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Effects of a Single Session of Prolonged Muscle Stretch on Spastic Muscle of Stroke Patients

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ABSTRACT

The control of spasticity is often a significant problem in the management of patients with spasticity. The aim of this study was to evaluate the effect of a single session of prolonged muscle stretch (PMS) on the spastic muscle. Seventeen patients with spastic hemiplegia were selected to receive treatment. Subjects underwent PMS of the triceps surae (TS) by standing with the feet dorsiflexed on a tilt-table for 30 minutes. Our test battery consisted of four measurements including the modified Ashworth scale of the TS, the passive range of motion (ROM) of ankle dorsiflexion, the H/M ratio of the TS, and the F/M ratio of the tibialis anterior (TA). The results indicated that the passive ROM of ankle dorsiflexion increased significantly ($p < 0.05$) compared to that before PMS treatment. Additionally, PMS reduced motor neuron excitability of the TS and significantly increased that of the TA ($p < 0.05$). These results suggest that 30 minutes of PMS is effective in reducing motor neuron excitability of the TS in spastic hemiplegia, thus providing a safe and economical method for treating stroke patients.

Key Words: prolonged muscle stretch, spasticity, H-reflex, motor neuron excitability

I. Introduction

Spasticity has been defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron (UMN) syndrome" (Lance, 1980). In other words, spasticity and flexor spasms of UMN syndrome are due to hyperactive spinal reflexes. Some hypertonus of the UMN syndrome is due to imbalance in descending motor control (Little and Massagli, 1998).

Increased motor neuron excitability has been postulated to be a factor contributing to spasticity (Angel and Hofmann, 1963; Ashby and Verrier, 1976; Ashby *et al*., 1987). Spasticity frequently interferes with the motor functions of patients with stroke, spinal cord injury, traumatic brain injury, multiple sclerosis and cerebral palsy. In neurological rehabilitation, the control of spasticity is often a significant problem. Clinicians and researchers have attempted to alter motor neuron excitability through a variety of methods, including pressure (Leone and Kakulka, 1988), electrical stimulation (Bajd *et al*., 1985; Levin and Hui-Chan, 1992), vibration (Gillies *et al*., 1969), muscle tapping (Belanger, 1989), massage (Sullivan *et al*., 1991; Morelli *et al*, 1999), cooling (Bell and Lehmann, 1987) and stretch (Burke *et al*., 1971; Odeen and Knutsson, 1981; Odeen, 1981; Robinson *et al*., 1982; Tremblay *et al*., 1990; Richards *et al*., 1991; Kunkel, 1993; Childers *et al*., 1999; Avela *et al*., 1999). Among them, stretch has been extensively used in clinics because it is safe, convenient and economical.

Numerous investigators (Burke *et al*., 1971; Odeen, 1981; Tremblay *et al*., 1990; Richards *et al*., 1991; Kunkel, 1993) have studied the effects of prolonged muscle stretch (PMS) on spastic patients with cerebral palsy (CP) by means of long-term application of plaster ankle casts. Such interventions have been shown to increase the passive range of motion (ROM) of the ankle joint, to reduce muscle tone and to change variables during gait. Odeen and Knutsson (1981) found that when a paraplegia patient received a prolonged plantarflexor muscle stretch on a tilt-table or by bracing for

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30 minutes, the resistance of passive ankle dorsiflexion decreased significantly. In an other study, Odeen (1981) reported that mechanical stretching of the hip adductor in CP subjects led to increased passive and active ROM of hip abduction and less myoelectric activity in the hip adductor during active hip abduction. Tremblay *et al*. (1990) found that when children with CP received PMS of plantarflexor on a modified tilt table for 30 minutes, the passive ROM of ankle dorsiflexion increased. The neuromuscular responses (torque and EMG) to passive movement of the ankle joint were also significantly reduced. Burke *et al*. (1971) studied the effects of static stretch on the plantarflexor in the prone position. Increasing passive dorsiflexion of the ankle resulted in progressive diminution of the H-reflex. These studies suggest that PMS can reduce spasticity and improve the function of the antagonist, but they do not describe the changes in reflexes. Furthermore, the effective duration of a single PMS applied to spastic muscle has not been studied.

Thus, this study was designed to evaluate changes in motoneuron excitabilities and to clarify the effective duration resulting from a single session of PMS applied to the spastic muscle. The hypothesis was that the motoneuron excitability of the soleus muscle and that of the TA muscle would be changed by PMS. We also wanted to determine the relationships between the clinical assessment scale and motoneuron excitabilities, and those between passive ROM and motoneuron excitabilities.

II. Materials and Methods

Seventeen subjects (14 males and 3 females) between the ages of 33 and 79 years (56.76 ± 11.62) with spastic hemiplegia due to cerebrovascular accidents participated in the study. The average time since the onset of hemiplegia was 25.8 ± 21.82 months. The subjects were selected for the study on the basis of having spasticity in a lower limb, a minimum of 0° of passive ankle dorsiflexion, no pain in the lower limb and no history of a previous neurological disorder (Table 1).

Table 1. Subject Characteristics

No.	Sex	Side of hemiplegia	Age (yr)	Duration of static Hemiplegia (m)[a]	Modified Ashworth scale
1	M	L	59	4.55	1
2	M	L	68	36.52	2
3	M	R	41	38.57	1
4	M	R	60	33.87	1
5	M	R	53	79.68	3
6	M	R	33	9.77	2
7	M	R	44	27.68	3
8	F	R	59	15.84	3
9	M	R	49	19.29	1
10	M	L	65	71.65	4
11	M	R	62	3.77	2
12	F	R	57	39.3	1
13	F	L	79	10.77	2
14	M	L	60	12.77	2
15	M	L	70	7.77	1
16	M	R	65	3.5	3
17	M	L	41	23	2
Mean			56.76	25.8	2
SD			11.62	21.82	0.94

[a]m = month

1. Procedure

All the subjects were subjected to three tests, including a pre-test, post-test and post-45 minute test. Each test consisted of four measurements: (1) the modified Ashworth scale; (2) passive ROM of ankle dorsiflexion; (3) maximal amplitude of the H-reflex for soleus muscle as a percentage of the M response (H/M ratio); (4) amplitude of the F wave for tibialis TA muscle as a percentage of the M response (F/M ratio).

First, the spasticity level of the TS was measured according to the modified Ashworth scale (Bohannon and Smith, 1987) by a senior therapist. The scale of the modified Ashworth scale ranged from 0 to 5: grade 0 indicated no increase in muscle tone, and grade 5 indicated the affected part or parts were rigid in flexion or extension. Then the passive ROM, based on an average of 3 measurements, for dorsiflexion was recorded using a goniometer. The axis of the goniometer was fixed on the lateral malleolus. The angle between the fibula shaft and the fifth metatarsal bone was measured. A right angle between the fibula shaft and the fifth metatarsal bone was defined as 0° of dorsiflexion. After passive ROM measurements, were performed, the electromyograph system (Viking IV D, Nicolet Biomedical Inc., WI, U.S.A.) was used to perform nerve stimulation and reflex recording. The skin resistance overlying the soleus muscle and TA muscle was made as minimal as possible by shaving the area and brushing it with alcohol. The muscle responses were recorded through two silver-chloride EMG electrodes, nine mm in diameter, coated with conducting cream. A ground electrode was located on the lateral aspect of the lower leg. The procedures for recording the H/M ratio and F/M followed accepted guidelines as described below:

A. H/M Ratio

H and M response were evoked using a rectangular voltage pulse, 1 ms in duration, by a stimulator. The stimulator was placed over the tibial nerve at the popliteal fossa. The recording electrode was placed along the longitudinal axis of the calf muscle. The active electrode was placed on half the distance from the tibial tubercule to the medial malleolus, and the reference electrode was placed 3 cm distally from the active electrode. The intensity of the stimulation was gradually increased to record maximal H-reflexes, followed by maximal M responses. The peak-to-peak amplitude of the maximal H-reflex that was elicited without a direct muscle response was recorded. Then, we adjusted the intensity so that the maxi-

mal M response was present. The H/M ratio was calculated by dividing the mean value of 3 H-reflexes by the mean value of 3 M responses.

B. F/M Ratio

F waves of TA muscle were evoked using a rectangular voltage pulse, 0.1 ms in duration, by the stimulator. The stimulator was placed over the deep peroneal nerve behind and proximal to the fibular head. The recording electrodes were placed over the belly of the TA muscle, 3 cm away along the longitudinal axis and near midline. F-waves were elicited by means of supramaximal intensity. The F/M ratio was calculated by dividing the mean value of 10 F-waves by the mean value of 3 M-responses.

When these tests had all been completed, the subjects were positioned on a tilt-table. The table was tilted 85° relative to horizontal, and straps were used to offer adequate support (Fig. 1). The ankle joints were placed at maximal dorsiflexion for each patient depending upon their ROM. According to previous studies (Odeen and Knutsson, 1981; Tremblay *et al*., 1990), the duration of the treatment time was set at 30 minutes. All the tests performed before treatment was performed were repeated immediately after treatment and again 45 minutes after treatment.

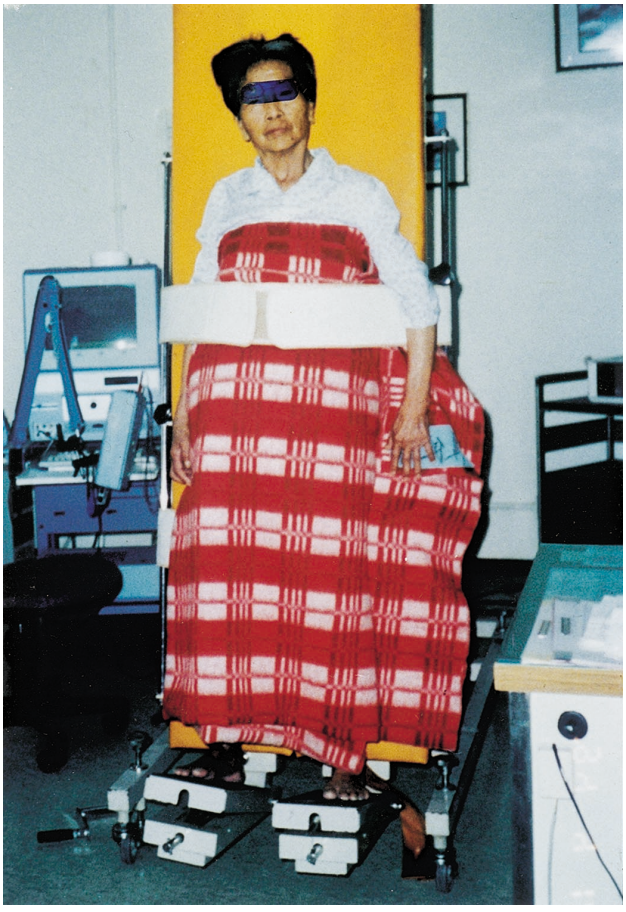

Fig. 1. Prolonged muscle stretch on a titled table.

2. Data Analysis

For data analysis, the differences in muscle tone (modified Ashworth scale of the TS) and ROM of dorsiflexion between different times before, after, and 45 minutes after treatment were compared by means of repeated measures analysis of variance at a significance level of 5%. The motoneuron excitabilities including the H/M ratios of TS and F/M ratios of TA at three different times were also compared. Moreover, the correlation between the H/M ratio and ROM of dorsiflexion, and the correlation between the ROM of dorsiflexion and the muscle tone were determined.

III. Results

With regard to resistance to passive dorsiflexion of the ankle joint based on the modified Ashworth scale, all the subjects suffered from an increase in muscle tone of the lower limb. Six subjects were grade 1. Six subjects were grade 2. Four subjects were grade 3. One subject was grade 4. (Table 1)

1. Muscle Tone and ROM of the Ankle Joint

A modified Ashworth scale was used to evaluate the muscle tone of the ankle plantarflexor. We found that the scores for pre-treatment (2 ± 0.9), post-treatment (1.2 ± 0.4) and 45 minutes post-treatment (1.5 ± 0.9) were not significantly different ($p > 0.05$) (Fig. 2). However, there were significant differences between pre-treatment (15.1 ± 7.5), post-treatment (20.2 ± 6.4) and 45 minutes post-treatment (16.0 ± 6.7) for the ROM of ankle dorsiflexion ($p < 0.05$) (Fig. 3).

2. Motor Neuron Excitability

There were significant differences in the H/M ratios between pre-treatment ($42.8\% \pm 22.3\%$) and post-treatment ($29.2\% \pm 17.3\%$) ($p < 0.05$). The 45-minute post-treatment

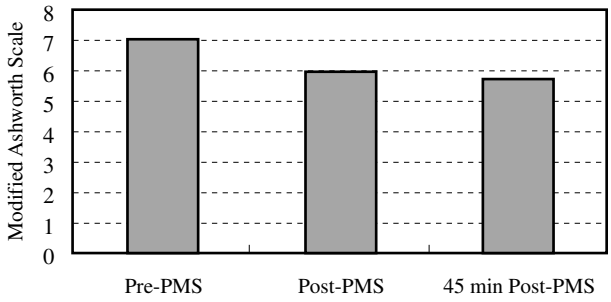

Fig. 2. Means of a modified Ashworth scale before, after, and 45 min after prolonged muscle stretch. The means before, after, and 45 min after prolonged muscle stretch were not significant ($p > 0.05$).

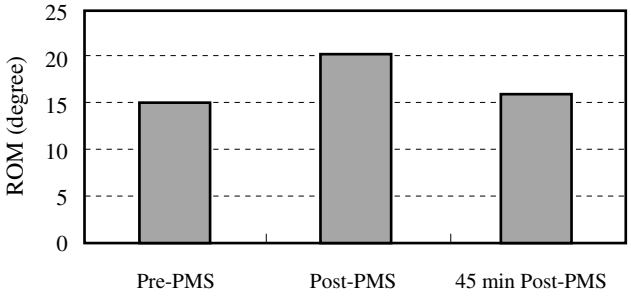

Fig. 3. Means of the ROM before, after, and 45 min after prolonged muscle stretch. The ROM after and 45 min after prolonged muscle stretch did not incredse significantly as compared to that pre-PMS ($p > 0.05$).

H/M ratio (28.5% ± 20.7%) also decreased significantly compared to the pre-treatment ratio ($p < 0.05$). However, the H/M ratios of post-treatment and 45 minutes post-treatment showed no significant difference ($p > 0.05$) (Fig. 4).

The F/M ratios of TA between pre-treatment (5.4% ± 3.3%) and post-treatment (11.8% ± 8.6%) were significantly different ($p < 0.05$). The F/M ratio 45 minutes post-treatment also increased significantly as compared to the pre-treatment ratio ($p < 0.05$). There was no significant difference between the F/M ratios post-treatment and 45 minutes post- treatment ($p > 0.05$) (Fig. 5).

3. Correlation Coefficient

The H/M ratio of the TS and the ROM of ankle dorsiflexion exhibited no significant correlation coefficient ($r = –0.414$, $p = 0.098$). There was no significant correlation coefficient between the H/M ratio of the soleus muscle and the muscle tone of the TS ($r = 0.098$, $p = 0.708$). In addition, the muscle tone of the TS and the ROM of dorsiflexion exhibited no significant correlation coefficient ($r = 0.286$, $p = 0.266$).

IV. Discussions and Conclusions

1. Changes of ROM and Muscle Tone

There was a significant increase in the ROM of ankle dorsiflexion after 30 minutes of prolonged muscle stretching. The reason may be that the motoneuron excitability of the TS decreased after a prolonged muscle stretch. Based on the modified Ashworth scale, there was no significant difference in muscle tone after 30 minutes of PMS. Although this scale is of ten used in clinics, the present study revealed that it could not reflect neurophysiological changes in a spastic muscle. This would be that the results of MAS included the changes of neurophysiology and biomechanics (muscle property) and the subjective expressions of therapists in clinics. A goal of further PMS studies could be to identify changes in neurological and biomechanical effects on spastic muscle, respectively.

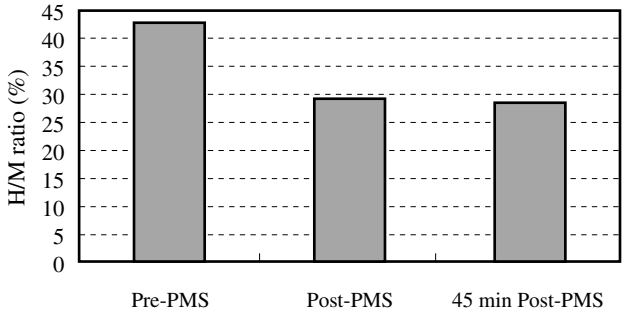

Fig. 4. Means of the H/M ratios of soleus muscle before, after, and 45 min after prolonged muscle stretch. The H/M ratios after and 45 min after prolonged muscle stretch decreased significantly as compared to that pre-PMS ($p > 0.05$).

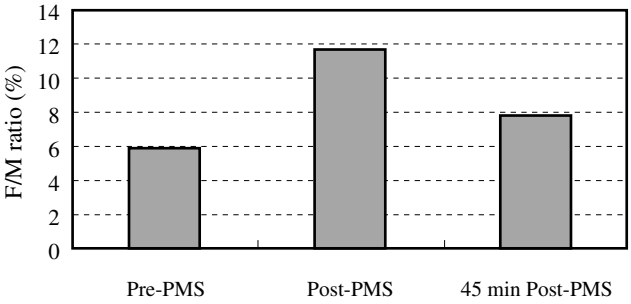

Fig. 5. Means of the F/M ratios of TA before, after, and 45 min after prolonged muscle stretch. The F/M ratios after and 45 min after prolonged muscle stretch were increased significantly as compared to the pre-PMS ($p > 0.05$).

2. Effects on Motor Neuron Excitability of the TS

Odeen and Knutsson (1981) recorded the resistance to passive movements of the ankle joint in paraparetic patients. The subjects stood on a tilt-table with ankle dorsiflexed, with the ankle plantarflexed, or with the ankle braced in maximal dorsiflexion for 30 minutes. They found these three procedures all resulted in a reduction in muscle tone. The largest reductions were obtained with stretch imposed upon the calf muscles. In the same year (Odeen, 1981), they also found that the active and passive ROM of hip abduction increased when the patient underwent a long-term muscle stretch of the hip adductor, and that the EMG activity of the hip adductor decreased when the hip was abducted.

Tremblay *et al*. (1990) recorded torque value and EMG activity while the ankle joint was passively or actively dorsiflexed or plantarflexed after a prolonged muscle stretch. The torque value and EMG activity of passive dorsiflexion and plantarflexion decreased significantly after treatment.

The characteristics of spasticity could be related to changes in neurophysiology and muscle properties. Most previous PMS studies (Odeen and Knutsson, 1981; Odeen, 1981; Tremblay *et al*., 1990) used EMG activity, active/passive ROM or resistance of the joint to evaluate the effect of PMS on

spastic muscle. These results usually included the effects of neurological and biomechanical factors, and were affected by the connecting tissue around the joints. It was, therefore, difficult to identify changes in neurological and biomechanical effects on spastic muscle. In the present study, we used the H/M ratio to measure changes in motor neuron excitability, which made changes in the mechanism of spasticity more clear from the neurological standpoint.

Kunkel (1993) found that "standing" in a frame did not alter the latency and amplitude of the H-reflex during a 6-month study protocol in which a "standing" program of 45 minutes twice daily was employed. However, the treatment time for that study was longer than that of other studies (Odeen and Knutsson, 1981; Odeen, 1981; Tremblay *et al*., 1990). This result differed from those of other studies, possibly due to an insufficient angle of ankle dorsiflexion in Kunkel's study.

Richards *et al*. (1991) recorded the EMG activities of the calf muscle and the tibialis anterior during walking. The subjects underwent 30 minutes of standing on a tilt-table. The results indicated that the EMG activities before and after 30 minutes of standing during walking showed no significant differences. However, they were evaluated under dynamic conditions, whereas the other studies conducted evaluation under static conditions.

In past studies (Odeen and Knutsson, 1981; Tremblay *et al*., 1990), an obvious decrease of spasticity was observed after a single session of treatment. Tremblay *et al*. (1990) recorded EMG activities after 25 and 35 minutes of treatment. They found that the inhibitory effects lasted for up to 35 minutes after cessation of a prolonged muscle stretch. In our study, we found that these effects could last for at least 45 minutes after treatment. The H/M ratios 45 minutes post-treatment and those immediately post-treatment showed no significant difference ($p < 0.05$). This result indicated that the motor neuron excitability at the end of treatment was on a level with that 45 minutes post-treatment.

Why could the H-reflex be changed by PMS? The mechanisms involved conld be as follows (Odeen and Knutsson, 1981; Odeen, 1981; Tremblay *et al*., 1990; Richards *et al*., 1991). (1) The Ib afferent fiber: in this case, the Golgi tendon organ would be fired while the calf muscle is stretched. Then the impulse would be transmitted by the Ib afferent fiber through the interneuron thus, inhibiting the αMN. (2) Another possibility is the II afferent fiber: in this case, the muscle spindle of the calf muscle would be fired while the muscle is stretched. The impulse would be transmitted by the II afferent fiber through the spinal cord, thus, inhibiting the neuron excitability of αMN. Further experiments are needed to establish the possible mechanism responsible for the PMS induced inhibitory effects.

3. Effects on Motor Neuron Excitability of the TA

In the present study, we found that the F/M ratios of the TA increased significantly while the H/M ratios decreased. Richards *et al*. (1991) found that the mean stance and swing phase EMG activation level of the TA was lower in the post-tests, but that there was no significant difference. The study by Trembaly *et al*. (1990) showed that the EMG response of passive movement at 30°/s and at 60°/s of the TA decreased significantly. However, their evaluation methods were different from ours.

4. Correlations of Motoneuron Excitability, Passive ROM of Ankle Dorsiflexion and Muscle Tone

Levin and Hui-Chan (1993) found that there was no significant correlation between the H/M ratio of the TS and the muscle tone based on evaluation performed using a modified Ashworth scale. Comparing the H/M ratio of the TS with the passive ROM of ankle dorsiflexion, no significant correlation was found in the present study. Neither was significant correlation found between the H/M ratio of the TS and the muscle tone. We believe that the H/M ratio is a neurophysiological expression, but the others (passive ROM and muscle tone) may be influenced by the elasticity of the muscle (Richards *et al*., 1991).

PMS can improve the ROM of the ankle joint and the motor neuron excitabilities of TS and TA. This is a safe and economical method for treating stroke patients. However, this treatment standing for a long time and may not be suitable for all patients. We suggest that further studies showld develop a spasticity measurement system, which could not only be used as a PMS treatment device in the semi-supine position, but also could be used to quantify the analysis of changes of spastic hypertonia.

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