

EFFECTS OF PROLONGED MUSCLE STRETCH ON REFLEX AND VOLUNTARY MUSCLE ACTIVATIONS IN CHILDREN WITH SPASTIC CEREBRAL PALSY

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ABSTRACT. We studied the short term effects of a single session of prolonged muscle stretch (PMS) on reflex and voluntary muscle activations in 22 children with spastic cerebral palsy (CP) assigned to an experimental ($n=12$) and a control group ($n=10$). Children of the experimental group underwent PMS of the triceps surae (TS) by standing with the feet dorsiflexed on a tilt-table for 30 min, whereas children of the control group were kept at rest. The effects were determined by measuring the associated changes in torque and in electromyographic (EMG) activity of the TS and tibialis anterior (TA) muscles during both passive ankle movements and maximal static voluntary contractions. The results indicate that PMS led to reduced spasticity in ankle muscles as demonstrated by the significant reductions ($p < 0.05$) of the neuromuscular responses (torque and EMG) to passive movement. These inhibitory effects lasted up to 35 min after cessation of PMS. In addition, the capacity to voluntarily activate the plantar flexors was significantly ($p < 0.05$) increased post-PMS, but the capacity to activate the dorsiflexors was apparently not affected. These findings suggest that repeated sessions of PMS may have beneficial effects in the management of spasticity in children with CP.

Key words: cerebral palsy, spasticity, stretch reflex, exercise therapy, electromyography, muscle contraction, ankle muscle.

The control of spasticity is often a significant problem in the management of children with cerebral palsy (CP). For instance, a variety of muscle relaxants have been tried over the years but their usefulness has been limited because of undesirable side effects (6). Chronic cerebellar stimulation has also been used to reduce spasticity and improve voluntary control in patients with CP (5) but this procedure has led to conflicting results and remains highly controversial (25). Passive stretching techniques have also been used for many years in the management of spasticity in children with

CP. For instance, the use of slow prolonged and sustained stretch of hypertonic muscles has been advocated for inhibition of hypertonic muscles (2, 13) and when applied over a longer period for the reduction of muscle contractures (24, 27, 28).

In children with CP, the effects of prolonged muscle stretch (PMS) have been studied mainly by means of long term applications of ankle plaster casts. Such cast applications have been shown to increase passive range of motion (28, 29), to improve spatio-temporal characteristics during gait (1, 29), and to reduce muscle tone (9, 26). Although these studies suggest that some improvement in motor function can be obtained with cast applications, they do not describe underlying changes in reflex and voluntary muscle activations in the muscle submitted to prolonged stretch. On the other hand, Odéen & Knutsson (21) have reported prolonged stretch of triceps surae, while lying or weight-bearing on a tilt table for 30 min, to lead to significantly reduced resistance to passive ankle movements in adult paraparetic patients. In a second study, Odéen (20) reported that mechanical stretch of spastic adductor muscles led to increased passive and active hip motion and less antagonist muscle coactivation during active hip abduction. These studies suggest that PMS can reduce spasticity and subsequently improve antagonist function during voluntary contraction in patients with spastic paresis.

In the present study, we used an approach similar to that used by Odéen & Knutsson (21) to apply PMS in paraparetic patients, to study the effects of PMS in a group of children with spastic CP. Our purpose was to evaluate the short term effects of a single session of PMS on reflex and voluntary muscle activations in children with spastic CP. The research hypothesis was that PMS would significantly reduce passive restraint and reflex activation of the ankle plantar flexors

Table I. Characteristics of children in the experimental and control groups

Group	Age (years)	Height (cm)	Weight (kg)	Diagnosis ^a
Experimental (n=12)				
Mean	7.0	111.7	20.9	
SD	2.6	19.4	6.2	8 D, 2 T, 2 H
Range	3-11	63-138	12-38	
Control (n=10)				
Mean	5.9	110.3	20.3	
SD	2.4	19.4	6.3	5 D, 5 H
Range	3-9	85-127	12-28	

^a D: diplegia, H: hemiplegia, T: tetraplegia.

(muscle group submitted to PMS) as well as significantly improve capacity to voluntarily activate the antagonistic muscle group (TA). A preliminary account of this study has been presented at the Xth World Confederation for Physical Therapy congress (17).

METHODS

Subjects

Twenty-two children with CP participated in the study. These children were recruited from the population of a paediatric rehabilitation center in Quebec City. Children were selected using the following criteria: (a) age (between 3 and 14 years); (b) a medical diagnosis of spastic CP including diplegia, tetraplegia or hemiplegia; (c) no surgical procedure to the triceps surae; (d) no fixed deformities of the ankle joint; and (e) ability to cope with the protocol requirements. Informed consent was obtained from the parents before the child was included in the study. The children were then assigned to an experimental (EXP) group or a control (CTL) group according to diagnosis and disability. Although these assignments were made randomly the absence of stratification for the plegia is responsible for a larger number of hemiplegics in the CTL group. This fact was taken into account in the statistical analysis. Characteristics of children in the EXP and CTL groups are given in Table I.

Procedures

1. Pre-test evaluations

1.1. *Passive movements.* Neuromuscular responses to passive movements were measured with the child sitting on a specially designed chair with the hip and knee flexed at 100 and 90 degrees respectively. The right foot was attached to a footplate and the ankle axis was aligned with the rotational axis of a Kin-Com dynamometer (Chattecx Corporation, Chattanooga, TN 37405, USA). Surface electrodes were placed on specific locations over the tibialis anterior (TA) and triceps surae (TS) muscles. The electromyographic activity (EMG) was first amplified and recorded as raw EMG, and then rectified, time averaged (time constant 20 ms) and recorded on a Grass model 7D polygraph and concomitantly with a PDP-11/23 PLUS DIGITAL (Digital Equipment Cor-

poration Maynard, MA 01754, USA) computer. The child was instructed to relax and neuromuscular responses (torque and EMG) were measured during five successive movements imposed by the dynamometer at 30°/s, 60°/s and 120°/s. For each movement cycle, the ankle was displaced from 35 degrees of plantar flexion to -5 degrees of dorsiflexion and back to plantar flexion, with a one second interval between each change of direction. Torque, ankle angle and EMG signals were simultaneously recorded on the polygraph and the computer for subsequent analysis.

1.2. *Voluntary contractions.* After completion of the passive movements, torque and EMG activity were recorded during static voluntary contractions against the dynamometer with the ankle in 10 degrees of plantar flexion. For plantar flexion (PF), the child was instructed to push as hard as possible on the footplate and for dorsiflexion (DF) to raise the foot. Three PF and DF attempts were recorded with each voluntary contraction lasting about 3 s and interspersed by 5 s rest periods.

2. Prolonged muscle stretch (PMS)

Children of the EXP group underwent prolonged muscle stretch (PMS) of the triceps surae muscle by standing with the feet dorsiflexed on a modified tilt table for 30 min. The tilt table was equipped with a plexiglass table to secure the child as well as to allow reading and other activities during the PMS session. Children of the CTL group were placed in a sitting position for an equivalent period and engaged in similar activities.

3. Post-test evaluations

Following the PMS session or the rest period, measures of torque and EMG activity were repeated for passive movements and for static contractions in both groups. The post-test evaluation began within five minutes after the end of the respective procedure applied in each group. In addition, to determine the duration of the effects of PMS, in some children passive movement tests at 60°/s were repeated 25 (n=18) and 35 (n=14) min after the end of the PMS session or the rest period (60P2 and 60P3).

4. Data analysis

4.1. *Torque and EMG-angles curves.* Analysis of the torque and EMG values measured during the passive movements

Table II. Velocity sensitivity ratios of the triceps surae (TS) and the tibialis anterior (TA) during passive movements

Velocity sensitivity ratios were calculated as: total EMG area evoked at 120°/s ($\mu\text{V}\cdot\text{s}$)/total EMG area evoked at 30°/s ($\mu\text{V}\cdot\text{s}$). DF: dorsiflexion, PF: plantar flexion

Group ^a	TS		TA	
	DF	PF	DF	PF
Experimental				
Mean	2.26	1.81	2.14	3.04
SD	1.11	0.83	1.27	2.27
Control				
Mean	1.86	1.43	1.38	1.73
SD	0.49	0.53	0.54	1.15

^a $n=10$ for each group.

was performed in three steps. The first step consisted of a visual inspection of each polygraphic record to choose movement cycles free of signal artifacts or influenced by the child's behavior (these were easily identified as irregularities in the traces). In the second step, three representative movement cycles were selected for averaging. Finally, values of torque and EMG activity were displayed graphically as mean torque-angle and EMG-angle curves for each movement direction and each velocity. These curves were used to analyze individual responses.

4.2. *Work total calculation.* To determine changes in the resistance to passive movement, the total energy (W_{tot}) required to displace the ankle was computed for each movement velocity. The W_{tot} was obtained by integrating the resistive torque through a 40 degree DF-PF movement cycle. The W_{tot} reflects the energy absorbed by both the TS and TA during passive movements and has been reported to be a good measure to determine changes in the active reflex component of muscle tone (14). W_{tot} was chosen as the dependent variable when comparing changes in resistance to passive movements both within and between groups.

4.3. *Changes in EMG responses.* To avoid direct comparison between absolute EMG levels, EMG ratios were devised to permit comparison between muscles and between subjects. The ratios were: 1) a velocity sensitivity ratio, which compares the EMG (area under the EMG-angle curve) elicited during passive movements at 120°/s with that elicited at 30°/s, for the same movement direction (DF or PF); and 2) a post-test/pre-test ratio, which compares the EMG (area under the EMG-angle curve) elicited in post-tests with that elicited in pre-tests for the same movement direction and the same velocity. The velocity sensitivity ratio measured the sensitivity of the EMG responses to increased movement velocity while the post-test/pre-test ratio determined the magnitude of change in the EMG responses elicited by the passive movements after the EXP or CTL procedure.

4.4 *Voluntary contractions.* To determine changes in static strength and muscle activations, the best of three attempted contractions was selected on the basis of the torque produced.

The amplitude of the torque and EMG activity, sampled at 100 Hz by the computer, were then averaged over a one second period for each subject to yield mean torque and EMG values. EMG values were then transformed into post-test/pre-test ratios for comparison between groups while the mean torque values were considered the representative voluntary torque capacity value.

4.5. *Statistical analysis.* Non parametric tests were used to compare the changes in the values for the different variables. The Mann-Whitney U-test was used to compare differences between the groups while the Spearman rank order correlation test evaluated the relationships between different variables. The level of significance was set at $p<0.05$ for all tests performed.

RESULTS

1. General responses to passive movements

All but 1 of the 22 children were able to relax their leg muscles during passive dorsiflexions (DF) and plantar flexions (PF). In a second child, part of the data was discarded because technical problems invalidated measurements at 30°/s. Thus, the final analysis included the data from 21 children, 11 in EXP group and 10 in CTL group. Analysis of individual torque- and EMG-angle curves revealed two characteristic patterns in the responses to the passive movements. First, the responses (EMG and torque) increased with increasing movement velocity. This velocity sensitivity was present in one or two muscles in most children. As shown in Table II, the velocity sensitivity ratios in children of both groups were highest in the TS and the TA during passive lengthening (DF and PF respectively). Such velocity sensitive responses suggest that spasticity is present not only in the TS, as expected, but also in the TA. Secondly, the presence of concomitant activation in the shortening muscles was revealed. Moreover, as demonstrated by the velocity sensitivity ratios (higher than one), the concomitant response in the shortening muscles were also velocity sensitive. Although the mean value of the velocity sensitivity ratios for all four conditions (DF: lengthening of the TS and shortening of the TA; PF: lengthening of the TA and shortening of the TS) was generally lower in the CTL group, this difference was not significant ($p>0.05$, U-test).

2. Individual responses to PMS

Although PMS led to reduced neuromuscular responses to passive movements in most children, there were variations in the way the muscle activation patterns were modified. Examples of such modifications are given in Fig. 1 during high velocity passive move-

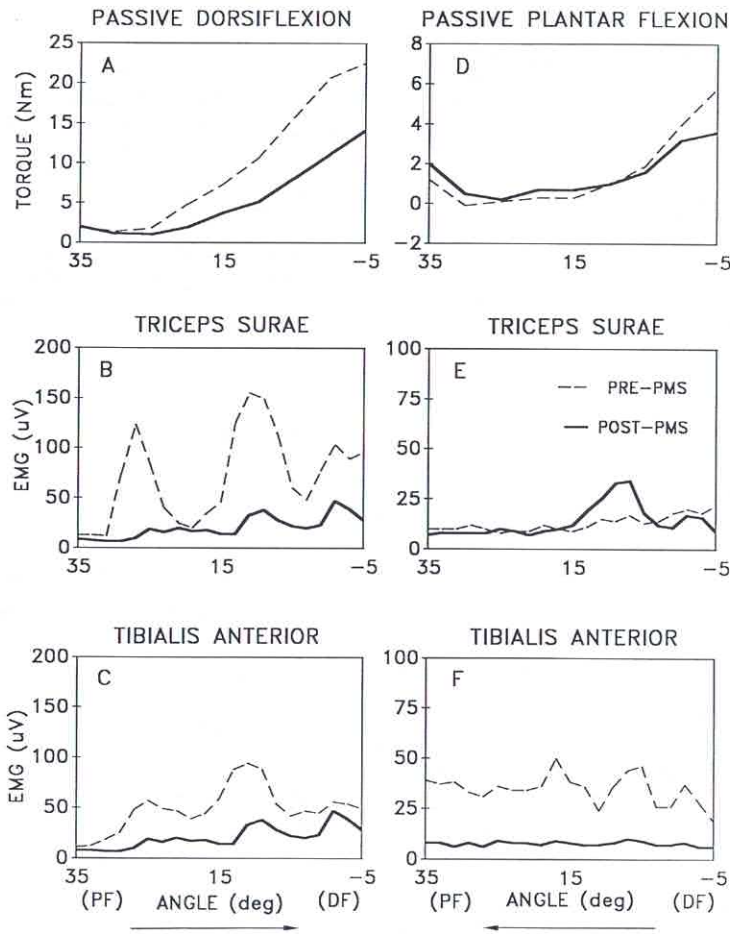


Fig. 1. Torque (Nm) and EMG (μV) responses obtained pre- (---) and post-PMS (—) during passive dorsiflexion (A, B and C) and passive plantar flexion (D, E, and F) at $120^\circ/\text{s}$ in a diplegic child of EXP group. Note the decreases in torque and EMG responses post-test in both movement directions.

ments. As can be seen in this figure, the resistive torque (Fig. 1 A) and the EMG in both the TA and TS (Figs. 1 B and 1 C) during passive DF at $120^\circ/\text{s}$ decreased after PMS. The torque also decreased, but less during passive PF (Fig. 1 D), and activations of the TS and TA (Figs. 1 E and 1 F) also decreased. Such reductions of both torque and EMG responses were seen in 4 of the 11 children following PMS. In the seven other children, the effects of PMS were less clear cut. For instance, a slight torque reduction could be combined to decreased activations in either the TS or TA, whereas in one child no changes occurred after PMS. Individual responses to PMS are illustrated in Fig. 2. In this figure, changes in W_{tot} and EMG responses to passive movement at $60^\circ/\text{s}$ are given for each subject of both groups. As can be seen (Fig. 2 A), the W_{tot} decreased in most children in the EXP group but the amplitude of change varied within the group. In most subjects, the changes in TS and TA activa-

tions (Figs. 2 B and 2 C) tend to be similar in terms of amplitude and direction. This tendency was confirmed ($p < 0.05$, Spearman rank order correlation test) by correlating concomitant EMG change scores (post/pre ratio) in the TS and TA activation elicited by passive dorsiflexion ($r = 0.50$) and plantar flexion ($r = 0.62$) at $60^\circ/\text{s}$. Significant relationships ($p < 0.05$) between TS and TA activation changes were also found at $120^\circ/\text{s}$ (DF: $r = 0.86$; PF: $r = 0.66$) but not at $30^\circ/\text{s}$ (DF: $r = 0.48$; PF: $r = 0.43$). The existence of such correlations indicates that a decreased response in one muscle was usually accompanied by a concomitant reduction in its antagonist during passive movements at $60^\circ/\text{s}$ and $120^\circ/\text{s}$.

The individual changes in neuromuscular responses measured in the CTL group are given for comparison in Figs. 2 D, 2 E and 2 F. It can be seen that, while the W_{tot} is almost unchanged in three children after the rest period, the W_{tot} decreased

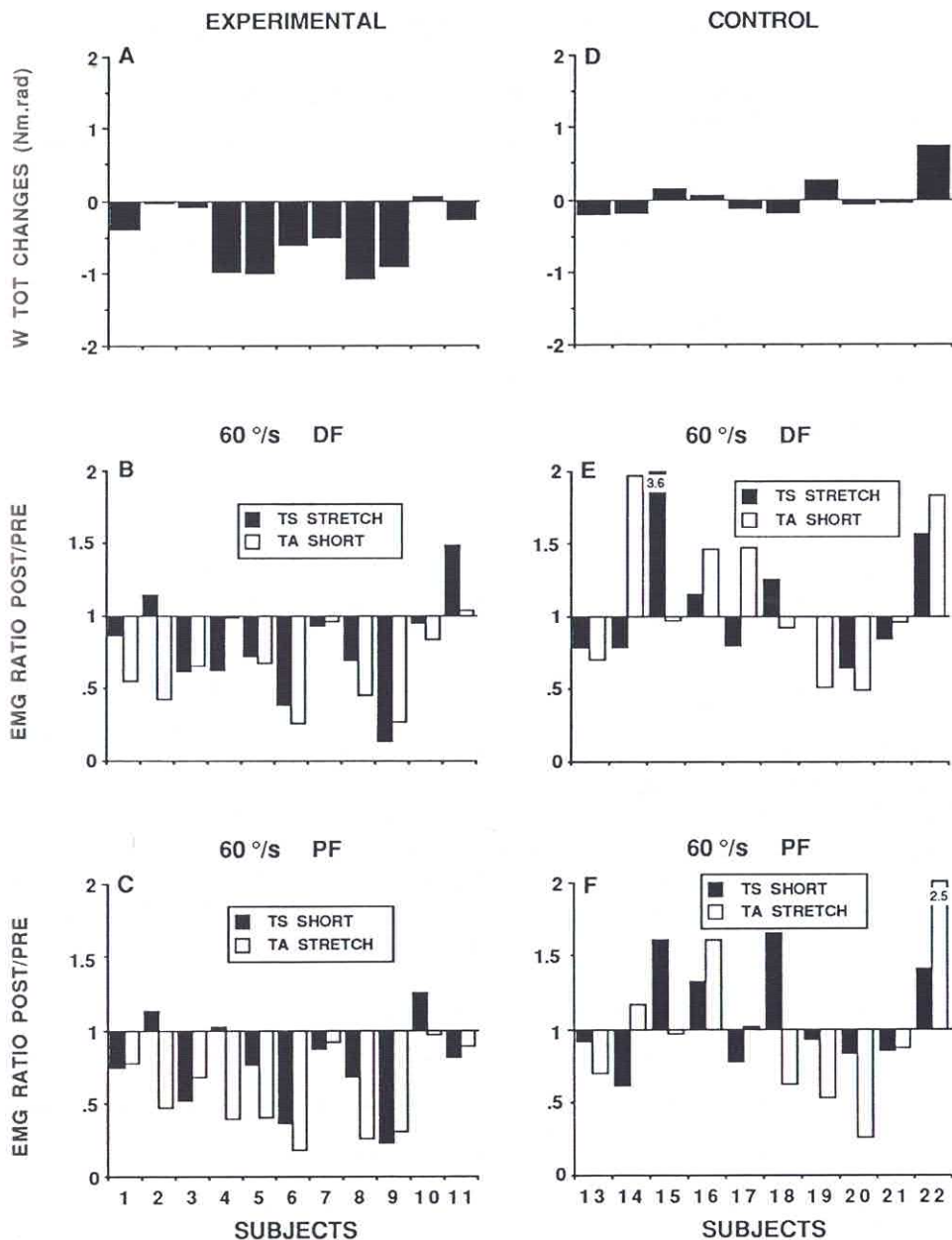


Fig. 2. Comparison of the individual changes in the neuromuscular responses to passive movements at 60°/s for both groups. Bars in A and D represent differences between pre- and post-test values for the total energy (W_{tot}). Bars in B, C, E and F represent the concomitant changes in EMG, calculated

as post/pre ratios of EMG responses in the triceps surae (TS) (dark columns) and tibialis anterior (TA) (open columns) during passive dorsiflexion (DF) and plantar flexion (PF) respectively.

slightly in 4 children and increased in 3 others (Fig. 2D). Such interindividual variations are also found in the EMG ratios illustrated in Figs. 2E and 2F. Moreover, the concomitant changes in TS and

TA activations are often in opposite directions for both passive DF (Fig. 2E) and PF (Fig. 2F), which is in contrast with the changes observed in the EXP group. Indeed, the concomitant changes in TS and

Table III. Changes of the total energy (W_{tot}) in NM·rad required to displace the ankle during passive movements at different velocities

Changes are given as mean (± 1 SD) differences between pre-test and post-test values obtained immediately after prolonged muscle stretch and 25 (60P2) and 35 min later (60P3)

Velocity (°/s)	Experimental			Control		
	n	Mean	SD	n	Mean	SD
30	10	-0.29*	0.38	10	-0.01	0.21
60	11	-0.51**	0.43	10	+0.05	0.29
120	11	-0.67*	0.84	10	-0.12	0.58
60P2	9	-0.68*	0.71	9	-0.01	0.24
60P3	8	-0.82*	0.73	6	-0.17	0.31

* $p < 0.05$, ** $p < 0.01$, Mann-Whitney U-test.

TA were not correlated at any movement velocity tested. This lack of correlation reflected the large response variations observed within the group.

3. Group responses

Changes in resistive torque as measured by W_{tot} values are given in Table III. Pre-test values were not statistically different between groups at all three movement velocities. Immediately after PMS, the W_{tot} was reduced ($p < 0.05$, U-test) in the EXP group as compared to the CTL group for all movement velocities, the most significant effect ($p < 0.01$) being at 60°/s (Table III). Significant reductions were still

present 25 and 35 min later (Table III). Associated changes in EMG responses of the TS and TA during passive movements are given as mean EMG ratios in Table IV. As seen in this table, the EMG ratio values were generally smaller in the EXP group for all movement conditions. Comparisons between groups indicated that EMG responses in the TS and TA during passive lengthening were reduced ($p < 0.05$) at 30°/s and 60°/s, but not at 120°/s (Table IV). Moreover, both muscles exhibited a reduced EMG response ($p < 0.05$) during passive shortening in most conditions (Table IV). Finally, the post-test evaluations (60P2 and 60P3) showed that lower EMG responses

Table IV. Changes in EMG responses to passive movements at different velocities

Changes are given as mean (± 1 SD) EMG post/pre ratios: total EMG area evoked post-test ($\mu V \cdot s$)/total EMG area evoked pre-test ($\mu V \cdot s$). Abbreviations as in Table II, number of subjects for each velocity as in Table III

Velocity (°/s)	Experimental				Control			
	TS		TA		TS		TA	
	DF	PF	DF	PF	DF	PF	DF	PF
30	0.74*	0.79	0.59*	0.59*	0.97	0.99	0.92	1.03
	(0.21)	(0.22)	(0.22)	(0.21)	(0.13)	(0.13)	(0.32)	(0.41)
60	0.78*	0.68*	0.65*	0.57*	1.25	1.09	1.13	1.03
	(0.35)	(0.30)	(0.27)	(0.26)	(0.83)	(0.35)	(0.53)	(0.61)
120	0.77	0.65*	0.54*	0.55	0.92	0.91	0.89	1.08
	(0.31)	(0.32)	(0.20)	(0.39)	(0.17)	(0.13)	(0.37)	(0.91)
60P2	0.69*	0.58*	0.60	0.58	1.11	1.00	0.99	0.79
	(0.53)	(0.19)	(0.26)	(0.21)	(0.47)	(0.43)	(0.59)	(0.50)
60P3	0.66*	0.57*	0.64	0.62	1.04	0.90	0.62	0.57
	(0.56)	(0.33)	(0.49)	(0.39)	(0.41)	(0.24)	(0.21)	(0.24)

* $p < 0.05$, Mann-Whitney U-test.

Table V. Changes in torque (Nm) and EMG activation during static voluntary contractions

Changes in torque are given as mean (± 1 SD) differences between pre-test and post-test values. Changes in EMG activation are given as mean EMG post/pre ratios: mean EMG activity produced post-test ($\mu\text{V}\cdot\text{s}$)/mean EMG activity produced pre-test ($\mu\text{V}\cdot\text{s}$). Abbreviations as in Table II

	Experimental			Control		
	<i>n</i>	Mean	SD	<i>n</i>	Mean	SD
<i>Plantar flexion</i>						
Torque	8	+2.57	3.51	9	+0.40	2.13
EMG, TS	8	1.67*	0.59	9	0.96	0.25
<i>Dorsiflexion</i>						
Torque	9	-0.07	0.65	7	+0.29	0.66
EMG, TA	9	1.42	0.67	7	1.07	0.27

* $p < 0.05$, Mann-Whitney U-test.

persisted up to 35 min in both TS and TA, but were significantly lower ($p < 0.05$) only in the TS (Table IV).

4. Voluntary contractions

Most children were able to voluntarily activate their plantar flexor ($n=17$) and dorsiflexor ($n=16$) muscles to produce torque during static contractions. The ability to produce plantar flexion and dorsiflexion torque in pre-tests was comparable for the children of both groups. Table V gives the changes in torque and EMG activation values obtained in both groups. Following the PMS session, 7 of the 8 children of EXP group had increased torque capacity during PF which was related to significantly ($p < 0.05$) increased activation of the TS as compared to children in the CTL group (Table V). It is worth noting that only in children with improved TS voluntary activation, were EMG responses in TS reduced during passive DF for all movement velocities. During static DF, although 4 children showed an increased torque capacity, the other 5 demonstrated a decreased capacity and changes were comparable to those observed in CTL group. In the CTL group, torque and EMG activations remained relatively unaltered after the rest period in most cases, both for static DF and PF (Table V) contractions.

DISCUSSION

This study clearly shows that a single 30 min session of PMS can reduce spasticity in children with CP. This finding is consistent with the PMS-induced in-

hibitory effects reported in spastic paraparetic patients by Odéen & Knutsson (21). In the present study, the duration of the PMS-induced inhibition was shown to last at least 35 min. These short-term effects, however, may have been of a longer duration since PMS-induced inhibition lasting up to four hours has been reported in adult paraparetic patients (21). Although statistically significant for passive movements at 30°/s and 60°/s, the lowered EMG responses of both the TS and TA were not significant during passive lengthening at 120°/s. Such a finding may either indicate that PMS-induced inhibition was less effective at a higher velocity or that the effects in the EXP group were too small in comparison to those in the CTL group. The latter possibility is more likely given the fact that reductions of EMG responses at 120°/s were even larger than those at 60°/s (Table IV). On the other hand, the finding that EMG responses were either slightly reduced or more variable for the TS and the TA respectively in children of CTL group suggests that inhibitory effects were not strong enough given the variability of EMG responses at 120°/s. The latter observation underlines the importance of control values when studying the specific effects of a therapeutic procedure in spastic patients. Whether repeated PMS sessions may have more extensive inhibitory effects cannot be excluded given the cumulative effects reported in spastic adult patients submitted to repeated PMS applications (19, 20).

What is the basis of the PMS-induced inhibition? As revealed by the decreased EMG activations during passive movement, these reductions are more likely

related to changes in the active reflex component of muscle tone than to the passive visco-elastic component. Modifications in the visco-elastic muscle properties usually require repeated sessions of stretching over an extended period of time (15, 23) and therefore, their contribution might have been negligible. Another finding which also supports the contribution of an underlying change in the reflex component is that reductions were also found in the antagonistic muscle group (TA) not submitted to PMS.

Some suggestions have been made as to possible neural sources of the PMS-induced effects. For instance, reflex inhibition mechanisms mediated by tendon organ receptors or secondary spindle afferents have been advocated to explain reductions of passive restraint in plantarflexors in spastic paraparetic patients (21). These proposed mechanisms are incompatible, however, with the current views on the properties of both tendon organs and spindle secondaries (3). A contribution of non spindle group II, III and IV muscle afferents, or flexor reflex afferents, would be more consistent with a reflex inhibition mechanism in the light of recent experiments. These non-spindle afferents, originating from free nerve endings in muscle, have been shown to be sensitive to stretch and light pressure (22). Moreover, these endings are thought to mediate, at least in part, the clasp-knife inhibition occurring when a spastic extensor muscle is passively stretched beyond a certain length (4). Thus, inhibitory effects resulting from the activation of these afferents by the PMS procedure might be implicated in the reduced reflex responses observed in the plantar flexors (extensors) but they cannot account for the reductions seen in the dorsiflexors since the latter muscles (flexors) might have been facilitated by such activation.

More recently, evidence that changes in the mechanical properties of the intrafusal muscle fibres and in the sensitivity of the receptor terminals are implicated in the modifications of reflex responses following changes in muscle length (either passive or active) have been reported both in animals (30) and humans (11, 12). For instance, Williams (30) showed a significant reduction of the sensitivity of spindle primary endings in rat muscles chronically immobilized in the extended position. In human subjects, stretch receptor sensitivity of finger flexors was found to be reduced following a 5 s passive finger extension and conversely increased after active finger flexion (12). These changes in spindle sensitivity were accompanied by changes in the stretch reflex which varied in

relation to changes in inherent muscle stiffness. Such a mechanism might be related to the lowered EMG responses observed in the plantar flexors held in a lengthened position for 30 min, but again, it could not account for the effects observed in the dorsiflexors kept shortened by the PMS procedure.

In fact, the lower EMG responses which were also found in the dorsiflexor group following PMS could either suggest that sustained muscle stretch in one muscle group induces non specific inhibitory effects or that alterations from the plantar flexor inflow somehow affects the excitability of the motoneurons supplying the dorsiflexor muscles. The latter possibility is more likely, given the evidence of strong spinal excitatory connections between ankle extensor and flexor motoneurons in children with spastic CP (10, 18). Evidence of such reciprocal excitation in ankle antagonistic muscles has also been found in some children evaluated in our laboratory (Tremblay et al., unpublished observations). Thus, if the assumptions about such reciprocal excitatory connections are correct, it would explain why reductions of TS responses (as discussed above) were accompanied by concomitant reductions in TA responses, as indicated by the correlations found during passive movements at 60°/s and 120°/s. On the other hand, the contribution of a non specific inhibition cannot be ruled out since some children exhibited reductions of EMG responses in TA without accompanying changes in TS. Non specific inhibitory effects spreading proximally and distally have been previously reported in spastic patients following reduction of peripheral inflows by blocking nerve fibers in one limb (7). One of the mechanisms underlying such non specific inhibitory effects might be long loop reflexes (8). Further experiments are required for establishing the possible mechanisms underlying the PMS-induced inhibitory effects.

The fact that significant improvement in voluntary muscle activation was found in the plantar flexors but not the dorsiflexors is somewhat in contrast with the finding of Odéen (20) that PMS-induced inhibition leads to better antagonist function. Comparison between our results and those of Odéen is difficult, however, because effects were studied in different muscles and during static rather than dynamic conditions which enhance the effects of antagonist coactivations. According to Knutsson & Mårtensson (16), three basic mechanisms might contribute to motor deficits in spastic paresis under dynamic conditions: prime mover dysfunction, spastic reflexes and antagonist coactivation. The fact that lowered EMG

responses during passive movements were found in all children displaying increased TS activation during static PF may indicate a relationship between the reduction of spastic reflexes and improved volitional control. Thus, it could be hypothesized that reduction of spastic reflexes somehow promotes a better access to the motoneuron pool during voluntary activation. Conversely, because voluntary activations did not improve for the dorsiflexors, it could be postulated that in these muscles, weakness is more likely associated with a deficient activation of the prime movers. Further investigations of the dynamic motor capacity in children with spastic CP are warranted to gain a more thorough understanding of the interactions between PMS-induced inhibition and voluntary activation.

The results of the present study have important clinical implications for the treatment of children with spastic CP. They confirm that PMS can be used to obtain short term reduction of spasticity and that this is associated with improved voluntary muscle activation. A major advantage of PMS over other types of therapy (e.g. drug therapy), is that no undesirable side-effects are produced. In addition, these results indirectly support some of the clinical observations reported in the literature about reduction of muscle tone after manual stretching applications (13) or after casting therapy (1, 29). It remains to be determined whether repeated sessions of PMS can induce long-lasting improvement in motor function in children with CP. The results of the present study suggest that repeated use of PMS over several weeks would have beneficial effects on spasticity and possibly abnormal movement patterns in children with spastic CP.

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