

Effects of thoraco-lumbar electric sensory stimulation on knee extensor spasticity of persons who survived cerebrovascular accident (CVA)

Ray-Yau Wang, PhD; Rai-Chi Chan, MD; Mei-Wun Tsai, MS

Faculty of Physical Therapy, National Yang-Ming University, Shih-Pai, Taipei, Taiwan; Department of Physical Medicine and Rehabilitation, Veterans General Hospital, Taipei, Taiwan; Faculty of Physical Therapy, National Yang-Ming University, Shih-Pai, Taipei, Taiwan

Abstract—Spasticity is mostly due to an excess of impulses to alpha motor neurons partly resulting from a change of interneuron activity. Low threshold afferent has been reported to change the activity in interneuron. The purpose of this study is to investigate the effects of surface spinal paravertebral stimulation on knee extensor spasticity. Ten survivors of stroke, with knee extensor spasticity, received electric stimulation for five 45-minute periods through surface electrodes applied to the skin in the twelfth thoracic and first lumbar areas. The electric stimulations had an amplitude modulated alternating current (AC), with a carrier frequency of 2500 Hz, modulated to “beats” frequency of 20 Hz. Stimulation amplitude was raised to elicit sensory stimulation. The pre- and post-treatment evaluation included the modified Ashworth scale, active torque during controlled knee movements at various velocities, and electromyographic (EMG) activity during the torque measurements. Our results indicate that nine of ten subjects demonstrated a decrease in the modified Ashworth scale post-treatment. The EMG activity of the spastic quadriceps during active knee flexion was decreased post-treatment as compared with the value before treatment. The active torque value of knee flexion or extension at 30°, 60°, or 90°/sec of angular velocity did not change significantly post-treatment. A trend of increasing spastic quadriceps EMG activity with respect to the angular velocity during an active knee flexion was established, with Ashworth scale considered. The level of EMG activity is higher

when the Ashworth scale is higher. According to our results, the surface paravertebral sensory stimulation was effective in reducing quadriceps muscle spasticity of the subjects. Both the modified Ashworth scale and the EMG activity of spastic quadriceps during eccentric contraction are suggested as sensitive tools for measuring spasticity of persons who survived cerebrovascular accident (CVA).

Key words: CVA, electric sensory stimulation, EMG, knee extensor spasticity, stroke.

INTRODUCTION

Spasticity, a velocity-dependent response to passive muscle stretch, is present in most persons with stroke (1). Spasticity may affect or interact with a variety of voluntary movement parameters (2). These may include force and torque production, rate of torque production, adaptability of selected measures of performance, ability to reciprocate movement, accuracy, and movement speed. Improvements in selected measures of performance were correlated to a reduction in spasticity (2).

The use of electric stimulation to temporarily reduce spasticity has been reported (3,4). Visser et al. demonstrated an anti-spastic effect on the calf muscles by stimulating the peroneal nerve(s). Such an effect is ascribed to the activation of the spinal interneuronic system (5). However, the decrease of spasticity by way of indirect activation of Golgi tendon organs through the electrically elicited contraction and the resultant tension in the spastic muscles has been reported (6). There is also reported evidence that low threshold afferent input can change the activity in interneurons and/or alpha motoneurons via segmental,

This material is based on work supported by the National Science Council of the Republic of China, and awards from the Medical Research and Advancement Foundation in memory of Dr. Chi-Shuen Tsou (ROC) to R-Y Wang.

Address all correspondence and requests for reprints to: Dr. Ray-Yau Wang, Faculty of Physical Therapy, National Yang-Ming University, 155, Sec 2, Li-Nong St., Shih-Pai, Taipei, Taiwan, Republic of China. email: rywang@ym.edu.tw

propriospinal, or supraspinal pathways (7). The improvement of knee flexion torque was obtained by applying low intensity electric stimulation on the skin of low-cervical and high-thoracic levels in persons with multiple sclerosis (8). Our previous study also indicated that surface paraspinal stimulation was effective in reducing calf muscle spasticity measured by passive ankle dorsiflexion torque in persons who survived cerebrovascular accident (CVA), commonly called "stroke" (9).

Evaluation of pre- and post-treatment procedures helps to establish the efficacy of various treatments in managing spasticity (9). However, quantification of spastic hypertonia remains a difficult and controversial problem (10). The Ashworth scale has been used clinically and is one of the standardized tests to measure spasticity (11). Torque measurements during velocity controlled joint movements can be used to quantify changes in velocity and length-dependent reflex activity of persons with spasticity (12). The EMG activity indicates the state of activation of the contractile element (13). Thus, we used a modified Ashworth scale, active torque, and EMG activity for spasticity measurement.

This study investigated the effects of surface electric sensory stimulation over the paravertebral region on knee extensor spasticity. An additional question posed was whether there is an indication of qualitative trend between the modified Ashworth scale and the EMG activity of the spastic quadriceps during active knee flexion.

METHODS

Subjects

Ten subjects, one female and five males with left hemiplegia, and four males with right hemiplegia were selected through non-probability accidental sampling (**Table 1**). Criteria for subject selection included the following: medically stable (per physician's consent), ability to follow commands, extensor spasticity in the lower limb, no history of a previous neurological disorder, and no pain or sensory impairment in the lower limb. Subjects were advised as to the nature of the study and gave their informed consent. The age range was from 29 to 80 years, with a mean of 57 years. The subjects were all ambulatory with assistive devices. The range of time since onset of stroke was between 8.6 and 18.4 months, with a mean of 12.5 months. The magnitude of spasticity was determined by the modified Ashworth scale (11) as shown in **Table 2**. Subjects were tested during passive knee flexion for quadriceps spasticity in the supine position. Six subjects scored grade 2 and four scored grade 3.

Treatment: Electric Stimulation

An electrode was placed on each side of the spine (5 cm apart) over the paravertebral skin at the twelfth thoracic and first lumbar vertebral levels. The self-adhesive electrodes were of rectangular shape (4.5 cm x 9 cm). The electric pulses were generated from amplitude modulated

Table 1.
General information and modified Ashworth scale.

Subj	Sex	Site	Type	Age	Time	Device	Ashworth	
							Pre	Post
1	F	R	isc	29	8.6	NBQC	2	2
2	M	R	isc	43	9.7	WBQC	3	1
3	M	R	isc	72	15.1	WBQC	2	1
4	M	L	isc	57	10.2	WBQC	3	1
5	M	L	hem	68	17.5	WBQC	3	1
6	M	R	isc	57	14.2	WBQC	3	1
7	M	L	isc	80	9.6	Cane	2	1
8	M	R	isc	46	18.4	WBQC	2	1
9	M	L	isc	68	10.3	NBQC	2	1
10	M	L	isc	46	11.4	Cane	2	1

Subj=subject; Site=lesion site, right (R) or left (L) of middle cerebral artery; Type=lesion type, ischemic (isc) or hemorrhagic (hem); Age in years; Time=time post-stroke, in months; Device=assistive device; NB=narrow-based; WB=wide-based; QC=quad cane; Cane=straight cane; Ashworth=modified Ashworth scale; Pre=pre-treatment; Post=post-treatment.

Table 2.
Modified Ashworth scale for grading spasticity (11).

Grade	Description
0	No increase in muscle tone.
1	Slight increase in muscle tone, manifested by a catch and release, or by minimal resistance at the end of the range of motion when the affected art(s) is moved in flexion or extension.
2	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance through the remainder (less than half) of the range of movement (ROM).
3	More marked increase in muscle tone through most of ROM, but affected part(s) easily moved.
4	Considerable increase in muscle tone, passive movement difficult.
5	Affected part(s) rigid in flexion or extension.

alternating current (AC) with a carrier frequency of 2500 Hz, modulated to deliver "beat" at a frequency of 20 Hz (3,4). The stimulation amplitude was adjusted for each subject to produce only sensory stimulation, and not to cause muscle contraction. Such a stimulation pattern was applied continuously for 45 minutes. The equipment used in this study was a dual channel multifrequency stimulation system (Prostim, Ever Prosperous Instrument, Inc. Taipei, Taiwan, ROC) (9).

Measurements

A commercially available computerized isokinetic dynamometer (Kin-Com model 500 H, Chattecx Co., Chattanooga, TN, USA) was used in this study. Peak torques during knee flexion and extension were determined at preset angular velocities of 30°, 60° and 90°/sec. Three consecutive torque amplitudes were recorded at each angular velocity, and averaged to yield the peak torque amplitude for analysis. A Nicolet Viking electromyographic (EMG) system model IIe was used to monitor EMG activities during active knee movements at angular velocities of 30°, 60°, and 90°/sec. Pairs of disposable Ag/AgCl surface electrodes with 10-mm radius were placed on the quadriceps and hamstrings muscles. Quadriceps active electrodes were placed 12 cm and 16 cm above the edge of the knee cap, and hamstring active electrodes were placed 12 cm and 16 cm above the popliteal fossa with the ground electrode on the forearm of the same side. The skin impedance was kept below 5k Ohm (checked by a built-in impedance meter in an EMG

machine). Bandwidth of the frequency filter was 5 to 500 Hz. With EA-100 amplifier, the display gain was set at 200 μ V to 500 μ V and the sweep speed at 200 msec/div with a total epoch of 4 seconds to include the EMG activities during velocity controlled joint movement at various velocities. All the EMG activities were rectified and integrated and were converted to digital data by a set-in AD converter with the sampling rate of 1024 samples/sec. The root mean square EMG (RMS EMG) was used as the measurement parameter. The cut-off criteria of automatic amplitude pick up was set at 20 μ V, so that the onset and termination of EMG activity easily could be determined.

The subjects were examined by an independent physical therapist. Each subject was seated in a chair with thighs and hips restrained by straps and legs hanging over the edge of the seat. The knee was maintained at 90° of flexion. First, measurements of the active knee extension torque (from 90° of flexion to full extension) in Newton meters (Nm) were recorded at 3 angular velocities, 30°/sec, 60°/sec and 90°/sec. Three measurements were taken at each velocity with a 30-second rest period between measurements. Additionally, surface EMG activities were recorded simultaneously from the quadriceps and hamstrings activation during the torque measurements. Second, measurements of active knee flexion torque (from full extension to 90° of flexion) were also recorded at 3 angular velocities, 30°/sec, 60°/sec and 90°/sec. Three measurements were taken and then averaged at each velocity, with a 30-second rest period between measurements. The surface EMG activities were recorded simultaneously from the hamstrings and quadriceps activation during the torque measurements. Finally, the spasticity of the subject's affected knee extensors was assessed three times, according to the modified Ashworth scale, by another physical therapist who was blinded to the torque and EMG data. After the initial measurements were taken, all subjects received treatments for five days. Another set of measurements was documented one day after the subjects completed the five treatments.

Statistical Analysis

Means and standard deviations were calculated for all measured variables. Treatment means were tested for homogeneity with a two-way analysis of covariance (ANCOVA). The differences between means of each of the measured variables were tested for significance by Scheffe's multiple range test or paired t-test. A difference between two means was considered to be statistically significant when p was less than 0.05.

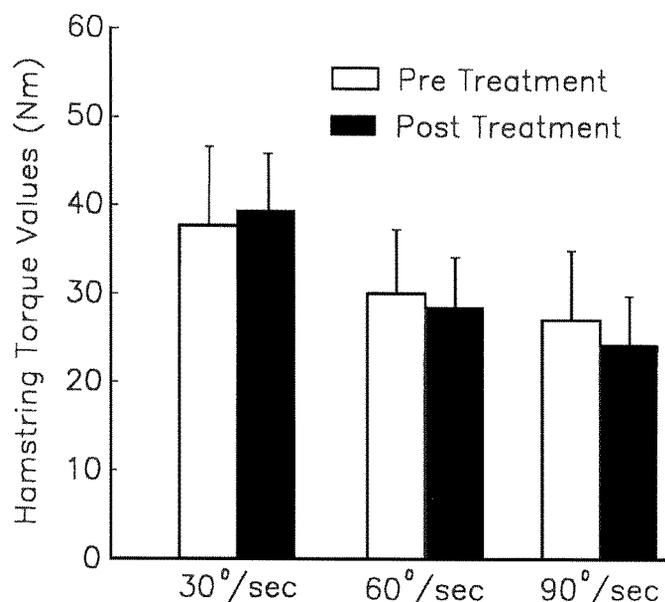


Figure 1. Effect of surface electric sensory stimulation on the thoraco-lumbar region on hamstrings torque values (Nm) during active knee flexion at 30, 60, and 90°/sec of angular velocity.

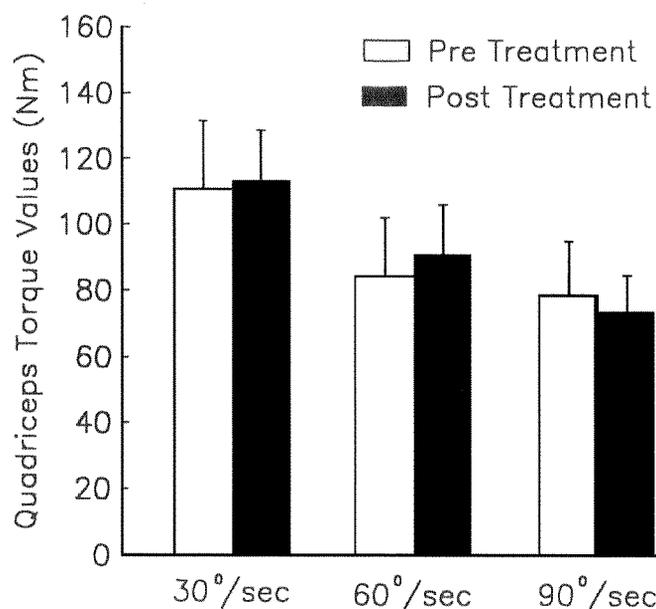


Figure 2. Effect of surface electric sensory stimulation on the thoraco-lumbar region on quadriceps torque values (Nm) during active knee extension at 30, 60, and 90°/sec of angular velocity.

RESULTS

Nine of the ten subjects demonstrated a decrease in the modified Ashworth scale during the passive knee flexion movement post-treatment. There was a reduction of one point in five of the patients, and a reduction of two points in four patients (**Table 1**). The mean torque values of active knee flexion (hamstrings) at 30°, 60°, and 90°/sec angular velocity after 5 electric stimulation treatments were not different from the values before treatment (**Figure 1**). Similarly, no differences were found between pre- and post-treatments in active knee extension torque (quadriceps) at 30°, 60°, and 90°/sec angular velocity (**Figure 2**). The hamstrings EMG activity levels during active knee flexion after treatment were not different from the levels before treatment (**Figure 3**). However, post-treatment, the EMG activity of quadriceps muscle activity during active knee flexion was lower than that before treatment at 90°/sec angular velocity ($t = -2.39, p < 0.05$) as shown in **Figure 4**. This post-treatment EMG activity level was also lower than pre-treatment at 30° and 60°/sec angular velocity, but not to a significant level (**Figure 4**). Both quadriceps (**Figure 5**) and hamstrings (**Figure 6**) EMG activity levels during active knee extension post-treatment did not change

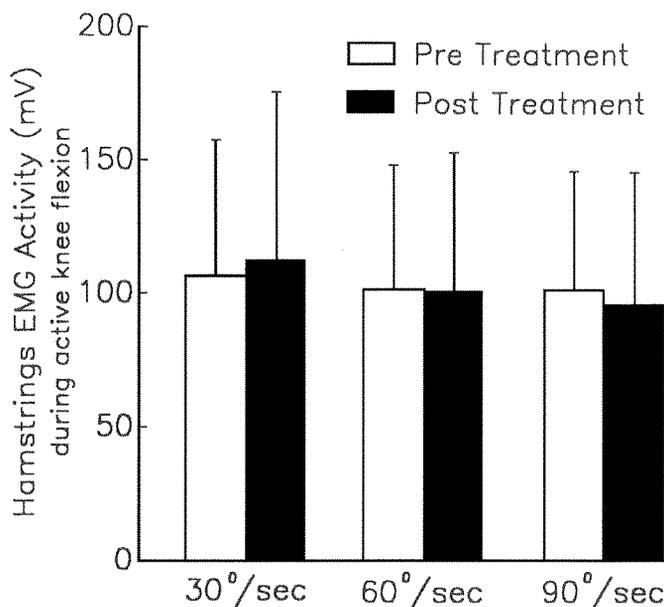


Figure 3. Effect of surface electric sensory stimulation on the thoraco-lumbar region on EMG activity of hamstring muscle during active knee flexion at 30, 60, and 90°/sec of angular velocity.

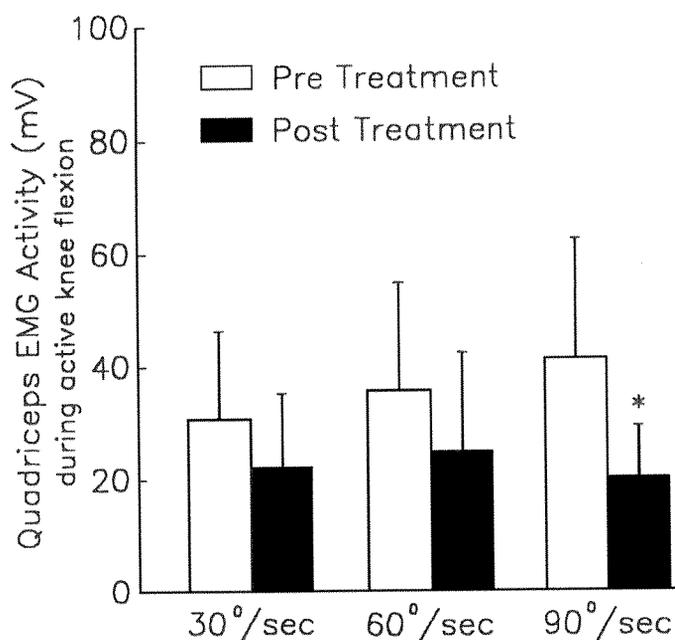


Figure 4. Effect of surface electric sensory stimulation on the thoraco-lumbar region on EMG activity of quadriceps muscles during active knee flexion at 30, 60, and 90°/sec of angular velocity. * $p < 0.05$ versus before treatment.

significantly as compared with the EMG activity level before treatment at 30°, 60°, or 90°/sec. **Figure 7** shows that the EMG activity increased linearly with the angular velocity, at two separate Ashworth scales 2 and 3. The EMG jumped consistently from a lower Ashworth scale 2 to a higher Ashworth scale 3 ($p < 0.05$).

DISCUSSION

Our main finding is the demonstration that 5 sessions of 45 minutes of surface electric sensory stimulation on the skin of the thoraco-lumbar area significantly decreased knee extensor spasticity in persons with stroke. Both the modified Ashworth scale and the EMG activity of spastic quadriceps during active knee flexion are sensitive tools for documenting changes in spasticity across treatment. A trend of the two measurements is also established.

The abnormal increase of muscle tone in persons with stroke is mostly due to an excess of impulses to alpha-motoneurons, depending on gamma-dynamic hyperfacilitation, a reduction of the presynaptic inhibition, or a change of the activity of the Renshaw interneurons (14). This alteration may be traced back to a loss of supraspinal control (14). In these conditions, treatment can

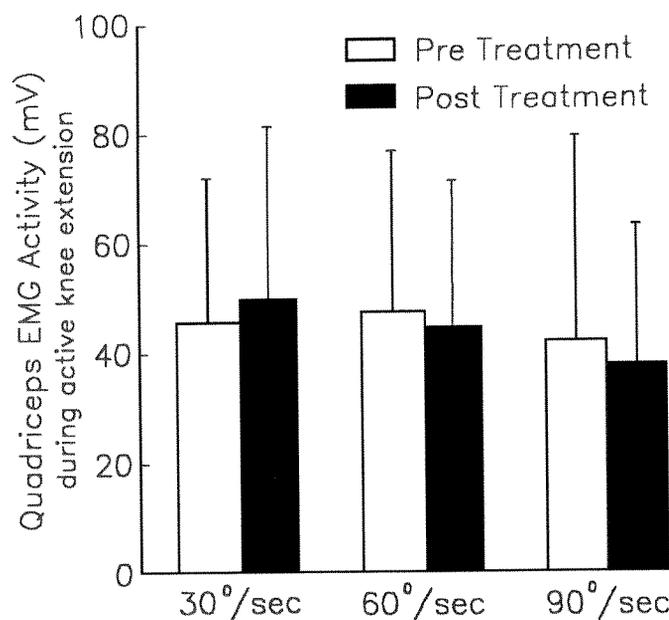


Figure 5. Effect of surface electric sensory stimulation on the thoraco-lumbar region on EMG activity of quadriceps muscle during active knee extension at 30, 60, and 90°/sec of angular velocity.

be regarded as useful when it is able to modulate the excess of impulses or to modulate the interneuronic system. Segmental input arising through electric stimulation may be one way of “switching on” presynaptic inhibitory mechanisms (15,16). The electric stimulation parameters used in this study may activate large diameter afferents of the paravertebral region, which may modulate the interneuronic activities of several spinal segments, and in turn decrease spasticity of the lower limb (9,15). After five electric sensory stimulation treatments, the decrease of spasticity in persons with stroke was noted, as indicated by the modified Ashworth scale and the EMG activity during eccentric contraction at a higher movement velocity.

Studies have demonstrated that the hyperactive stretch reflex can directly affect voluntary movement (17,18). In concentric contraction, the antagonists are stretched by the movement. This stretch usually leads to coactivation of the antagonist through stretch reflexes (19). In persons with spasticity, the spastic antagonist muscle restrains the joint movements considerably, because of the increased magnitude of the hyperactive stretch reflex (19). This spastic antagonist restraint increases as the movement velocity increases implicating the stretch reflex as velocity

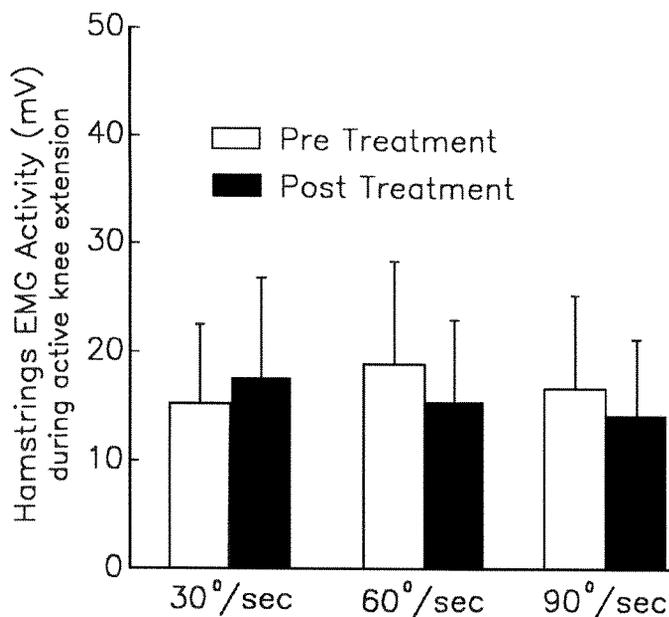


Figure 6. Effect of surface electric sensory stimulation on the thoraco-lumbar region on EMG activity of hamstring muscle during active knee extension at 30, 60, and 90°/sec of angular velocity.

dependent (19). Corcos found that the increased stretch reflex activity of calf muscles was the major factor in restraining dorsiflexion in his sample of subjects (18). Electric stimulation was proposed to reduce spasticity and improve control of stretch reflex in patients with stroke (3,7). Electric stimulation has been applied to antagonists of the spastic muscles for reciprocal inhibition, or directly to spastic agonist muscles to obtain a fatigue effect (3,7). Electric stimulation can also be used to decrease spasticity by modulating the transmission of afferent or efferent impulses (3,4). The underlying mechanisms may be due in part to sensory habituation resulting from generalized desensitization of the spinal pathway (3,4) or to an enhancement of presynaptic inhibition of spastic muscles (4,8). Our data suggest that after surface paraspinal electric sensory stimulation, the EMG activity of the spastic quadriceps during active knee flexion had decreased, especially at the higher velocity (90°/s), indicating the possible effect of our treatment on the hyperactive stretch reflex through sensory habituation, or enhancement of presynaptic inhibition, or both. However, the active torque

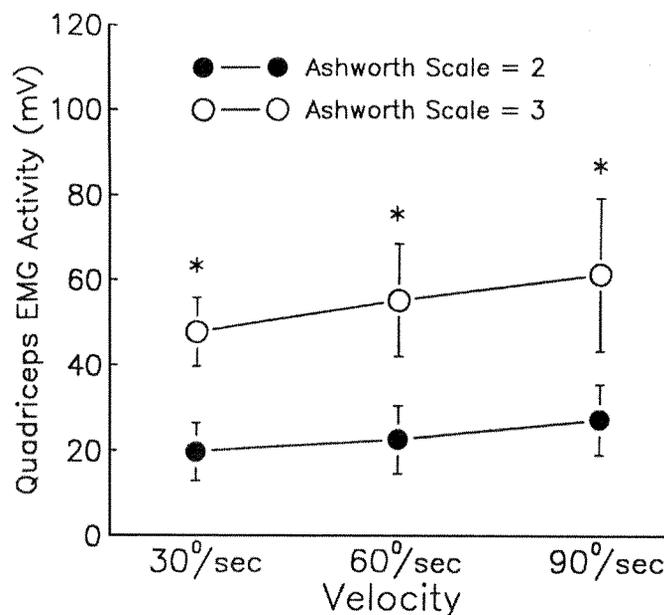


Figure 7. Quadriceps EMG activity with respect to the Ashworth scale during active knee flexion at 30, 60, and 90°/sec of angular velocity. * $p < 0.05$ versus Ashworth scale 2.

or the EMG activity of hamstrings during velocity controlled active knee flexion did not change significantly, suggesting that the EMG activity of the eccentric contraction of the quadriceps is more sensitive in detecting changes of spasticity than is the peak voluntary torque or the EMG activity of the hamstrings during active knee flexion with controlled velocity. Conversely, according to our previous results, the peak torque during passive, velocity-controlled movement was more sensitive for measuring ankle-calf-muscle spasticity than was the surface EMG activity during passive ankle dorsiflexion (9). Taking these facts together, we suggest that torque generated by spastic muscles during passive movement, EMG activity of spastic eccentric contraction, and a modified Ashworth scale are all useful measuring tools in tracking the temporal dependent magnitude of spasticity in persons who survived stroke.

A trend was established between the spastic quadriceps EMG activity and the angular velocity during active knee flexion, with the Ashworth scale considered. The EMG activity increases linearly with respect to the angular

velocity, with a drastic jump of the EMG activity when the Ashworth scale increases. The modified Ashworth scale was found to be correlated positively with the ankle passive torque, and such correlation was also velocity dependent according to our previous study (9).

Inadequate torque production is a common clinical finding in the presence of spasticity in persons with stroke (19). The restraint of voluntary movement can be attributed to abnormal muscle timing and errors in central activation (17,18). Knutsson demonstrated a decrease of voluntary torque with increasing velocity of movement in the spastic agonist muscle (19). The decrease of spastic quadriceps torque was also found with increasing velocity during active knee extension movement in our study. The active torque values of spastic knee extensors or the EMG activity of quadriceps during velocity controlled active knee extension had not been altered post-treatment, suggesting that the surface paraspinal sensory electric stimulation on the skin of the thoraco-lumbar area may not be able to change the recruitment pattern or the central activation pattern in spastic muscles.

In conclusion, this study adds ample support to the beneficial effects of surface paraspinal thoraco-lumbar sensory stimulation in the treatment of knee extensor spasticity in persons who survived CVA. There is a trend of the increasing of the spastic quadriceps EMG activity with respect to the angular velocity, with consideration of the Ashworth scale. Both the modified Ashworth scale and the EMG activity of spastic quadriceps eccentric contraction are suggested as sensitive tools for measuring spasticity of persons who survived CVA.

ACKNOWLEDGMENTS

The authors wish to thank Mr. Al Vendouris for his editing and English language consultancy.

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Submitted for publication January 1, 1999. Accepted in revised form May 3, 1999.