

Marquette University

e-Publications@Marquette

Biomedical Engineering Faculty Research and Publications

Biomedical Engineering, Department of

1-2005

A Physiologically Based Clinical Measure for Spastic Reflexes in Spinal Cord Injury

Ela N. Benz

Rehabilitation Institute of Chicago

T. George Hornby

Rehabilitation Institute of Chicago

Rita K. Bode

Rehabilitation Institute of Chicago

Robert A. Scheidt

Marquette University, robert.scheidt@marquette.edu

Brian D. Schmit

Marquette University, brian.schmit@marquette.edu

Follow this and additional works at: https://epublications.marquette.edu/bioengin_fac



Part of the [Biomedical Engineering and Bioengineering Commons](#)

Recommended Citation

Benz, Ela N.; Hornby, T. George; Bode, Rita K.; Scheidt, Robert A.; and Schmit, Brian D., "A Physiologically Based Clinical Measure for Spastic Reflexes in Spinal Cord Injury" (2005). *Biomedical Engineering Faculty Research and Publications*. 162.

https://epublications.marquette.edu/bioengin_fac/162

Marquette University

e-Publications@Marquette

Biomedical Engineering Faculty Research and Publications/College of Engineering

This paper is NOT THE PUBLISHED VERSION; but the author's final, peer-reviewed manuscript. The published version may be accessed by following the link in the citation below.

Archives of Physical Medicine and Rehabilitation, Vol. 86, No. 1 (January 2005): 52-59. [DOI](#). This article is © Elsevier and permission has been granted for this version to appear in [e-Publications@Marquette](#). Elsevier does not grant permission for this article to be further copied/distributed or hosted elsewhere without the express permission from Elsevier.

A Physiologically Based Clinical Measure for Spastic Reflexes in Spinal Cord Injury

Presented in part to the American Spinal Injury Association, May 19, 2001, Long Beach, CA, and the American Physical Therapy Association's Combined Sections, February 15, 2003, Tampa, FL.

Ela N. Benz

Sensory Motor Performance Program, Rehabilitation Institute of Chicago, Chicago, IL

T. George Hornby

Sensory Motor Performance Program, Rehabilitation Institute of Chicago, Chicago, IL

Department of Physical Medicine and Rehabilitation, Northwestern University, Chicago, IL

Rita K. Bode

Center for Rehabilitation Outcomes Research, Rehabilitation Institute of Chicago, Chicago, IL

Robert A. Scheidt

Center for Rehabilitation Outcomes Research, Rehabilitation Institute of Chicago, Chicago, IL

Department of Physical Medicine and Rehabilitation, Northwestern University, Chicago, IL

Department of Biomedical Engineering, Marquette University, Milwaukee, WI

Brian D. Schmit

Sensory Motor Performance Program, Rehabilitation Institute of Chicago, Chicago, IL

Department of Physical Medicine and Rehabilitation, Northwestern University, Chicago, IL
Department of Biomedical Engineering, Marquette University, Milwaukee, WI

Abstract

Benz EN, Hornby TG, Bode RK, Scheidt RA, Schmit BD. A physiologically based clinical measure for spastic reflexes in [spinal cord injury](#). Arch Phys Med Rehabil 2005;86:52-9.

Objective

To test the validity of the Spinal Cord Assessment Tool for Spastic reflexes (SCATS), a clinical tool intended to rate spastic [motor behavior](#) after spinal cord injury (SCI).

Design

By using correlational analyses, the SCATS was validated using concurrent measurements of kinematics and electromyograms and traditional assessments of [spasms](#) and spastic [hypertonia](#).

Setting

Research laboratory (kinematics and electromyography) and outpatient medical clinic (traditional measures of spastic hypertonia).

Participants

Eleven people with SCI were used for kinematic and electromyographic measurements. Seventeen people with SCI were used for comparison with other clinical scales.

Interventions

Not applicable.

Main outcome measures

Kinematic and surface electromyographic measurements of the tested lower extremity were used to quantify magnitude and/or duration of motor behaviors, and the Penn Spasm Frequency Scale (PSFS) and the Ashworth Scale were used to measure spasm frequency and resistance to joint movement for the hip flexors, knee flexors, and ankle plantarflexors, respectively. Concurrently, the SCATS was used to assess the [clonus](#) response to an imposed ankle dorsiflexion, the flexion response to a stimulus to the foot, and the knee extensor activity in response to an imposed leg extension. Each component of the SCATS was compared with the Ashworth Scale, the PSFS, and kinematic and electromyographic measurements by using the Spearman rank correlation test.

Results

Clonus, [flexor spasm](#), and extensor spasm responses measured by using the SCATS correlated significantly with kinematic and electromyographic recordings ($P<.01$). Significant correlations were also observed between the SCATS extensor spasms and the Ashworth scores for hip and knee flexors and for ankle plantarflexors ($\rho=.98$, $.88$, $.61$, respectively). Also, SCATS flexor spasms and SCATS clonus scores correlated significantly with some of the Ashworth scores. Only SCATS clonus scores correlated significantly with spasm frequency measures ($\rho=.59$, $P<.05$).

Conclusions

The SCATS produced a valid measure of 3 distinct types of spastic motor behaviors in SCI and may provide a complementary tool for measuring spastic hypertonia. Such a measure is valuable because current assessment tools do not differentiate between the different types of spastic motor behaviors that manifest after SCI.

Distinguishing the 3 spastic reactions using an efficient and valid clinical tool may help guide management of spastic hypertonia in SCI.

Keywords

Muscle hypertonia, Muscle spasticity, Rehabilitation, Spinal cord injuries

People With [Spinal Cord Injury](#) (SCI) often experience exaggerated reflex responses affecting muscles that are deficient in voluntary control. These reflexes arise from both proprioceptive and exteroceptive inputs, are described as the positive signs of the upper [motoneuron](#) syndrome, and manifest as [spasticity](#) and dystonia.[1, 2, 3](#) The functional implications of these spastic [motor behaviors](#) are underscored by patient and clinician reports that spasticity interferes with mobility and activities of daily living (ADLs).[3](#)

Attempts to quantify spastic [hypertonia](#) in SCI have had mixed results. Assessment tools used most often by clinicians to measure spastic hypertonia are the Ashworth Scale[4](#) and the Modified Ashworth Scale (MAS), which measure spastic behaviors based on the following traditional definition: “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.”[5](#)(p485) This definition focuses on resistance to movement at a single joint, which is primarily sensitive to stretch reflex hyperexcitability. The MAS is effective for globally assessing spastic hypertonia in patients with tetraplegia[6, 7, 8](#); however, limitations to the Ashworth scales have been expressed.[9, 10, 11](#)

In addition to [stretch reflex](#) excitability, spastic [motor behavior](#) in SCI also includes hyperexcitable interneuronal reflexes involving multiple joints (ie, spasms).[12, 13, 14, 15, 16](#) To address involuntary motor behaviors that incorporate multijoint [spasms](#), a scale devised by Penn et al was developed by using patient reports of spasm frequency.[17](#) Other investigators[18](#) have addressed the multidimensional nature of spinal spastic [hypertonia](#) by combining clinical and self-report measures. Despite recognition that the manifestation of spastic motor behaviors in SCI is more complex than measures of spasm frequency and resistance to single joint movement, a standardized, simple clinical measure that encompasses the primary spastic reactions has yet to evolve.

The multidimensional nature of reflex hyperexcitability in SCI is often shown by 3 distinct types of [spasms](#) that are reported by patients and clinicians: [clonus](#) (particularly at the ankle), [flexor spasms](#), and extensor spasms.[3](#) Clonus is an involuntary rhythmic muscle contraction occurring at a frequency of 3 to 8Hz, and it is typically elicited by rapid [passive movement](#) of the ankle into dorsiflexion.[19](#) Flexor spasms are described as multijoint flexion movements of the leg after various cutaneous stimuli,[13](#) and extensor spasms are multijoint leg extension movements often evident after changes in hip position.[15](#) Because each of these types of spastic responses involves different reflex pathways, and therefore different pathophysiologic mechanisms, a clinical tool that is easily administered and that is sensitive to the type of spastic [hypertonia](#) seen in SCI would be of considerable value to clinicians.

To address the need for a clinical scale to measure [spasms](#) and spastic [hypertonia](#) in SCI, the Spinal Cord Assessment Tool for Spastic reflexes (SCATS) was developed. The goal of this study was 2-fold. The first goal was to validate the SCATS by using kinematic and electromyographic measurements to quantify magnitude and/or duration of [motor behaviors](#) and to establish their correlation with clinician-determined spasm ratings. Second, we aimed to show that the SCATS would differentiate among the 3 types of spastic reflexes, and we wished to compare the SCATS to other clinical measures of spastic hypertonia that are commonly used in the clinical setting, including the Ashworth Scale and the Penn Spasm Frequency Scale (PSFS).[17](#)

Methods

Participants

All subjects were recruited from the inpatient and outpatient clinics of the Rehabilitation Institute of Chicago. Inclusion criteria for all components of our study included history of SCI, age 16 to 65 years, and patient or clinician report of spastic [motor behaviors](#). Exclusion criteria included acute orthopedic injuries, heterotopic ossifications, [decubiti](#), and acute urinary tract infections. Informed consent was obtained for each subject, and all procedures were conducted in accordance with the Helsinki Declaration of 1975 and approved by the institutional review boards of Northwestern University and Marquette University. All subject demographics are summarized in [table 1](#).

Table 1. Subject Descriptions for Comparison of the SCATS With Kinematic Electromyographic Measures and for Comparison of the SCATS With the Ashworth Scale and PSFS

Subject No.	ASIA Class	Injury Level	Age (y)	Spasticity Medication	Months Postinjury	SCATS Clonus	SCATS Flexion	SCATS Extension
Kinematic and electromyographic measures								
1	A	T3	45	None	48	2	1	1
2	D	T4	40	None	46	2	1	0
3	A	C7	35	40mg baclofen tid	30	0	2	1
4	A	T4	35	None	30	3	2	2
5	A	C5	17	None	3	0	0	0
6	D	T9	45	20mg baclofen tid	360	0	1	0
7	A	C5	22	10mg baclofen tid	40	0	2	2
8	A	C6	47	None	24	1	1	1
9	A	C6	30	10mg baclofen qid	18	0	1	1
10	A	C5	44	None	24	0	3	0
11	A	T4	40	None	360	0	1	1
Ashworth Scale and PSFS								
12	A	C5	28	20mg baclofen qid	48	0	1	0
13	A	C6	52	10mg diazepam tid	264	0	3	0
14	D	C5	32	10mg baclofen tid	180	0	2	2
15	C	C6	39	40mg baclofen qid	192	1	0	2
16	A	T10	48	None	372	3	3	3
17	B	C6	40	40mg baclofen qid	204	0	0	0
18	A	T3	51	40mg baclofen qid	24	0	3	0
19	A	C7	30	40mg baclofen qid	36	1	0	0
20	A	C45	63	40mg baclofen tid	360	0	1	0
21	C	C7	35	None	30	3	1	3
22	A	T4	62	None	180	0	0	0
23	A	T7	39	10mg baclofen tid	270	2	3	0
24	A	C5	22	None	40	0	2	2
25	A	T3	45	None	48	2	1	1
26	A	C6	45	None	120	1	2	1
27	A	C7	47	None	130	2	1	1

Abbreviations: ASIA, American Spinal Injury Association; qid, 4 times daily; tid, 3 times daily.

For kinematic and electromyographic analysis, 11 subjects were recruited via flyers posted in the testing facility, and a telephone interview was conducted to ensure compliance with all inclusion and exclusion criteria. For comparison with the Ashworth Scale and the PSFS, 17 subjects were recruited via referral by their primary care physician during outpatient medical visits.

Instruments

The SCATS

The development of the SCATS was based on previous clinical measures of spastic [hypertonia](#) that use a physical examination to assess involuntary motor behaviors, as well as recent laboratory measurements of [clonus](#), flexor, and extensor [spasms](#).^{15, 16, 19} Specific techniques for measurement of these spasms follow.

SCATS: clonus

Clonus of the plantarflexors was quantified in response to a rapid passive dorsiflexion of the ankle ([fig 1A](#)). The ankle was dorsiflexed at an angle that triggered clonus, and the duration of clonic bursts was timed. An ordinal rating from 0 to 3 was determined by the duration of clonic activity where 0 is no reaction; 1 is mild, clonus was maintained less than 3 seconds; 2 is moderate, clonus persisted between 3 and 10 seconds; and 3 is severe, clonus persisted for more than 10 seconds.

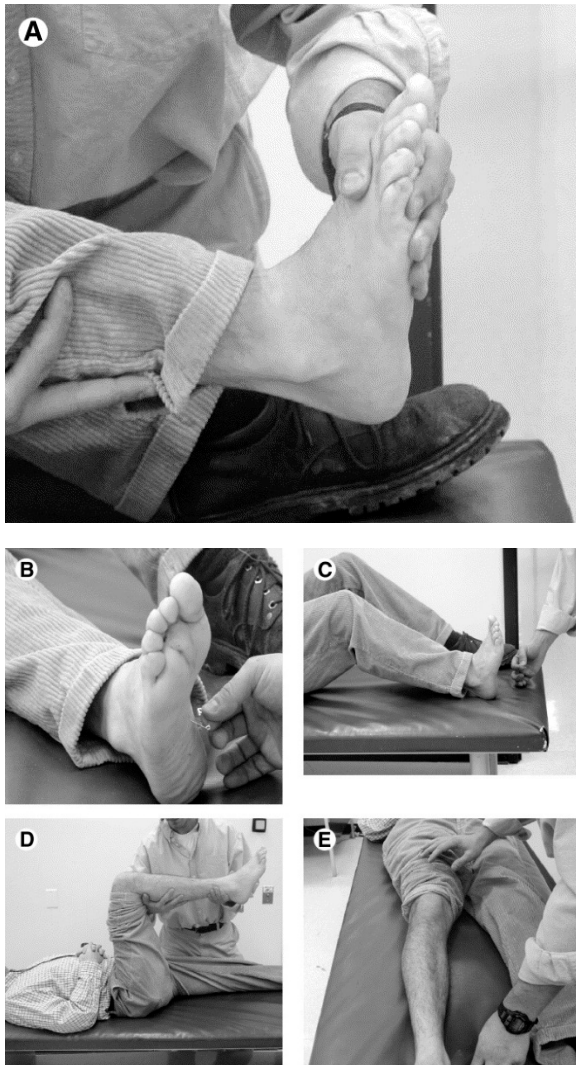


Fig 1. Procedures for the SCATS measurement. (A) [Clonus](#) was assessed by using an imposed dorsiflexion movement of the ankle, with 1 hand placed under the calf and the other placed on the foot.

(Continued) (B, C) The flexor response to a pinprick perturbation to the bottom of the foot was assessed visually. (D, E) The response to simultaneous hip and knee extension was observed to document extensor [spasms](#). The starting position of the movement is shown at the left, and the terminal position is shown on the right.

SCATS: flexor spasms

With the knee and hip extended to 0°, the clinician applied a pinprick stimulus for 1 second to the medial arch of the subject's foot ([fig 1B](#)). Excursion of the big toe into extension, ankle dorsiflexion, and knee and hip flexion were visually observed for severity. The rating scale consisted of a score from 0 to 3, where 0 is no reaction to stimulus; 1 is mild, less than 10° of excursion in flexion at the knee and hip or extension of the great toe; 2 is moderate, 10° to 30° of flexion at the knee and hip; and 3 is severe, 30° or greater of knee and hip flexion.

SCATS: extensor spasms

With the contralateral limb extended, the tested knee and hip were positioned at angle of 90° to 110° of hip and knee flexion, and then both joints were simultaneously extended. One hand cupped the heel while the other was placed on the outside of the thigh ([fig 1C](#)). Once a reaction was elicited, the duration of visible muscle contraction in the quadriceps muscle was measured by observing superior displacement of the [patella](#). The timed scale (0–3) that was used for clonus was also applied to the timed extensor spasms.

Kinematic measurements

An Optotrak 3020 motion analysis system^a was used to measure the kinematics of each perturbation by using 12 markers placed on the following [anatomic landmarks](#): second distal [interphalangeal joint](#), first metatarsal head, [lateral malleolus](#), tibial shank, patella, lateral [femoral condyle](#), lateral quadriceps, [greater trochanter](#), anterior superior iliac spine, bisection of the pelvis at [iliac crest](#), xiphoid process, and a hand width under the [axilla](#). Marker location data were digitally sampled at 1000Hz for 20 seconds by a personal computer (PC), for each trial.

Electromyograms

Electromyograms (EMGs) were recorded from the tibialis anterior, soleus, medial gastrocnemius, [vastus](#) medialis, rectus femoris, medial hamstrings, adductors, and contralateral rectus femoris. Active surface electrodes^b were applied to lightly abraded skin over the respective muscles. Electromyographic signals were amplified (10,000×) and sampled at 1000Hz by using the same PC used to acquire the kinematic data.

Ashworth Scale

The Ashworth Scale⁴ was used to quantify tone of the ankle plantarflexors, knee flexors, and hip flexors. This scale uses ratings from 0 to 4 to assess the resistance to a movement applied throughout the entire range of motion. The scores correspond to 0 being no increase in tone; 1 being a slight increase in tone, giving a catch when the limb is moved into flexion or extension; 2 being a more marked increase in tone but limb easily flexed; 3 being a considerable increase in tone, with [passive movement](#) difficult; and 4 being a limb rigid in flexion or extension.

Penn spasm frequency scale

The PSFS¹⁷ was also used to quantify [spasticity](#) in 17 participants. This scale is based on self-reporting by using a scale from 0 to 4, with the following rankings: 0, no spasm; 1, mild spasms induced by stimulation; 2, infrequent full spasms occurring less than once per hour; 3, spasms occurring more than once per hour; and 4, spasms occurring more than 10 times per hour.

Procedures

Two experimental paradigms were used in this study: (1) to assess the validity of the SCATS measurements based on concurrent kinematic and electromyographic measurements and (2) to estimate the relation of this new [clinical assessment tool](#) to those often used in a clinical setting.

Kinematic and electromyographic measurements

In the first set of experiments, 11 subjects with SCI of 3 months to 10 years in duration were assessed to validate the SCATS measurements. Repeated trials of each of the SCATS tests (clonus, [flexor spasms](#), extensor spasms) were performed by the same tester on each subject's right lower extremity ([fig 1](#)). Perturbations intended to elicit clonus, flexor spasms, and extensor spasms were applied sequentially, and each battery of tests was repeated 10 times, with simultaneous measures of SCATS scores, kinematic data, and electromyographic responses.

Comparison with other clinical scales

In the second set of experiments, a comparison of the SCATS with other clinical scales was conducted in 17 subjects with SCI during routine outpatient medical visits. A single [physical therapist](#) administered 3 measures of spastic motor behaviors, including the PSFS, Ashworth Scale, and SCATS. The Ashworth Scale was measured in the right lower extremity for knee and hip extension and ankle dorsiflexion. Subjects were between 6 months and 10 years postinjury ([table 1](#)), with mixed injury levels (American Spinal Injury Association scale) and were taking various medications to control their upper [motoneuron](#) symptoms. Each test was performed once.

Data analysis

Kinematics

Three-dimensional, lower-extremity kinematics obtained via infrared marker data were used to calculate the approximate angles between adjacent limb segments during SCATS testing. This analysis was conducted by using an assumption of rigid body motion. A vector representing the orientation of each body segment relative to the laboratory coordinate frame was defined using markers fixed to the proximal and distal anatomic landmarks of each body segment: torso, pelvis, thigh, shank, and foot. The angle between adjacent limb segments (ie, the joint angles) was estimated by using the vector dot product:

$$\theta_{\text{joint}} = \bar{\mathbf{q}}_1 \cdot \bar{\mathbf{q}}_2 / |\bar{\mathbf{q}}_1| |\bar{\mathbf{q}}_2|$$

Concurrent validity was established by using a correlation analysis of kinematic and electromyographic signals with SCATS measurements. The time trajectory of each joint angle was displayed for analysis by a trained clinician; changes in joint angle were quantified by using an interactive, cursor-based analysis tool developed by using custom-designed Matlab software.⁵ To assess flexor spasms after pinprick stimulation of the medial arch, joint angle changes in the hip, knee, and ankle were calculated by taking the difference between these angles measured before and after stimulation. For quantification of ankle clonus after rapid, passive ankle dorsiflexion, the duration and frequency of clonic bursts were determined by using the same graphics tool. The number of beats per second and the duration of the response were calculated as a measure of clonus.

Electromyograms

The durations of knee (vastus medialis) and ankle (soleus, medial gastrocnemius) extensor EMGs were used to provide objective verification of clinicians' assessments of extensor spasm duration and clonus duration during SCATS testing. EMGs were band-pass filtered (cutoff frequencies, 20Hz, 2000Hz), amplified (gain, 10,000), and low-pass filtered (cutoff frequency, 500Hz), before being sampled at 1000 samples per second. The same cursor-based tool used to analyze joint kinematics was applied in the analysis of EMG durations. The time trajectory of

each electromyographic signal was displayed for every trial. Visual estimation of the onset and terminus of soleus and medial gastrocnemius EMGs was used. Spasm duration was calculated directly from these measures.

Correlation of SCATS and kinematic and electromyographic parameters

A correlational analysis was conducted between the clinician-measured SCATS score and magnitude or duration of the associated kinematic or electromyographic parameter. The extensor SCATS score was also correlated with vastus medialis electromyographic activity; the flexor SCATS with excursion of the ankle, knee, and hip into flexion and the sum of the joint angles; and the clonus SCATS with the duration of the EMG of the medial head of the gastrocnemius. The 10 trials of data for each subject were pooled across the 11 subjects, and a Spearman rank correlation test was used to obtain correlations between the SCATS and the targeted kinematic or electromyographic parameter ($\alpha=.05$). The SCATS score was then calculated in each category based on the kinematic and electromyographic data, and the correlational analysis was repeated (Spearman rank correlation test at $\alpha=.05$).

Repeatability of test perturbations

The mean and standard deviation (SD) of the input perturbations were calculated for each test as follows: for clonus, we measured the input ankle angular perturbation magnitude and the rate of change of this perturbation; for the extensor spasm stimulus, we measured the extension speed at the hip and knee, as well as the position of the hip and the knee where spasm was elicited.

Comparison with Ashworth Scale and PSFS

To determine correlations between the SCATS, Ashworth Scale, and PSFS scores, a Spearman rank correlation analysis was conducted ($\alpha=.05$). Specifically, all flexor, extensor, and clonus ratings were compared with Ashworth scores determined during ankle dorsiflexion and hip and knee extension. Further comparisons were made between individual SCATS ratings and the subject-reported PSFS rating.

Results

Validation using kinematic and electromyographic analysis

An example of typical input and output responses for each spastic response is given in [figure 2](#). [Figure 2A](#) shows the rapid, passive ankle dorsiflexion, followed by rhythmic medial gastrocnemius-soleus electromyographic bursting and ankle plantarflexion movements typical of [clonus](#). Rapid dorsiflexion movements were necessary to generate clonus in all subjects who manifested such behaviors (4/11 subjects). Mean amplitude of the manual dorsiflexion perturbations \pm SD was $21^\circ \pm 5.6^\circ$, generated at a mean angular velocity of $166^\circ \pm 61^\circ/\text{s}$, with differences likely arising from velocity-dependent spastic responses typically observed with rapid stretch of plantarflexors in people with SCI. Similarly, [figure 2C](#) shows the rapid passive hip and knee extension, with a mean angle change of $108.81^\circ \pm 12.3^\circ$ at the hip and $106.99^\circ \pm 8.81^\circ$ at the knee. The average peak speed of the perturbation was $89.15^\circ \pm 18.5^\circ/\text{s}$ at the hip, which was followed by prolonged knee extensor and ankle plantarflexor electromyographic activity in 7 of 11 subjects tested. Objective measurements of [flexor spasm](#) activity after pinprick, as shown in [\(fig 2B\)](#), were collected by using hip, knee, and ankle angle trajectories after medial arch stimulation. A typical electromyographic response from the lower-extremity musculature is also shown in this figure.

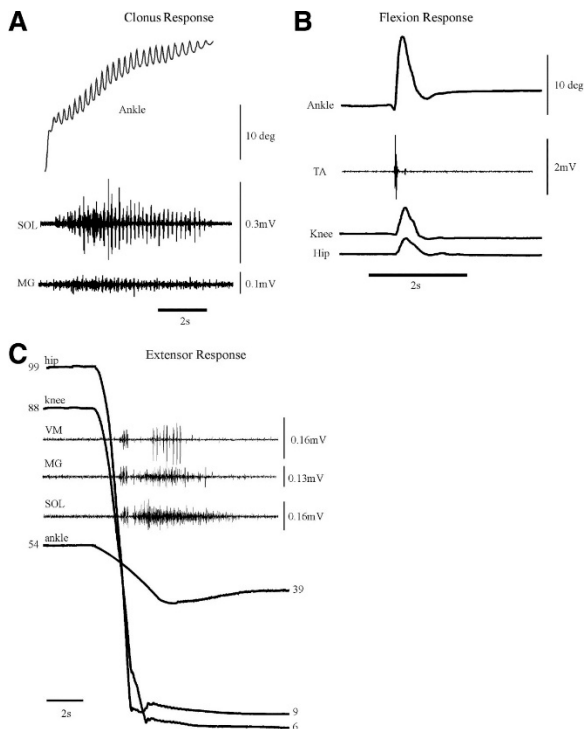


Fig 2. Kinematic and electromyographic measurements for [clonus](#), [flexor spasms](#), and extensor [spasms](#) are shown. (A) The top trace shows the ankle position during a clonus perturbation and response. Dorsiflexion is upward. The initial movement perturbation is followed by clonic oscillations of the ankle. The response ankle movements are accompanied by bursts of activity in the plantarflexors (below). (B) The ankle, knee, and hip flexion responses to a pinprick stimulus applied to the foot are shown along with the electromyographic signal from the tibialis anterior. (C) The extension movement and ensuing electromyographic response of the leg extensors is shown. The initial and final joint flexion angles are indicated on the position traces. Extension of the leg was followed by knee and ankle extensor activity, which was observed as a displacement of the [patella](#). Abbreviations: MG, medial gastrocnemius; SOL, soleus; TA, tibialis anterior; VM, [vastus](#) medialis.

All SCATS measures correlated significantly with the corresponding EMG or kinematic measurement and with SCATS scores identified from electromyographic or kinematic data, as summarized in [table 2](#).

Table 2. Correlation of the SCATS and Kinematic and Electromyographic Measures

Measure 1 (laboratory based)	Measure 2 (clinical measure)	ρ	P
Vastus medialis duration	Extensor SCATS	.90	<.001
Soleus duration	Extensor SCATS	.70	<.001
Extensor SCATS	Extensor SCATS	.94	<.001
Medial gastrocnemius duration	Clonus SCATS	.69	.002
Clonus SCATS	Clonus SCATS	.90	<.001
Ankle excursion angle	Flexor SCATS	.69	<.001
Knee excursion angle	Flexor SCATS	.81	<.001
Hip excursion angle	Flexor SCATS	.82	<.001
Flexor SCATS	Flexor SCATS	.87	<.001

Validation of the SCATS with the Ashworth Scale and PSFS

The outcomes of statistical comparisons between the SCATS and the Ashworth Scale yielded variable results ([table 3](#)). Specifically, comparison of Ashworth scores from the hip, knee, and ankle all correlated

significantly with SCATS extensor [spasm](#) scores. Conversely, SCATS flexor spasm ratings correlated significantly only with hip flexor Ashworth scores, while SCATS clonus scores were correlated only to knee and ankle Ashworth measures. In consideration of the established validity of SCATS (as determined earlier), the lack of consistent correlations between SCATS and Ashworth scores indicates differences in the manifestation of spastic responses across this subject population.

Table 3. Spearman Rank-Order Correlation of the SCATS, Ashworth Scale, and PSFS

	Ashworth Hip	Ashworth Knee	Ashworth Ankle	SCATS Clonus	SCATS Flexion	SCATS Extension
PSFS	.43	.43	.51	.59*	.41	.40
Ashworth hip		.90 [†]	.67*	.56	.55*	.98 [†]
Ashworth knee			.77 [†]	.65*	.47	.88 [†]
Ashworth ankle				.60*	.40	.61*
SCATS clonus					.35	.59*
SCATS flexion						.56*

*Significant at $P < .05$.

†Significant at $P < .01$.

Comparison of PSFS ratings with SCATS scores revealed a significant correlation only with the SCATS clonic responses, which indicates that clonus may play a substantial role in patient reports of spasm frequency. Conversely, considering the preponderance of flexor and extensor spasm behaviors, as shown in the study, patient subjectivity may play a substantial role in the perception of the severity of spastic [motor behaviors](#).

Discussion

The results of our study are promising for the use of the SCATS to quantify spastic [motor behaviors](#) in SCI. The SCATS provided a valid estimate of [clonus](#), flexor, and extensor [spasms](#), as evidenced by the kinematic and electromyographic analyses. Comparison of the SCATS with commonly used [clinical assessment tools](#) revealed correlation coefficients of variable significance. Extensor spasms correlated highly with Ashworth scores, whereas [flexor spasms](#) and clonus scores correlated to a lesser extent.

The SCATS may have advantages over previously used measures of spastic [hypertonia](#) for subjects with SCI. The clinical scales most widely used by clinicians, in particular the Ashworth Scale and the MAS, classify [spasticity](#) as a single-joint, velocity-dependent resistance to movement. Although these scales provide valid estimates of spastic hypertonia, their reliability has been questioned.^{10, 20, 21} In addition, multijoint flexor and extensor spasms, which are prevalent in SCI, are not accounted for in the Ashworth Scale and MAS. The SCATS is a more comprehensive clinical scale for spastic hypertonia in SCI that includes measurements of multijoint spastic motor behaviors. As a result, the SCATS provides additional valuable information about the occurrence of different types of spastic motor behaviors, and the incidence of different components of the SCATS may reflect patient-specific physiologic differences.

The PSFS is a more general scale than the Ashworth, and theoretically it includes many types of spastic motor behaviors. Surprisingly, the PSFS only correlated significantly with SCATS clonus scores, which may indicate a substantial role of clonus in the perception of the occurrence of spasms. These results suggest that the PSFS does not adequately record flexor and extensor spasms, which may only be triggered during specific ADLs. Flexor and extensor spasms may be used by patients to assist in functional tasks in a somewhat controlled manner and thus may not be included in the patient reports. These observations suggest that the PSFS is useful

for identifying the frequency of problem-causing spasms; however, it does not appear to distinguish between the types of spastic reflexes that are elicited. As a result, it would be beneficial for clinicians to gather more data from both measures.

Although the SCATS reliably distinguishes between different types and severity of spastic reflexes in SCI and can be administered quickly in the clinic, it ideally should be combined with other measures. For example, the SCATS fails to account for the patient's own perspective of the severity of spasticity. We propose that the SCATS be integrated into a multidimensional approach to the evaluation of spastic hypertonia by using patient and clinician reports. This approach would provide both objective measures of spastic hypertonia and subjective, patient-based information that could be acquired within a timeframe appropriate for a clinical setting.¹¹ The SCATS augments the data provided by the Ashworth Scale and the PSFS by providing information about the type of spastic hypertonia and the underlying physiology.

Because of the variability of SCATS scores among subjects, differences in manifestation of spastic motor behaviors likely reflect differences in the pathophysiologic mechanisms that underlie the spasms. For example, clonus is believed to occur when there is recurrent excitation of a hyperactive [stretch reflex](#) and/or activity of a central oscillator that excites the motor neurons.^{8, 22, 23} Conversely, flexor spasms are often triggered by cutaneous stimuli and may be attributed to an increased excitability of the [interneurons](#) of the [flexor reflex](#) pathway.^{13, 24} The association of flexor spasms with the flexion [withdrawal reflex](#) has been shown by decreased thresholds for activation of the flexion withdrawal reflex in SCI and by the presence of a large amplitude and duration of [flexor muscle](#) activity when a cutaneous stimulus is applied.¹³ Finally, extensor spasms, which are often triggered by extension of the hips,³ may be mediated by increased excitability of the interneuronal circuitry normally used for increasing limb stiffness during standing or during the stance phase of locomotion.¹⁵ The SCATS provides a mechanism to accurately assess targeted spastic behaviors that are associated with specific physiologic mechanisms.

Improved understanding of the predominant types of spastic motor behaviors is important to spasticity management, because different types of spasms have been found to interfere with functional mobility in various ways. Patients report that clonus interferes with function primarily associated with wheelchair propulsion and transfers. Flexor spasms are reported to interfere with function during sleep, bed positioning, and transfers.^{3, 6} Flexor spasms have also been clinically observed to interfere with ambulation. Extensor spasms, which occur most frequently, cause the most discomfort and produce the greatest interference with function during transfers and wheelchair propulsion.^{3, 6} However, people have also reported that extensor spasms can be helpful, especially when performing lower-extremity dressing.

We believe that knowledge of the type of spastic motor behaviors and its severity will facilitate informed decisions about the administration of pharmacologic and [physical treatment](#) approaches. For example, clinical practice suggests that clonus can be minimized by positioning a footplate on the wheelchair that puts the ankle at an angle past that at which the hyperactive reflex is elicited ($\approx 2^\circ$ from maximum dorsiflexion).²⁵ Furthermore, evaluation of force distribution along the plantar aspect of the foot when prescribing shoes or ankle-foot [orthoses](#) could minimize flexor spasms. Extensor spasms may be minimized by changes in the wheelchair seat angle (dump), to ensure that hip angle does not change during wheelchair propulsion, on uneven terrain, or with upper-extremity activity. In addition, alternative transfer techniques that do not require a change in hip angle could be implemented. With a greater understanding of the physiologic mechanisms underlying spasms and spastic hypertonia, and with clinical tools designed to assess accurately the various motor patterns seen after SCI, effective pharmacologic interventions can be prescribed. For instance, knowledge of whether the spasms are related to motoneuronal or interneuronal hyperexcitability may enable targeting of specific serotonergic or noradrenergic pathways.²⁴ During pharmacologic treatment, the SCATS could also be used, as clinicians use the Ashworth Scale, to assess the efficacy of the drug.^{26, 27, 28}

It should also be noted that the SCATS is intended primarily for people with SCI and may not be suitable for use with other populations that exhibit spastic hypertonia such as those with traumatic brain injury, stroke, or [cerebral palsy](#). The extensor and flexor reflexes that are common in SCI are less prominent in people with more cephalad injuries.

Conclusions

The SCATS provides a valid method for measuring spastic [motor behaviors](#) in the clinical setting and distinguishes among different types and severity of spastic reflexes. The SCATS may be a useful adjunct to self-report scales and may have a significant impact on guiding both functional and pharmacologic management of [spasticity](#) in SCI.

References

1. Young RR, Wiegner AW. Spasticity. *Clin Orthop* 1987;Jun(219): 50-62.
2. Meythaler JM. Physiology of spastic hypertonia. *N Am Clin Phys Med Rehabil* 2001;32:2099-109.
3. Little JW, Micklessen P, Umlauf R, Brittel C. Lower extremity manifestations of spasticity in chronic spinal cord injury. *Am J Phys Med Rehabil* 1989;68:32-6.
4. Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner* 1964;192:540-2.
5. Lance JW. Symposium synopsis. In: Feldman RG, Young RR, Koella WP, editors. *Spasticity: disordered motor control*. Chicago: Yearbook Medical Publishers; 1980. p 485-94.
6. Skold C, Levi R, Seiger A. Spasticity after traumatic spinal cord injury: nature, severity and location. *Arch Phys Med Rehabil* 1999;80:1548-57.
7. Meythaler JM. Spastic hypertonia. Appendix. *Phys Med Rehabil Clin North Am* 2001;12(4):953-6.
8. Hinderer SR, Dixon K. Physiologic and clinical monitoring of spastic hypertonia. *Phys Med Rehabil Clin North Am* 2001;12(4): 733-45.
9. Skold C, Levi R, Seiger A. Spasticity after traumatic spinal cord injury: nature, severity and location. *Arch Phys Med Rehabil* 1999;80:1548-57.
10. Hass BM, Crow JL. Towards a clinical measurement of spasticity? *Physiotherapy* 1995;81:474-9.
11. Blackburn M, van Vliet P, Mockett SP. Reliability of measurements obtained with the modified Ashworth scale in the lower extremities of people with stroke. *Phys Ther* 2002;82:25-34.
12. Kuhn RA. Functional capacity of the isolated human spinal cord. *Brain* 1950;73:1-51.
13. Shahani BT, Young RR. The flexor reflex in spasticity. In: Feldman RG, Young RR, Koella WP, editors. *Spasticity: disordered motor control*. Chicago: Yearbook Medical Publishers; 1980. p 287-95.
14. Meythaler JM, Guin-Renfroe S, Brunner RC, Johnson A, Hadley MN. Intrathecal baclofen for spastic hypertonia from stroke. *Stroke* 2001;32:2099-109.
15. Schmit BD, Benz EN. Extensor reflexes in human spinal cord injury: activation by hip proprioceptors. *Exp Brain Res* 2002;145: 520-7.
16. Schmit BD, McKenna-Cole A, Rymer WZ. Flexor reflexes in chronic spinal cord injury triggered by imposed ankle rotation. *Muscle Nerve* 2000;23:793-803.
17. Penn RD. Intrathecal baclofen for severe spasticity. *Ann N Y Acad Sci* 1988;153:157-66.
18. Priebe MM, Sherwood AM, Thornby JI, Kharas NF, Markowski J. Clinical assessment of spasticity in spinal cord injury: a multi-dimensional problem. *Arch Phys Med Rehabil* 1996;77:713-6.
19. Hidler J, Rymer WZ. A simulation study of reflex instability in spasticity: origins of clonus. *IEEE Trans Rehabil Eng* 1999;7:327-40.
20. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth Scale of muscle spasticity. *Phys Ther* 1987;67:206-7.

21. Pandyan AD, Johnson GR, Price CI, Curless RH, Barnes MP, Rodgers H. A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measures of spasticity. *Clin Rehabil* 1999;13:373-83.
22. Gottlieb GL, Agarwal GC. Physiological clonus in man. *Exp Neurol* 1977;54:616-21.
23. Rossi A, Mazzocchio R, Scarpini C. Clonus in man: a rhythmic oscillation maintained by reflex mechanisms. *Electroencephalogr Clin Neurophysiol* 1990;75:56-63.
24. Heckman CJ. Alterations in synaptic input to motoneurons during partial spinal cord injury. *Med Sci Sports Exerc* 1994;26:1480-90.
25. Meinders M, Price R, Lehmann JF, Questad KA. The stretch reflex response in the normal and spastic ankle: effect of ankle position. *Arch Phys Med Rehabil* 1996;77:487-92.
26. Katz RT, Rovai GP, Brait C, Rymer WZ. Objective quantification of spastic hypertonia: correlation with clinical finding. *Arch Phys Med Rehabil* 1992;73:339-47.
27. Sampaio C, Ferreira JJ, Pinto AA, Crespo M, Ferro JM, Castro-Caldas A. Botulinum toxin type A for the treatment of arm and hand spasticity in stroke patients. *Clin Rehabil* 1997;11:3-7.
28. Meythaler JM, Guin-Renfroe SG, Hadley MN. Continuously infused intrathecal baclofen for spastic/dystonic hemiplegia: a preliminary report. *Am J Phys Med Rehabil* 1999;78:247-54.

Suppliers

- a. Northern Digital Inc, 103 Randall Dr, Waterloo, ON N2V 1C5, Canada.
- b. Model 2.1; DelSys Inc, 650 Beacon St, 6th Fl, Boston, MA 02215.
- c. Version 6.1; The MathWorks Inc, 3 Apple Hill Dr, Natick, MA 01760-2098.