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The effect of cooling on muscle co-ordination in spasticity: assessment with the repetitive movement test

J. HARLAAR†*, J. J. TEN KATE†, A. J. H. PREVO‡, T. W. VOGELAAR† and G. J. LANKHORST†

† Department of Rehabilitation Medicine, Free University Hospital, De Boelelaan 1117, 1081 HV Amsterdam, The Netherlands

‡ Rehabilitation Centre, Rembrandtkade 10, Utrecht, The Netherlands

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Abstract

Purpose: Cooling muscles might produce a temporary reduction of spasticity. This study investigated muscle co-ordination in spasticity under the influence of cooling.

Methods: A repetitive movement (RM-) test of the ankle was used, while measuring the angle and surface-electromyography (EMG) of the m. tibialis anterior and m. triceps surae. Ensemble averaging provided quantified measures of muscle activation. Sixteen patients with spasticity in their lower extremity due to stroke or spinal cord injury participated in the study. Physical examination and the RM-test was done before and after cooling the m. triceps surae for 20 minutes by cold-packs.

Results: The results show that Achilles hyperreflexia and clonus were abolished in all, and all but one patient, respectively. The EMG of the m. triceps surae, acting as a prime mover, was increased ($p = 0.028$). However, this improved muscle co-ordination resulted in just a slightly increased active range of motion (less than 2 degrees at $p = 0.049$).

Conclusion: Apparently, the increase in excitability of the alpha motoneuron pool in voluntary movements of patients with spasticity is not followed by an improvement in the ability to move.

Introduction

The effect of cooling muscles to temporarily decrease hypertonia in spasticity is a well known phenomenon.[1–3] As such, it is advocated as a component of therapeutic interventions and mainly applied because of its facilitating effect. However, it does not hold for all patients as sometimes after cooling the muscle no decrease in hypertonia is observed.[3] This difference in reaction is sometimes referred to as *cryo-positive* and *cryo-negative*.[4,5] Originally, this so-called cryo-test was thought to identify increased fusimotor drive as the specific pathogenesis of spasticity. However, although the pathophysiological mechanisms underlying spasticity are very complex and still not fully understood, the concept of increased fusimotor drive in spasticity is now obsolete.[6–9]

The excitability of spinal motor neurons is influenced by various descending pathways that either work directly or influence the inhibition or facilitation of the interneurons within spinal reflex pathways.[6,9,10] A decrease in presynaptic inhibition that facilitates the segmental reflex arc might also contribute to spasticity.[10–12] Insofar as these mechanisms contribute to spasticity, their effect is proportional to the amount of afferent input from muscle spindles. Therefore, reducing the sensitivity of the muscle spindles to stretch by cooling[13,14] is likely to be the mechanism which is responsible for reducing hyperreflexia in spasticity.[15]

Up to now the effect of cooling in spasticity has been described mainly in terms of altered T-, H- and M-reflexes. Unfortunately, these results cannot be generalized to impairments in co-ordination of movements under voluntary control. In some subjects with spasticity, involuntary stretch reflexes in antagonist (i.e. lengthening) muscles are inhibited by voluntary effort of the agonist,[16–17] whilst in others the restraining co-contraction is increased.[16,18] Furthermore, decreased spindle-sensitivity might also affect the output of a voluntary shortening muscle. Altogether, the effect of the suppression of spindle activity through cooling in active motions is not straightforward. As voluntary movements are closely related to functional activities, a better understanding of the effects of cooling on voluntary movements may give insight into the mechanisms of spasticity that contribute to functional disability in a patient.

* Author for correspondence; e-mail: j.harlaar@azvu.nl

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Voluntary control implies the involvement of supraspinal processes, so a standardization of supraspinal drive is necessary in order to study the effect of cooling on active motion in spasticity. McLellan and Sahrmann used a simple cyclic motor task to reveal the phase-patterns of contraction and co-contractions in spasticity for the knee and elbow, respectively.[17–19] The aim of the present study was to quantify the muscle activation patterns in a repetitive movement test and, subsequently, to evaluate the different reactions of muscle co-ordination on cooling of the m. triceps surae in spasticity.

Methods and materials

SUBJECTS

Patients who were clinically classified as having hypertonic musculature of the lower extremity combined with a spastic equinovarus of the ankle were selected. However, if the patient could not perform voluntary movement of either knee or ankle, the subject was excluded from participation in the study. Other reasons for exclusion were: suspected pathologies of the peripheral nerve, sympathectomy, allergic reactions to cooling, or the syndrome of M. Raynaud (excessive vasoconstriction as a reaction to the exposure to cold).

The study was approved by the Medical Ethics Committee of the University Hospital and informed consent was obtained from all participants. Sixteen patients (10 male, 6 female) participated in the study, 10 of whom had hemiplegia as a result of stroke. In 6 patients the impaired function was due to dysfunction of the spinal cord (congenital paraparesis, spinal cord injury or MS).

PROCEDURE

Before treatment, a complete physical examination and the repetitive movement test were carried out. Subsequently, the m. triceps surae was cooled by a 20 minute application of cold-packs, while the patient was resting in a comfortable chair.[20] The cold-packs were cooled to a temperature of -12 °C. Skin temperature (at a central location of the muscle belly) was measured by a thermocouple before and immediately after the application. All tests were repeated after the treatment. The whole procedure lasted for approximately one hour.

CLINICAL EXAMINATION

The patient was positioned in a chair with knees and hips at approximately 90° of flexion, and their feet hanging down. Achilles tendon and knee tendon reflexes were tested on the affected side and scored on a 5 point scale. Ankle clonus was rated on a 6 point scale (table 1). Standing and walking was assessed in a qualitative way.

Table 1 Clinical examination scoring scales

	Reflex		*Clonus*
0	No reflex	0	No clonus
1	Light reflex	1	Hard to elicit
2	Normal	2	Present; < 3 repetitions
3	Exaggerated	3	Present; < 10 repetitions
4	Strongly exaggerated	4	Present; $\geqslant 10$ repetitions
		5	Spontaneous

THE REPETITIVE MOVEMENT TEST

The patient was seated in a specially constructed high and stable chair, and was instructed to perform a repetitive maximal dorsal flexion of the foot at the affected side. The movement was self-paced (at a comfortable rhythm) and was performed during 30 seconds. This test was called the – dorsal – repetitive movement test (RM-test). Subsequently, the patient was asked to perform the RM-test in a plantar direction.

The recording of the RM-test involved the instrumentation shown in figure 1. The movement was recorded by means of an electro-goniometer, with two plastic arms fixed to the lateral side of the ankle, the fixed part just above the malleoli and the moving part lateral to the fifth metatarsale. These arms could mutually rotate by a precision turn-potentiometer (linearity: 1% full scale). This variable resistor provides an electrical signal, proportional with the angle of rotation. The axis of rotation was aligned to the distal part of the lateral malleolus. After fixation, but before recording, the offset of the goniometer was calibrated at 0° plantar flexion.

Also during the test surface EMG was recorded of m. tibialis anterior and m. triceps surae. The location of the bipolar leadoff was at the centre of the muscle belly, the orientation of the line connecting the pair of electrodes being perpendicular to the transverse plane. The location was carefully marked, so that the exact electrode position could be reproduced after cooling. The circular electrodes measured 6 mm in diameter with a centre-to-centre distance of 20 mm, the reference electrode being 15 mm away from the two others. These three electrodes were integrated in the housing of a small pre-amplifier (Medelec AE15), a configuration which assured a noise

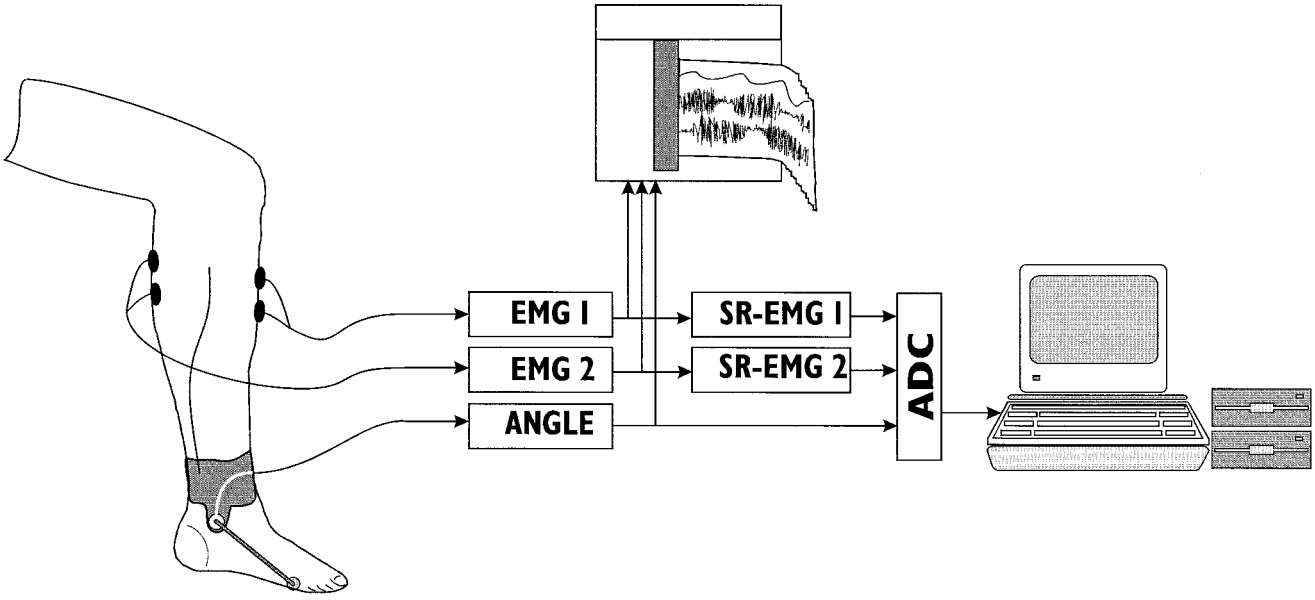

Figure 1 Instrumentation for the measurement of the repetitive movement test ADC: Analog-to-Digital Converter; SR-EMG: Smoothed Rectified EMG.

and artefact free EMG-signal. This signal was high-pass filtered (20 Hz 6dB/oct), amplified (Medelec AA6T) and displayed on an oscilloscope. Before the actual recording the gain of the amplifier was set to the maximal value at which there was no clipping of the signal. The EMG signal was also recorded on an ink-writing X-t recorder, together with the goniometer signal (figure 1). In addition, the EMG was processed into the SR-EMG (Smoothed Rectified EMG) using a two-phase rectifier and a first-order low-pass filter (τ = 200 ms, i.e. 0.8 Hz) (Medelec I7) (figure 2a).

The SR-EMG signals and the goniometer signal were recorded in a computer system, by means of an A/D converter (8 bits, 60 Hz) and stored on a floppy disk for off-line analysis. (Apple II+ computer & Digilog ADC16; the software was written in MS-FORTRAN under the CP/M operating system).

In order to obtain a comprehensive view of the activation patterns of both agonist and antagonist muscles, ensemble averaging of the cyclic movement was performed (figure 2b). This was achieved in three steps. Firstly, *segmentation* of the cyclic signal into N segments, N being the number of repetitions during the test. Subsequently, all segments were time-normalized by linear interpolation using a time base of 0–100% cycle time. Finally, the ensemble-averaged signals (and the standard deviation per %-cycle-time) were calculated, printed and saved for further parameterization. This procedure was followed to obtain a more reliable estimate of muscle activation levels eliminating the cycle-to-cycle variation of muscle activation.[21]

PARAMETERS

The following parameters were derived for performance of the movement (from the average goniometer signal):

Frequency:	average frequency of the (self-paced) repetitive movement [/min.]
Score:	total amount of movement during 30 seconds [°]
Rom:	average range of motion per cycle [°]

The following parameters were derived from the muscle activity level (from the average SR-EMG signal):

Max:	maximum of average SR-EMG when the muscle is acting as an agonist [μV]
Min:	minimum amount of average SR-EMG [μV], (i.e. bias activity)
Mod:	modulation of the signal: (MAX − MIN)/MAX [%]

Both parameters were derived before (A) and after (B) cooling. In order to normalise for inter-individual differences, an additional parameter was defined:

Rel: MAX/((MAXA + MAXB)/2) [%].

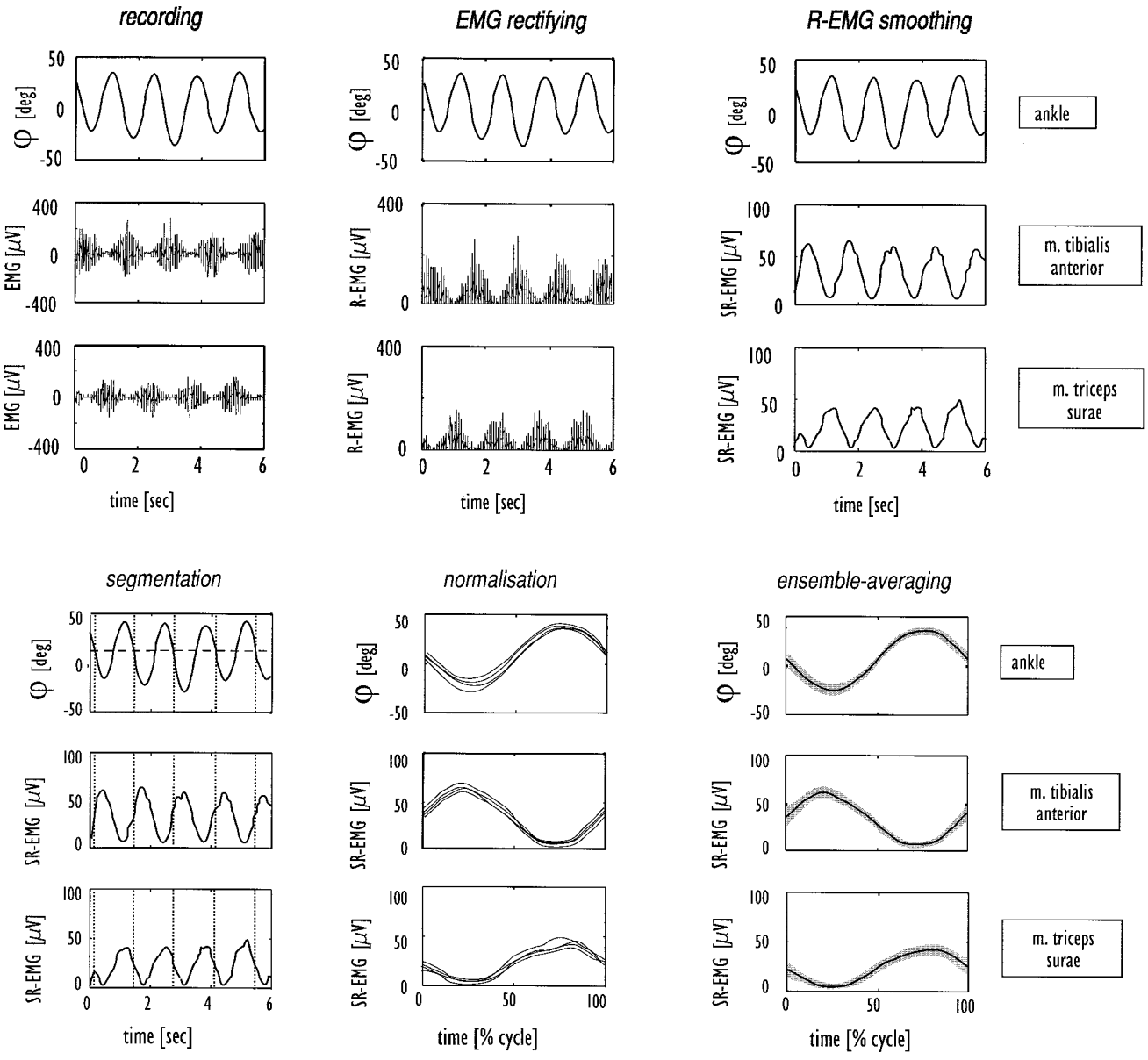

Figure 2a On-line signal processing.
Figure 2b Off-line signal processing.

Each parameter was obtained for the m. tibialis anterior (TA) and the m. triceps surae (TS).

STATISTICS

All test parameters were compared before and after cooling. Statistical tests were carried out to control for the two-tailed level of significance, set at $p = 0.05$. A Student's t-test for paired observations was applied for the performance and muscle activation parameters. The results of the clinical examination were evaluated with the Mann-Whitney test, for paired observations. Calculations were performed in SPSS.

Results

The skin temperature, due to the cooling procedure, dropped by 16.8 ± 2.3 °C, which shows that the procedure was quite effective in decreasing skin temperature. This amount of skin cooling corresponds with a drop in muscle temperature of approximately 5 °C.[20] The effect

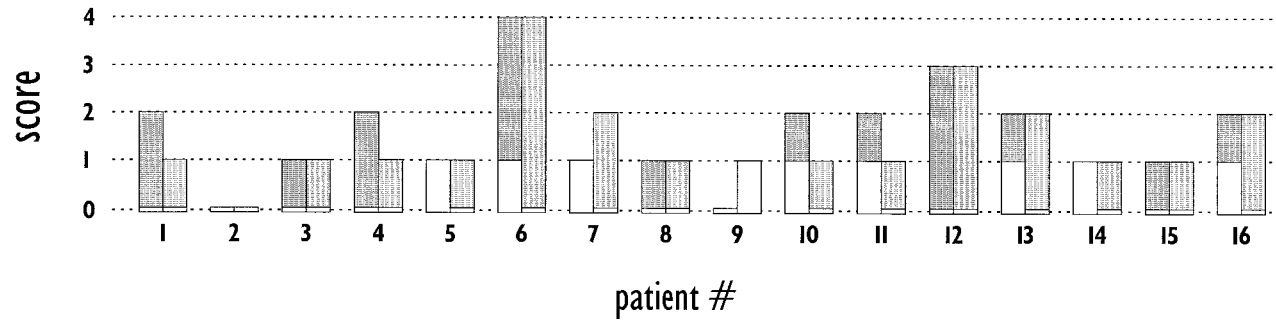

Figure 3 Changes in clinical assessments after cooling per subject (N = 16). Initial scores are shown as bar height, the shaded area represents the decrease in score after cooling. Abscissa: patient number. Ordinate: reflex/clonu score (table I). Left side of bars: Achilles tendon reflex. Right side of bas: ankle clonus.

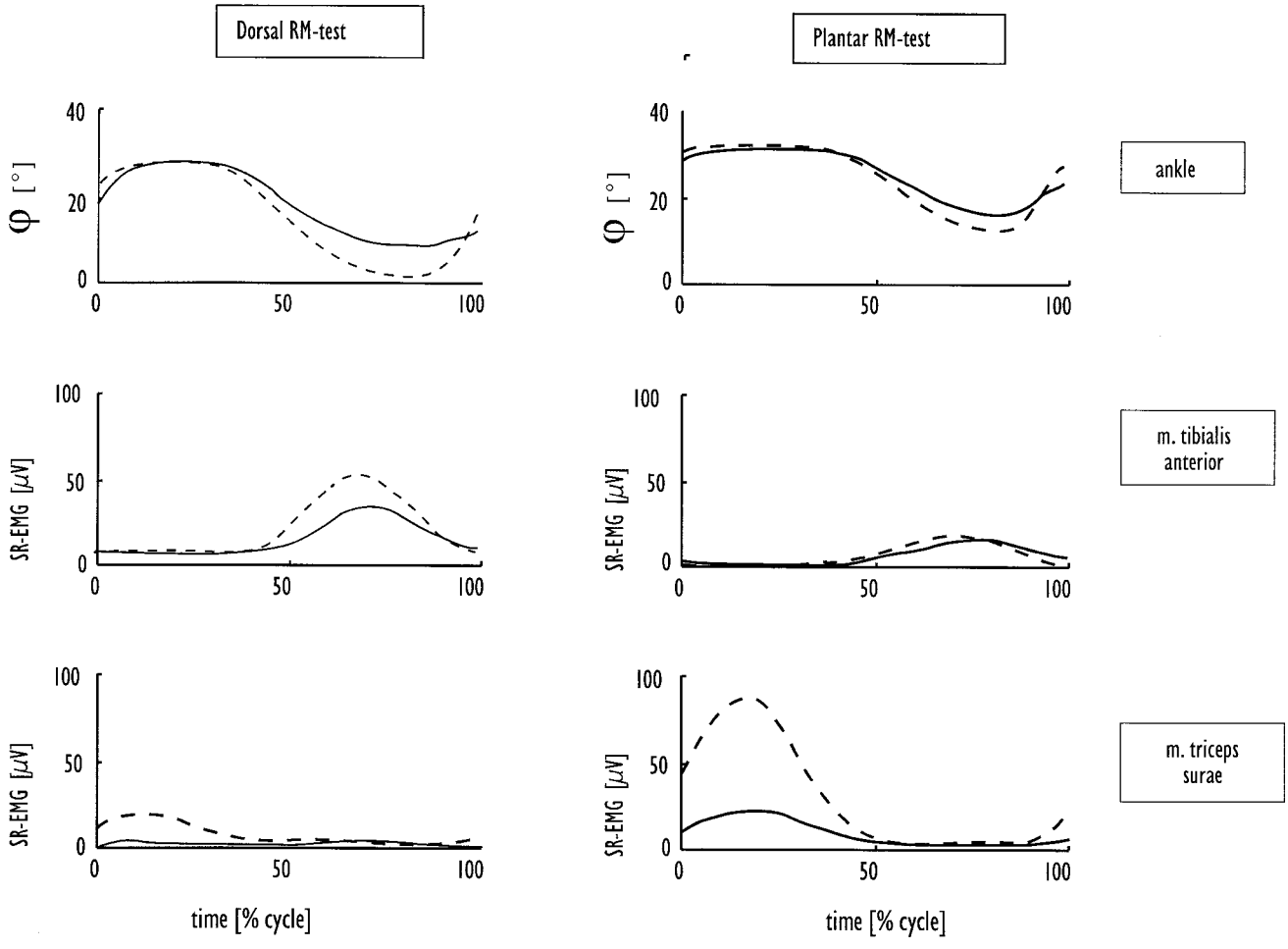

Figure 4 Typical result of the repetitive movement test after signal-processing. Ankle-angle is in degrees of plantar flexion. Solid line: before cooling. Dashed line: after cooling.

on the knee tendon reflex, initially normal for all but 2 patients, was minimal, but the effects on Achilles tendon reflex and ankle clonus were significant. An overview is shown in figure 3. All 5 patients who showed no response on Achilles tendon reflex were assessed as grade 0 or 1, including the 2 non-responders with respect to the ankle clonus.

A typical result of the repetitive movement test is shown in figure 4. This patient showed not only an increase in the SR-EMG of the m. triceps surae, but also

Table 2 Group means, mean difference (standard deviation), and levels of significance of performance parameters of the repetitive movement tests, before and after cooling m. triceps surae. Significant effects are marked with an asterisk

		Group mean (s.d.)			
Parameter	*Applied at*	*Before*	*After*	*Mean difference*	*p-value*
Passive ROM	Dorsal RM	22.7 (4.6)	23.8 (3.1)	1.1 (3.7)	0.255
[deg]	Plantar RM	21.4 (3.7)	22.3 (2.8)	0.8 (2.6)	0.218
Active ROM	Dorsal RM	15.8 (6.8)	16.0 (7.3)	0.2 (5.0)	0.872
[deg]	Plantar RM	12.6 (4.3)	14.4 (6.2)	1.8 (3.4)	0.049*
Frequency	Dorsal RM	22.2 (4.8)	24.0 (3.6)	1.8 (3.1)	0.036*
[/min.]	Plantar RM	25.5 (5.6)	24.0 (4.9)	−1.4 (4.1)	0.177
Score	Dorsal RM	295 (154)	336 (183)	41 (105)	0.146
[deg]	Plantar RM	224 (114)	294 (168)	70 (124)	0.040*

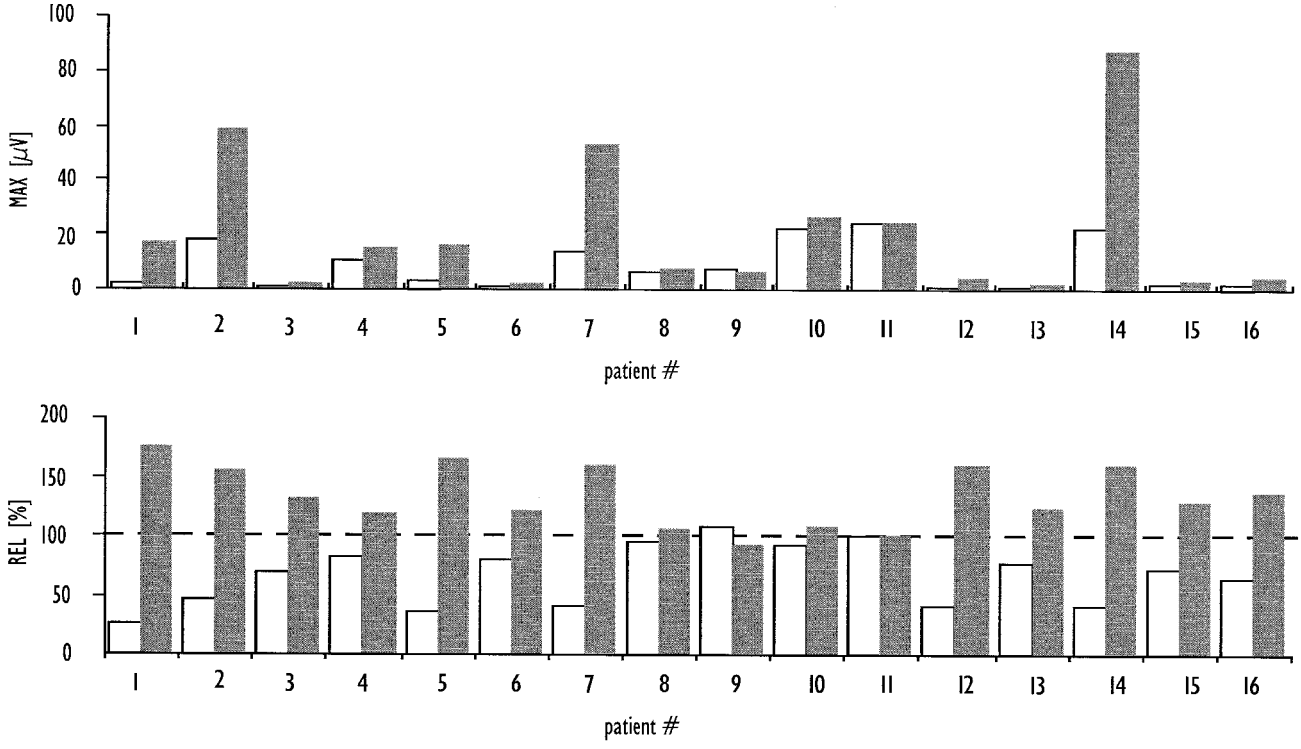

Figure 5 Effect of cooling on the muscle activation per subject, as absolute SR-EMG (upper part of figure) and relative change (lower part of figure). Before cooling is shown as open bars, after cooling is shown by filled bars.

of the m. tibialis anterior, which was not the case in all patients.

The parameters that characterize the performance of the repetitive movement did not change dramatically under the influence of cooling the m. triceps surae. Small changes which just reached the level of significance were seen in the active range of motion and the score of the plantar repetitive movement, as well as the frequency of the dorsal repetitive movement (table 2).

From figure 5 it can be seen that the effect of cooling apparently increased the activation level of the m. triceps surae, being the muscle that was cooled. The effect on the absolute levels is shown per patient in figure 5. It can also be seen from this figure that the subject-specific levels of SR-EMG vary considerably. Therefore it was decided to express the change as a percentage of the mean pre- and post-cooling SR-EMG level per patient. After this normalization procedure the effect of cooling emerged

Table 3 Group means, mean difference (standard deviation), and levels of significance of muscle activation parameters of the agonist muscles: m. tibialis anterior (dorsal RM-test) and m. triceps surae (plantar RM-test). Values are shown before and after cooling m. triceps surae. Significant effects are marked with an asterisk

		Group mean (s.d.)			
Parameter	*Applied at*	*Before*	*After*	*Mean difference*	*p-value*
Maximum SR-EMG	m. tibialis ant.	22.3 (25.3)	26.6 (26.3)	4.3 (11.4)	0.153
[μV]	m. triceps surae	8.7 (8.7)	20.5 (24.9)	11.8 (19.3)	0.028*
SR-EMG modulation	m. tibialis ant.	71.9 (27.1)	79.1 (24.3)	7.17 (17.9)	0.130
[%]	m. triceps surae	57.8 (30.3)	66.5 (29.7)	8.7 (15.1)	0.035*
SR-EMG relative level	m. tibialis ant.	86.5 (32.8)	113.5 (32.8)	26.9 (65.5)	0.121
[%]	m. triceps surae	67.1 (25.5)	133.0 (25.5)	65.9 (51.0)	0.000*

clearly (figure 5). In table 3, group mean activation parameters on both m. tibialis anterior and m. triceps surae are shown. All parameters for the m. triceps surae increased significantly, but the increase in all parameters of m. tibialis anterior did not reach a significant level.

Discussion

The superficial cooling of m. triceps surae has an apparent effect on spasticity, as it is observed by common clinical assessment during physical examination.[1,3] This was confirmed in this study. Hyperreflexia of the Achilles tendon was eliminated in all patients, and clonus disappeared in all but one patient. Knee tendon reflexes were scarcely affected, indicating a local effect of cooling. The elimination of hyperreflexia of the Achilles tendon and ankle clonus might be an important improvement for the patient. However, this effect is only temporary, and will last for only two hours at the most. Patients might wish to use this easily applicable method when they need relief for a short period of time. The effect might also be beneficial when the hyperreflexes and/or clonus hinder a therapeutic intervention, e.g. the application of a peripheral nerve block[22] or a casting procedure in the manufacture of an ankle-foot-orthosis.

In addition to this well-known clinical fact, we attempted to reveal some changes in muscle activation and performance in repetitive movement of the ankle, i.e. the RM-test. For this purpose the Smoothed Rectified EMG (SR-EMG) was recorded as a measure for the relative level, or envelope, of EMG muscle activation. The additional process of ensemble averaging thus further averages the cycle-to-cycle variation of muscle activation. It must be stressed that an adequate description of the recording and signal-processing techniques used is necessary, as seemingly minor changes in these techniques might significantly affect the parameters that are based on it.[23]

The results of this study show that an increased agonist EMG-activity of the m. triceps surae is observed after cooling. However, this does not result in better performance on the RM-test. Only a minor, clinically non-relevant increase in range of motion in the direction of plantar flexion was seen, which could be explained by a higher muscular force of the m. triceps surae as a result of higher activation. In only one patient a slight co-contraction of the m. triceps surae during dorsal flexion was seen. As a result of cooling, this co-contraction diminished. This phenomenon is compatible with the idea that co-contraction of the m. triceps surae is due to disinhibition of reflexes, triggered by muscle spindle activity following lengthening of the muscle. This induced co-contraction would be reduced after decreasing the sensitivity of muscle spindles by cooling the muscle, an effect that was seen in some patients in a study by Knuttson.[3] However, in the present study this was certainly not a general observation. An increase in dorsal flexion, which would have been of functional relevance, did not occur. In the light of these observations it might be hypothesised that limited dorsal flexion is not due to hyperreflexia of the m. triceps surae, but is caused by mechanical factors, e.g. a shortened m. triceps surae complex.[9,24–27]

An increase in SR-EMG of the m. triceps surae was not observed in all patients. Apparently there is no uniform response to cooling muscles in patients with spasticity. Post-hoc analysis showed no correlation of this response with the response to clinical examination. Functional improvement was not systematically assessed, but it was noticed that only patients who were hindered by a severe ankle-clonus improved their gait after the cooling. At this point it is not clear how the RM-test of the ankle, following cooling the m. triceps surae in spastic patients might contribute to clinical decision-making.

An explanation of the increased activation of the m.

triceps surae might be twofold. Firstly, due to the decrease in temperature, the electronic and/or the electro-physiological properties of the muscle tissue might be changed, so that a higher electrical signal is measured at the same level of activation of the muscle. Studies that describe the effect of cooling on the EMG, using stimulation to control the level of activation, are scarce. In anaesthetized cats it was found that both EMG and muscle force increased with temperature reduction, the EMG increase being less variable.[28] In contrast, the M response in normal human m. triceps surae was found to decrease after cooling the m. triceps surae.[15,20] Cooling slows down the chemical and electrophysiological processes along the muscle fibre, which results in a decrease of the velocity of the motor unit action potential, and thus a lower, but prolonged M-response.[29,30] The effect on the interferenced signal (i.e. the surface EMG of gross muscle contraction) will be a lowering of the bandwidth of the EMG.[29,31,32] The SR-EMG, being an estimation of the root mean square value of the EMG, is unaffected by a shift in the EMG power spectrum.

A second explanation of the higher levels of EMG after cooling focuses on spinal nerve activity. Cooling of the skin at the m. triceps surae affects the sensory inflow from skin receptors which increases the H reflex[20,33] or leaves it unaffected.[15] Cooling of the muscle decreases the H-reflex,[20] but leaves the H/M ratio unaffected.[15] As the sensitivity of muscle spindles to stretch is decreased at a lower temperature,[13,14] the Achilles tendon reflex (T-reflex) is decreased.[15,20] These results indicate that the excitability of the alpha-motorneuron pool is unchanged under the influence of muscle cooling. However, this is found in normal subjects without voluntary effort. There is no *a priori* reason why this should be generalized to patients with spasticity, and to situations in which supraspinal drive is present. For example. Sinkjær *et al.* showed the H-reflex modulation to be a function of excitation level in patients with spasticity.[34]

Placing normal subjects in a low ambient temperature showed a doubling of the m. soleus EMG in a functional task.[35] Long-term exposure to low ambient temperature (muscle temperature decreased by 5 °C) showed variable effects in the EMG of upper-arm muscles, with a decreased performance.[36] On the other hand Mucke and Heuer found unchanged mechanical output with a strong decrease in the EMG after cooling.[29] It can be concluded that the current literature on the effects of muscle-cooling on surface EMG cannot be unambiguously phrased to explain the effects that were found in this study. Meanwhile, the most plausible cause of the increase in SR-EMG of m. triceps surae after cooling in patients with spasticity, is an increased level of excitability of the alpha-motorneuronpool in voluntary movements.

References

1 Miglietta O. Action of cold on spasticity. *Am. J. Phys. Ther.* 1973; **52**: 198–205.
2 Hedenberg L. Functional improvement of the spastic hemiplegic arm after cooling. *Scan. J. Rehab. Med.* 1970; **2**: 154–158.
3 Knuttson E. Topical cryotherapy in spasticity. *Scan. J. Rehab. Med.* 1970; **2**: 159–163.
4 Knuttson E, Lindblom U. Mårtensson A. Differences in effects in gamma and alpha spasticity induced by the GABA derivative baclofen (lioresal). *Brain* 1973; **96**: 29–46.
5 Mårtensson A. Antispastic medication; *a review. Scan. J. Rehab. Med.* 1981; **13**: 143–147.
6 Pierrot-Deseilligny E, Mazieres L. Spinal mechanisms underlying spasticity. In: Delwaide PJ, Young RR (eds). *Clinical Neurophysiology in Spasticity*. Amsterdam: Elsevier Science Publishers BV, 1985; 63–76.
7 Burke D. Mechanisms underlying the tendon jerk and H-reflex. In: Delwaide PJ, Young RR (eds). *Clinical Neurophysiology in Spasticity*. Amsterdam: Elsevier Science Publishers BV, 1985; 55–62.
8 Young RR, Wiegner AW. Spasticity. *Clin. Orthop.* 1987; **219**: 50–62.
9 Davidoff RA. Skeletal tone and the misunderstood stretch reflex. *Neurology* 1992; **42**: 951–963.
10 Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurements (review article). *Archives of Physical Medicine and Rehabilitation* 1989; **70**: 144–155.
11 Faist M, Mazevet D, Dietz V, Pierrot-Deseilligny E. A quantitative assessment of presynaptic inhibition of Ia afferents in spastics. Differences in hemiplegics and paraplegics. *Brain* 1994; **117**: 1449–1455.
12 Milanov I. A comparative study of methods for estimation of presynaptic inhibition. *Journal of Neurology*. 1992; **239**: 287–292.
13 Eldred E, Lindsey DF, Buchwald JS. The effect of cooling on mammalian muscle spindles. *Experimental Neurology* 1960; **2**: 144–157.
14 Mense S. Effect of temperature on the discharges of muscle spindles and tendon organs. *Pflügers Arch.* 1978; **374**: 159–166.
15 Bell KR, Lehmann JF. Effect of cooling on H- and T-reflexes in normal subjects. *Archives of Physical Medicine and Rehabilitation* 1987; **68**: 490–493.
16 Knuttson E, Mårtensson A. Dynamic motor capacity in spastic paresis and its relation to prime mover dysfunction, spastic reflexes and antagonist co-activation. *Scan. J. Rehab. Med.* 1980; **12**: 93–102.
17 McLellan DL. Co-contraction and stretch reflexes in spasticity during treatment with baclofen. *J. Neurol. Neurosurg. Psychiat.* 1977; **40**: 30–38.
18 Knuttson E, Richards CL. Different types of disturbed motor control in gait of hemiplegic patients. *Brain* 1979; **102**: 405–430.
19 Sahrmann SA, Norton BJ. The relationship of voluntary movement to spasticity in the upper motor syndrome. *Ann. Neurol.* 1977; **2**: 460–465.
20 Knuttson E, Mattson E. Effects of local cooling on monosynaptic reflexes in man. *Scan. J. Rehab. Med.* 1969; **1**: 126–132.
21 Kleissen RFM, Zilvold G. Estimation uncertainty in ensemble averaged surface EMG profiles during gait. *J. Electromyogr. Kinesiol.* 1994; **4**: 83–94.
22 Glenn MB. Nerve Blocks. In: Glenn MB, Whyte J (eds). *The Practical Management of Spasticity in Children and Adults*. Philadelphia-London: Lea & Febiger, 1990; 227–258.
23 Winter DA. Overall principle of lower limb support during stance phase of gait. *J. Biomech* 1980; **13**: 923–972.

24 Dietz V, Quitern J, Berger W. Electrophysiological studies of gait in spasticity and rigidity. (Evidence that altered mechanical properties of muscle contribute to hypertonia). *Brain* 1981; **104**: 431–449.
25 Halar EA, Stolov WC, Venkatesh B, Brozovich FV, Harley JD. Gastrocnemius muscle belly and tendon length in stroke patients and able bodied persons. *Archives of Physical Medicine and Rehabilitation* 1978; **59**: 476–484.
26 Hufschmidt A, Mauritz KH. Chronic transformation of muscle in spasticity: a peripheral contribution to increased tone. *J. Neurol. Neurosurg. Psychiat*. 1985; **48**: 676–685.
27 Thilman AF, Fellows SJ, Garms E. Mechanism of spastic muscle hypertonus. *Brain* 1991; **114**: 233–244.
28 Engbaek J, Skovgaard LT, Friis B, Kann T, Viby-Mogensen J. Monitoring of the neuromuscular transmission by electromyography (I). Stability and temperature dependence of evoked EMG response compared to mechanical twitch records in the cat. *Acta Anaesthesiologica Scandinavica*. 1992; **36**: 495–504.
29 Mucke R, Heuer D. Behaviour of EMG-parameters and conduction velocity in contractions with different muscle temperatures. *Biomedica Biochimica Acta* 1989; **48**: S459–S464.
30 Bertram MF, Nishida T, Minieka MM, Janssen I, Levy CE. Effects of temperature on motor unit action potentials during isometric contraction. *Muscle & Nerve* 1995; **18**: 1443–1446.
31 Hägg GM. Interpretation of EMG spectral alterations and alteration indexes at sustained contraction. *J. Appl. Physiol*. 1992; **73**: 1211–1217.
32 Holewijn M, Heus R. Effects of temperature one electromyogram and muscle function. *European Journal of Applied Physiology & Occupational Physiology* 1992 **65**: 541–545.
33 Urbscheit N, Bishop B. Effects of cooling on the ankle jerk and h-response. *Phys. Ther*. 1970; **50**: 1041–1049.
34 Sinkjær T, Toft E, Hansen HJ. H-reflex modulation during gait in multiple sclerosis patients with spasticity. *Acta Neurologica Scandinavia* 1995; **91**: 239–246.
35 Winkel J, Jorgensen K. Significance of skin temperature changes in surface electromyography. *European Journal of Applied Physiology & Occupational Physiology* 1991; **63**: 345–348.
36 Oksa J, Rintamaki H, Makinen T, Hassi J, Rusko H. Cooling-induced changes in muscular performance and EMG activity of agonist and antagonist muscles. *Aviation Space & Environmental Medicine* 1995; **66**: 26–31.