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EFFECTS OF A SINGLE SESSION OF PROLONGED PLANTARFLEXOR STRETCH ON MUSCLE ACTIVATIONS DURING GAIT IN SPASTIC CEREBRAL PALSY

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ABSTRACT. Activations of the triceps surae (TS) and tibialis anterior (TA) muscles during gait were studied in children with spastic cerebral palsy (CP) immediately before and after 30 min of standing on a tilt-table with the ankle dorsiflexed to stretch the TS in the experimental group ($n=8$) or after a rest period in the control group ($n=11$). The EMG activity from the TS and TA was recorded concomitantly with electronic footswitch signals by a computer. Video records were made of the sagittal gait movements. Effects of PMS were determined by comparing change scores for selected spatiotemporal and muscle activation parameters between the groups. The change scores defined for the muscle activations were: a post-test/pre-test ratio of the EMG activity in specific segments of the gait cycle and a locomotor spasticity index. PMS did not significantly ($p>0.05$) affect any of the spatiotemporal parameters nor did it alter any of the TS and most of the TA activation parameters or the SI indexes for the TS and TA. Only the TA post/pre activation ratio for the 0–16% segment of the gait cycle was smaller ($p<0.01$), indicating a decrease in TA activation post-PMS.

Key words: cerebral palsy, spasticity, gait, muscle stretch.

Prolonged muscle stretch (PMS) denotes a maintained stretching of spastic muscles by manual or mechanical means. Therapists make extensive use of inhibiting postures and manual stretching of hypertonic muscles to reduce muscle tone and subsequently encourage active movements (5). The term "inhibitive cast" has been applied to specialized casts of various materials used to maintain the foot, ankle and toes in a position thought to inhibit abnormal spastic reactions when the child is standing or walking. In one of the few quantitative studies, Tardieu et al. (30) reported an increased range of passive ankle movements after three weeks in a cast. As a whole, however, the literature on the effects of such "inhibitive casts" consists of short anecdotal reports (1, 11, 13, 29) or case studies (8, 15, 16, 36) without control groups, in which semi-quantitative evaluation procedures are used.

More recently, Watt et al. (32) on the basis of subjective scores of gait changes from video records reported that 3 weeks of inhibitive casting led to a significant improvement in foot-floor contacts that was maintained up to 2 weeks after cast removal but was lost in retests 5 months later. In another study, Bertoti (4), found stride length measured from footprint records to be significantly longer in a group of CP children after 10 weeks of wearing a short leg cast as compared to a control group. Lastly, in a detailed biomechanical analysis of the use of an ankle-foot orthosis to control knee hyperextension, Simon et al. (27) reported that such braces corrected the knee hyperextension in all but one of 15 children and that in 3 the correction was maintained even after the brace was removed. Semi-quantitative analysis of the muscle activations failed to discern changes in the triceps surae activation with and without the brace although compensatory changes were noted in the quadriceps and gluteus maximus muscles. It could be that the semi-quantitative analysis procedures failed to reveal the amplitude modulations necessary to characterize the changes in EMG. It is also possible that the largely preprogrammed muscle activations during gait are less sensitive to peripheral manipulations.

Odéen & Knutsson (23) showed how prolonged muscle stretch of spastic plantarflexor muscles of adult paraparetic patients could diminish passive restraint. Furthermore, by comparing the effects of plantarflexor stretch in the lying and standing positions with the aid of a tilt table they demonstrated the superiority of stretch when combined to the standing position over stretch alone. These results strongly supported the well known beneficial clinical effects on spasticity in paraparetic patients obtained by standing on a tilt table or with the aid of a special

standing apparatus (for a review see Odéen 21, 22). One can question whether such inhibitive effects can be expected to occur on muscle activations during gait following PMS given the different control mechanisms involved in the genesis of the semiautomatic gait activations as compared to reflex or voluntary activations performed in the sitting or lying position (18).

In the present study the short-term effects of a single session of PMS of the plantarflexors were evaluated on activations of these same plantarflexors and the antagonist dorsiflexors during gait in children with spastic cerebral palsy using a control group design. The spastic plantarflexors were stretched by standing on a tilt table for 30 min with the feet dorsiflexed by an adjustable footplate as described by Knutsson & Odéen (23). In a parallel study these same children were shown to have significantly decreased spastic restraint in both the stretched and shortened muscles by this procedure and increased capacity to voluntarily activate the plantarflexors following this stretching procedure (19, 20, 31). Preliminary findings were reported at the Xth World Confederation for Physical Therapy Congress in Sydney (26).

Table I. *Subject characteristics*

D = diplegia, H = hemiplegia

Case	Age (yrs)	Sex	Height (cm)	Weight (kg)	Diagnosis
Expermental group (n=8)					
1	7	F	117	18	D
2	5	F	112	17	D
3	7	M	123	26	D
4	11	F	115	20	D
5	6	F	119	25	D
6	9	F	130	24	D
7	4	M	104	17	H
8	3	F	90	12	H
Mean ± SD	7 ± 3[a]		114 ± 12	20 ± 5	
Control group (n=11)					
9	4	M	97	14	D
10	8	F	127	28	D
11	5	M	108	19	D
12	13	M	139	29	D
13	5	F	106	19	H
14	8	F	121	23	H
15	3	F	85	12	H
16	9	M	127	28	H
17	4	F	103	16	D
18	9	F	127	27	H
19	4	M	114	23	D
Mean ± SD	7 ± 3		114 ± 16	22 ± 6	

[a] Values give mean ± 1 SD.

METHODS

Subjects

Nineteen (19) children with spastic cerebral palsy were recruited from the population of children treated at the Cardinal Villeneuve Rehabilitation Centre in Quebec City. The children (12 diplegics and 7 hemiplegics), aged 3 to 13 years old, met the following inclusion criteria: absence of surgery to the legs, clinical evidence of spasticity in the plantarflexors, capable of walking 10 M unassisted and mentally capable of participating in the tests. They were divided into an experimental (EXP) and a control group (CTL). These groups are unbalanced, however, because random allocation of the subjects, made for a parallel study (31) that included non-walkers was not made in predetermined blocks stratified for disability. The effects of the unequal number of diplegics and hemiplegics on the results were statistically evaluated. Prior to acceptance into the study, the children were evaluated by a neuropediatrician to confirm the diagnosis and then parental or guardian informed consent was obtained. Subject characteristics are given in Table I.

Procedures

1. *Recording of gait movements and muscle activations.* To record muscle activations, surface electrodes were placed on the triceps surae (TS) and tibialis anterior (TA) muscles of one leg. Movement artifacts were reduced by connecting short electrode leads to miniature preamplifiers which were connected to a battery and an electrode box carried at the waist, and then by means of a 10 m shielded cable to the electrode selector unit of a Grass (Grass Corporation, Quincy, Mass., USA) polygraph. The myosignals were amplified and recorded to check for movement artifacts prior to being rectified, time averaged (time constant 20 ms) and fed to a PDP 11/23 Plus Digital (Digital Equipment Corporation, Maynard, Ma 0174, USA) computer for recording (sampling frequency = 100 Hz) and analysis. The EMG activity was synchronized to the gait cycle by electronic footswitches attached to the heel, midfoot and toe of each shoe. The children were requested to walk along an 8 m walkway at free speed. Sampling of EMG and footswitch signals was automatically started and stopped as the child interrupted beams from photoelectric cells placed 4 m apart. Concomitant video records were made of the sagittal gait movements. Gait records were taken twice: a pre-test just prior to the treatment and a post-test made about 5 min after the treatment. At least 10 gait cycles were recorded for each test.

2. *Experimental and control treatments.* Following completion of the gait pre-tests, the EMG electrodes and footswitches were left in place and the children received either the EXP or CTL treatment for 30 min. Children in the EXP group stood upright with the help of a modified tilt table. Hip and knee positions were controlled by special supports and stretch of the plantarflexors was maintained at a comfortable level by keeping the ankle in maximal dorsiflexion by means

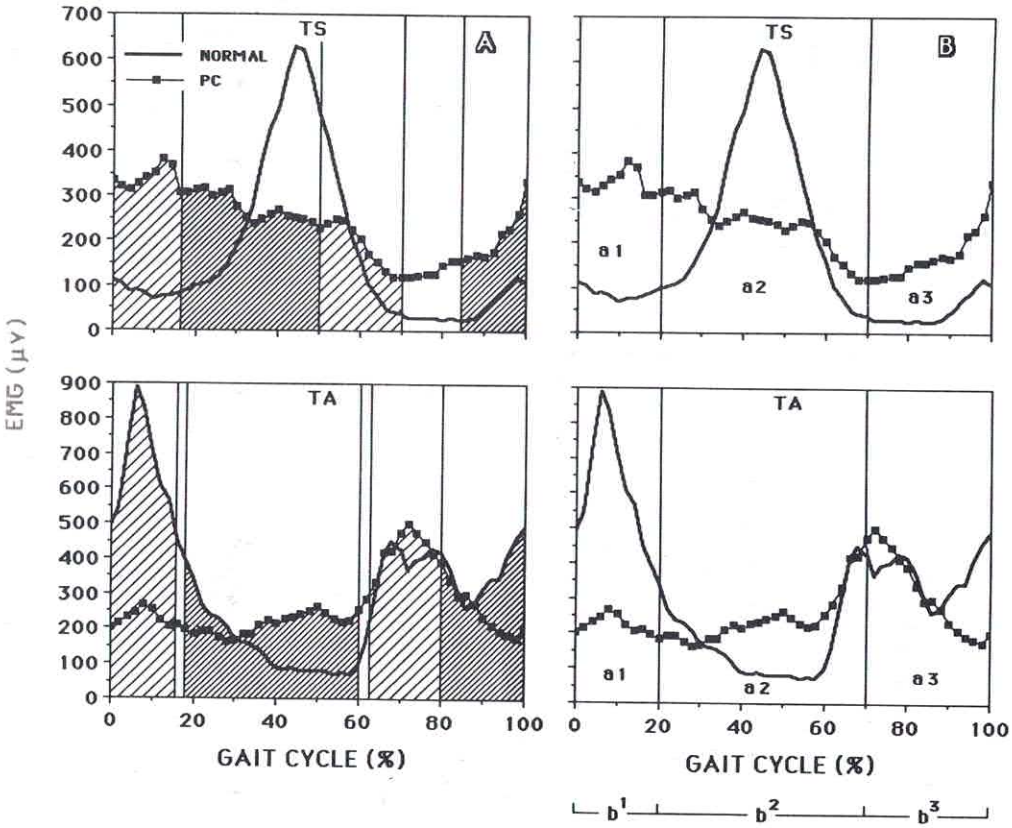

Fig. 1. Comparison of mean activation profiles of the triceps surae (TS) and tibialis anterior (TA) muscles during gait in a 4-year-old diplegic child and a normal child of the same age to illustrate: A) analysis of specific segments of the activation profile and B) subdivision of the activation profile for calculation of the locomotor spasticity index.

of an adjustable footplate. While standing, the children in the EXP group were engaged in educational activities. Between tests, children in the CTL group were seated and also engaged in educational activities.

3. *Analysis.* Temporal parameters were derived from the footswitch signals and computed by the computer program as was the average gait velocity. For each subject, mean muscle activation profiles obtained during the pre- and post-tests were graphically represented as the amplitude of the EMG in μv at each 2% of the gait cycle. Mean activation profiles were then calculated for each group. The gait movement patterns were qualitatively analyzed by visual appraisal of the video records by experienced evaluators. Movement changes were used to help interpret the data but were not considered primary outcome variables.

Treatment effects between the groups were defined by comparing change scores for selected gait parameters. Selected spatiotemporal parameters were: cycle duration, % stance time, average velocity and cadence. The muscle activation profiles were statistically analyzed in two ways. First the activation profiles were divided into 6 segments: 0–16%, 16–50%, 18–60%, 60–70%, 62–80% and 84–100% of the gait cycle (Fig. 1 A). The area under the activation profile for each of these segments became a specific EMG parameter for the statistical analysis. EMG change scores were defined by post/pre EMG ratios for each segment.

Secondly, the spastic locomotor disorder index (SI) proposed by Fung & Barbeau (12) was calculated for the TS and TA (see Fig. 1 B). The gait cycle is divided into two equal parts representing periods when the muscle is activated ("on") and relatively non-activated ("off"); thus for the TS and TA the gait cycle is subdivided into periods b1 (0–20%), b2 (20–70% and b3 (70–100%). The area (a) under the muscle activation profile for each of these periods is calculated to give $a1$, $a2$ and $a3$. The SI is defined as the ratio of the EMG area in the "off" periods to that in the "on" periods. Thus for the TS the SI = $(a1+a3)/a2$ and for the TA the SI = $a2/(a1+a3)$. Fung & Barbeau (12) reported SI values of $0.12+0.04$ ($n=5$) and 0.20 ± 0.05 ($n=5$) for the medial gastrocnemius and TA, respectively, in adult normal subjects. The SI indexes for the activation profiles illustrated in Fig. 1 are 0.99 (spastic) and 0.22 (normal) for the TS and 0.87 (spastic) and 0.37 (normal) for the TA. For the statistical analysis the SI change score was equal to the difference in SI between pre- and post-tests.

Differences between the groups for the spatiotemporal and EMG change scores were statistically evaluated by the Mann-Whitney U test with the significance level set at $p<0.05$.

RESULTS

1. *Spatiotemporal parameters*

Table II gives the spatiotemporal parameters for the pre- and post-tests as well as the change scores. Comparison of the change scores for the different parameters failed to reveal statistically significant ($p > 0.05$) differences.

2. *Gait movements*

Eight of the 12 diplegic children initiated the stance phase with forefoot (midfoot and toe footswitches) contact. Of these, 8 had foot equinus throughout the stance phase accompanied by knee hyperextension in 3 cases and by excessive knee flexion in 3 other cases. Those with knee hyperextension had foot drag in the swing phase. In 3 of the diplegic children the gait dysfunction was less severe; the stance phase was initiated with heel contact and relatively minor abnormalities occurred in the ankle and knee movements throughout the gait cycle. Six of the 7 hemiplegic children initiated the stance phase with forefoot contact while only 1 had heel contact. In the stance phase, knee hyperextension was observed in 3 (accompanied by foot equinus throughout stance in 1 child) and excessive knee flexion during stance in 1, while toe drag during swing occurred in 6 of the hemiplegic children.

Visual inspection of the video records did not reveal systematic changes between the tests although minor changes were noted in some children. Furthermore, it was not possible to define systematic differ-

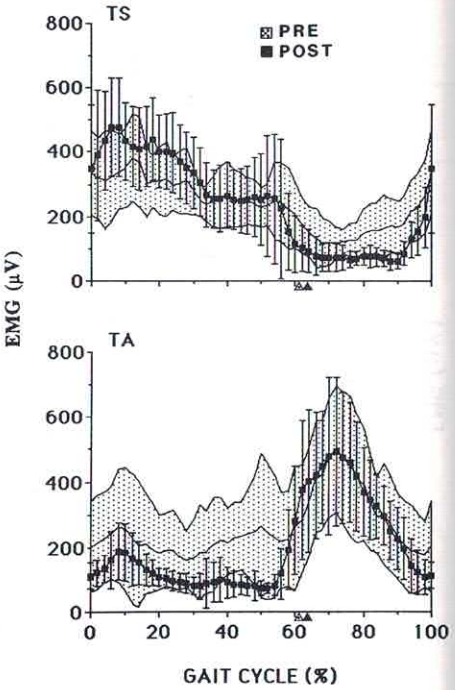

Fig. 2. Comparison of the muscle activation profiles pre (shaded area = ±1 SD, $n = 20$ gait cycles) and post- (square indicate mean and vertical bars ± 1 SD, $n = 20$ gait cycles) 10 min of muscle stretch in a 4-year-old diplegic child. The amplitude (in uv) of the muscle activations in the triceps surae (TS) and tibialis anterior (TA) on the *Y*-axis is given relative to the gait cycle on the *X*-axis. Cadence = 163 and 178 steps/min in pre- and post-tests, respectively. Arrow indicate end of stance phase.

Table II. *Spatiotemporal gait parameters obtained in pre- and post-tests for both groups of CP children*

	Experimental group ($n=8$)			Control group ($n=11$)		
	Pre-test	Post-test	Change score[a]	Pre-test	Post-test	Change score
Stride length (cm)	66[b] (12)	67 (12)	2 (4)	78 (13)	79 (18)	2 (11)
Cycle duration (ms)	1 033 (187)	995 (265)	−39 (113)	1 095 (158)	1 035 (141)	−61 (145)
Cadence (steps/min)	122 (24)	132 (32)	10 (12)	112 (17)	118 (17)	6 (16)
Velocity	65 (9)	72 (12)	7 (8)	77 (15)	77 (16)	5 (17)
% Stance	60 (8)	61 (8)	−1 (8)	61 (5)	62 (5)	1 (2)

[a] Change score: difference between pre- and post-test scores.
[b] Values give mean ± 1 SD.

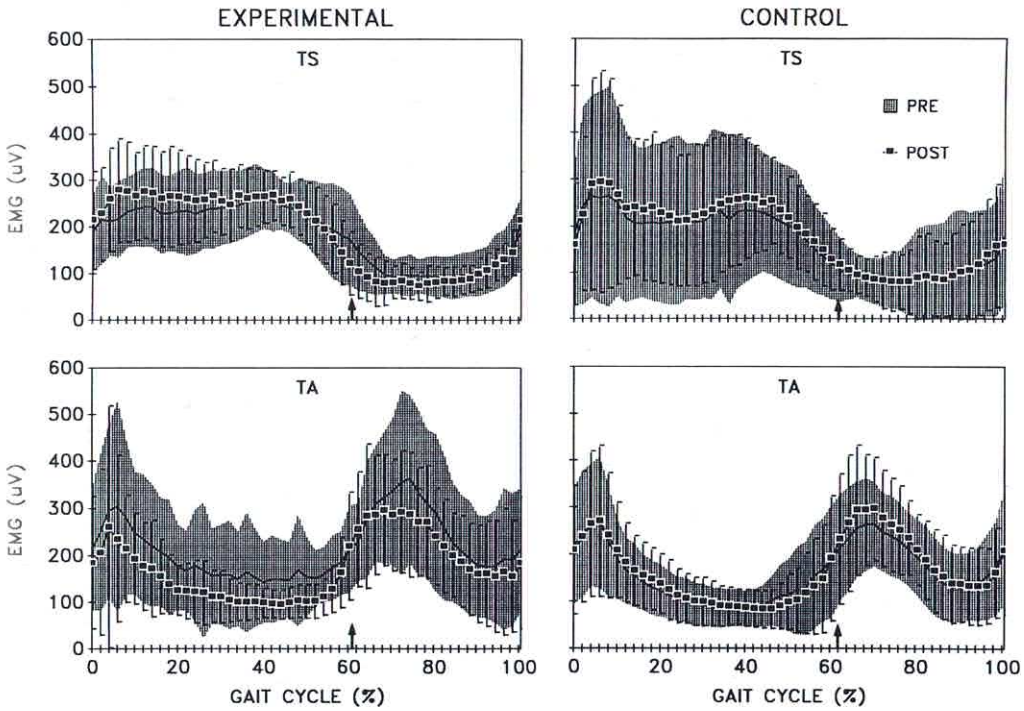

Fig. 3. Comparison of muscle activation profiles during gait in the 2 groups of subjects in pre- and post-tests. EMG amplitude (in uv) on the *Y*-axis is given relative to the gait cycle (*X*-axis) for the triceps surae (TS) and tibialis anterior (TA) muscles. Values give mean ± 1 SD, $n=8$ for the experimental group and $n=11$ for the control group. Arrows indicate end of stance phase.

ences between the tests from analysis of the computer printouts of the footswitch contacts.

3. *Muscle activations*

To illustrate the individual results, the activation profiles of the TS during gait of one of the children in the EXP group are illustrated in Fig. 2. This diplegic child walked with flexed knees and initiated the gait cycle with forefoot contact. As can be seen in the figure, the mean activation profiles of the TS and TA post-PMS generally fall within the dispersion of pre-test values and in this case the variability of the EMG measures was less in the TS during swing and in the TA during stance post-PMS. The SI for the TS was 0.99 in the pre-test and 0.95 in the post-test while for the TA it was 0.87 and 0.67 in the pre-test and post-test, respectively.

Fig. 3 gives the EXP and CTL group mean activation profiles during gait for the TS and TA in pre- and post-tests. Only minor changes can be discerned in post-test activation profiles of the CTL group. In the EXP group, the mean TS activation is slightly higher in early stance and lower in late stance in the post-test but these changes are within the dispersion of the pre-test values. In the TA, the mean stance and swing phase activation level is lower in the post-tests but again within the dispersion of pre-test values. To verify the statistical significance of these small changes, EMG post/pre ratios for specific segments under the activation curves were compared (see Methods). These EMG post/pre ratios are given in Table III. The only significant difference between the respective post/pre ratios (change scores) between the EXP and CTL groups was the smaller ratio ($p<0.01$) for the TA from 0–16% of the gait cycle, indicating a decrease in EMG activity in this part of the gait cycle post-PMS. Examination of individual values revealed that 7 of the 8 children in the EXP group (in comparison to 3 of 11 in the CTL group) had a post/pre EMG ratio = < 1.0 for the TA in this segment of the gait cycle.

4. *Spastic locomotor disorder index (SI)*

Table IV gives the mean values and dispersions of the spasticity locomotor disorder index (SI) calculated in

pre- and post-tests as well as the change scores for each group. In the EXP group the SI for the TS in the pre-test ranged from 0.50 to 1.05 while in the CTL group it ranged from 0.38 to 1.66. Although not statistically different ($p > 0.05$), the SI index was higher and more variable in the CTL group in the pre-tests. Only small changes occurred in the SI index in post-tests and the change scores were similar in both groups ($p > 0.05$). The mean SI index for the TA was remarkably similar in both groups (range in EXP group = 0.59–1.00 and in CTL group = 0.42–1.65) although its variability was larger in the CTL group. The SI change scores between pre- and post-tests for both groups were similar ($p > 0.05$).

5. *Effect of disability*

Because the groups were not evenly matched for disability (hemi or diplegia), the effect of disability on the different parameters was examined. This analysis revealed that the area under the EMG segment from 50–70% of the gait cycle was greater ($p < 0.01$) in the group of diplegic children in pre-tests. No other spatiotemporal or EMG parameter differed with disability in pre-tests. Analysis of the effect of disability on change scores again pointed to the 50–70% segment, revealing lower ($p < 0.02$) post/pre EMG ratios for the diplegic group thus suggesting that the prolonged late stance TS activation is reduced after PMS.

DISCUSSION

In this study the effects of a single session of prolonged stretch of the plantarflexors on activations of these same muscles and their antagonists during gait were studied in children with spastic CP. Despite in-depth analysis of the EMG activation profiles, the only significant finding was a lower TA activation from 0–16% of the gait cycle post-stretch. This lower early stance TA activation post-stretch did not, however, alter the SI index (12), possibly reflecting its lack of sensitivity. Since prolonged stretch of the TS reduced spastic restraint during passive ankle move-

Table III. *Mean post/pre EMG ratios calculated for different segments of the activation profiles during gait for the triceps surae (TS) and tibialis anterior (TA) muscles of both groups of CP children*

Segment of gait cycle	0–16%	16–50%	18–60%	50–70%	62–80%	84–100%
TS						
EXP ($n=8$)	1.14[a]	1.07	–	0.91	–	1.06
CTL ($n=11$)	1.12	1.16	–	1.12	–	1.11
TA						
EXP ($n=8$)	0.79*	–	0.88	–	1.02	0.90
CTL ($n=11$)	1.04	–	0.97	–	1.10	1.01

[a] Values give mean post/pre EMG ratio for each segment.
*$p < .01$; Mann-Whitney U-test.

Table IV. *Comparison of the mean locomotor spasticity index calculated for the triceps surae and tibialis anterior muscles in pre- and post-tests for both groups of CP children*

	Experimental group ($n=8$)				Control group ($n=11$)		
	Pre-test	Post-test	Change score[b]	Statistical comparison[c]	Pre-test	Post-test	Change score
Triceps surae	0.74[a] (0.20)	0.79 (0.23)	0.05 (0.13)	NS	0.82 (0.39)	0.76 (0.26)	−0.06 (0.18)
Tibialis anterior	0.78 (0.13)	0.76 (0.17)	−0.02 (0.17)	NS	0.77 (0.32)	0.71 (0.23)	−0.06 (0.15)

[a] Values give mean ± 1 SD for spasticity index calculated as defined by Fung & Barbeau (1989).
[b] Change score: difference between pre- and post-tests.
[c] Differences between change scores were not significant; $p > 0.05$ (Mann-Whitney U-test).

ments in the same children (31), it was also expected to reduce spasticity during walking. Indeed, on the basis of gait studies in adult spastic patients (6, 9, 17, 18, 33) and in CP children (2, 3, 17, 26, 27), diminished spasticity could be expected to affect the timing and amplitude of the TS activation.

The present findings clearly show that the response of semiautomatic gait muscle activations (14) do not respond in the same way as reflex and voluntary activations to prolonged muscle stretch. These CP children had significantly decreased resistance to passive movements and lower reflex EMG activations as well as improved voluntary activation of the TS immediately after and up to 35 min after receiving prolonged stretch to the plantarflexors by similar methods on a different day (31). This disparate response suggests that inhibitory effects of the stretching procedure may be specific to the type of activation or behavior (reflex, voluntary or semi-automatic). It could be postulated, for instance, that descending excitatory influences during gait implicate a different neuronal circuitry that bypasses the pathway through which the inhibitory influence of stretching is exerted. Indirect support for this postulate comes from the presence of spasticity in a given muscle during passive movements but not during gait (18, 27).

On the basis of the present results, it is not possible to ascertain if the spasticity during gait was altered. One way to answer this question would be to measure the H or stretch reflex during gait (7, 28, 35). It is possible, however, that prolonged stretch led to a decrease in spasticity but that it did not induce functional changes during gait. In fact, given the high resistance of abnormal gait in spastic CP children to pharmacological therapy (17), likely because of the preponderance of excessive coactivation of antagonist muscles (2, 17, 18), it is possible that one 30 min session is insufficient to modify muscle activations during gait in spastic CP. In support of this view is the finding that improvements in ankle movements or spatiotemporal gait parameters induced by an ankle foot orthosis are time dependent and can take a few weeks to appear (4, 27, 29, 32). Lastly, it can be argued that even if spasticity was reduced, muscle activation profiles during gait may never be changed or normalized in spastic CP given the underlying lesion in an immature brain. Two observations made in our laboratory, however, indicate otherwise. First, the finding that the use of a walker in a spastic diplegic child allowed a more normal activation pattern of the TS and TA to emerge (25) and second, the remodelling of the TS activation profile during gait of a 3-year-old hemiplegic girl after wearing an ankle foot orthosis for 3 months (10).

The predicted beneficial effect on the TA, shortened by the procedure, was increased early stance and late swing phase activation. Not only did this not occur but in fact the only significant finding post-stretch was a decreased TA early stance phase activation. It is known that the TA activation burst in early stance becomes more prominent with maturation as normal infants acquire a definite heelstrike (24). It is thus not surprizing to see an even lower TA activation burst in spastic CP who lack dorsiflexion at the end of the swing phase and initiate the gait cycle with full foot or toe contact (34). On the other hand, it is more difficult to explain the further decrease in this activation burst post-stretch. Could the lower activation level be a response of the usually elongated TA (by the action of the spastic plantarflexors) to the unaccustomed shortened position held for 30 min while the TS is being stretched? This change in muscle length may affect the sensitivity of the muscle receptors so that the excitability of the TA is much reduced, resulting in a still lower activation level in early stance.

Since the control group had proportionally more hemiplegic cases than the experimental group, one can argue that the disability level may have skewed the response to the inhibitory procedure. This may have been the case for the 50–70% segment of the TS activation profile and it is possible that a larger proportion of diplegics may have led to significant changes for this EMG segment. In general, however, statistical evaluation of the effects of disability level on the changes in the parameters chosen to represent the effects of the stretching procedure showed that disability per se was not the determining factor in the differences in group results.

In summary, the results of the present study clearly indicate that a single session of TS stretching in the upright position does not produce a functional improvement in the gait pattern. These findings do not mean that long-term muscle stretch cannot affect muscle activations during gait but only that 30 min is not enough. Further studies are needed to determine the intensity and duration of the minimal effective stretching stimulus since casts and ankle orthoses which apply a stretch over longer periods can induce change. The results also demonstrate the stability or reproducibility of the gait activations in spastic CP when the recording electrodes are left in place. Finally, the present study emphasizes the importance of a

control group and the use of "change" scores in the statistical analysis. The interpretation of the effects of the stretching procedure can be quite different when comparing pre- and post-test results within groups or when looking only at the experimental group.

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