

Antispastic Effect of Penile Vibration in Men With Spinal Cord Lesion

Line Læssøe, MD, Jens Bo Nielsen, Fin Biering-Sørensen, MD, Jens Sønksen, MD

ABSTRACT. Læssøe L, Nielsen JB, Biering-Sørensen F, Sønksen J. Antispastic effect of penile vibration in men with spinal cord lesion. *Arch Phys Med Rehabil* 2004;85:919-24.

Objective: To evaluate the possible antispastic effect of penile vibratory stimulation (PVS) in men with spinal cord lesion (SCL).

Design: Unblinded, before-after trial.

Setting: Ambulatory care.

Participants: Nine men with SCLs from C2 to T8 were randomly allocated into 2 groups.

Intervention: Twenty-four hours of electromyographic recordings from the quadriceps and tibialis anterior muscles were taken, followed by PVS or no treatment and another 24 hours of electromyographic recordings. The presence of electromyographic activity of an amplitude 4 times the baseline, with a duration of more than 5 seconds, was taken to signify a spasm. The number of spasms per hour was calculated before and after PVS and no treatment. Spasticity was evaluated by the Modified Ashworth Scale (MAS).

Main Outcome Measure: Reduction in spasticity and spasms.

Results: The electromyographic data showed a significant reduction in the frequency of leg spasms up to 3 hours ($P < .05$). Significantly decreased spasticity, as evaluated by MAS, was found immediately after vibration ($P < .01$).

Conclusions: PVS may be useful as an antispastic therapy.

Key Words: Ejaculation; Electromyography; Rehabilitation; Spasm; Spinal cord injuries; Vibration.

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MEN WITH SPINAL CORD LESION (SCL) often have severe disabilities, such as urinary, bowel, and sexual dysfunction; inability to walk; and spasticity and/or spasms in the lower extremities. The disability depends on the location and completeness of the SCL. Spasticity is clinically characterized by increased muscle tone and hyperactive tendon reflexes. Spasms are sudden, involuntary muscle contractions.

Spasticity and spasms in the legs may limit daily activities for the man with SCL and are usually treated with physiotherapy (PT) and spasmolytic agents.¹ Spasmolytics are often ineffective and have many potential side effects, including

drowsiness. More optimal means of reducing spasticity would therefore be of great value.

Penile vibratory stimulation (PVS) is used by men with SCL to obtain ejaculation for the purpose of fertilization. To obtain reflex ejaculation by penile vibration, it is necessary to use sufficient vibration amplitude (amplitude, ≥ 2.5 mm) and to have an intact reflex arc.² Vibration-induced afferent nerve stimulation is transmitted via the pudendal nerve to the sacral spinal cord (S2-4) and from there to the T10-L2 segments. The efferent innervation of the ejaculatory organs is via the hypogastric and pudendal nerves.³

It has been suggested that electroejaculation may lead to reduced spasticity in men with SCL.⁴ Electroejaculation must be performed by a physician and is therefore not practical for home use. Unlike electroejaculation, PVS is easily applied by the man with SCL himself and can be performed at home as often as wanted.

Because all our experiences related to reduced spasticity after ejaculation induced by PVS, our hypothesis was that ejaculation is necessary to reduce spasticity. We wanted to investigate this systematically; therefore, the penis and not a more convenient site was chosen as the site to stimulate by vibration. General muscle relaxation and reduction of leg spasms after ejaculation induced by PVS have also been reported by men with SCL and observed clinically.⁵ This was also found in the pilot study for our investigation. However, these studies did not provide conclusive evidence of the antispastic effect of PVS. In our study, we monitored leg spasms by electromyographic measurements and evaluated the extent of spasticity using the Modified Ashworth Scale (MAS) before and after penile vibration.

METHODS

Ethics

Our study was approved by the local ethics committee. All volunteers received written and oral information and gave their informed consent. The experiments were performed according to the Declaration of Helsinki and later amendments.

Participants

Nine men with SCL were included. The inclusion criterion was men with SCL who had reported spasticity and/or leg spasms. Their ages ranged from 27 to 67 years. Time since SCL ranged from 4 months to 50 years. Their level of lesion ranged from C2 to T8. Six had motor complete lesions, and 3 were motor incomplete. Completeness of the lesions was classified according to the American Spinal Injury Association (ASIA) Impairment Scale⁶ (table 1).

Design

This was an unblinded, crossover study, and each person served as his own control. All participants were allocated randomly to 2 study groups, with 48 hours of electromyographic measurement from the lower extremities. Initially, in both groups, 24 hours of electromyographic recordings were taken, followed by either PVS or no treatment. Subsequently,

From the Department of Urology and Clinic for Para- and Tetraplegic, Rigshospitalet (Biering-Sørensen); Copenhagen University Hospital, Copenhagen (Læssøe, Sønksen); and Department of Neurophysiology, Panum Institute, University of Copenhagen, Copenhagen (Nielsen), Denmark.

Supported by Multicept A/S, Hørsholm, Denmark.

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit on the author(s) or on any organization with which the author(s) is/are associated.

Reprint requests to Line Læssøe, MD, Dept of Urology 2112, Rigshospitalet, University of Copenhagen, Blegdamsvej 9, DK-2100 Copenhagen, Denmark, e-mail: llaessoe@rh.dk.

0003-9993/04/8506-8311\$30.00/0

doi:10.1016/j.apmr.2003.08.082

Table 1: Characteristics of the Men With SCL Participating in the Project, Including Use of Spasmolytic Agents, Prophylactic Nifedipine During the PVS, and Whether Antegrade Ejaculation Was Obtained During Stimulation

Person	Age (y)	Time Since Lesion	Level and Type of Spinal Cord Lesion*	Type of Lesion	Spasmolytic Agents	Prophylactic (nifedipine)	Antegrade Ejaculation
1	27	1y	C4, class A	Traumatic	Yes	Yes	Yes
2	28	4mo	T6, class A	Traumatic	Yes	No	No
3	50	5mo	T6, class D	Nontraumatic	Yes	Yes	No
4	34	16y	C5, class A	Traumatic	Yes	Yes	Yes
5	67	50y	C2, class C	Nontraumatic	Yes	No	No
6	35	8y	T2, class A	Traumatic	Yes	Yes	Yes
7	46	28y	T6, class D	Traumatic	Yes	No	Yes
8	34	14y	C6, class A	Traumatic	No	No	No
9	29	24y	T8 plus cerebral contusion, class A	Traumatic	Yes	Yes [†]	No

*ASIA classification⁶: class A, complete (no motor or sensory function is preserved in the sacral segments S4–5); class C, incomplete (motor function is preserved below the neurologic level, and more than half of key muscles below the neurologic level have a muscle grade <3); class D, incomplete (motor function is preserved below the neurologic level, and at least half of key muscles below the neurologic level have a muscle grade ≥3).

[†]Subject 9 stated that his level of lesion was C6; therefore, he received prophylactic nifedipine. Afterward the level was found to be T8, according to his medical record.

another period of 24 hours of electromyographic recording was done. After at least 1 week, the 48 hours of electromyographic measurement were repeated. However, those men who had PVS received no treatment and those men who had no treatment received PVS (fig 1).

Penile Stimulation by Vibration

A handheld vibrator^a was used; on this vibrator it is possible to read the exact vibration amplitude (millimeters) and frequency (hertz). Ejaculation was the desired endpoint, so the penis was the site to stimulate. Stimulation was performed using a vibrating disk (diameter, 3.5cm) made of hard plastic placed against the frenulum of the penis until antegrade ejaculation was obtained or for a maximum stimulation period of 5 minutes. No investigation was made to find out if retrograde

ejaculation had occurred. An amplitude of 3.0mm, with a frequency of 100Hz, was used.² The only major complication of PVS is the risk of autonomic hyperreflexia in persons with a high-level (above T6) lesion. This condition was prevented by using a calcium-antagonist (10–20mg of nifedipine)⁷ prophylactic, applied sublingually 15 to 20 minutes before the vibration procedure in all SCL men with a high-level lesion, unless they were familiar with the PVS procedure and had no signs of autonomic dysreflexia.

Electromyographic Measurements of the Spasm Frequency in the Lower Extremities

Electromyographic activity was recorded using monopolar surface electrodes (Blue sensor, disposable electrodes, type NF-50-K; quantity, 12) placed over the bellies of the quadriceps femoris and tibialis anterior muscles bilaterally. The reference electrode was placed over the major trochanter on the left side. The signals were sampled and amplified (1000–5000×) using a Biosaca^b ambulatory, 8-channel, electromyography recorder. The sampling frequency rate was 128Hz. A 10-Hz high-pass filter was applied before subsequent data analysis. All wires from the electrodes to the amplifier were taped^c to the skin, to minimize movement artifacts. The surface electrodes remained in the same position during the 48 hours of recording sessions. The electromyography recorder was placed in a small bag, which hung on the wheelchair or lay on a table near the bed. The men with SCL were all encouraged to maintain their daily activities with a normal level of physical activity, including PT. Also, they were told to keep a diary and to include all incidents that differed from their normal program, such as extraordinary physical activity or hours spent at rest in their bed. The medication was kept constant during the study period.

To analyze the electromyographic recordings, software built for that purpose (programmed in Matlab^d) was used to automatically detect the occurrence of electromyographic activity in any of the 4 leg muscles from which measurements were taken. The chosen criterion for a spasm was electromyographic events with activity exceeding 4 times the baseline and with a duration of more than 5 seconds. It was subsequently verified visually whether the identified electromyographic activity reflected genuine electric activity from the muscles or artifacts due to movement of the electrodes or connecting wires.

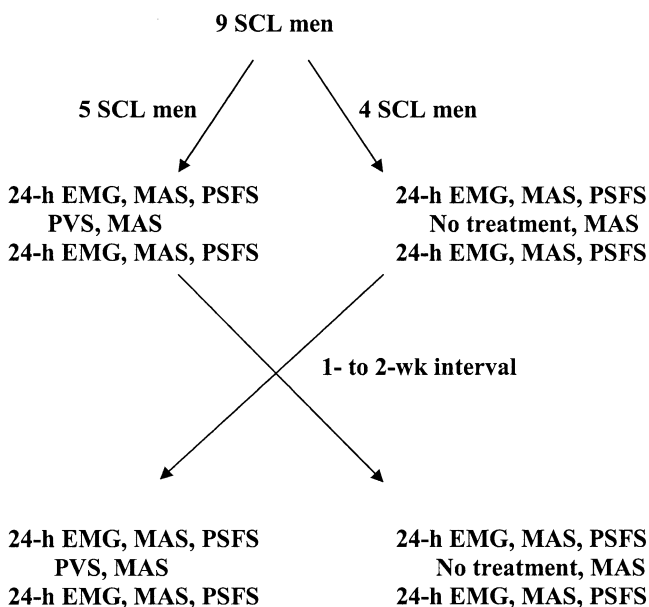


Fig 1. Design of the study. Men with SCL were evaluated with electromyographic (EMG) recordings, the Modified Ashworth Scale, and the Penn Spasm Frequency Scale (PSFS).

Table 2: Clinical Assessment of Spasticity Using the MAS, Before PVS and No Treatment, Immediately After, and 24 Hours After PVS and No Treatment

Person	MAS*					
	Before PVS	Immediately After PVS	24 Hours After PVS	Before No Treatment	Immediately After No Treatment	24 Hours After No Treatment
1	3	0	2	3	3	4
2	3	2	3	1	1	1
3	4	1	3	3	4	4
4	3	2	2	5	4	4
5	1	0	2	3	3	2
6	1	0	1	1	1	1
7†	0	0	0	0	0	0
8	4	3	3	3	3	3
9	4	1	4	4	4	4
Mean	2.6	1.0‡	2.2	2.6	2.6	2.6

NOTE. The assessment was a total evaluation of the muscle tone in the flexors and extensors of the knee and ankle.

*Range of the MAS is 0 to 5: 0, no increase in muscle tone; 1, slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion (ROM) when the affected part is moved in flexion or extension; 2, slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM; 3, more marked increase in muscle tone through most of the ROM, but affected part easily moved; 4, considerable increase in muscle tone, passive movement difficult; 5, affected part rigid in flexion or extension.

†Subject 7 was clinically graded 0 on all occasions, but self-reported spasticity nevertheless.

‡ $P = .009$.

Clinical Assessment of Spasticity

A clinical assessment of the spasticity was made by the same physician, according to the MAS⁸ (table 2) at study entry (baseline), 24 hours immediately after PVS or no treatment, and again after 48 hours. The whole assessment included an evaluation of the muscle tone in the flexors and extensors of the knee and ankle.

In addition, the men with SCL gave their subjective evaluation of the effects of the treatment using the Penn Spasm Frequency Scale⁹ (PSFS) (table 3) to grade the spasm frequency.¹⁰ This evaluation was conducted for the first 24 hours before PVS or no treatment and then again 24 hours after this.

Role of the Funding Source

This study was a part of a doctoral project funded by Multiccept A/S. The funding source had no involvement in the study design; in the collection, analysis, and interpretation of data; in

the writing of the report; or in the decision to submit the article for publication.

Statistics

Because initial analysis showed that the effect of treatment was seen only within 3 hours of treatment, the statistical analysis was restricted to 5 hours before and after treatment.

Statistically significant changes in the number of electromyographic bursts, in MAS scores, and in PSFS scores after penile vibration were determined using the Wilcoxon signed-rank test.

A P value of less than .05 was considered significant.

RESULTS

The electromyographic recordings from subject 8 failed because of a technical problem. The recordings from the remaining 8 men with SCL were technically satisfactory.

The analysis software detected an average of 30 ± 21 electromyographic events per hour in the 8 men with SCL before vibration. The number of electromyographic events varied very significantly from 1 hour to the next in the same subject (mean variability, 10 electromyographic events/h) as well as between subjects (mean variability, 28 electromyographic events/h). Nevertheless, in all 8 men, there was a reduction in the mean number of electromyographic events in the initial 3 hours after vibration, as compared with the mean number of electromyographic events before vibration, and this reduction was statistically significant ($P < .05$). In figure 2A, the mean number of electromyographic events per hour in the 8 subjects in the first 5 hours after vibration is expressed as a percentage of the mean number of electromyographic events per hour before vibration. The largest reduction occurred in the first hour after vibration, after which the events gradually decreased until no significant effect was observed in the third hour after vibration. A similar reduction was not observed after no vibration (fig 2B).

The clinical evaluation showed a significant decrease in muscle tone after PVS, as evaluated by the MAS ($P < .01$) (table 2). When the subjects were clinically evaluated again 24 hours later, this reduction in muscle tone had vanished. The

Table 3: Evaluation of Men With SCL of the Effects of Treatment, Using the PSFS to Grade Spasm Frequency

Person	PSFS*			
	Before PVS	24 Hours After PVS	Before No Treatment	24 Hours After No Treatment
1	3	3	3	3
2	4	3	3	4
3	3	3	3	4
4	4	2	4	4
5	3	3	3	3
6	3	3	3	3
7	3	3	3	3
8	2	3	4	4
9	3	2	4	3
Mean	3.1	2.8	3.3	3.4

*Range of the PSFS is 0 to 4: 0, no spasms; 1, mild spasms induced by stimulation; 2, infrequent full spasms occurring $< 1/h$; 3, spasms occurring > 1 but < 10 times/h; 4, spasms occurring > 10 times/h.

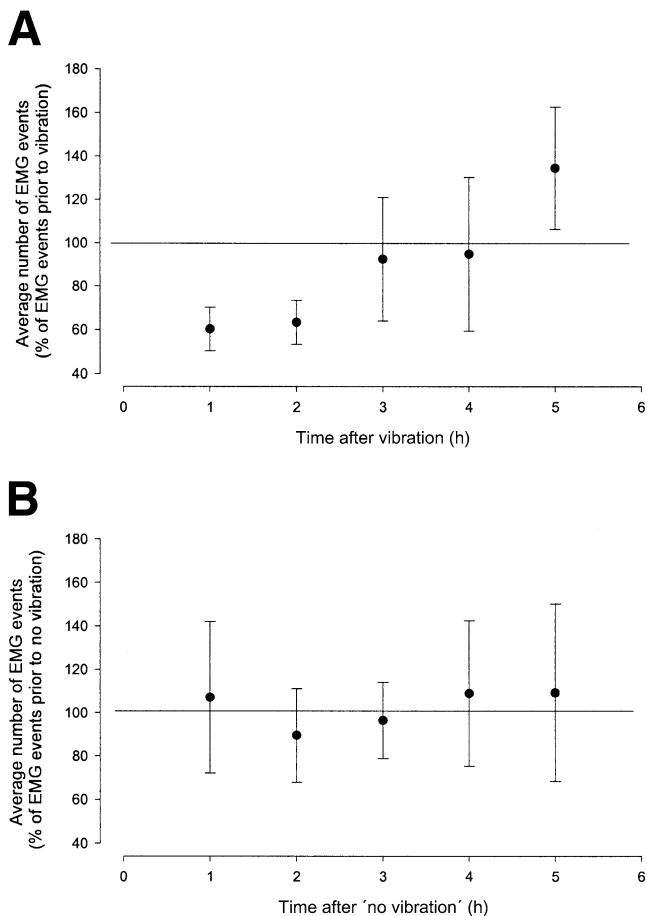


Fig 2. Decrease in the number of electromyographic events after vibration in men with SCL. The graphs show the average number of electromyographic events (defined as electromyographic activity exceeding 4 times the baseline activity and lasting >5s) as a percentage of the control number of events after either (A) vibration or (B) no vibration in the 8 men with SCL. The number of electromyographic events per hour was calculated for each man with SCL in a control period before either vibration or no vibration. The number of electromyographic events for each hour after vibration or no vibration was then expressed as a percentage of this control number of electromyographic events. Finally, the data from all subjects were pooled to obtain the graphs. The vertical bars signify 1 standard error of the population mean.

subjects spontaneously reported that they experienced a relaxation in the legs and a reduction in the spasm frequency after vibration, and there was a tendency toward a decrease in the number of spasms according to the PSFS. However, this did not reach statistical significance ($P=.26$) (table 3).

DISCUSSION

In our study, we showed that PVS produces a significant reduction in the number of involuntary bursts of electromyographic activity and muscle tone in leg muscles. Subjects also reported that their leg muscles were more relaxed immediately after vibration and that the number of spasms was reduced. When subjects used the PSFS for more than 24 hours, this reduction did not reach statistical significance.

Many subjects scored 3 on the PSFS (corresponding to >1 spasm to ≤ 10 spasms/h) both before and after PVS, yet at the same time all subjects experienced a reduction in the number of spasms, although it was not sufficient to change their scoring

on the PSFS. A 3 on the PSFS may conceal a marked reduction in the number of spasms (eg, a reduction from 10 to 6 spasms/h, corresponding to the 40% reduction observed in the number of electromyographic events) (fig 2A). We therefore believe that the most likely reason why we found no significant reduction in the number of spasms when using the PSFS is that it provides too crude a measure of the number of spasms and does not precisely enough reflect the experience of the subjects.

Measuring the number of involuntary electromyographic events provides an alternative, objective source of information about spasm frequency. However, this method is not without problems. First, it is unclear as to the extent to which the electromyographic activity reflects the clinically perceived spasms. In many cases, electromyographic activity was likely insufficient to produce an actual movement of the leg or the muscle and therefore insufficient to produce a clinical spasm. The fact that most men with SCL scored 3 on the PSFS, although the program detected on average 30 electromyographic events per hour, confirms this. Furthermore, we were able to record only from 4 muscles, and it may be that the clinically perceived spasms did not involve activation of these specific muscles. Although the electromyographic events provide information about involuntary muscle activity, they do not reflect exactly the clinically perceived spasm activity.

A second problem with the electromyography method, in the way we used it, is that we had no control of the factors that might have provoked the electromyographic activity in the men with SCL. They were instructed to maintain the same daily activities on each of the 4 days during which recordings were made, and they were asked to keep a diary to document this; otherwise there was no surveillance of the subjects during the electromyographic measurements. Some of the electromyographic activity was probably provoked by external factors—such as toilet visits, movement from wheelchair to bed, and handling by health care personnel—and the diaries provide some information about this. However, it was not possible to establish a clear correlation between such factors and the electromyographic events. What is important for this study is that there was no difference in the number of these external stimuli on the 4 days of the study. Furthermore, although we cannot fully exclude this possibility, we find it unlikely that a change in the number and/or nature of external stimuli should be responsible for the observed reduction in the number of electromyographic events after PVS.

A third problem with the electromyography method is the very large variability. It was not surprising to find a large interindividual variability, because it is well known that spasm frequency may vary significantly among men with SCL. It was, however, more surprising that the number of electromyographic events varied markedly from 1 hour to the next in the same person without any evident change in the number of external stimuli, as documented by the diaries that the subjects kept. This variability makes it difficult to use the method to evaluate the effect of therapeutic interventions, and it explains why a significant reduction in the spasm frequency was not found in individuals but was found only when pooling the data from all 8 subjects. At the same time, the fact that we observed a statistically significant reduction, despite the large variability within and between individuals, suggests that the reduction in the number of electromyographic events, induced by PVS, was indeed of a very significant magnitude (average 40% reduction in the 8 subjects).

The reduction in muscle tone, as evaluated by the MAS, was also very remarkable, corresponding on average to a normalization of the muscle tone from a state of clearly pathologically increased muscle resistance. The MAS is widely accepted as

the most optimal scale for evaluation of the extent of muscle tone, although it is not without problems. In relation to our study, the main problem with the MAS is that the evaluation of muscle tone is based on the examining clinician's evaluation of the response to passive manipulation of a person's legs. A precise estimate based on this evaluation requires a very experienced examiner, and expectations of the outcome may easily influence the evaluation. Optimally, the evaluation should be performed by a clinician who was blinded to whether the person had the stimulation intervention or not, or alternatively, the muscle tone should be objectively evaluated by biomechanic measurements, but this was not possible in our study. Nevertheless, given the very significant reduction in the MAS score and the agreement with the changes in the electromyographic events and the person's subjective perception, we believe that the change in the MAS score reflects a reduction in spasticity.

In 5 men (subjects 2, 3, 5, 8, 9) antegrade ejaculation did not occur by PVS after 5 minutes of stimulation, and no investigations were made to confirm whether retrograde ejaculation had occurred. In these cases, the desired endpoint of stimulation leading to an antegrade ejaculation was not reached, but in all cases a reduction in spasticity was nevertheless found by the MAS. The best antispastic effect was seen in the tetraplegic persons with complete lesions—the same group that was able to ejaculate by PVS most often and that had the most severe spasms and spasticity in the lower extremities. Whether it is necessary to stimulate until an antegrade ejaculation is unknown and requires further investigation, but the results indicate that a more distinct effect is obtained when that point is reached.

The mechanism for the antispastic effect of PVS is an intriguing aspect of our study. One possibility is that a humoral factor is released in relation to ejaculation, which exerts a general muscle relaxant effect. Identification and isolation of this factor could be of importance in the therapy of spasticity. Alternatively, and probably more likely, the afferent nerve activity generated by vibration and/or the neuronal activity generated in relation to ejaculation may have influenced the neuronal circuits in the lumbar spinal cord, which are involved in the pathophysiology of spasticity. In the cat, pudendal afferents have been shown to project to spinal neurons involved in the control of leg muscles.^{11,12} Several different mechanisms have been suggested to be involved in the pathophysiology of spasticity, including decreased presynaptic inhibition of primary afferents,^{13,14} decreased reciprocal inhibition,¹⁵ and increased Ib excitation.¹⁶ It would seem likely that the activation of pudendal afferents elicits changes in the transmission in these spinal pathways and thereby reduces spasticity. Activation-dependent changes in transmission in the pathway-mediated disynaptic reciprocal inhibition has been shown.^{15,17} Crone et al¹⁵ observed that spastic patients who received chronic stimulation of the peroneal nerve tended to have larger reciprocal inhibition of the soleus muscle evoked by stimulation of the peroneal nerve than did patients who received no chronic stimulation, and they suggested that this reflected an activity-dependent increase in the transmission in the pathway. Our observations may reflect a similar strengthening of inhibitory pathways. We aim in future studies to investigate the significance of this for the observed antispastic effect of PVS, by measuring the transmission in various spinal pathways before and after PVS.

Regardless of the mechanism underlying the observations, the main observation of our study is that PVS may be an efficient way of reducing spasticity in men with SCL with no or very few side effects. Our study indicates that the antispastic

effect of PVS lasts for about 3 hours, but it also raises the possibility that longer-lasting effects may be observed when PVS is repeated. This is also an issue that we aim to investigate in future studies.

Because it was anticipated that a similar effect would be seen in women with SCL, after vibration on the clitoris, as was seen in men after PVS (ie, contractions of the abdominal muscles, gooseflesh, limb movements), the study was originally planned to include women with SCL. However, women with SCL were generally averse to participating in the study. Because the compliance of women with SCL was so poor, a meeting was arranged, between approximately 20 women with SCL and the persons responsible for the project, to discuss the treatment. The general attitude toward vibration on the clitoris was that it was not an acceptable method to use as a treatment of physical problems, despite the fact that it could be an alternative to spasmolytic drugs. The women with SCL expressed concern that vibration on the clitoris would involve and activate both psychologic and sexual trauma and feelings, and without any offer of therapy to address these issues, they did not feel comfortable with the project. Certainly, these statements should be taken into consideration in the design and planning of future studies of this kind involving women with SCL.

In the end, only 2 women with SCL participated and completed the study. Although the symptom reaction in women with SCL to vibratory stimulation on the clitoris is more difficult to evaluate because there is no ejaculation present as a sign of orgasm, it seemed that the women achieved a reduction in their spasm frequency and spasticity, but this requires further investigation.

CONCLUSIONS

Our study showed a significant effect of PVS on spasm frequency in the lower extremities, evaluated by electromyographic measurements before and after vibration. Furthermore, a clinical effect was found using MAS that reflected reduced spasticity in the lower extremities immediately after PVS. This suggests that PVS may be useful as an antispastic therapy, at least in some men with SCL, and unlike electroejaculation, penile vibration has the advantage of being easily applied by the subject himself at home.

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