Relief of hemiparetic spasticity by TENS is associated with improvement in reflex and voluntary motor functions

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Summary Our previous studies showed that a single 45 min application of transcutaneous electrical nerve stimulation (TENS) prolonged soleus H and stretch reflex latencies in hemiparetic subjects. In addition, 9 daily 30 min TENS applications enhanced vibratory inhibition of the H reflex and tended to decrease hyperactive stretch reflexes. These findings suggested that longer-term TENS may be effective in reducing hemiparetic spasticity. Our present objectives were 2-fold: to determine whether longer-term repetitive TENS stimulation would lead to a reduction in clinical spasticity in hemiparetic subjects, and whether such a reduction could be associated with a decrease in stretch reflex excitability and an improvement in voluntary motor function.

We compared the effects of 15 daily 60 min TENS treatments over a 3 week period, with those of placebo stimulation applied to the common peroneal nerve of the affected leg in similar groups of spastic hemiparetic subjects. Our test battery consisted of 5 measurements which assessed (1) clinical spasticity scores, (2) maximal H reflex to M response ratios, (3) vibratory inhibition of H reflex, (4) stretch reflexes, and (5) maximal voluntary isometric plantarflexion and dorsiflexion, in standing.

In contrast to placebo stimulation which produced no significant effects, repeated applications of TENS over time decreased clinical spasticity (P < 0.05), and increased vibratory inhibition of the soleus H reflex (P = 0.02) after 2 weeks. These changes occurred with a substantial improvement in voluntary dorsiflexion force up to 820%, but not plantarflexion force. They were followed by a reduction in the magnitude of stretch reflexes (P = 0.05) in the spastic ankle plantarflexor, concomitant with a decrease in the EMG co-contraction ratios after a further week of stimulation.

Our results thus indicated that repeated applications of TENS can reduce clinical spasticity and improve control of reflex and motor functions in hemiparetic subjects. Furthermore, the underlying mechanisms may be due partly to an enhancement in presynaptic inhibition of the spastic plantarflexor, and partly to a possible “disinhibition” of descending voluntary commands to the paretic dorsiflexor motoneurons.

Key words: Spasticity; TENS; Electrical stimulation; Hemiparesis; Motor control; Reflexes

Pathophysiological mechanisms aside, there is abundant evidence that low threshold afferent input can reduce the ongoing activity in interneurons and/or alpha motoneurons via segmental, propriospinal or supraspinal pathways (Pierrot-Deseilligny and Maziers 1984). Such a possibility has been demonstrated in the cat where descending commands were modified by activity in cervical propriospinal neurons during target-reaching tasks (Alstermark et al. 1981). Similarly, electrical stimulation (0.6 × motor threshold) of low threshold afferents from the median and ulnar nerves has been found to inhibit Ia afferent mediated excitation in reflexly and voluntarily activated wrist flexor muscles (Malmgren and Pierrot-Deseilligny 1988a,b). Furthermore, dorsal column stimulation has been shown to inhibit transmission in spinothalamic tract pathways via both pre- and post-synaptic inhibitory mechanisms (Foreman et al. 1976). Indeed, there is considerable supraspinal and peripheral afferent convergence onto spinal interneurons mediating presynaptic inhibition (Baldissera et al. 1981). If
presynaptic inhibition is indeed diminished in spasticity (cf., Ashby and Verrier 1976; Burke 1988), then segmental input arising through natural or electrical stimuli may be one way of “switching on” presynaptic inhibitory mechanisms.

Now, remote subcutaneous electrical stimulation of the contralateral wrist has been found to suppress ankle clonus in spastic multiple sclerosis patients (Walker 1982). This inhibition developed gradually — 1 h after 60 min of stimulation — and outlasted the period of stimulation for up to 3 h in some instances. According to other clinical reports, low-intensity transcutaneous electrical nerve stimulation (TENS) also reduced spasticity in 3 of 6 spinal cord injured patients (Bajd et al. 1985), and improved bladder and motor functions, specifically the knee flexion torque (by 13.6–41.9%), in the majority of the multiple sclerosis patients studied (Fredriksen et al. 1986). These effects are reported whether TENS was applied directly over the spastic muscles for 20 min (Bajd et al. 1985), or continuously on the skin overlying the dorsal columns at low cervical–high thoracic level for 2 weeks (Fredriksen et al. 1986). However, these studies have not taken into consideration possible placebo effects. Moreover, the mechanisms by which TENS could be exerting its effects on spasticity and movement control remain unclear. Some evidence is emerging that it may be acting via a combination of several mechanisms. Possible enhancement of presynaptic inhibition was suggested by our finding of a significant increase in the amount of vibratory inhibition of the H reflex in spastic hemiparetic subjects following 9 daily 30 min TENS applications to the low back (Hale and Chan 1986b). Alternatively, the release of inhibitory neuromodulators or opioids may account for the prolonged influence of TENS observed in pain management (Salar et al. 1981; Almay et al. 1985).

Based on these findings, we recently studied the effects of 45 min of TENS, applied either segmentally to the ipsilateral common peroneal nerve or heterosegmentally to the contralateral median nerve in spastic hemiparetic subjects (Levin and Chan 1989). When compared with placebo stimulation applied for the same duration, we found that TENS stimulation produced a significant prolongation of H and stretch reflex latencies in the spastic calf muscles, for up to 60 min post stimulation. These effects were observed regardless of whether the simulation was applied segmentally to the leg, or heterosegmentally to the contralateral wrist. Our findings thus suggested that a single session of TENS could lead to a prolonged increase in stretch reflex threshold levels in hemiparetic spasticity, which could be independent of segmental reciprocal inhibitory mechanisms.

Considering that the disorder in spasticity may be partly related to enhanced stretch reflex excitability (Feldman 1986; Powers et al. 1988), the above findings led us to speculate that repeated TENS applications over a period of time (weeks) could result in a significant reduction of clinical spasticity. We set out to investigate this possibility in the present study. The effects of 15 daily 60 min TENS stimulation on subjective spasticity, hyperactive stretch reflexes and maximal voluntary isometric ankle contractions were studied in spastic hemiparetic subjects. These results were compared with those of placebo stimulation applied for the same period to a similar group of patients, in an attempt to monitor possible changes in these measures due to mental set and/or time-related changes.

More specifically, the objective of our present study was to investigate whether repetitive low-threshold different stimulation (TENS) over 2 or 3 weeks would lead to a reduction in clinical spasticity in hemiparetic subjects. A further objective was to find out whether such a reduction was associated with a decrease in stretch reflex excitability, an increase in presynaptic inhibitory mechanisms and/or an improvement in voluntary motor function. The findings from this study have been communicated in abstract form (Chan and Levin 1990).

Methods and materials

Patients (n = 13; age 59.1 ± S.D. 13.6 years) with spastic hemiparesis due to cerebrovascular accidents were selected for the study based on the following criteria: spasticity in the lower limb; a minimum of 10° of passive ankle dorsiflexion; no history of a previous neurological disorder; no pain or major sensory impairment in the lower limb. They were advised as to the nature of the study and gave their informed consent. Clinical assessment of spasticity comprised Achilles tendon jerks, resistance to passive ankle dorsiflexion, and the amount and duration of ankle clonus. This evaluation was done by the same examiner using a 4-point scale for “clonus,” and a 5-point scale for the other two indexes. Because the score for “resistance to passive dorsiflexion” most closely represents tone (Berardelli et al. 1983), it was doubly weighted. These 3 scores were then summed to generate the “total spasticity score” (Table I). Scores ranging from 0–9, 10–12, and 13–16 corresponded to mild, moderate and severe spasticity respectively. Initially, the subjects were randomly allocated to either a treatment (n = 7) or a placebo group (n = 6). Since the placebo stimulation was later found to have no effect, 4 of these 6 patients then underwent a further 3 weeks of TENS stimulation, increasing the number of patients in the treatment group to 11.
**Experimental protocol**

Most subjects were tested twice, at least 1 week apart, for baseline measurements (Table I) and to determine data reproducibility. The test battery consisted of 5 measurements: (1) clinical spasticity scores; (2) maximal amplitude of the H reflex as a percentage of the maximal M response (H/M; Schieppati 1987); (3) amount of inhibition of the H reflex during vibration (Hvib/Hctl; Desmedt and Godaux 1978); (4) excitability of the soleus stretch reflex in terms of latency, onset angles and magnitude (SR/M area); and (5) maximal voluntary isometric plantarflexion and dorsiflexion (MVC) of the ankle (EMG and force) in standing. The testing procedure was as follows: spasticity about the ankle joint was first assessed with the subject seated. They then stood on a force platform (see below) and were instructed to generate sustained plantarflexing or dorsiflexing MVCs for about 2 sec following a **response signal**, which was preceded at 500 msec by a **warning signal**. In each ensemble of 6 trials, 2 “catch trials” were randomly interspersed to ensure that subjects would not anticipate the response signal. During a “catch trial,” no response signal was presented following the warning signal, so that only baseline EMG and force were recorded. All in all, 4 dorsiflexing and 4 plantarflexing MVCs were collected from both the affected and non-affected legs. These measurements were followed by reflex testing with the subjects lying in a semi-supine position (see below).

Following the initial testing session(s) and the allocation to either treatment or placebo group, subjects and/or their family members were instructed in the use of a portable TENS stimulator. Stimulation (0.125 msec square pulses, 99/sec, 2 × sensory Th for TENS and essentially zero, 0.1 × Th for placebo), was applied via a Selectra 7720 stimulator (Medtronic) by surface electrodes (3.8 cm × 5.1 cm) attached to the skin over the common peroneal nerve (L4-S2) posterior to the head of the fibula on the hemiparetic leg. This nerve supplies the muscles antagonistic to the spastic calf muscles. The mean sensory threshold was determined as the average of 3 trials in which the intensity of the

| TABLE I |
| A comparison of baseline clinical features, reflex profile and maximal voluntary force between spastic hemiparetic subjects in TENS treatment and placebo groups. |

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Reflex profile</th>
<th>Maximal contraction</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>Etiology</td>
<td>Time since injury (months)</td>
</tr>
<tr>
<td><strong>TENS treatment group</strong></td>
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<tr>
<td>75</td>
<td>L.CVA</td>
<td>18</td>
</tr>
<tr>
<td>49</td>
<td>L.CVA</td>
<td>27</td>
</tr>
<tr>
<td>67</td>
<td>L.CVA b</td>
<td>8</td>
</tr>
<tr>
<td>31</td>
<td>L.CVA</td>
<td>25</td>
</tr>
<tr>
<td>76</td>
<td>R.CVA b</td>
<td>43</td>
</tr>
<tr>
<td>43</td>
<td>L.CVA</td>
<td>8</td>
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<tr>
<td>67</td>
<td>R.CVA</td>
<td>23</td>
</tr>
<tr>
<td>57</td>
<td>L.CVA</td>
<td>85</td>
</tr>
<tr>
<td>73</td>
<td>R.CVA b</td>
<td>26</td>
</tr>
<tr>
<td>58</td>
<td>R.CVA</td>
<td>16</td>
</tr>
<tr>
<td>47</td>
<td>L.CVA b</td>
<td>11</td>
</tr>
<tr>
<td>Placebo group</td>
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<td>67</td>
<td>L.CVA b</td>
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<td>47</td>
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<tr>
<td>10.6</td>
<td>R.CVA b</td>
<td>17.2</td>
</tr>
</tbody>
</table>

<sup>a</sup> Spasticity score: 0–9 = mild spasticity; 10–12 = moderate spasticity; 13–16 = severe spasticity.
<sup>b</sup> Four subjects participated in both groups after no placebo effect was observed.
<sup>c</sup> Mean.
<sup>d</sup> S.D.

SR = stretch reflex; PF/DF = plantarflexion/dorsiflexion; L/R CVA = left/right cerebrovascular accident.
stimulation was gradually increased to a level when the subject first reported a faint tingling sensation in the cutaneous distribution of the nerve. Subjects were informed that they may or may not feel any sensation associated with the stimulation. Under these conditions, sub-threshold TENS was considered “placebo” stimulation. The stimulation (or placebo) was applied for 60 min per weekday. This treatment time was considered optimal, based on findings from previous studies of the effects of a single session of electrical stimulation in spastic patients (Walker 1982; Levin and Chan 1989). Compliance with the treatment protocol was verified by inspection of a log book kept by the subject or his/her family. To assess the time course of the effects of stimulation, the 5 measurements described above were repeated after 2 and 3 weeks of stimulation (i.e., days 15 and 22, maximum of 15 treatments).

Reflex testing

Subjects reclined in a semi-supine position with the knee in 30° flexion and the ankle in the neutral position fixed by sanplint casts and velcro straps. This was done to ensure a constant initial length of the ankle muscles — a known variable affecting the H reflex (e.g., Robinson et al. 1982). The axis of rotation of the footplate was aligned to that of the ankle joint (Inman 1976). Ankle joint angles were recorded with a potentiometer (Beckman 5311) mounted on the same axis. H reflexes were elicited via a 2 cm cathode (single 1 msec pulses, 0.1/sec) placed on the skin overlying the posterior tibial nerve in the popliteal fossa for selective stimulation of the nerve trunk (Hugon 1973). The reference electrode (20 cm²) was positioned superior to the patella. For each stimulus intensity investigated, 10 responses were averaged by a PDP11-23 plus computer. The intensity of the stimulation was gradually increased to record maximal H reflexes, followed by maximal M responses. It was then decreased to elicit control H reflexes (Hctl), set at approximately 30% of the maximal M across all subjects. Vibration was applied by placing a vibrator (Ling Dynamics 101) at a right angle to the skin overlying the inferior third of the Achilles tendon. Vibration parameters (100 Hz, 2 mm peak-to-peak amplitude) were optimal to produce maximal inhibitory effects on the soleus H reflex (De-smedt and Godaux 1978). After accommodation (1 min), 10 H reflexes were elicited while the vibration was continued. At the end of vibration, H reflexes were monitored until they returned to 90% of the control value. Stretch reflexes were elicited by rapidly dorsiflexing the ankle by a mechanical stretching device which provided a fast consistent profile of a 30° ramp displacement (see Results). Subjects were instructed not to intervene voluntarily. The device consisted of a footplate attached to an overhead bar by springs and a mechanical stop which arrested dorsiflexion at 10° past the neutral position. The stretch was applied from a position of 20° plantarflexion when background EMG was quiet. To allow sufficient recovery in the reflex pathways, 30 sec was allowed between trials.

MVC testing

Subjects stood on a customized force platform that had a back support, to which the body was secured by a wide strap. This provided balance stabilization for the subject, so that the weight was equally distributed on each leg. To further minimize postural destabilization while performing contractions with the non-affected leg, subjects grasped hand railings situated on either side of the apparatus. They stood with ankle joints in the neutral position (90°), and with feet fixed by heel cups and velcro straps to footplates located 30.5 cm apart. Subjects maintained their knees in slight flexion. In this position, most of the plantarflexing force would be generated by the soleus muscle, although contributions from other plantarflexors could not be entirely eliminated. The height of the platform was adjusted, so that the ankle joint aligned coaxially with the axis of rotation of the footplate. Note that the standing position was chosen because it represents a functionally important stage in the recovery of locomotion in hemiplegic patients.

Forces applied to the footplate were transmitted by a rigid transverse bar to a force transducer (Lebow 3132) positioned under the right or the left footplate for independent measurement of plantarflexing or dorsiflexing force generated by either leg (sampling rate 1250/sec). Force data were proportional to torques, since for our apparatus, the distance from the axis of rotation to the site of force recording was fixed. H reflexes, M responses, stretch reflexes and voluntary EMG activities were recorded with bipolar silver-silver chloride surface electrodes (Medi-Trace 1801) positioned on the soleus and tibialis anterior muscles, with a reference electrode over the head of the fibula. EMG signals were filtered (10–500 Hz), amplified (gain 1000 for H reflex and 5000 for the other signals) with Disa 15C01 amplifiers, sampled (2000 and 1250/sec, respectively) and stored for further analysis. Signals were recorded for 200 msec after the H reflex stimulation and from 500 msec before, to 900 msec after the ankle displacement for stretch reflexes.

Data analysis

Maximal M response areas were computed by rectifying and integrating the M wave over a time window spanning the response onset to its offset (i.e., the point at which the trace exceeded, or returned to 3 S.D. of the baseline). H reflex amplitude or area values were normalized by the maximal M response (i.e., H/M...
amplitude; cf., Angel and Hofmann 1963; Schieppati 1987). H reflexes during vibration were normalized by Hctl (Hvib/Hctl). In this way, mean values could be compared across subjects. EMG signals from stretch reflex trials were analyzed in terms of latency, onset angle, response duration, and area values. Trials were rejected if the subject intervened voluntarily or was unable to relax completely. EMG signals were first rectified and background baseline activity removed. EMG latency was determined from the time when the displacement surpassed 2° to the time when the EMG signal exceeded 3 S.D. of the baseline value. The latency was then used to determine the onset angle from the displacement trace. EMG areas were calculated as the integral of the EMG over the 150 msec following its onset. These were expressed as percentages of the maximal M response areas evoked in the same muscles (SR/M). For isometric MVCs of the ankle muscles, soleus and tibialis anterior EMG signals, as well as plantarflexing and dorsiflexing force, were measured from 500 msec before to 2 sec following the response onset. Maximal EMG areas and force were calculated in a 500 msec window placed where the force trace reached a plateau. EMG co-contraction ratios were then computed as ratios of the EMG area when the muscle acted as an antagonist to that when it acted as an agonist. Note that both EMG and force measurements from the affected leg of our hemiparetic subjects demonstrated high reproducibility in our previous study. Specifically, agonist EMG area, maximal force and EMG co-contraction ratios during dorsiflexing contractions all revealed high interclass correlation coefficients of \( r = 0.91, 0.95 \) and 0.99 respectively, when the same patients were tested on different days (Levin and Chan 1990).

Differences between the baseline data from the 2 groups of subjects were tested by Student's \( t \) tests. Post-stimulation changes for each of the above measures were expressed as raw data, or computed as percentages of the control values for each subject. The effects of TENS on each measure were compared with pre-stimulation control values or with placebo stimulation, using repeated measures and randomized ANOVAs respectively. Differences between individual pairs of means were determined via "least significant difference" tests. A significance level of 0.05 was used for all tests.

Results

Characteristics of spastic hemiparetic subjects and similarity between groups

Based on our clinical evaluation of the subjects comprising the treatment group, 3 had mild, 4 had moderate, and 4 others had severe spasticity (Table I). In the placebo group, 1 subject had mild, 2 had moderate and 3 had severe spasticity. Of relevance is the finding that mean control composite spasticity scores did not differ between the 2 groups of subjects, being 11.2 ± 2.9 and 11.8 ± 2.5 respectively \( (t = -0.102, P > 0.90) \); see also the histograms on the left side of Fig. 1). Furthermore, there were no initial differences in control H/M, Hvib/Hctl, and stretch reflex values between the TENS and placebo groups \( (P > 0.10, \text{Table I}) \); nor in the mean control values of maximal plantarflexing and dorsiflexing force \( (P > 0.05, \text{Table I}) \); nor in the EMG co-contraction ratios during ankle dorsiflexion, the latter being 50.0 ± 21.4% and 66.1 ± 11.2% respectively for the TENS and placebo groups \( (P > 0.05) \). When all these data from the placebo group were compared to the original TENS group \( (n = 7) \), there were also no initial differences. In other words, all available data confirmed that the patients receiving TENS were similar to those receiving placebo stimulation.

Effects of TENS and placebo stimulation on spasticity scores and H reflexes

The mean control and post-stimulation spasticity scores after 2 and 3 weeks of TENS (filled columns) or placebo (open columns) stimulation for each group are presented in Fig. 1. A decrease in spasticity was evident after 2 weeks of TENS but not placebo stimulation. However, an additional week of TENS stimulation did not lead to a further reduction in spasticity scores. Of clinical importance is the finding that the reduction in spasticity following 2 and 3 weeks of TENS was significant when contrasted with placebo.
stimulation ($F = 4.66, \ p < 0.05$). This finding was also significant when the data from the original 7 members of the TENS group were considered. This result is particularly remarkable in view of the consistency of the spasticity scores after 2 and 3 weeks of placebo stimulation (Fig. 1, open columns).

Surprisingly, neither TENS nor placebo stimulation had any significant effect on the H/M ratios, despite the latter's tendency to decrease after 2 weeks of TENS (Fig. 3). On the other hand, $H_{vib}/H_{ctl}$ ratios did show significant trends after TENS stimulation. Examples of $H_{vib}/H_{ctl}$ ratios are shown in Fig. 2 for one subject with severe spasticity, who received both TENS (subject 9 in Table I) and placebo (subject 5 in Table I) treatments. $H_{vib}/H_{ctl}$ ratios in this subject were highly consistent when measured 1 week apart,
being 30.4% and 29.7% on the 2 occasions (Fig. 2A and B). Furthermore, they showed a significant reduction following TENS but not placebo stimulation (Fig. 2C–F). It is noteworthy that this improvement was evident in 7 of the 11 subjects receiving TENS treatment. Table II contrasts the effects of stimulation on the reflex measures between treatment and placebo groups. In 2 subjects of the treatment group, the reduction in Hvib/Hctl ratios after 3 weeks of TENS was marked (from 29.8% to 3.1% in subject 7; and from 96.3% to 2.7% in subject 10; Table II). For the 4 subjects in whom these ratios did not change, one was among the oldest subjects (75 years, subject 1), one had very long-standing hemiplegia (85 months, subject 8), and one had variable responses (subject 4). The 4th subject (subject 3 in the treatment group and subject 1 in the placebo group) had very consistent results in all 6 testing sessions.

Table II showed that the mean group Hvib/Hctl ratios were significantly decreased (F = 7.08, P = 0.02) following either 2 or 3 weeks of TENS (from 57.7% to 39.6% and 42.5%) but not placebo stimulation (from 71.6% to 71.0% and 68.1% respectively). Correspondingly significant changes were found when the placebo group was compared to the original TENS group (F = 4.56, P = 0.05). In the latter comparison, the Hvib/Hctl ratio was decreased from 49.3% to 30.8% and 30.5% after 2 and 3 weeks of TENS respectively. The changes in the placebo and in the whole TENS (n = 11) groups are graphically depicted in the histograms of Fig. 3 as percentages of their respective control values. In the figure, the mean Hvib/Hctl ratios following 2 and 3 weeks of TENS stimulation were 70.3 ± 35.0% and 68.9 ± 40.6% respectively, as compared to placebo stimulation (104.7 ± 30.3% and 92.6 ± 23.8% respectively). In other words, whereas 2 weeks of TENS significantly reduced the Hvib/Hctl ratio, an additional week of treatment produced no further reduction in this ratio.

Effects of TENS and placebo stimulation on stretch reflexes

In order to compare treatment effects on the stretch reflexes, we had to make sure that the stretch perturbation applied to the subjects was constant across days, by evaluating its reproducibility when patients were tested on different occasions. Our results showed that the mean velocities of stretch ranged from 450.2 ± 58.8°/sec to 511.0 ± 77.5°/sec, and were not significantly different across days (P > 0.10). These findings demonstrated the consistency of the stretch input across different testing sessions, thereby making it valid to compare treatment effects on the stretch reflexes over time.

We found that TENS, but not placebo stimulation, tended to delay the onset and significantly decreased the magnitude of stretch responses. More specifically, following 3 weeks of TENS treatment, stretch reflex onsets were prolonged to 134.6 ± 69.7% when compared with placebo stimulation (105.9 ± 31.1%). The apparent prolongation of stretch reflex onsets following TENS, however, was non-significant, possibly due to the high inter-subject variability in this measure. On the other hand, SR/M areas were significantly reduced from 50.3 ± 25.7% to 34.6 ± 20.2% (49.6 ± 20.3% to 33.4 ± 19.8% in the original TENS group) after 3 weeks of TENS treatment, whereas compared with placebo stimulation (from 68.0 ± 27.8% to 68.5 ± 26.7%; F = 4.03, P = 0.05, Table II). Note that, unlike the findings on spasticity scores and Hvib/Hctl ratios, this reduction was not apparent after 2 weeks of treatment.

Effects of TENS and placebo stimulation on maximal isometric voluntary contractions

In a previous study, we found that overall EMG and force data recorded from the non-affected legs of 13 hemiparetic subjects did not differ from those obtained in the legs of 7 age-matched normal subjects (Levin and Chan 1990). In that study, agonist EMG areas during plantarflexing and dorsiflexing contractions were found to be 81.4 ± 29.0 μV.sec and 161.1 ± 46.3 μV.sec respectively in the non-affected leg of hemiparetic subjects, and were thus similar to the values of 95.8 ± 36.5 μV.sec and 168.0 ± 119.9 μV.sec observed respectively in the normal subjects. Maximal force was also similar, being 25.7 ± 10.1 kg and 10.4 ± 3.2 kg for the plantarflexors and dorsiflexors respectively in the non-af-
fected legs of hemiparetic subjects, as compared to 29.1 ± 8.5 kg and 13.7 ± 6.0 kg for the plantarflexors and dorsiflexors respectively in normal subjects. Finally, EMG co-contraction ratios during dorsiflexing contractions were 31.6 ± 13.9% on the non-affected side of hemiparetic subjects which, again, did not differ from the 20.3 ± 8.1% observed in the normal limbs. Therefore, we felt justified in comparing the effects of TENS and placebo stimulation on these 3 measurements between the affected and non-affected legs of our hemiparetic subjects.

The most striking result of our TENS treatment was a marked improvement in dorsiflexing force in the affected legs of hemiparetic subjects. This effect is illustrated by the data obtained from one subject in Fig. 4. In this subject, the mean control maximal force was 1.4 kg which, after 2 weeks of treatment, increased to 3.4 kg. By the end of 3 weeks of treatment, there was an even greater increase in force to 7.4 kg.

Following 3 weeks of TENS stimulation, mean maximal dorsiflexing force had increased to 5.5 ± 2.9 kg in the TENS group (5.8 ± 2.9 kg in the original TENS group of 7 subjects) but only to 3.7 ± 2.9 kg in the placebo group. Compared to control, this improvement was significant for the TENS (F = 4.40, P < 0.05) but not for the placebo group (F = 1.09, P = 0.37). Fig. 5 shows the changes in maximal plantarflexing and dorsiflexing force as percentages of their pre-stimulation control values for both groups. For dorsiflexion, the improvement in force across the TENS group was remarkable for the affected but not the non-affected leg. It ranged from 5% to 820% of individual control values in the 11 subjects tested. Although plantarflexing force showed a slight improvement following TENS and placebo stimulation in both the affected and non-affected legs, the magnitude of the improvement was similar for both types of stimulation, with no significant difference being found between group mean values.

Fig. 4. Four dorsiflexion trials in one subject with moderate spasticity (A) control, (B) after 2 and (C) 3 weeks of TENS stimulation. In this subject, maximal isometric force generated by the dorsiflexors increased from a mean of 1.4–7.4 kg after 3 weeks of treatment.
EMG co-contraction ratios during dorsiflexion and plantarflexion were very stable across days (interclass correlation coefficient = 0.99 and 0.97 respectively). In the group receiving TENS treatment, the mean co-contraction ratio during dorsiflexion was $50.0 \pm 21.4\%$ prior to treatment and decreased to $42.0 \pm 16.5\%$ and then to $38.1 \pm 20.4\%$ after 2 and 3 weeks of treatment, respectively. During plantarflexion, the ratio (control $= 51.0 \pm 30.7\%$) remained stable after 2 weeks of stimulation ($49.2 \pm 32.8\%$) but decreased significantly to $32.1 \pm 18.3\%$ at the end of 3 weeks. In contrast, placebo stimulation had no effect on co-contraction ratios for either type of contraction.

Discussion

Our main findings from this study demonstrate that repeated TENS applications significantly improved clinical spasticity, concomitantly with reflex and voluntary motor control in spastic hemiparetic subjects. Specifically, TENS reduced spasticity scores (Fig. 1), increased vibratory inhibition of the H reflex (Figs. 2 and 3, and Table II), decreased the magnitude of soleus stretch reflexes (Table II), improved dorsiflexing force (Figs. 4 and 5) and decreased EMG co-contraction ratios. The results of this study confirmed our earlier findings in spastic hemiparetic subjects of enhanced vibratory inhibition of the H reflex following 9 daily 30 min TENS treatments to the low back (Hale and Chan 1986b). In that study, although non-significant, trends towards reduced H/M ratios and stretch reflex magnitude were also reported.

Our 2 studies in patients with spastic hemiparesis agree with 2 previous reports of therapeutic TENS interventions in spasticity of different origins. In one report, 20 min of TENS applied segmentally over the L3/4 dermatome in spastic spinal cord injured subjects, resulted in a marked immediate decrease in knee extensor spasticity as measured by pendulum testing in 3 of the 6 patients examined (Bajd et al. 1985). Similarly, TENS stimulation applied continuously to the skin overlying the dorsal columns of the spinal cord (low cervical, high thoracic region) for 2 weeks, improved bladder function and voluntary knee flexor (and, at higher speed, extensor) torque in 18 or 27 multiple sclerosis patients (Fredriksen et al. 1986). Regrettably, the possibility of placebo effect has not been addressed in these clinical studies, as is the case in the present study.

Now, spasticity has been attributed to hyperactivity and abnormal organization in mono- and polysynaptic reflex pathways (Lance 1980; Nichols 1989). Among other factors, this may result from a lowering of stretch reflex thresholds in spastic muscles (Feldman 1986; Powers et al. 1988). Indeed, H and stretch reflex latencies, which may reflect thresholds, are decreased in spasticity, while stretch reflex magnitudes (H/M ratios and SR/M areas) are enhanced (Ashby and Verrier 1976; Hale and Chan 1986a). Despite the obvious hyperactivity in stretch reflexes, we had found neither latency nor magnitude measures to be correlated with the severity of clinical spasticity in spastic hemiparetic patients in our previous studies (Hale and Chan 1986a; Levin and Chan 1989). However, a significant correlation was found between the severity of spasticity and
the reduction in dorsiflexing force (Levin and Chan 1990), suggesting that an amelioration of spasticity could be manifested by an improvement in voluntary dorsiflexion. This indeed was what we found in the affected leg of hemiparetic subjects after 2 and 3 weeks of TENS application to the common peroneal nerve (Fig. 5).

The exact time course of these improvements cannot be judged based on the few studies to date. However, these results concur with the suggestion that there may be a critical duration for disinhibition or restitution of function following multimodal stimulation (Walsh and Cummins 1976). The critical time period is unknown. In a previous study, we demonstrated that a single 45 min TENS treatment applied to the ipsilateral leg or the contralateral wrist delayed H and stretch reflex latencies without decreasing the magnitude of the reflex response (Levin and Chan 1989). Indeed, increases in stretch reflex thresholds, as reflected by prolonged reflex latencies, may be the first changes observed following a brief intervention to reduce spasticity. However, for a noticeable therapeutic effect, a reduction in reflex magnitude would also be expected. According to the present results and our previous study on the influence of 30 min of daily TENS for 9 days (Hale and Chan 1986b), it appeared that 2 weeks of 60 min of daily TENS were necessary for a decrease in clinical spasticity (Fig. 1) and an increase in dorsiflexing force to become significant (Fig. 5), in addition to the increase in vibratory inhibition of the H reflex (Figs. 2–3 and Table II) found in this and our earlier study (Hale and Chan 1986b). Furthermore, a reduction in both stretch reflex magnitudes (Table II) and EMG co-contraction ratios become evident only after a further week of TENS stimulation (Fig. 4). In summary, these results suggest that the initial improvements in spasticity and dorsiflexing force after 2 weeks of TENS stimulation, may have been due in part to an enhanced inhibition of the H reflex. These changes were followed by a reduction in the magnitude of the plantarflexor stretch reflexes and EMG co-contraction ratios evident only after 3 weeks of stimulation.

The effects of long-term low-threshold afferent stimulation on transmission in pathways mediating spasticity have not previously been investigated. However, the TENS parameters used in the present study (low intensity, high frequency) have been shown to activate mainly large diameter afferents (Levin and Chan 1988). Taken together with our present findings, repetitive stimulation of large diameter afferents via TENS over a prolonged period of time (weeks) could lead to a reduction in spasticity simultaneously with an improvement in voluntary motor control. Several mechanisms may explain these effects. The finding that 2 weeks of TENS enhanced vibratory inhibition of the H reflex concurs with that of our previous study when 30 (versus 60) min of TENS was applied over a 2 week period (Hale and Chan 1986b). These results pointed to the possible involvement of an enhanced presynaptic inhibitory mechanism in mediating the effect. Although the evidence is indirect, vibratory inhibition of the H reflex has been attributed in part to presynaptic inhibition of group Ia terminals (Gillies et al. 1969; Burke et al. 1976). Thus, increased vibratory inhibition of the H reflex by repetitive TENS may have been exerted via an enhancement of presynaptic inhibition, which has been shown to be suppressed in hemiplegic spasticity (Ashby and Verrier 1976).

It was interesting that TENS treatment resulted in a striking increase in the strength of the dorsiflexors but not that of the plantarflexors. The increase in dorsiflexing force, however, was still well below the mean value generated by the non-affected legs of the same subjects (Levin and Chan 1990). This improvement in voluntary dorsiflexor function cannot be explained by spontaneous recovery, since all subjects were long past the time in which the majority of recovery of function occurs (Moskowitz et al. 1972). Certainly, placebo stimulation had no effect on these parameters (Fig. 5). Furthermore, none of the subjects was undergoing other treatment or training during the study period. Such an improvement also cannot be explained by alterations in motor unit properties, known to be changed only in the spastic extensor but not in the weak flexor muscles (Edstrom 1970; Sica and Sanz 1976).

As partly alluded to in the Introduction, at least 2 concurrent mechanisms may contribute to the reflex and voluntary abnormalities in spasticity. On the one hand, decreased stretch reflex thresholds may result in hyperactive segmental reflex excitability. On the other, it may be assumed that part of the motor deficit in the weak dorsiflexors in hemiplegia is related to a lack of or decrease in descending excitation to flexor motoneurons (Burke 1988). Thus, in spasticity, hyperactive segmental activity could effectively “mask” or override any underlying descending motoneuronal excitation, for example, through a stronger reciprocal Ia inhibition from the spastic plantarflexors as observed by Yanagisawa et al. (1976). This would result in an even more marked decrease in dorsiflexing force. Repetitive TENS stimulation may have improved dorsiflexor function by increasing presynaptic inhibition, followed by decreasing stretch reflex hyperexcitability of the plantarflexor (soleus) concomitant with reducing the EMG co-contraction ratio. These mechanisms probably served to “disinhibit” or “unmask” underlying neural pathways to dorsiflexor motoneurons. At the same time, as our results indicate, the recovery of force would not be complete, since the descending command remains impaired. Furthermore, the long time course of stimula-
tion needed to produce the desired effect is suggestive that the changes may have been mediated by plastic mechanisms. Such possibilities include sprouting of intact descending pathways making new synapses with motoneurons (Goldberger and Murray 1978), and/or unmasking or reorganization of somatosensory-motor cortical connections (Bach-y-Rita 1981).

Clinical implications

Low-intensity TENS is a clinically accessible, electrical stimulation technique currently used in the physiotherapeutic treatment of pain disorders. This study reports that repeated applications of TENS decreased clinical spasticity and hyperactive reflexes of the spastic plantarflexors and improved motor function of the paretic dorsiflexors in chronic spastic hemiparetic subjects. It is important to note that these changes were directly a result of the TENS intervention, since no effects were evident following placebo stimulation. Furthermore, these patients had reached a stable state, showing no improvement prior to TENS intervention. The use of TENS, therefore, appears to be an effective adjunct in the non-surgical treatment of spasticity and the retraining of motor functions in hemiparetic patients. In contrast to dorsal column stimulation via implanted electrodes, it has the added advantage of being non-invasive.

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References


