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Altered motor control and spasticity after spinal cord injury: Subjective and objective assessment

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Abstract—This study of measures of spasticity, or altered motor control, compares the clinically used Ashworth scale with a method based on surface electromyographic (sEMG) recordings called brain motor control assessment (BMCA) in a group of 97 subjects with spinal cord injury (SCI) and varying levels of motor dysfunction. In this paper, we describe how sEMG-derived scores relate to the severity of spasticity as judged clinically. When sEMG data from passive movements from the BMCA were analyzed by Ashworth category, we found that when the sEMG data were averaged for a limb, there was a significant difference between scores for those with Ashworth 0 vs. 2 and 3, and 1 vs. 2 and 3 ($p<0.001$), but not between 0 and 1. Analysis of the individual muscle scores improved the discrimination between Ashworth categories. Superiority of sEMG data over Ashworth category as an objective quantification of altered motor control ("spasticity") is argued.

Key words: *classification, computer-assisted, electromyography, muscle spasticity, signal processing, spinal cord injuries.*

INTRODUCTION

Spasticity is a common sequellae of spinal cord injury (SCI): along with paralysis and pain, it is a major complaint of individuals after SCI. Levi and colleagues reported 68 percent of those persons experienced spasticity and 41 percent of those experiencing pain or limitation of activities as a consequence of that spasticity (1). Even among those with American Spinal Injury Association (ASIA) impairment scale scores D or E, 16 percent had problems with neurological deterioration or spasticity (2). While many means of treatment of spasticity are being explored (3–10), the existing options for such treatment are limited. In spite of the best efforts to manage spasticity clinically, it remains an important problem for individuals after SCI (11,12). Advances in, or selection of, management methods are complicated by the absence of appropriate means for objectively and quantitatively monitoring spasticity and upper motor neuron dysfunction (13).

A widely accepted definition of spasticity was presented by Lance (14), namely that spasticity is a "velocity-dependent increase in tonic stretch reflexes ... with exaggerated tendon jerks ... as one component of the upper motor neuron syndrome". This definition has been defended as "the only motor neuron symptom which, so far, has responded to drug therapy" (15). Young broadened the definition to include the Babinski response, velocity-dependent increase in tonic stretch reflexes, exaggerated phasic stretch reflexes, hyperactive cutaneous reflexes, increased autonomic reflexes, and abnormal postures to describe manifestations of excessive involuntary motor activity, which he defined as spastic paresis (16). It is quite common for discussions of spasticity to implicitly use the broader definition (17). However, there is general agreement

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that, at present, there exists no ideal "gold standard" against which to compare putative systems for assessing the spastic individual (16,18).

The Ashworth scale (19) is most commonly used clinically. It was initially intended as part of a triad to standardize observations of "spastic weakness" in multiple sclerosis along with spasms and individuals' self-reports. The scale was subsequently modified to increase its resolution at low levels of perceived resistance in the upper limb of individuals after stroke (20). In spite of the inclusion of spasm frequency and self-report as important components of the clinical picture in Ashworth's original paper, clinical reports tend to be non-comprehensive in scope, which may limit their utility (21). In a recent study of 15 subjects with motor complete SCI, Sköld and colleagues sought to validate use of the Ashworth scale by comparing scores with surface electromyographic (sEMG) measures (22). However, Haas et al. concluded that the Ashworth scale is of limited use in the assessment of spasticity in the lower limb of individuals with SCI (23).

Previous attempts to objectively assess spasticity through a quantitative method have focused upon either direct or indirect measurement of forces generated by muscles during movement (24–27) corresponding to the definition presented by Lance (15) or on evaluation of excitability of the stretch reflex loop (28–32), in an attempt to address underlying mechanisms of spasticity. However, these quantitative methods have not gained wide acceptance for a variety of reasons. There is a perception that quantitative measures do not correlate well with clinical measures (8). A partial explanation for this lack of correlation may be found in the argument by Priebe et al. that the various clinical measures used assess different aspects of spasticity (21). The fact that any quantitative measure does not correlate with a particular clinical measure, then, should not be particularly surprising. Furthermore, some of the quantitative methods suggested are difficult to employ in a routine clinical setting, requiring expensive equipment, extensive set-up, or both.

We have described a multi-channel sEMG technique, which provides a basis for comprehensive assessment of spasticity (33). We called this method brain motor control assessment (BMCA) to indicate the importance of descending control even in apparently paralyzed subjects (34,35). This method, which is in many ways similar to a comprehensive, clinical neurological examination carried out with sEMG recording, provides objective, quantifiable and reproducible data (36) regarding the subject's altered motor control, or motor behavior, under a variety of conditions. Quantitative measures derived from sEMG data along with clinical measures were used to build a model relating the two (37). Analysis of this model indicated that, if properly combined, these sEMG measures are related to the Ashworth classifications, and can be used to predict the clinical findings.

To further explore the utility of the sEMG method to characterize altered motor control (spasticity) in subjects after SCI and as an initial effort to develop an index of that altered control based on the sEMG measurements, we report sEMG observations during passive limb movement. We analyzed this data from 97 subjects with SCI to investigate the utility of two different combinations of the data sets in differentiating among the subjectively determined Ashworth categories for these subjects who exhibited varying degrees of spastic behavior and spasticity.

METHOD

Subjects

A convenience sample of 95 male and 2 female veterans with cervical or thoracic SCI were studied. The time since injury ranged from 0.5 to 39 years (7.7±7.8). The subjects ranged in age from 21 to 82 (mean 45). Because we are interested in muscle activity in the lower limbs, the study was designed to exclude subjects with extensive lower motor neuron injuries. Sixty-two of the subjects had cervical lesions, and the remainder, thoracic.

Clinical Examination

All subjects were examined in the same laboratory environment. The examination was in two parts, carried out after placement of surface electrodes over major muscle groups of the lower limbs. The first part consisted of a standardized clinical examination. Sensory and motor neurological levels for left and right sides and degree of completeness (AIS) was determined using the International Standard for Neurological and Functional Classification of Spinal Cord Injury (38,39).

The Ashworth Scale (19) was used to assess muscle tone in the lower limb while moving the hip and knee together in a single maneuver. A single score was given to the full maneuver (flexion and extension) for each limb. Subjects with Ashworth ratings of 0 to 3 were included. Subjects with Ashworth scores of 4 were excluded due to the inability of the technologist conducting the neurophysiological study to complete the hip and knee flexion maneuver reliably, thereby underestimating the (potential) sEMG activity, and

to the limited number of subjects with such scores (2) from the original sample of subjects.

Recording

The second part of the examination, the brain motor control assessment (BMCA) has been extensively described elsewhere (33) but major points are repeated here. Pairs of recessed, silver-silver chloride surface electrodes were centered on identified reference points (40), with 3-cm separation between the electrodes. The skin was lightly abraded to obtain a pair-wise electrode impedance less than 5 KΩ. The quadriceps electrode pair (Q) was placed on the anterior aspect of the thigh midway between the superior border of the patella and the anterior superior iliac spine. The adductor electrodes (A) were placed midway between the medial femoral epicondyle and the pubic tubercle. The hamstrings electrodes (H) were placed at the midpoint of a line between the fibular head and the ischial tuberosity, with the hip and knee flexed, allowing access to the posterior portion of the thigh. The tibialis anterior electrodes (TA) were placed four finger breadths below the tibial tuberosity and one finger breadth lateral to the tibial crest. The triceps surae pair (TS) was placed one hand breadth below the popliteal crease on the midline of the calf.

sEMG signals were amplified using Grass 12A5 amplifiers (Astro-Med, Inc., West Warwick, RI 02893) with a gain of 5,000 and a bandwidth of 40 Hz to 600 Hz (-3 db) and were digitized at 1,800 samples per second (s/s) per channel with 12-bit accuracy using the CODAS data acquisition system (DATAQ Instruments, Inc. Akron, OH 44333). After calibration, data were continuously recorded for the approximately 1 hr required for the electrophysiological data collection. Four additional channels were used at various points in the protocol, two level sensor outputs to document thigh, leg or foot movement, a transducer channel to document tendon tap force, and an event mark to denote timing of protocol commands.

Protocol

Data were collected in strict accordance with a protocol (33), beginning with 5 min of relaxation followed by reinforcement maneuvers, voluntary maneuvers, passive maneuvers, tendon taps, clonus, application of vibration, and finally, plantar stimulation. Each subject received the identical protocol in the identical order, administered by a highly experienced BMCA technologist. For this paper, responses to the three repetitions of passive movement of the right hip and knee are reported. The maneuver consisted of hip and knee flexion together (first phase) then extension (second phase). Each phase was maintained for a minimum of 5 s to provide time for the subject's responses to plateau, and to allow a constant analysis time to be used.

Data Reduction

Clinical data were scored according to published clinical scales as previously described (21). The originally recorded, full bandwidth sEMG data were reduced using a root mean square (RMS) algorithm (41) to an sEMG envelope with an effective sampling rate of 20 s/s. This envelope data was the basis for subsequent processing. We then averaged activity for each muscle channel over a 5-s window, corrected for the baseline by subtraction of the average activity in the 1 sec immediately preceding the maneuver (36). The sEMG envelope data for each maneuver were thereby reduced to a vector of 10 response elements from left and right lower limbs, in which each element corresponded to the activity of an individual muscle to a phase of a maneuver. As unilateral maneuvers were examined and analyzed in this paper, only the right five elements are presented in this paper. In this manner, we derive a five-element response vector (RV) in which each element represents the response of a single muscle (Q, A, H, TA, TS) to one phase (flexion or extension), presented in units of μV_{RMS}. This response vector was also reduced to a single variable by averaging these five elements to create the average limb response (ALR), again with units of μV_{RMS}. Thus, we represented the sEMG response to each phase of each maneuver from all five muscles as either a single, scalar quantity (ALR) or a vector (RV) to reduce the data to a manageable size from the original, nearly 200 Mbytes of data.

Data Analysis

Two separate analyses of variance were conducted utilizing these sEMG data. As the "expected" activity in a healthy subject is zero, for the first analysis, we chose to use the ALR, which was analyzed using Ashworth categories (4 levels) and phase of movement (2 levels) as independent variables providing a 4x2 design with one dependent variable.

The second analysis was a multivariate analysis using the five elements of the RV as dependent variables. The Ashworth categories (4 levels) and flexion and extension (2 levels) served as independent variables providing a 4 x 2 design with 5 dependent variables. The individual response elements (muscles) were included to determine if there are differences in the patterns of motor activity between the

Table 1.
Subject characteristics by Ashworth score.

		Ashworth 0	1	2	3
Gender	M	24	33	22	16
	F	1		1	
ASIA	A	12	14	9	6
	B	10	10	5	3
	C		6	7	4
	D	3	3	2	3
Level	Cer	12	21	17	12
	Thor	13	12	6	4
Motor	Mean	41.5	35.9	28.5	34.9
	SD	20.0	21.8	20.1	21.1
Age	Mean	42.3	46.1	49.4	43.7
	SD	13.2	13.3	17.1	13.7
Time	Mean	7.6	8.4	6.4	8.6
	SD	9.4	7.7	6.5	7.7

M=male; F=female; ASIA=ASIA impairment score; Level=injury level; Cer=cervical; Thor=thoracic; Motor=total motor score; SD=standard deviation; age in years; Time=time since injury, in years.

Ashworth categories. These data were analyzed using multivariate general linear model procedure utilizing type III sums of squares.

RESULTS

Subject Characteristics

The average ASIA motor score in the study group was 18.3 (SD 11.6) and ranged from 0 to 47. The extent of injury was assessed using the ASIA impairment scale (AIS). Subjects in this study exhibited a range of severity of lesions, with 42 percent classed as AIS A, 29 percent as AIS B, 18 percent as AIS C and 11 percent, AIS D. The main grouping variable in this study was the subject's Ashworth score. There were 25 subjects in group 0, 33 in group 1, 23 in group 2, and 16 in group 3. There is no significant difference on any of the subject descriptors between the Ashworth groups. **Table 1** shows the descriptors arranged by Ashworth category.

Qualitative Evaluation

The clinical hallmark of spasticity, as ordinarily construed, is the resistance to passive stretch. In individuals with spasticity after spinal cord injury, passive movements may activate the muscles stretched as evident both in the perceived resistance to stretch and in the electromyographic activity in those muscles. To illustrate the type of surface electromyographic (sEMG) activity elicited by these passive maneuvers, we present responses in two subjects (**Figures 1** and **2**). Flexing the hip and knee resulted in reproducible activation of the H, with accompanying resistance to movement, indicated by the rating of 3 on the Ashworth scale (**Figure 1a**). Similarly, the second or extension phase of the movement, stretched and activated the Q (**Figure 1b**). Note that one of three flexion trials resulted in activation of the TA, and all three extension phase movements activated the TS (**Figure 1b**), although the ankle joint was not directly manipulated by the technologist in the maneuver. This subject presents one of many possible variations of responses to passive manipulation of the hip and knee.

While the clinical examiner may perceive resistance in the form of opposing torque generated about the joint or joints moved, the sEMG signals reveal any spread of activation to other muscles in the same limb or even to muscles of the contralateral limb, as is illustrated in **Figure 2** in another subject exhibiting spastic behavior. In this subject, passive hip and knee flexion movement resulted in relatively low amplitude sEMG signals, in particular, less activity in the H (**Figure 2a**) compared to the first subject's responses shown in **Figure 1a**. This lower activity may correspond to the lower Ashworth score (1/4) for this subject. However, activity was also evident in TA and TS on all three repetitions in this subject. Furthermore, the extension movement elicited a great deal of activity in the second subject, although much of the activity occurred after the end of the movement (**Figure 2b**). Thus, the sEMG data generated during passive movement usefully describe the behavior of the spinal motoneuron pools during and after the conclusion of such movements, for a period of time selected in the analysis (here 5 s).

Data Reduction

As described in the Methods, the sEMG waveform data are reduced to a response vector (RV) by computing the average of the root mean square (RMS) voltage during the 5 s following initiation of the maneuver. In **Table 2** are presented the RVs derived from the exemplary data for the maneuvers pictured in **Figures 1** and **2**. These response vector elements are the average sEMG voltages (μV_{RMS}) calculated across 5 s for each muscle and across the three repetitions shown in the figures. This unilateral maneuver resulted in primarily unilateral muscle activity in both

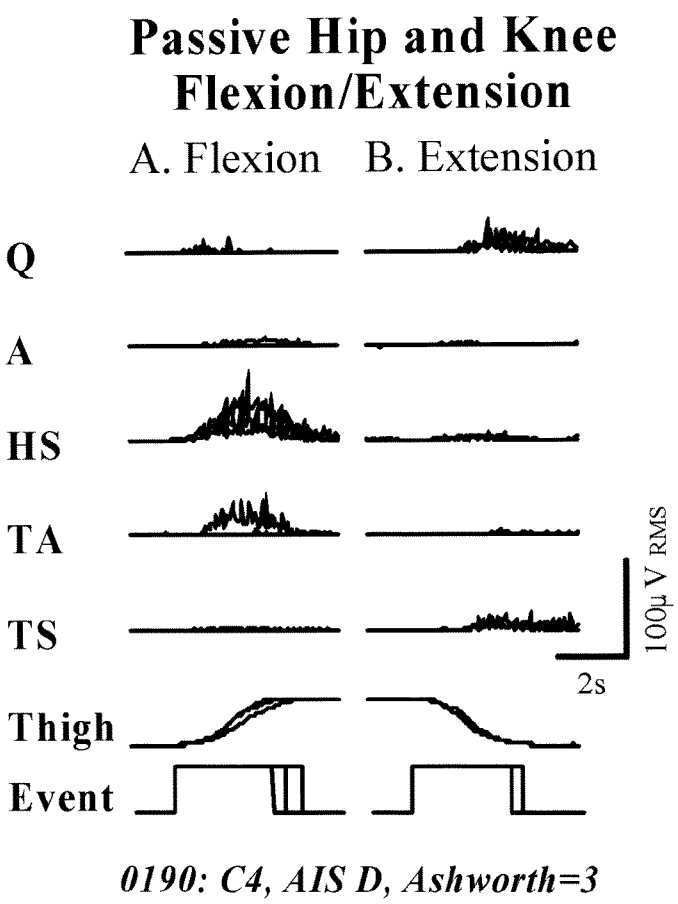

Figure 1.
sEMG Waveforms during Repeated Passive Hip/Knee Flexion and Extension.
a) Flexion of the hip and knee resulted in very little recorded activity in this subject, primarily in the H. This activity declined somewhat in the H, and diminished to essentially zero after the first movement in the TA.
b) Extension of the hip and knee from flexion to the starting position resulted in very little activity. However, the clinical evaluation rated this subject 3/4 on the Ashworth scale for passive movement of the right hip and knee together. Data in this and the subsequent figure are from quadriceps (Q), adductors (A), hamstrings (H), tibialis anterior (TA) and triceps surae (TS) muscles. The thigh position is indicated (flexion upward, extension downward). Finally, the event marker denotes the examiner's indication of the beginning and end of each phase of the movement. The data shown are three superimposed sets of waveforms, plotted from the data extracted to compute the average activity for each muscle, with the initial second being baseline, and the last 5 s, the response to the maneuver. Shown at the bottom of the figure is the study number, the level of the lesion (motor, right), the ASIA impairment scale and in this case, the Ashworth score for passive right hip and knee flexion and extension.

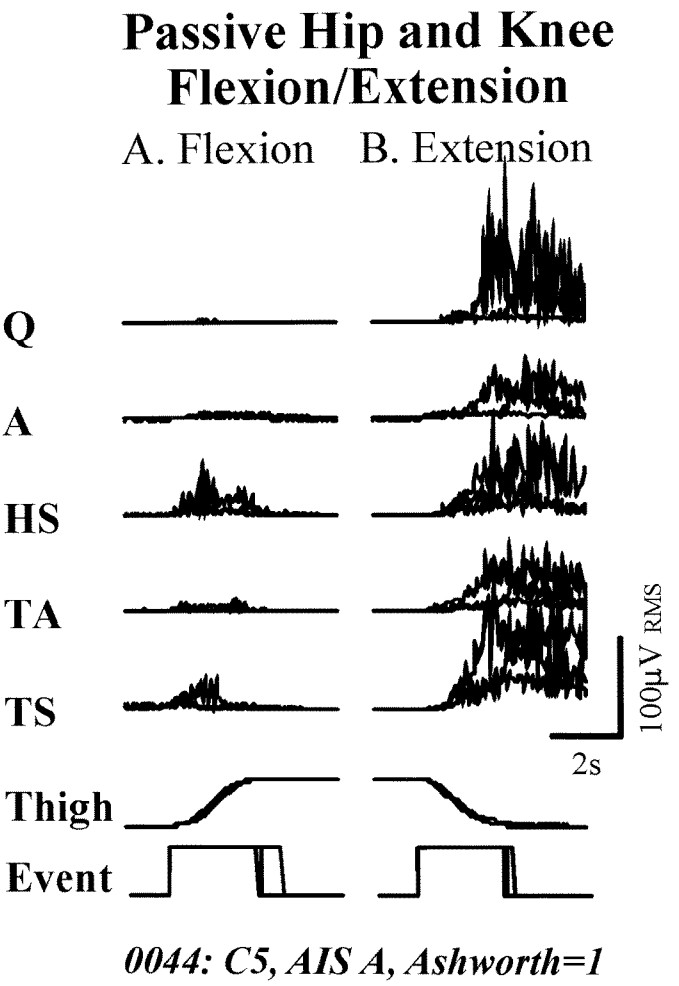

Figure 2.
sEMG Waveforms during Repeated Passive Hip/Knee Flexion and Extension.
a) This AIS A subject was scored as 1/4 for right hip and knee movement. Hip flexion elicited a relatively small response, primarily in the RHS.
b) However, hip extension generated widespread activity in all recorded ipsilateral muscles, with some spread contralaterally and even to trunk muscles. Note that there was some counter-balancing activation of flexors and extensors during extension, which may have contributed to the relatively low perceived Ashworth score.

subjects. As this was frequently the case, inclusion of response vector elements from the contralateral side muscles would have diminished any discriminative power of the response vector. Thus, in this paper, we deal only with (right side) muscles unilateral to the passive movement.

Quantitative Analysis

Analyzing the combined average limb response (cALR) for flexion and extension together for all 97 subjects revealed that the cALR is significantly different between

Table 2.
Response vectors from responses pictured in **Figures 1** and **2**.

Fig. Man.	Right hip/knee 1a Flex	1b Ext	2a Flex	2b Ext
Q	0.4	2.9	0.2	16.2
A	1.7	0.7	1.8	11.5
H	11.3	1.1	4.0	16.8
TA	2.9	0.6	1.0	13.8
TS	0.1	2.3	0.3	30.1
ALR	3.3	1.5	1.5	17.7

Fig.=Figure; Man.=maneuver; Ext=extend; Q=quadriceps electrode pair; A=adductor electrodes; H-hamstrings electrodes; TA=tibialis anterior electrodes; TS=triceps surea pair; ALR=average limb response.

Ashworth categories (F=19.59, df 3 and 178, p< 0.001). However there was no significant difference for the ALR between the flexion and extension phases of the maneuver. Nor was there a significant interaction between Ashworth Category and phase of maneuver. Multiple comparisons revealed that the ALR was sufficient to distinguish between Ashworth category three and any other category. It was not sufficient to distinguish between groups 0, 1, or 2 at all. **Figure 3** shows the box plots for ALR. The substantial overlap shown between the groups could be the reason for this lack of discriminant power.

For the second analysis, multivariate tests show that overall, there are significant differences in the five RV elements between Ashworth categories (F=5.01, df 15 and 480, p<0.001) and between flexion and extension (F=7.56, df 5 and 174, p<0.001). In addition, there is a significant interaction effect for Ashworth by movement (F=2.11, df 15 and 480, p=0.008).

Univariate tests indicate that all five RV elements contribute to the observed significant differences between Ashworth categories (p<0.001), with F values of 8.44, 8.72, 16.95, 9.22, and 12.91 for Q, A, H, TA, and TS, respectively, df 3 and 178. Only the Q (F=247.14, df 1 and 178, p<0.001) and TA (F=871.00, df 1 and 178, p<0.001) elements were significantly different between the flexion and extension phases of the maneuver. The significant interaction effect was influenced by the TA element alone (F=314.34, df 3 and 178, p<0.001). The importance of this interaction is lessened by virtue of the fact that the TA does not contribute to the resistance perceived by the clinician when examining hip and knee flexion and extension.

The significant main effect for the Ashworth categories is depicted in **Figure 4.** This figure shows the general increase in RV elements (in this case, averaged across flexion and extension phases) corresponding to the Ashworth category. Multiple comparisons were computed to determine which elements distinguished between which Ashworth categories. None of the five elements was able to distinguish between Ashworth categories 0 and 1. The H and Q elements distinguished between groups 0 and 2. All five elements were significantly different between both categories 0 and 3 and categories 1 and 3. Only Q was significantly different between categories 1 and 2. Finally, all elements except Q were significantly different between categories 2 and 3. **Table 3** summarizes these differences.

DISCUSSION

sEMG-Based Measures

By recording sEMG data it is possible to directly measure the behavior of the neuromuscular system in a variety of conditions. We propose that these measures are relevant to the subject's underlying pathophysiological, spastic condition. Proper interpretation of the quantitative

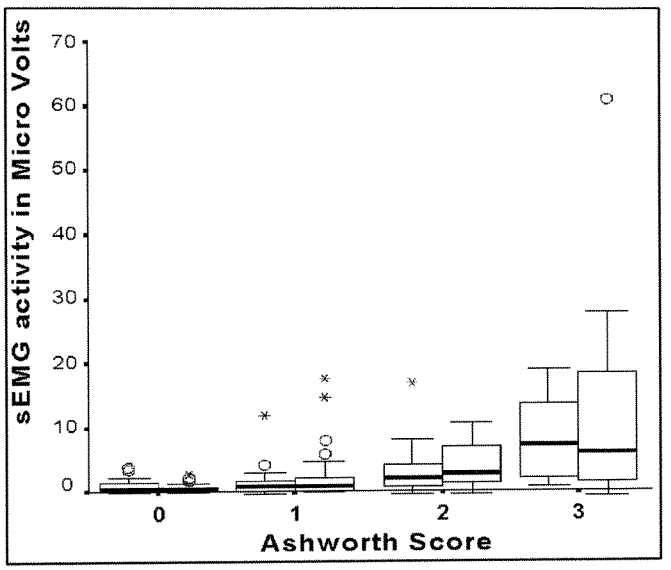

Figure 3.
Average sEMG Activity
Box plots representing the recorded sEMG activity averaged over all five muscles by phase of movement for Ashworth categories. The box represents the interquartile range which contains the 50% of values. The whiskers are lines that extend from the box to the highest and lowest values, excluding outliers. A line across the box indicates the median. For each score, the left box represents the flexion movement, and the right, extension.

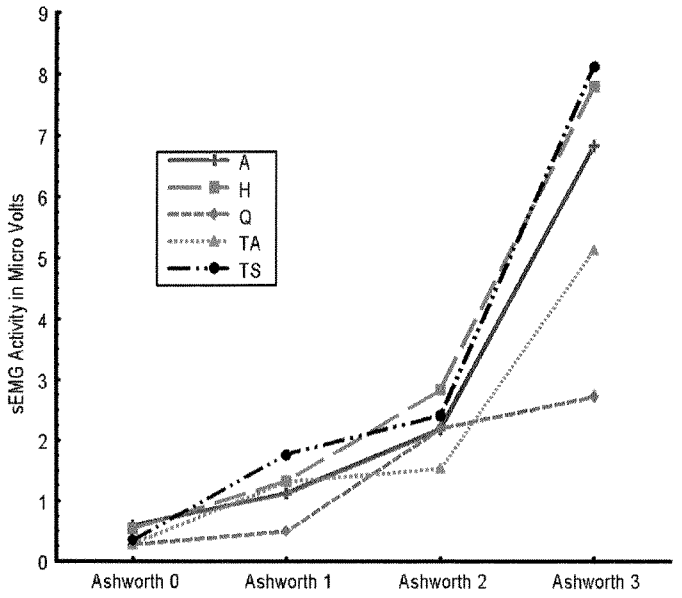

Figure 4.
Individual Muscle sEMG Activity Recorded sEMG activity for the individual muscles averaged across the phases of movement by Ashworth category illustrating the main effect for Ashworth groups.

sEMG data requires knowledge of the recording situation and most importantly, full appreciation of the event or maneuver leading up to the recorded activity. Employment of fully documented "controlled inputs" makes it possible to interpret the results of the recording (42).

The premise upon which this paper is based is that altered motor control following SCI or spastic paresis (8) is most often and most directly evident as increased excitability of spinal motoneuron pools (43–45). This increased excitability is manifested in a variety of features, including exaggerated phasic and tonic reflexes, spasms and altered voluntary control (46). Wiesendanger stated that spasticity "is characterized by altered activity patterns of motor units occurring in response to sensory and central command signals which lead to co-contractions, mass movements and abnormal postural control" (47). McLellan suggested "excessive and inappropriate muscular activation occurring in association with the upper motor neuron syndrome" as a functional definition of spasticity (48).Regardless of the mechanism (loss of inhibition, changed properties, etc.), motoneuron hyperexcitability is undoubtedly centrally involved in all these manifestations of spasticity.

Table 3.
Muscles that demonstrate significant differences between Ashworth categories in multiple comparisons.

	Ashworth Group		
Ashworth Group	0	1	2
1		—	—
2	H,Q	Q	—
3	H,Q,A,TS,TA	H,Q,A,TS,TA	H,A,TS,TA

H=hamstrings electrodes; Q=quadriceps electrode pair; A=adductor electrodes; TS=triceps surea pair; TA=tibialis anterior electrodes.

The use of sEMG makes it possible to examine the behavior of a large set of muscles in each subject. We chose to present the temporal patterns of activity in each muscle resulting from these maneuvers in terms of the RMS envelope of activity as the most direct and meaningful expression of the motor control itself (36). We selected the average sEMG voltage expressed in μV_{RMS} during a defined time window as the measure of maneuver response in each muscle. By combining this activity from multiple muscles, we create a response vector for each phase of each maneuver. This response vector represents an expansion beyond what would be perceived by a clinical examiner, who is only asked to evaluate the resistance to muscle stretching.

We have previously described the methods employed in documenting these sEMG features (33) and showed that these methods generate consistent and reliable data using the average limb response (ALR) when applied in a population of stable SCI subjects (36). The consistency of the data suggests that the variability in the examination induced by the recording technologist is well within acceptable limits (**Figures 1** and **2** show three superimposed trials of manually executed passive movements). Consistency of the speed of passive movement was evident in the movement duration and pattern of motion sensor output. Furthermore, prior applications have demonstrated the sensitivity of sEMG measures to induced changes (49,50).

Using an informatics approach, we were able to show good agreement between clinical scales and the overall average sEMG activity (37). In another study we found that, out of all the clinical and instrumental approaches taken, sEMG measures yielded the best agreement with the subjects' self perceptions of spasticity (51). The main purpose of the present work is to further validate the use of sEMG activity as a measure of altered upper motor neuron dysfunction commonly termed spasticity.

Confounding Errors

There are a number of potential sources of systematic errors for both clinical and electrophysiological measures. Errors in clinical scales have been discussed elsewhere (52,53), and will not be further discussed here. Systematic errors in the BMCA measurements include 1) improper implementation of the passive maneuver by the BMCA technologist examiner, 2) improper recording or 3) improper analysis procedures. The high degree of reproducibility of the mechanograms shown in **Figure 1** along with the previously reported consistency of the data (36) suggest that well-trained examiners are able to maintain a standard protocol, thereby arguing against improper implementation of the maneuvers. However, in those subjects with very strong spasticity (Ashworth 4), the examiner may not be able to overcome the resistance, thus under-estimating the magnitude of the condition in the recorded sEMG signal. The method of analysis chosen, namely, the calculation of the average activity of each muscle during each phase of each maneuver, is adequate to describe the excess response to the maneuvers in this paper in subjects with spastic paresis, although the baseline-corrected response may or may not yield appropriate values for other forms of motor disorder such as dystonia.

The most important source of systematic error in recording is that arising from the arbitrary scaling imposed by the biophysics of the sEMG recording itself. This uncontrolled scale factor requires further efforts to fully resolve, such as transformation of the sEMG data into the motor neuron activity domain, expressed as the pooled firing rate (54). The present work assumes an identity matrix transformation, which accounts for the measurement units only, that is, that the sEMG data is essentially equivalent to the pooled firing rate (which, of course, cannot be precisely correct). An absolutely correct transformation would require the ability to directly measure the activity of the spinal motor pool. In the absence of this ability, one can estimate the number of motor units in the muscle, and use that information along with specific anatomic features of a particular muscle (skin thickness, muscle size, placement relative to the electrodes, etc.) to compute an approximate transformation. The general agreement between the electrophysiological and clinical measures suggests that this scaling problem may be a second-order, rather than a primary, problem. Furthermore, such scale transformations do not affect the utility of sEMG data in following changes in serial studies in single subjects, in which such factors can be shown to cancel out (55).

Comparison of Clinical and sEMG Measures

In spastic subjects, passive movement may generate activity in the stretched muscle, as seen in **Figure 1a**. Such activity, when it occurred only in the muscle stretched, could also be perceived by the clinician examining the subject. However, in other subjects, the recorded activity was less directly linked to the movement, in that it occurred prominently in muscles other than those stretched, and its time of occurrence did not necessarily correspond with the movement, but rather was triggered by the movement (**Figure 2b**). The method of registering the response to passive movement intentionally included both such instances as equally relevant to understanding of the subject's condition. Inevitably, this leads to a lack of agreement in specific instances between the perceived response as judged clinically, and the sEMG values, as evident in **Figure 2**.

When the individual muscles are not averaged, but are considered as a response vector, the discriminant power is enhanced over that of the average limb response. Thus, even when considering the response to passive limb movement, the spastic subjects respond in distinctive patterns much more complicated than might be supposed from the single clinical score.

Variability

Another reason for any differences between two methods may be that the subject changed in response between the observations of the clinician and the electrophysiological recordings. A high degree of variability has been attributed to quantitative measures of spasticity (56). This variability has many sources, including higher brain function and emotions (57). Wiesendanger (47) pointed out the necessity to understand tone as a part of postural control, and that it must have prospective as well as reactive components. Ultimately, such control is the expression of the brain (58). Spasticity and related UMN dysfunction should be recognized as a motor behavior. Thus, to control the variability, both behavior of the subject and external stimuli (acoustic, etc.) must be controlled, as well as such obvious factors as bladder filling or infections, decubitus ulcers, and so forth (48). In other words, much of the reported "variability" is in fact a manifestation of residual brain control, rather than "random noise," and thus can be controlled by taking that residual control into consideration. In a study in stroke subjects, Katz and colleagues reported much less variability than was commonly expected when using an instrumental approach

to assessment of spasticity (13). In our case, the clinical and electrophysiological portions of the study were done in the same session, within 15 min of each other, further limiting the variability. We also make great efforts to control the testing environment, to separate the subject from any extraneous stimulation.

There is a general perception, perhaps because of the variability in motor control observed when studies are not well-controlled, that sEMG data is highly variable. However, we previously demonstrated that the methods used in this study are quite reproducible, with test-retest correspondence as high as 0.97 with averaging (54). In other words, systematic placement of electrodes at defined landmarks, full compliance with a comprehensive protocol, appropriate averaging of data and careful attention to the environment, immediate history, medications, etc., will yield a consistent motor response and hence reproducible results. The key here is that the sEMG data reflect the expressed motor control, and by controlling the sources of variability in motor control, one can gain a representative and reproducible impression of that control through the sEMG data.

Increased Mechanical Resistance (and Low sEMG Values)

It is instructive to examine cases in which the two methods yield contradictory results. First, consider the case where the Ashworth score is two or greater and yet the ALR or response vector has a very low value. If assessment of resistance to passive stretch and recording of sEMG occurred simultaneously, and sEMG activity was absent but there was increased resistance to the passive movement, it is likely that the increased resistance was due to viscoelastic properties of the muscle, connective tissue, or joint (59). Individuals with little or no sEMG response to passive stretch do not exhibit "spasticity" as defined by Lance (14). In other words, the absence of sEMG response to passive stretch demonstrates that there is no neuromuscular response to that maneuver, thereby objectively documenting that aspect of the individual's condition.

Increased sEMG (but Low Ashworth Scores)

In general, the observed sEMG activity may be expected to correlate with externally perceivable forces. However, the net force expressed at any joint is a result of combined forces produced by all of the muscles active at that joint. If the averaged sEMG activity is on the order of 2 μV or more, and the corresponding Ashworth score is 0 or 1, passive stretch may have induced approximately equal amounts of sEMG activity in antagonistic muscles (e.g., **Figure 2b**). A balance of activity in flexor and extensor muscles can result in complete or partial obliteration of any external, net torque even in the presence of substantial activity in opposing muscles, just as two weights on opposite sides of a frictionless pulley can result in no net torque (60,61). In such an instance, net torque badly underestimates the total neuromuscular activity induced by the maneuver, regardless of the means of assessing that torque.

What Is the "Right" Answer?

We propose that the "right" answer is the one that is most useful in the management of spastic paresis, for example, in decision making regarding pharmacological interventions. Considering the situation above, in which clinical and sEMG measures did not agree, selection of such interventions intended to modify neuromuscular functions is more appropriately based on the sEMG measurements rather than on perception or measurement of increased torque. Based upon the reasoning outlined above, the sEMG result more closely corresponds to the inappropriate neuromuscular function than does the torque-based result. Assessments that evaluate the effectiveness of a particular intervention based only on net torque may occasionally yield confounding results compared with assessments that are based on the total neuromuscular activity evoked by this particular maneuver. This would be true if an intervention disproportionately reduced flexor or extensor activity, for example. If a torque measure were used to titrate the dosage of medication in such a case, the measure might appear to at first grow worse then better as the dosage successively reduced activity in one, then the other muscle.

CONCLUSIONS

The lack of an acceptable "gold standard" for assessment of spasticity prevents establishment of criterion-related validity for sEMG measures. The ability to objectively record the sEMG data and reduce to numbers does not equal the ability to assess spasticity in the subjects in whom the measures were taken (62). Nevertheless, the presented data correlate to a limited degree with accepted clinical scales, and thereby achieve at least some degree of criterion-related validity. To achieve content validity, measures of spasm activity and interference with voluntary control must also be incorporated into a scoring system along with stretch reflexes. Such measures are a part of the BMCA examination as currently constituted, and their analysis will be dealt with in a future paper.

In summary, the multi-channel, sEMG approach described in this paper provides an important method of assessing motor behavior under a variety of circumstances. Data obtained are quantitative, reproducible, and clinically relevant. This technique may be directly applied to repeated observations on the same subject to describe changes in motor control with treatment (e.g., as the uncertainty of the inverse problem may be assumed a constant). Indeed, individual data sets provide much more information about the subjects than can be presented in limited space. Remaining problems with this technique include the identification of appropriate coefficients that will permit transformation of the sEMG data into an absolute, pooled firing rate data space, taking into consideration biophysical differences among subjects. The fact that the data, as currently processed, seem to yield valid results suggests that neglecting this theoretically important transformation is not a major source of error. Nevertheless, continuing efforts should be made to improve the absolute accuracy of these data through such transformations, and continued studies are needed to identify the limits of accuracy of the data, and to demonstrate the relevance of the derived scores.

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