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EFFECTS OF TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION ON H-REFLEX AND SPINAL SPASTICITY

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ABSTRACT. The purpose of this study was to investigate the short-term effects of transcutaneous electrical nerve stimulation (TENS; 99 Hz; 250 ms pulses) on H-reflex and spinal spasticity. Considering the reflex hyperexcitability commonly displayed in spinal cord-injured subjects, it was hypothesized that repetitive low threshold afferent stimulation would have an inhibitory effect on the triceps surae H-reflexes which could also be reflected by a decrease in plantarflexor spasticity. Clonus, Achilles tendon reflex and modified Ashworth evaluations were performed on 14 spinal cord-injured subjects prior to and after 30 minutes' application of TENS. Non-parametric statistical analyses ($n = 14$; $\alpha = 0.05$) failed to reveal significant effects of TENS on H-reflex amplitude. However, there was a significant decrease in scores for the Achilles tendon reflex and the modified Ashworth test. The clonus score decreased in most subjects post-TENS, although not in a statistically significant manner. The present *pilot results* thus suggest that TENS appears to be effective in reducing spinal spasticity, as measured clinically.

Key words: H-reflex, rehabilitation, spasticity, spinal cord injury, TENS.

INTRODUCTION

Repetitive low threshold stimulation, applied either transcutaneously or percutaneously, has been shown to modulate the soleus (SO) H-reflex amplitude in healthy and chronic pain subjects (9, 11–13, 27) as well as to inhibit clonus in subjects with spastic paraparesis (34–36) and to reduce spasticity in hemiplegic (20) and spinal cord-injured subjects (1, 5). Levin & Hui-Chan (20) reported, in hemiparetic subjects, an increased amount of vibratory inhibition of the H-reflex after repeated daily applications of transcutaneous electrical nerve stimulation (TENS) (99 Hz; 0.125 ms pulse duration; 2 X sensory threshold for TENS) over the common peroneal nerve (CPN). Knee extensor spasticity, assessed by the pendulum test, was also shown to decrease in 50% of spinal cord-injured subjects after 20 minutes of TENS (100 Hz; 0.3 ms pulse duration; 50 mA). Recently, a significant decrease in the ankle viscoelastic stiffness, evaluated by the Spasticity Measurement System (17), following 20 minutes of surface electrical stimulation (30 Hz) over the tibialis anterior muscle was reported (31). However, because different spasticity measures, stimulation parameters or experimental paradigms were used, results of these studies can hardly be compared.

The short-term effects of different TENS parameters on motoneuronal excitability were recently reported in healthy subjects (11–13). In a study aimed at determining which of previously tested parameters (frequency of 50 or 99 Hz; over the sural or CPN) would be more effective in decreasing H-reflex amplitude in healthy subjects, a control session without TENS provided baseline values to determine the variability of the physiological responses (11). The presence of stable reflexes for the duration of this 45 minutes' control session strongly suggested the constancy of the supraspinal influences. This enabled the authors to assume that the different effects of treatments found in the experimental sessions were due to repetitive low-threshold afferent stimulation by TENS. Despite the variability of effects observed across subject, a tendency towards a decrease in the SO H-reflex amplitude in five out of eight subjects (11), and a significant increase in the gastrocnemius

lateralis (GL) H-reflex amplitude (12), were observed after 30 minutes of continuous TENS stimulation at 99 Hz over the CPN. Considering the very short latency (< 10 ms) between the TENS spikes and the H-reflex stimulus, the increase in the GL H-reflex amplitude could depict Ib facilitation from the antagonist muscles (26). Presynaptic inhibition of the SO H-reflex would be induced at longer condition-test latencies (between 25 and 60 ms; 23). The significant decrease ($p < 0.05$) in SO and GL H-reflex amplitude during discontinuous TENS (stimulation stopped 35 ms prior to each H-reflex stimulus) observed in healthy subjects after 30 minutes of stimulation (13) could thus depict an increase in presynaptic inhibition resulting from the repetitive stimulation of low-threshold afferents.

The purpose of this study was thus to investigate, in spinal cord-injured subjects, the short-term effects of TENS applied over the CPN on H-reflex and plantarflexor (extensor) spasticity at the ankle. Considering the reflex hyperexcitability commonly displayed in that population (19, 22, 33), it was hypothesized that low-threshold stimulation would have, as observed in healthy subjects, an inhibitory effect on the H-reflex amplitude of the triceps surae muscles (13). Furthermore, considering the reported effects of TENS on different spasticity indicators (1, 5, 20, 35, 36), it was believed that the inhibitory effects of TENS on reflex excitability would be reflected by a decrease in clinical spasticity scores.

METHODS AND MATERIALS

Fourteen spinal cord-injured subjects (13 males and 1 female) between the ages of 21 and 54 years ($\bar{X} \pm$ SD; 36 ± 10 years; 1.78 ± .08 m; 74 ± 15 kg) volunteered for the study. Most subjects had an incomplete lesion of the spinal cord at levels varying from C4–C5 to T12. Descriptive characteristics of the subjects are presented in Table I. This study was approved by the Rehabilitation Center Ethics Committee and subjects gave their informed consent.

Transcutaneous stimulation

A TENS stimulator (Medtronic Selectra; 99 Hz; 250 μs square pulse duration) and round carbon-rubber electrodes (4.9 cm^2) were used to deliver repetitive low-threshold afferent stimulation over the CPN, just behind the head of the fibula. The intensity of stimulation was 15 mA, which was twice the average perceptual threshold to sensation for TENS of healthy subjects (13). To avoid signal contamination by spike artifacts of the TENS stimulation, the repetitive low-threshold afferents' stimulation was discontinued 35 ms prior to the H-reflex stimulus (for a period of 95 ms) using an electronic delay device consisting of a solid state bi-directional signal switch with high voltage metal oxide semiconductor transistors (Hewlett Packard HSSR-8200; 200 V; 160 W) and a high-speed photosensitive drive circuit (50 μs).

Electrophysiological testing

Subjects were comfortably seated on an air-filled cushion (ROHO) with the trunk and upper extremities well stabilized,

Table I. *Characteristics of the subjects*

Age	Level	Time (months)	Etiology	ASIA scale	Ambulatory status
34	C4–C5	36	Trauma	C	W/C (motor)
37	C5–C6	14	Trauma	D	1 cane
54	T11–T12	7	Carcinoma	D	2 canes
35	C6–C7	2	Trauma	C	W/C
44	C5–C6	23	Trauma	C	W/C
34	C6–C7	12	Trauma	D	2 canes
48[1]	C7–C8	60	Carcinoma	B	W/C
49	C5–C6	26	Trauma	B	W/C
28	C6–C7	50	Trauma	C	W/C
23	T9–T10	18	Trauma	D	2 canes
31	N/A	4	Myelitis	C	W/C
21	C6–C7	46	Trauma	B	W/C
35	C5–C6	194	Trauma	C	W/C
36	C5–C6	171	Trauma	A	W/C

W/C = wheel-chair; N/A = non applicable, sequels caused by a cervical myelitis; ASIA = American Spinal Injury Association (A = no sensory or motor function below the level of the lesion; B = some sensory but no motor function below the level of the lesion; C = some motor function extending through S_4–S_5 but no active movement against gravity; D = some active movement against gravity).

[1] The only woman participating in the study.

and the right lower extremity immobilized at angles of 90° at the hip, 60° at the knee and 90° at the ankle. The skin temperature over the triceps surae muscles was monitored throughout the experimental session using a Thermal J42 Cyborg skin feedback thermometer. Bipolar electromyographic (EMG) electrodes with preamplifier at the recording sites were positioned in the midline below the junction of the gastrocnemii for the SO, and over the bellies of the GL, gastrocnemius medialis (GM) and tibialis anterior muscles. A cuff-type ground electrode was placed below the knee, above the EMG electrodes. Amplified EMG signals (CMRR: 120 dB; Z_{in}: 2 GW; gain: 1000), with a frequency band ranging from 10 to 1000 Hz, were used to record both the clonus and the reflexes. The data were digitized on-line at a sampling frequency of 5000 Hz using a data acquisition card mounted in a 386 IBM compatible computer and transferred to a digital computer to be processed.

The experimental procedures for reflex testing have been previously described in detail (13). H-reflexes were evoked every 30 seconds by stimulating the posterior tibial nerve in the popliteal fossa, using a 1 ms rectangular pulse (Grass stimulator; 14). The anodal electrode ($9 \times 12\,cm^2$) was placed above the patella, on the quadriceps muscle. The intensity of the test stimulus was chosen to elicit a peak-to-peak amplitude of approximately half the maximal SO H-reflex amplitude ($H_{max}/2$) as well as a small muscle compound action potential (M response) to monitor the consistency of the stimulation conditions (29).

After stabilizing the physiological response by evoking H-reflexes for 10 minutes, the maximal SO H-reflex (H_{max}) and M response (M_{max}) were measured. H-reflexes ($H_{max}/2$) were then recorded for 5 minutes before (control) and during the 30 minutes of TENS application. Once the TENS was discontinued, the reflex responses were recorded for a further 10 minutes before measuring the post-treatment SO H_{max} and M_{max} values.

For each muscle, an estimate of H-reflex amplitude was calculated every 5 minutes by averaging 10 consecutive H-reflex responses. Within each treatment period, estimates of peak-to-peak H-reflex amplitudes during (T5 to T30) and after (P5 and P10) TENS were normalized by expressing them as a percentage of the control estimate (Ctrl) to allow for within-treatment comparison. Pre- and post-TENS H_{max}/M_{max} ratios were also computed.

Clinical assessment

Considering the reflex hyperexcitability, as well as the enhanced muscle tone characterizing spasticity (16), the clonus, Achilles tendon reflex (ATR) and modified Ashworth (4, 6) tests were used to "clinically" describe the plantarflexor spasticity at the ankle. Summation of these item scores generated a global (composite) spasticity score ranging from 0 to 16 (20). High reproducibility of these clinical ratings of spasticity (Intra Class Correlation coefficient of 0.87) was previously shown in hemiplegic subjects tested on different days (21). The clinical evaluations were performed by a senior physical therapist, pre- and post-TENS, with the subjects still sitting in the experimental apparatus. The therapist was instructed to evaluate the clonus, ATR and resistance to passive movement of the ankle, as she normally would in her clinical practice. Clonus was scored on a four-point scale where 1 denoted no clonus and 4, sustained clonus. The ATR was assessed using a five-point scale where a score of 0 indicated the absence of response and a score of 4 described a maximally hyperactive response. Finally, muscle tone of the ankle extensor was graded on a double weighted modified Ashworth scale (4, 6, 20). A score of 0 meant no increase in muscle tone, whereas 8 corresponded to severe increase in tone of the affected limb (20).

Statistical analysis

The averaged SO M_{max} amplitude and the skin T° obtained at the beginning and at the end of the experimental session were compared (paired t-tests; $a = 0.05$) in order to verify the constancy of the recording conditions throughout the session.

Considering the free distribution of the data, non-parametric Friedman ANOVAs (37) were utilized to determine the presence of significant within-treatment effects ($\alpha = 0.05$), across subjects, on the H-reflex amplitude of a given muscle. In order to decrease the number of variables relative to the number of subjects, only the Ctrl, T10, T20, T30, and P10 estimates were compared within-treatment to determine whether the H-reflex amplitude of a given muscle was affected by transcutaneous stimulation over time. Non-parametric Wilcoxon signed-ranks tests (32) were used to evaluate any pre- and post-TENS changes on the clinical scores (clonus, ATR, Ashworth, spasticity) as well as on the H_{max}/M_{max} ratios.

Correlation analyses were used to establish the linear relationship between electrophysiological and clinical indicators of spasticity. In order to estimate the internal consistency (average correlation of an item with the other items in the scale) of the clinical scores (clonus, ATR and modified Ashworth), the reliability coefficient alpha (Cronbach's a) was determined from both the pre- and post-TENS scores (8, 24). Principal components analyses were also performed to establish the extent to which each clinical item contributed to the composite spasticity score (24, 25).

RESULTS

The averaged scores (±s.e.) of different spasticity variables obtained pre- and post-TENS as well as the results of Wilcoxon signed-ranks tests ($p < 0.05$) are summarized in Table II.

Electrophysiological indicators of spasticity

In 5 of the 14 subjects, either no reflex could be elicited or the H-reflex could not be stabilized. H-reflex data were thus collected and analysed in 9 subjects. For those 9 subjects, the small muscle compound action potential elicited by the test stimulus remained relatively stable throughout the experimental session. Paired t-tests failed to reveal any significant differences either between the SO M_{max} responses or skin temperature values obtained at the beginning and at the end of the experiment ($p > 0.05$).

Fig. 1 depicts the mean modulation of the triceps surae muscles H-reflex amplitude during 30 minutes of TENS applied over the CPN. Although the GM H-reflex tended to decrease, non-parametric Friedman

Table II. *Averaged scores (±s.e.) of different spasticity indicators obtained pre- and post-TENS, and results of Wilcoxon signed-ranks tests*

Variables (*n*)	Pre-TENS (s.e.)	Post-TENS (s.e.)	*p*-value
Clonus (14)	2.8 (±0.3)	2.5 (±0.3)	0.11
ATR (14)	2.9 (±0.3)	2.3 (±0.3)	0.01*
Ashworth (14)	3.1 (±0.4)	2.4 (±0.4)	0.04*
Spasticity (14)	8.7 (±0.8)	7.2 (±0.8)	0.01*
H-reflex (9)	1.0 (±0.0)	0.9 (±0.1)	0.89
H_{max}/M_{max} (8)	0.7 (±0.1)	0.7 (±0.1)	0.50

ATR = Achilles tendon reflex.
s.e. = standard error.
*$p < 0.05$.

ANOVAs did not reveal any significant effects of repetitive cutaneous stimulation on the H-reflex amplitude (χ^2 values of 2.9, 3.6 and 4.6 for the SO, GL and GM respectively; $p > 0.05$). However, as demonstrated in Fig. 2, there was, amongst subjects, a wide range of responses of the H-reflex amplitude to repetitive low-threshold afferents stimulation. While the reflex in one subject was inhibited, that of another was either non-responsive to or facilitated by the TENS. The inter-subject variability could thus explain the absence of overall significant treatment effects on this electrophysiological variable. Similarly, no difference in pre- and post-TENS H_{max}/M_{max} ratios was disclosed by the Wilcoxon signed-ranks test ($p = 0.5$).

Clinical indicators of spasticity

Wilcoxon signed-ranks tests revealed significant effects of TENS on the ATR ($p = 0.01$), the modified Ashworth test ($p = 0.04$) as well as on the composite spasticity ($p = 0.01$) score (Table II). Although there was no significant difference ($p = 0.1$) in the clonus scores pre- and post-TENS, a strong tendency toward a decrease in clonus was observed after 30 minutes of stimulation. The statistically significant response of the global spasticity score to TENS seems more reflective of the effects of repetitive stimulation on the ATR and muscle tone (as evaluated by modified Ashworth) rather than those on the clonus.

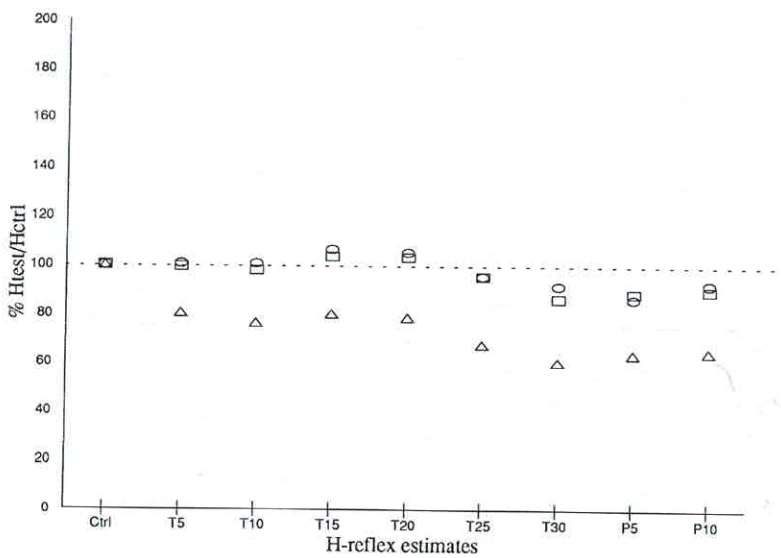

Fig. 1. Averaged modulation ($n = 9$) of the soleus (□), gastrocnemius lateralis (○) and gastrocnemius medialis (△) H-reflex amplitude 5 minutes prior to (Ctrl), during 30 minutes of (T5 to T30) and 10 minutes post (P5 and P10) TENS applied over the common peroneal nerve.

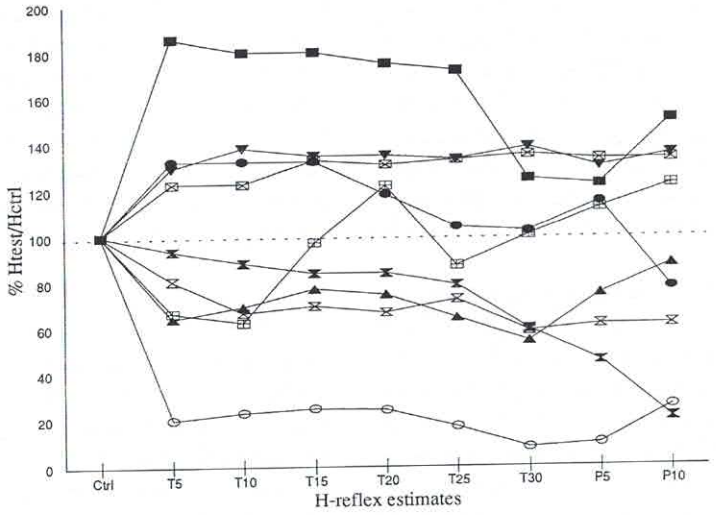

Fig. 2. Inter-subject variability depicted by the modulation of soleus H-reflex amplitude, in nine spinal cord-injured subjects, 5 minutes prior to (Ctrl), during 30 minutes of (T5 to T30) and 10 minutes post (P5 and P10) TENS applied over the common peroneal nerve.

Correlation analyses were done to better delineate the relationship between the electrophysiological and clinical indicators of spasticity. An inter-correlation matrix shown in Table III was drawn from the computed correlation analyses. No significant ($p > 0.05$) correlations between the H-reflex amplitude and any clinical indicators of spasticity were disclosed. From Table III, it is also obvious that all clinical measures are highly inter-correlated ($p < 0.005$) indicating that the severity of clonus varied with that of the muscle tone and of the ATR. The global spasticity score, a composite sum of the scores of three clinical evaluations, is also significantly correlated ($r = 0.94$, 0.95, 0.89) with each of its components. As reflected by Cronbach's α of 0.89 and 0.92 obtained from the pre- and post-TENS data respectively, the level of internal consistency is also very high between the different clinical tests (8, 24).

The principal-component analyses performed separately on the pre- and post-TENS data revealed a contribution of clonus, ATR and muscle tone by relating these three items into one factor which explains, in each data set, 86% (eigenvalue of 2.6) of the total variance in the data (25). It could thus be postulated, at a conceptual level, that this one-factor model could fit the concept of spasticity clinically evaluated, hereby defined as the summation of the clonus, ATR and modified Ashworth scores.

DISCUSSION

The present study failed to reveal any significant effects of TENS on H-reflex amplitude in spinal cord-injured subjects. The tendency toward a decrease in SO H-reflex amplitude during TENS observed in

Table III. *Inter-correlation matrix of the regressions performed between the clinical and electrophysiological indicators of spasticity obtained post-TENS*

Variables (n)	Clonus	ATR	Ashworth	Spasticity	H-reflex	H_{max}/M_{max}
Clonus (14)	1.00					
ATR (14)	0.88*	1.00				
Ashworth(14)	0.73*	0.77*	1.00			
Spasticity (14)	0.94*	0.95*	0.89*	1.00		
H-reflex (9)	0.36	−0.01	0.22	0.23	1.00	
H_{max}/M_{max} (8)	−0.40	0.19	−0.49	−0.33	−0.13	1.00

ATR = Achilles tendon reflex.
* $p < 0.05$.

healthy subjects (9, 11, 13, 27) could not be reproduced in spinal cord-injured subjects. Moreover, the reduction in clonus post-TENS previously described by Walker (35, 36) could not be reproduced in most subjects. However, there was a significant decrease in ATR, modified Ashworth and global spasticity scores. The latter results suggest that TENS could be effective in reducing clinical spasticity in spinal cord-injured subjects.

Considering both the reflex hyperexcitability displayed in spinal cord-injured subjects (19, 20, 22, 33) and the trends toward a decrease in SO H-reflex amplitude observed in healthy subjects during repetitive low threshold stimulation (9, 11, 13, 27), it was hypothesized that TENS would also have inhibitory effects on H-reflex amplitude in that population. The stability of both the skin temperature (2) and the M responses throughout a session (29) strongly suggests that modulation in the H-reflex amplitudes could be attributed to treatment effects rather than to variations in the testing conditions. However, due to wide inter-subject variability (Fig. 2), no overall significant effects of TENS on either the H-reflex amplitude during the stimulation or the H_{max}/M_{max} ratios obtained pre- and post-TENS were revealed. These findings are similar to previously published results in hemiparetic subjects failing to show, after 45 minutes of TENS, significant changes in vibratory inhibition of the SO H-reflex or in H_{max}/M_{max} ratios (15). The intra-subject variability may have further contributed to the overall absence of significant effects on H-reflex amplitude during TENS. The H-reflex stimuli was observed, at times, to elicit flexor or extensor spasms in some subjects. Moreover, as the subjects were instructed not to take their antispasmodic medication within the hour prior to the experimentation, some subjects had spasms during the course of data collection. Although, in healthy subjects, the stability of the H-reflex amplitude during a control session suggested that modulations observed during TENS consisted of treatment effects (11), the intra-subject variability of the H-reflex amplitude in some spinal cord-injured subjects may have been a confounding factor in the experimental design.

The present correlational data also support previous findings in hemiparetic subjects (15). The correlation analyses failed to reveal any significant correlations between the H-reflex amplitude or H_{max}/M_{max} ratio with the clonus, ATR, muscle tone (modified Ashworth) or global spasticity scores (Table III). Consequently, although the use of H-reflex to assess treatment effects on spasticity is still widely reported (10, 18, 28), the presence of wide inter- and intra-subject variability of H-reflex response to treatment as well as the absence of correlation with clinical indicators of spasticity question the value of H-reflex amplitude as an electrophysiological indicator of spasticity in clinical studies.

Clinical assessment of spasticity

The Cronbach's α computed to estimate the internal consistency of clinical scores provided a lower border of the reliability of the composite spasticity score of approximately 0.90. Consequently, the reliability coefficient for the clinical evaluation is equal to or greater than 0.90. Furthermore, the principal components analysis indicates that, indeed, it was useful to combine the clonus, ATR and modified Ashworth scores, said to reflect one factor, so as to arrive at a composite score which better describes the spasticity clinically evaluated. These results thus support the use of a global spasticity score such as described by Levin & Hui-Chan (20). However, it is important to keep in mind that the principal component analysis procedure was only exploratory and, consequently, may only serve as a basis for the formulation of hypotheses to be tested in a newly designed study using confirmatory factor analysis as well as a better defined theoretical context and a much larger sample of subjects.

The present study revealed, in spinal cord-injured subjects, a significant decrease in ATR and modified Ashworth scores reflected by a significant difference in the global spasticity score after 30 minutes of repetitive low-threshold cutaneous stimulation by TENS. The clonus decreased post-TENS in most subjects although not in a significant manner. Levin & Hui-Chan (20) also reported, in hemiparetic subjects, a reduction in clinical spasticity score as well as an improved control of motor functions after 2 weeks' daily applications of TENS. Considering the significant increase in vibratory inhibition observed in hemiparetic subjects (20), an increase in presynaptic inhibition (3) following repetitive low-threshold afferents stimulation could account for the decrease in clinical spasticity. Moreover, the absence of significant effects of TENS on H-reflex amplitude, although a significant reduction in clinical spasticity score was obtained, might reflect influences of repetitive stimulation of low-threshold afferent on the sensitivity of

the spindle receptors which are bypassed for the elicitation of the H-reflex. It now seems to be demonstrated that alpha and gamma motoneurons are controlled by pathways largely independent of each other (30). Since low-threshold afferents project to gamma motoneurons and modify their excitability, it is possible that TENS indirectly influences the responsiveness of the spindle receptors through its effects on gamma motoneurons. This would explain that the scores of the spasticity indicators involving changes in muscle length (ATR, modified Ashworth and clonus) were reduced while no significant decrease in H-reflex amplitude or H_{max}/M_{max} ratio was found.

The absence of a significance change of clonus in spite of a significance in Achilles tendon reflex could be explained by the fact that, if the dorsiflexion starting the clonus is increased, a sustained clonus can be elicited even after a relatively marked increase of its threshold. In the present study, clonus was subjectively evaluated as is commonly done in the clinical setting using an ordinate scale from 1 to 5. The clonus could have been more precisely assessed by determining its threshold for elicitation or by controlling the velocity and range of motion within which it was elicited. A large amplitude of movement may elicit sustained clonus even after its threshold is increased. Furthermore, it might also be assumed that repetitive stimulation of low-threshold afferent might decrease the sensitivity of the muscle spindles to phasic stretch, such as reflected by the significant reduction in ATR and modified Ashworth scores. It is now well acknowledged that there is no conclusive evidence of increased fusimotor drive in spastic patients (7). However, it is possible that an excessive muscle spindles responsiveness resulting from changes in the extrafusal muscle fibers would contribute to the increased muscle tone (7). The absence of significant effects on the clonus post-TENS supports a stronger effect of repetitive stimulation of low-threshold afferents on the dynamic fusimotor activity.

Clinical implications

The effects of short-term conditioning of low-threshold afferents on the recovery profile of the SO H-reflex have been extensively studied in man to determine both the effects and the time course of effects of cutaneous stimulation on motoneuronal excitability. TENS has been shown to have longer lasting modulatory effects on the SO H-reflex (9, 20, 27). Based on the tendency toward a decrease in SO H-reflex amplitude during TENS observed in healthy subjects (9, 11, 13, 27), and on the reflex hyperexcitability associated with spinal spasticity (19, 22, 33), the present study was aimed at investigating the effects of repetitive low-threshold cutaneous stimulation on H-reflex in spinal cord-injured subjects. Albeit interesting outcomes of the effects of repetitive stimulation of low-threshold afferent on clinical spasticity which support previously published results (1, 5, 20), the present results can only suggest that TENS appears to be effective in reducing spinal spasticity as measured clinically. This study cannot readily infer a causality between the application of TENS and the subsequent reduction in spasticity scores considering that other potentially confounding factors could have been responsible for the reduction in spasticity scores observed, post-TENS, in spinal cord-injured subjects. For this reason, the present study can only be considered as a pilot study. Further clinical studies, designed with control groups receiving placebo treatments, are needed to corroborate the present findings on the effects of TENS on spinal spasticity as well as to verify whether such a decrease in clinical spasticity has any functional outcomes.

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