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

G E Voerman,[1] J F M Fleuren,[1] L A C Kallenberg,[1] J S Rietman,[1,2] G J Snoek,[1] H J Hermens[1,2]

[1] Roessingh Research and Development, Enschede, The Netherlands; [2] Faculty of Electrical Engineering, Mathematics and Computer Science, University of Twente, Enschede, The Netherlands

Correspondence to:
Dr G E Voerman, Roessingh Research and Development, PO Box 310 7500 AH Enschede, The Netherlands;
g.voerman@iq.umcn.nl

Received 15 February 2008
Revised 29 July 2008
Accepted 8 August 2008
Published Online First
23 October 2008

ABSTRACT

Aim: 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.

Method: sEMG 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 the Visual Analogue Scale (VAS) for that particular activity. sEMG data were analysed 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: 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%.

Conclusions: 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.

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 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 "standardised" 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 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 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 analysed 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 scrutinise the

Table 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

AS, Ashworth scale.

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 AND MATERIALS

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 1.

Measurement protocol

Each subject was measured at 2 or 3 days, with a cumulative minimum of about 10 h, 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, pregelled ARBO H93, interelectrode distance 24 mm) placement were performed according to international guidelines for sensor placement.[16] The 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 frequency 1024 Hz; manufactured by TMSi, Oldenzaal, The Netherlands) by means of cables taped to the skin (fig 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]

Figure 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 artefacts are likely to occur.[18] Beginnings and endings of bursts of muscle activity were subsequently detected using custom-made software based on the Approximated Generalised 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 judgement.[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 s 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 artefacts.

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 each of 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 min 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 inter-related, 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). An 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" (dichotomised 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; ie, 3%). The percentage of agreement between experts and algorithm was thus high, that is 97%.

Description of data

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

"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%, eg, getting dressed), "being active" (13%, eg, performing sports), "stable body position" (7%, eg, working behind computer), "therapy" (5%, eg, occupational and physical therapy), and "other" (4%, eg, emotional conversations, clinical evaluations). The sEMG burst data for each group of activities are provided in table 3.

sEMG components defined by PCA

PCA results indicated the extraction of seven components, as for these components, eigenvalues were >1 (fig 3). Inspection of the component loadings indicated that all variables were strongly loaded on one component only (see table 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 4).

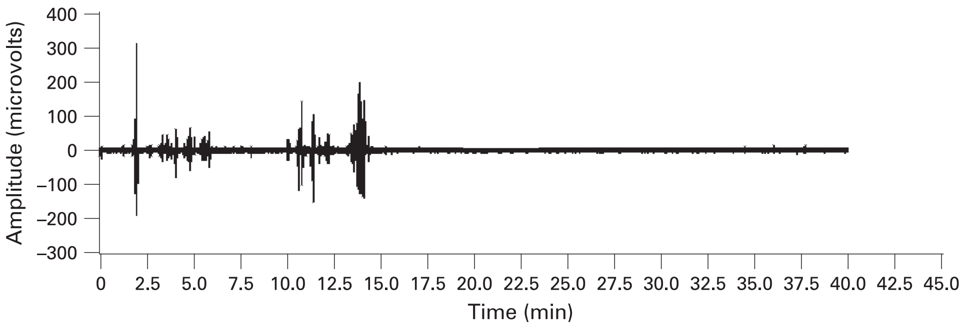

Figure 2 Muscle activity of the rectus femoris during activities of daily life.

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.

Association between VAS, sEMG components and context

Seven fixed factors (the seven components) and one random factor (ie, 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 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 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 to 56.8) and "other" (39.0; 0 to 74) significantly higher VAS scores were reported compared with activities classified as "stable body position" (12.5; 1 to 41.5), while "activities of daily living" (16.5; 3 to 39.3), "being active" (16.0; 6 to 34), and "therapy" (18; 0 to 55) did not (see table 5). This model (−2 log likelihood = 2273.6, compared with 2377.5 for model without fixed factors) explained 35% of the variance in 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

Table 2 Number of activities, related Visual Analogue Scale (VAS) scores, and number of hours recorded per patient

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

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

Table 3 Median and interquartile range scores for the separate surface electromyography variables

	Activity 1	Activity 2	Activity 3	Activity 4	Activity 5	Activity 0
Mean RMS RF	9.3 (6.5 to 12.7)	9.5 (6.5 to 12.5)	6.6 (5.5 to 9.8)	9.6 (5.7 to 14.5)	9.7 (7.4 to 15.5)	10.5 (9.4 to 11.9)
Mean RMS VL	9.9 (7.2 to 14.4)	8.4 (5.8 to 14.9)	6.2 (4.8 to 12.5)	12.8 (7.0 to 18.6)	9.2 (5.4 to 18.5)	8.8 (6.1 to 12.5)
Mean RMS AD	7.2 (5.2 to 11.2)	8.2 (5.6 to 10.6)	7.6 (5.0 to 10.2)	6.9 (5.6 to 14.2)	7.8 (6.1 to 14.5)	7.2 (5.8 to 10.1)
Mean RMS ST	8.4 (6.3 to 10.5)	9.2 (7.6 to 12.2)	6.8 (4.8 to 7.2)	5.6 (4.6 to 7.9)	7.4 (5.8 to 9.4)	6.6 (5.6 to 16.7)
SD RMS RF	5.9 (2.9 to 10.1)	5.7 (2.3 to 10.8)	4.8 (3.4 to 7.5)	6.2 (2.0 to 11.9)	8.8 (4.7 to 16.9)	6.5 (5.7 to 9.7)
SD RMS VL	6.7 (3.6 to 12.2)	7.3 (2.3 to 13.7)	5.0 (1.0 to 9.4)	6.9 (3.6 to 16.8)	5.0 (0.9 to 15.0)	5.8 (2.4 to 16.7)
SD RMS AD	4.0 (1.8 to 7.4)	4.4 (2.1 to 6.9)	3.7 (1.5 to 13.2)	4.6 (2.1 to 15.4)	5.8 (2.7 to 13.2)	3.5 (2.3 to 5.7)
SD RMS ST	4.4 (2.5 to 8.3)	6.7 (3.4 to 10.9)	2.6 (1.7 to 5.2)	2.8 (2.0 to 4.3)	3.3 (2.7 to 8.2)	3.6 (2.2 to 8.6)
No of bursts RF	4 (4 to 13)	5 (2 to 11.5)	11 (2.5 to 46)	7 (1 to 21.5)	8 (3.3 to 33.8)	7 (4 to 9)
No of bursts VL	13 (4 to 21.3)	8 (3 to 27.5)	54 (8 to 85)	26 (6 to 69.5)	12.5 (1.8 to 25.6)	8 (6 to 16)
No of bursts AD	6 (3.3 to 11)	5.5 (2 to 13.3)	5 (2 to 35)	13 (2 to 29.5)	6 (1.8 to 48.5)	3 (2 to 7)
No of bursts ST	7 (4 to 13)	6 (2 to 12)	7.5 (4 to 16.5)	7.5 (4 to 16.5)	15.5 (2.8 to 27.0)	6.5 (3.0 to 18.8)
Mean burst duration RF	1.22 (0.5 to 2.7)	1.0 (0.4 to 2.9)	0.9 (0.3 to 1.7)	0.8 (0.5 to 31.5)	1.0 (0.5 to 1.8)	2.9 (2.0 to 4.3)
Mean burst duration VL	0.7 (0.4 to 1.8)	2.3 (0.3 to 3.1)	0.4 (0.2 to 0.8)	0.7 (0.2 to 2.0)	0.6 (0.4 to 1.7)	1.8 (1.0 to 2.4)
Mean burst duration AD	3.2 (1.4 to 6.4)	2.1 (0.7 to 4.6)	2.1 (0.5 to 3.2)	3.2 (1.7 to 4.4)	3.1 (2.2 to 6.5)	4.5 (3.1 to 5.2)
Mean burst duration ST	5.3 (2.6 to 8.5)	3.1 (1.2 to 7.1)	2.5 (0.9 to 9.1)	3.4 (2.9 to 8.7)	3.0 (1.4 to 3.4)	4.6 (3.8 to 5.0)
SD burst duration RF	1.6 (0.6 to 2.7)	1.3 (0.4 to 2.7)	1.0 (0.3 to 2.0)	2.6 (1.1 to 175.4)	0.7 (0.2 to 2.9)	3.0 (2.2 to 4.9)
SD burst duration VL	1.2 (0.5 to 2.4)	1.1 (0.6 to 2.4)	0.7 (0.4 to 1.9)	3.1 (0.6 to 17.6)	0.5 (0.1 to 2.7)	2.3 (1.6 to 3.6)
SD burst duration AD	3.5 (1.7 to 5.7)	2.4 (1.3 to 3.8)	1.7 (0.5 to 3.8)	3.8 (2.1 to 5.5)	2.3 (1.1 to 4.1)	3.9 (1.5 to 5.0)
SD burst duration ST	5.1 (2.8 to 7.9)	3.5 (1.7 to 7.0)	39.5 (2.0 to 67.5)	4.8 (1.1 to 8.4)	4.1 (2.3 to 5.3)	3.7 (2.6 to 6.1)

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. AD, adductor group; RF, rectus femoris; RMS, root mean square; ST, semitendinosus; VL, vastus lateralis.

is associated with the (hyper)excitability of neural pathways due to loss of supraspinal control: increased alpha-motoneuron 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 hypothesise 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 the 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 hypothesised that spasticity would also be dependent on context variables like time of day and the activity that was being performed. Skold[17] showed, for instance, fluctuating hourly VAS ratings in cervical SCI patients. Results of the

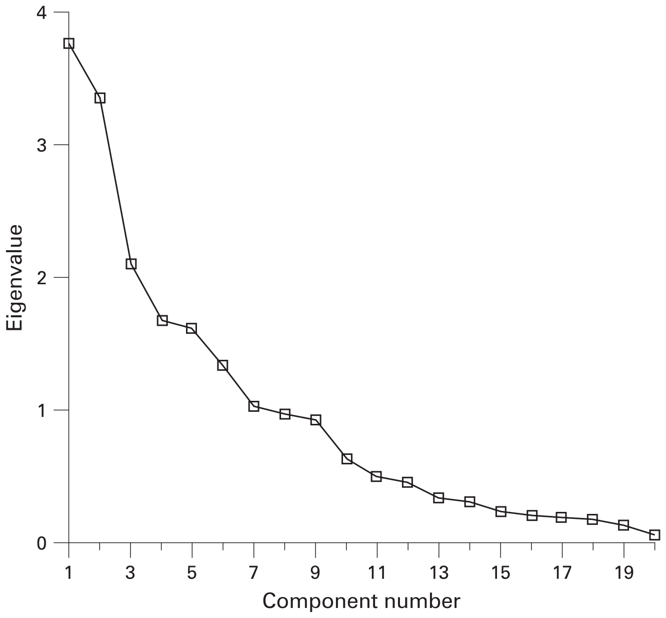

Figure 3 Scree plot.

Table 4 Loadings, percentage of variance for principal-components extraction and varimax rotation on sEMG variables

	Component 1	Component 2	Component 3	Component 4	Component 5	Component 6	Component 7
Mean burst duration AD	**0.686**	−0.080	−0.058	−0.046	−0.042	−0.042	0.299
Mean burst duration ST	**0.832**	−0.129	−0.004	0.176	0.082	0.001	−0.030
SD burst druration AD	**0.685**	0.006	0.088	0.020	−0.033	0.024	0.338
SD burst duration ST	**0.791**	−0.081	0.069	0.217	0.166	0.061	−0.093
Mean RMS RF	0.055	**0.789**	0.077	−0.225	0.076	0.166	0.055
Mean RMS AD	−0.256	**0.754**	0.129	0.205	0.084	0.025	−0.042
SD RMS RF	0.082	**0.795**	0.147	−0.261	0.070	0.120	0.120
SD RMS AD	−0.293	**0.718**	0.200	0.169	0.130	0.052	−0.042
No of bursts RF	0.127	0.180	**0.769**	−0.080	0.123	0.044	0.138
No of bursts VL	0.214	0.131	**0.693**	0.039	−0.161	−0.183	0.010
No of bursts AD	−0.071	0.138	**0.760**	0.038	0.039	0.200	0.032
No of bursts ST	−0.138	0.033	**0.760**	−0.006	−0.027	0.164	−0.034
Mean burst duration RF	0.164	−0.047	−0.015	**0.918**	0.061	0.022	0.119
SD burst duration RF	0.139	−0.049	0.006	**0.919**	0.078	0.027	0.130
Mean RMS ST	0.091	0.144	−0.103	0.075	**0.914**	0.077	0.015
SD RMS ST	0.044	0.123	0.085	0.059	**0.924**	0.066	−0.015
Mean RMS VL	0.018	0.098	0.046	−0.014	0.044	**0.923**	−0.070
SD RMS VL	0.022	0.187	0.190	0.065	0.104	**0.882**	0.034
Mean burst duration VL	0.103	−0.008	0.007	0.090	0.032	−0.002	**0.855**
SD burst duration VL	0.174	0.093	0.105	0.143	−0.031	−0.039	**0.839**
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.
AD, adductor group; RF, rectus femoris; RMS, root mean square; ST, semitendinosus; VL, vastus lateralis.

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-motoneuron 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 characterised 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 the sampling adequacy, sphericity, accumulated explained variance (ie, 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

Table 5 Multilevel models

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

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.

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 generalisable 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.

Acknowledgements: The authors would like to thank A Nene, V Erren-Wolters, H Witteveen, D Eilander, B Fleerkotte and K Groothuis-Oudshoorn for their contribution to this study.

Funding: This work is part of the Freeband AWARENESS project (http://awareness.freeband.nl). Freeband is sponsored by the Dutch government under contract BSIK 03025.

Competing interests: None.

Ethics approval: Ethics approval was provided by the Medical Ethics Committee of Roessingh, Enschede (NL).

Patient consent: Obtained.

REFERENCES

1. **Vanek, ZF**. Spasticity. August 2007. http://www.emedicine.com/neuro/topic706.htm (accessed 10 Oct 2007).
2. **Lance JW.** *Symposium synopsis*. Chicago: Year Book Medical Publishers, 1980:485–94.
3. **Pandyan AD,** Gregorič M, Barnes MP, *et al*. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil* 2005;**27**:2–6.
4. **Adams MM,** Hicks AL. Spasticity after spinal cord injury. *Spinal Cord* 2005;**43**:577–86.
5. **Voerman GE,** Gregorič M, Hermens HJ. Neurophysiological methods for the assessment of spasticity: the Hoffmann reflex, the tendon reflex, and the stretch reflex. *Disabil Rehabil* 2005;**27**:33–68.
6. **Pandyan AD,** Johnson GR, Price CI, *et al*. A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measures of spasticity. *Clin Rehabil* 1999;**13**:373–83.
7. **Lechner HE,** Frotzler A, Eser P. Relationship between self-and clinically rated spasticity in spinal cord injury. *Arch Phys Med Rehabil* 2006;**87**:15–19.
8. **Platz T,** Eickhof C, Nuyens G, *et al*. Clinical scales for the assessment of spasticity, associated phenomena, and function: a systematic review of the literature. *Disabil Rehabil* 2005;**27**:7–18.
9. **Wood DE,** Burridge JH, van Wijck FM, *et al*. Biomechanical approaches applied to the lower and upper limb for the measurement of spasticity: a systematic review of the literature. *Disabil Rehabil* 2005;**27**:19–32.
10. **Burridge JH,** Wood DE, Hermens HJ, *et al*. Theoretical and methodological considerations in the measurement of spasticity. *Disabil Rehabil* 2005;**27**:69–80.
11. **den Berg JP,** van Asbeck FWA. A 24-hour monitoring of spasticity. *J Rehabil Sci* 1991;**4**:46–8.
12. **Tepavac D,** Swenson JR, Stenehjem J, *et al*. Microcomputer-based portable long-term spasticity recording system [abstract]. *IEEE Trans Biomed Eng* 1992;**39**:426–31.
13. **Kleissen RFM,** Baardman G, Hermens HJ, *et al*. Long-term ambulatory recording of surface EMG in the assessment of spasticity. In: Anderson PA, Hobart DJ, Danoff JV, eds. *Proceedings of the International Society of Electrophysiological Kinesiology*. 1991:45–52.
14. **Stenehjem J,** Swenson JR, Grange TS. Spasticity evaluation by ambulatory EMG. *Adv Ext Control Hum Extrem* 1987;**IX**:385–9.
15. **Bacher M,** Scholz E, Diener HC. 24 hour continuous tremor quantification based on EMG recording. *Electroencephal Clin Neurophysiol* 1989;**72**:176–83.
16. **Hermens HJ,** Freriks LW, Disselhorst-Klug C, *et al*. Development of recommendations for SEMG sensors and sensor placement procedures. *J Electromyogr Kinesiol* 2000;**10**:361–74.
17. **Skold C.** Spasticity in spinal cord injury: self-and clinically rated intrinsic fluctuations and intervention-induced changes*. Arch Phys Med Rehabil* 2000;**81**:144–9.
18. **Hermens HJ,** Hutten MMR. Muscle activation in chronic pain: its treatment using a new approach of myofeedback. *Int J Indust Ergon* 2002;**30**:325–36.
19. **Staude GH.** Precise onset detection of human motor responses using a whitening filter and the log-likelihood-ratio test. *IEEE Trans Biomed Eng* 2001;**48**:1292–305.
20. **Takada K,** Yashiro K, Morimoto T. Application of polynomial regression modeling to automatic measurement of periods of EMG activity. *J Neurosci Meth* 1995;**56**:43–7.
21. **Marieb EN.** *Human anatomy & physiology*. Redwood City: Benjamin Cummings, 2001.
22. **Hoffman DS,** Strick PL. Step-tracking movements of the wrist. III. Influence of changes in load on patterns of muscle activity. *J Neurosci* 1993;**13**:5212–27.
23. **Cattell RB.** The scree test for the number of factors. *Multivar Behav Res* 1966;**1**:629–37.
24. **Tabachnick BG,** Fidell LS. *Using multivariate statistics*. Boston: Allyn & Bacon, 2001:607–75.
25. **Comrey AL,** Lee HB. *A first course in factor analysis*. 2nd edn. Hillsdale: Lawrence Erlbaum, 1992.
26. **Snijders T,** Bosker R. *Multilevel analysis. An introduction to basic and advanced multilevel modeling*. London: Sage, 1999:101.
27. **Mahoney JS,** Engebretson JC, Cook KF, *et al*. Spasticity experience domains in persons with spinal cord injury. *Arch Phys Med Rehabil* 2007;**88**:287–94.