van spasticiteit door de dag heen.
Tot nu toe is er in de medische literatuur geen overeenstemming over welke meetmethode het beste is in welke situatie. Wel is er toenemende consensus over het feit dat spasticiteit op meerdere niveau's gemeten zou moeten worden, waarin de verschillende uitingsvormen worden meegenomen en die de verschillende niveau's van het ICF raamwerk representeren.

In **hoofdstuk 1** wordt de (patho)fysiologie van normale en verhoogde spierspanning beschreven. Tevens worden de problemen bij het meten van spasticiteit in de klinische situatie benoemd en het doel van dit proefschrift geformuleerd. Het belangrijkste doel is om een bijdrage te leveren aan het ontwikkelen van een brede set van klinisch toepasbare meetinstrumenten voor spasticiteit, ter ondersteuning van klinische besluitvorming.
**Hoofdstuk 2** beschrijft de resultaten van een studie, waarin de invloed van houding en spierlengte op rek reflex activiteit wordt bestudeerd bij patiënten met spasticiteit als gevolg van een beroerte. Rek reflex activiteit werd gemeten bij negentien patiënten met bekende spasticiteit, met behulp van de Ashworth schaal, de pendulum test en passief opgelegde bewegingen van de benen, in zowel zit- als lighouding. Spieractiviteit werd geregistreerd met oppervlakte elektromyografie (EMG). De resultaten laten zien dat zowel klinische als neurofysiologische metingen van spasticiteit fors worden beïnvloed door verandering in houding en spierlengte. Alle gemeten spieren vertoonden meer rek reflex activiteit in de verlengde ten opzichte van de verkorte toestand. Het exact vastleggen van de houding van de patiënt is daarom essentieel voor de betrouwbaarheid van de metingen.
De studie die beschreven is in **hoofdstuk 3** heeft als doel om de waarde van passieve rek testen voor het meten van spasticiteit vast te stellen. Rek reflex activiteit van spastische spieren tijdens cyclische passieve bewegingen werd vergeleken met reflex activiteit tijdens vergelijkbare actieve bewegingen van het been. De

uitkomsten van reflex activiteit tijdens passieve en actieve testen bleken niet sterk gerelateerd te zijn. De spieractivatie tijdens rekfase liet verschillend gedrag zien in de verschillende situaties. Tijdens de actieve taak blijken andere mechanismen, zoals co-activatie en vertraagde start en beëindiging van spiercontractie, een rol te spelen. Daaruit wordt geconcludeerd dat de uitkomst van een passieve rek test alleen onvoldoende representatief is voor spasticiteit tijdens actief bewegen of voor bewegingssturing.

**Hoofdstuk 4** beschrijft de klinimetrische eigenschappen van de veel gebruikte klinische meetmethode, de Ashworth Schaal. In deze studie werden zowel de construct validiteit als de interbeoordelaars betrouwbaarheid van de Ashworth Schaal onderzocht, door gelijktijdig gebruik van oppervlakte EMG en dynamometrie. In het eerste deel van de studie scoren drie ervaren (para)medici spasticiteit in de buigspieren van de elleboog van negentien patiënten met een UMN letsel. Het tweede deel van de studie richt zich op de strekspieren van de knie. De studie laat zien dat de Ashworth Schaal onvoldoende valide en betrouwbaar is als meetmethode voor spasticiteit, zowel voor de bovenste als onderste extremiteit. Het is daarom belangrijk dat clinici zich goed bewust zijn van de beperkingen van deze schaal. Het wordt dan ook aanbevolen om het gebruik van deze schaal, zowel in de klinische praktijk als in de onderzoekssetting, te vermijden.

**Hoofdstuk 5** richt zich op de associatie tussen de ervaren mate van spasticiteit door de patiënt, gemeten met de Visual Analogue Scale (VAS), en de ervaren mate van ongemak als gevolg van spasticiteit, gemeten met de Borg schaal. Zesentwintig patiënten met een motorisch complete dwarslaesie werden gevraagd om een vragenlijst in te vullen. De studie toonde aan dat de ervaren mate van ongemak slechts matig gerelateerd was aan de ervaren mate van spasticiteit. De impact van spasticiteit op het dagelijks leven bleek gerelateerd aan de context waarin het optrad. De correlatie tussen VAS en de Borg schaal was het hoogst tijdens activiteiten met een 'stabiele lichaamshouding' en tijdens 'actief zijn'. Patiënten gaven aan dat het ervaren ongemak vaak geassocieerd was met praktische bezwaren, zoals verstoring van de nachtrust of onveilige situaties. Psychologische

factoren, zoals de mate van acceptatie van hun situatie of een negatief zelfbeeld, werden ook genoemd.

De invloed van contextuele factoren wordt sterk bevestigd door de bevindingen uit de studie die beschreven is in **hoofdstuk 6**. Dit hoofdstuk gaat in op het verband tussen de ervaren mate van spasticiteit door veertien dwarslaesiepatiënten, gemeten met de VAS, en objectieve spasticiteitmetingen met behulp van oppervlakte EMG tijdens dagelijkse activiteiten. De door de patiënten ervaren mate van spasticiteit bleek slecht marginaal gerelateerd aan spasticiteit, ofwel onwillekeurige spieractivatie, gemeten met continue EMG metingen.

**Hoofdstuk 7** behandelt de kwantificatie van onwillekeurige spieractivatiepatronen in de onderste extremiteiten van negentien patiënten met een motorisch complete dwarslaesie tijdens dagelijkse activiteiten. In deze patiëntengroep, met zelfgerapporteerde spasticiteit, bleken de gemeten spieren gedurende slechts een klein deel van de dag actief te zijn. Contextuele factoren bleken ook hier een rol te spelen: zowel de duur van spieractivatie als de mate van co-activatie van spieren waren groter tijdens specifieke activiteiten. Met name activiteiten waarbij een verandering van houding plaatsvindt, zoals het maken van een transfer, bleken meer spieractiviteit uit te lokken.

Het proefschrift wordt afgesloten met een algemene discussie in **hoofdstuk 8**, waarin bovenstaande bevindingen worden bediscussieerd en geïntegreerd. De nadruk ligt hierbij op de implicaties voor de klinische praktijk.

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# Over de auteur

# Over de auteur

Judith Fleuren werd geboren op 28 december 1968 te Nijmegen. Na het VWO (Augustinianum te Eindhoven) studeerde zij geneeskunde aan de Universiteit te Maastricht, waar in 1995 het artsdiploma werd behaald. Zij verrichte wetenschapsstages op het gebied van biomedische technologie (Technische Universiteit Eindhoven) en interne geneeskunde (Universitair Ziekenhuis Gent, België, en Universidad de la Frontera, Temuco, Chili).

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Belangstelling voor wetenschappelijk onderzoek was de reden om naar Enschede te vertrekken. In juli 2002 werd de opleiding tot revalidatiearts gestart in revalidatiecentrum Het Roessingh, in combinatie met promotieonderzoek (de zgn. aiosko-constructie) bij Roessingh Research and Development. In juni 2006 ontving zij in Turijn een prijs voor beste klinische presentatie tijdens het XVI$^{e}$ congres van de International Society for Electrophysiology and Kinesiology. In december 2006 haalde zij haar Master of Epidemiology, toegekend door de Vrije Universiteit van Amsterdam. Zij is geregistreerd als epidemioloog.

Judith Fleuren werkt sinds 1 juli 2009 als revalidatiearts in Het Roessingh, met als aandachtsgebied CVA. Zij blijft als onderzoeker verbonden aan Roessingh Research and Development.

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