J Rehabil Med 2002; 34: 25–32

Taylor&Francis healthsciences

EFFECTS OF FUNCTIONAL ELECTRICAL STIMULATION TRAINING FOR SIX MONTHS ON BODY COMPOSITION AND SPASTICITY IN MOTOR COMPLETE TETRAPLEGIC SPINAL CORD-INJURED INDIVIDUALS

Camilla Sköld,[1,2,3] Lars Lönn,[5] Karin Harms-Ringdahl,[2] Claes Hultling,[3] Richard Levi,[1,3,4] Mark Nash,[6] and Åke Seiger[1,3]

From the [1]Department of Clinical Neuroscience, Occupational Therapy and Elderly Care Research, Karolinska Institute, Stockholm, [2]Department of Physical Therapy, Karolinska Institute and Karolinska and Huddinge University Hospitals, Stockholm, [3]Spinalis SCI Research Unit, Karolinska Institute, Stockholm, [4]Frösunda Center, Solna, [5]Department of Radiology, Body Composition and Metabolism, Sahlgrenska University Hospital, Göteborg, Sweden [6]Department of Orthopedics and Rehabilitation and the Miami Project to Cure Paralysis, University of Miami School of Medicine, Miami, Florida, USA

The effect of functional electrical stimulation (FES) training on body composition, assessed by computed tomography, and the effect of spasticity, assessed by both objective and subjective measures, are evaluated. Fifteen motor-complete spinal-cord-injured men participated in the study. Eight of the 15 subjects undertook FES cycling 3 times weekly for 6 months. Whole body computed tomography scans evaluated changes in body composition. Simultaneous Modified Ashworth Scale and electromyography (EMG) measurements, resistive torque (Kin-Com) and EMG measurements, and self-ratings with Visual Analogue Scale during four consecutive days were used to evaluate changes in spasticity. Lower extremity muscle volume increased by an average of 1300 cm^3 ($p < 0.001$) in the training group compared to the control group, who experienced no change. Otherwise no changes in body composition were seen. Significant correlations (Spearman) were found between individual EMG activity recordings and movement-provoked Modified Ashworth Scale ratings in 26% of the test situations, irrespective of group and time. The objective and subjective evaluation of movement-provoked passive (viscoelastic) and active (spasticity-related) resistance remained unchanged.

Key words: SCI, Computer Tomography, muscle hypertrophy, spasticity, Modified Ashworth Scale, Visual Analogue Scale, EMG, resistive torque, FES.

J Rehabil Med 2002; 34: 25–32

Correspondence address: Camilla Sköld, RPT, PhD, FoUU-enheten Stockholms Sjukhem, Mariebergsgatan 22, SE-112 35 Stockholm, Sweden. E-mail:Camilla.Skold@neurotec.ki.se

Accepted June 26, 2001

INTRODUCTION

Spasticity is a common sequela of an upper motor neuron lesion associated with spinal cord injury (SCI) (1). In the prevalence-based study of SCI individuals in the greater Stockholm area, spasticity was reported to be problematic in 30% of the population (2). No curative treatment for SCI-related spasticity is currently available. Symptomatic intervention is therefore crucial.

Spasticity has been defined by Lance (3) to include both increased muscle tone (tonic stretch reflex) and increased tendon reflexes (phasic stretch reflex). The spasticity syndrome has been further extended to include increased exteroceptive reflexes (flexion reflex) and pathological radiation (clonus) (4, 5). These components constitute the operational definition of spasticity used in this study.

Spasticity has been shown to decrease transiently for up to several hours in response to various physiological interventions, such as cutaneous stimulation and muscle elongation. Several studies have shown cutaneous electrical stimulation to reduce various components of the spasticity syndrome and/or reduce the exteroceptive flexion reflex and the pathological radiation (clonus) (6–8). Muscle elongation intervention has been shown to result in a reduction both of phasic and tonic stretch reflexes (9, 10). Functional electrical stimulation (FES), probably interacting with all components of the syndrome, has been shown to reduce spasticity (11–13) in some studies, whereas in others changes varied (14) or even an increase in spasticity have been reported (15).

During the acute phase of SCI, dramatic changes in body composition occur. Over time, further changes are established due to the decreased level of activity in SCI individuals (16). The two main features are the dramatic decrease in lean body mass, i.e. skeletal muscle and bone mass, and the relative increase in adipose tissue mass (17). FES cycle ergometry has a multitude of effects on bodily functions, such as increased muscle mass and decreased fat tissue mass. Several authors have found increased thigh muscle mass (7% to 22%) as measured with computed tomography (CT) after FES exercise (6 to 12 weeks) (18–20). Mohr et al. (21) showed a 12% increase in thigh muscle mass after 1 year of FES training as measured with magnetic resonance imaging (MRI). Hjeltnes et al. (18) further reported significantly increased lean body mass by 1% and significantly decreased body fat by 2%, both measured with dual-energy X-ray absorptiometry. Because FES cycling causes changes in muscle that may affect patterns of spasticity, our

 ISSN 1650–1977

hypothesis was that FES-induced increase in muscle mass also increases the spastic force of paralysed muscles in individuals with motor complete cervical SCI.

The aim of this study was to test the hypothesis that FES cycle ergometry alters muscle mass, adipose tissue and spasticity in individuals with motor complete cervical SCI. Changes in body composition and regional tissue distribution were examined by a multicompartment technique based on CT. EMG activity, resistive muscle torque measurements and clinical and subjective ratings were combined to evaluate spasticity.

METHODS

Subjects

The mean age at injury for the 15 randomly selected male subjects with SCI was 33 years (range 21–48); mean time since injury was 9 years (range 1–21). Ten of the subjects were ASIA grade A and 5 were grade B. Two (both grade A) used Baclofen in doses of 20 to 60 mg/day orally. They had been on Baclofen medication for several years and were instructed not to modify their doses.

The Regional Human Ethics Committee of the Karolinska Institute approved the study.

Body composition

All patients were hospitalized during examination. Before and after the FES training period of 6 months, body weight was measured in the morning, after voiding, to the nearest 0.1 kg using a calibrated electronic precision tension scale (Violavågen, Botek, Sweden) mounted on a manual lift (Masterholds c750A, Cartel Ltd, UK, 1989). Body height was measured with the patient supine. Body mass index (BMI) was calculated as body weight (kg)/height $(m)^2$.

With the subjects in a recumbent position, tissue areas were determined with a CT scanner (Philips' Tomoscan 350, Philips Medical Systems, Best, The Netherlands). Twenty-two CT scans of the whole body were performed, out of which 9 determined the lower extremity (LE), as previously described (22). Parameters used were slice thickness 12 mm 302 mAs, exposure and scanning times 1.2 and 4.8s, respectively, 256*256 scan data and image matrix and field of view 480 mm (in-plane spatial resolution of 1.56 mm). The effective dose equivalent per examination was 2–4 mSv. The images from the CT scanner were transferred to a separate analysing unit (Philips' SAVS). Tissue areas were determined as previously described (22), with the following precision errors calculated from double determinations: subcutaneous adipose tissue (AT) (0.5%), intraperitoneal AT (4.6%), retroperitoneal AT (12.1%), the sum of intraperitoneal and retroperitoneal AT (1.2%), muscle plus skin (0.3%) and visceral organs (0.7%) (23).

With dual X-ray absorptiometry, body composition is determined as a three-compartment model: $BW = BF + BMC + LTM$. BF is body fat and BMC is bone mineral content. Lean tissue mass (LTM) consists of proteins, structural lipids, water, glycogen, non-osseous minerals and a small residual. $LTM + BMC$ is equal to fat-free mass (FFM) in traditional 2-compartment models. With an imaging technique such as CT, body composition is examined at the tissue and organ level (24). Tissue volumes can be calculated from tissue areas and distances between scans (23). Models at the tissue and organ level could be expressed as body volume = lean body volume + AT volume. The lean body volume can be analysed as several tissues and organs. Each component can be converted into mass by taking the corresponding tissue density into account (23).

Spasticity analyses

Each subject was tested before the training period, after 6 months of FES training and after 6 months of deconditioning. None of the subjects was stretched in connection with either testing or training. All tests took approximately 30–45 min each time. First, Modified Ashworth Scale (MAS) (25) rating of movement-provoked spasticity of the right lower extremity (LE) was performed, thereafter the isokinetic Kin-Com movement provocation of the right and left LE, respectively, and finally, MAS rating of movement-provoked spasticity of the left LE was performed.

Movement-provoked spasticity. The MAS ratings (25) of movement-provoked spasticity were performed during simultaneous surface recording of EMG activity of the rectus femoris and the lateral biceps femoris muscles with the subject lying supine. MAS is a 6-grade scale (0–5), with 0 indicating no muscle tone and 5 indicating a rigid limb. The EMG recordings were performed as previously described by our group (26). First flexing and then extending the knee once initiated the movement provocation at a velocity of approximately 350°/s, as estimated in an earlier study by a two-camera, 3-D video motion analysis system (26). During knee flexion movements, the hip was in an extended position, and during knee extension movements the hip was flexed 90° (Fig. 1).

The MAS and EMG values were recorded during 10 s. The EMG baseline for the 10-s recording period was defined as mean electrical activity before and after the movement-associated electrical activity. Peak electrical activity was defined as the highest voltage 1 to 3 s from the start of EMG recording. Duration of movement-associated electrical activity was defined as the time of continuous electrical activity above baseline.

Isokinetic movement provocation. Thereafter, isokinetic (Kin-Com dynamometer 125E Plus computer, CHATTECX Corp, Chattanooga Group, Hixon, TN, USA) movement provocation of resistive muscle torque was performed during EMG activity recordings. The subjects were tested seated inclined with a backrest giving a hip flexion angle of 40° during the test procedure (Fig. 1). Flexion and extension movement of the knee joint was performed at a velocity of 250°/s. Only 14 subjects were analysed due to incomplete resistive torque data of one of the FES training subjects.

The torque and EMG values were recorded during 0.3 s. The EMG baseline for the 0.3-s recording period was defined as the mean of the first 10 point estimates (0.02 s) plus 2 SD for each individual subject, side, movement and muscle group, respectively (27). Duration of EMG activity was defined as a minimum of 10 consecutive point estimates above baseline. Maximum EMG activity was defined as the highest voltage during the whole recording period. Actual EMG activity was defined as the EMG activity value at peak torque, i.e. the greatest resistance (Nm) during the whole recording period.

VAS ratings. Subjects rated their spasticity every other hour when awake during 4 consecutive days. The rating instruction was "rate your spasticity for the time period since you last rated" using a new ungraded form for each rating performed. The text "No Spasticity" was placed to the left of the 100 mm VAS scale and "Most Imaginable Spasticity" to the right. These ratings collectively described the intrinsic pattern of spasticity during the day (28).

FES cycle ergometry training

The subjects were placed in a sitting position on the FES bicycle (ERGYS I Clinical Rehabilitation System, Therapeutic Technology, Tampa, USA) consisting of a lower extremity ergometer, a stimulus control unit and a reclineable chair. A computer delivered and monitored electrical stimulation according to prescribed parameters such as individual pre-set threshold levels. Stimulation intensities ranged from threshold levels determined for each individual muscle group to elicit a palpable contraction up to a maximum of 130 mA. Before exercise, surface self-adhesive electrodes (CEFAR medical products ab/Form No. 28001/92, Sweden) were placed over motor points on the quadriceps, hamstrings and gluteal muscle groups of both legs. Two active electrodes and one reference electrode were applied over each muscle group. Six separate channels for sequential surface muscle stimulation were used during cycling with a computer-controlled closed loop system. Each channel supplied mono-phasic rectangular pulses lasting 350 µs, and delivered intermittent pulse trains of 30 Hz to each of the two active electrodes over a given muscle. The quadriceps muscle was thereby stimulated at 60 Hz and with intensity ranging from the pre-set threshold levels to 130 mA. A pedal position sensor allowing continued calculation

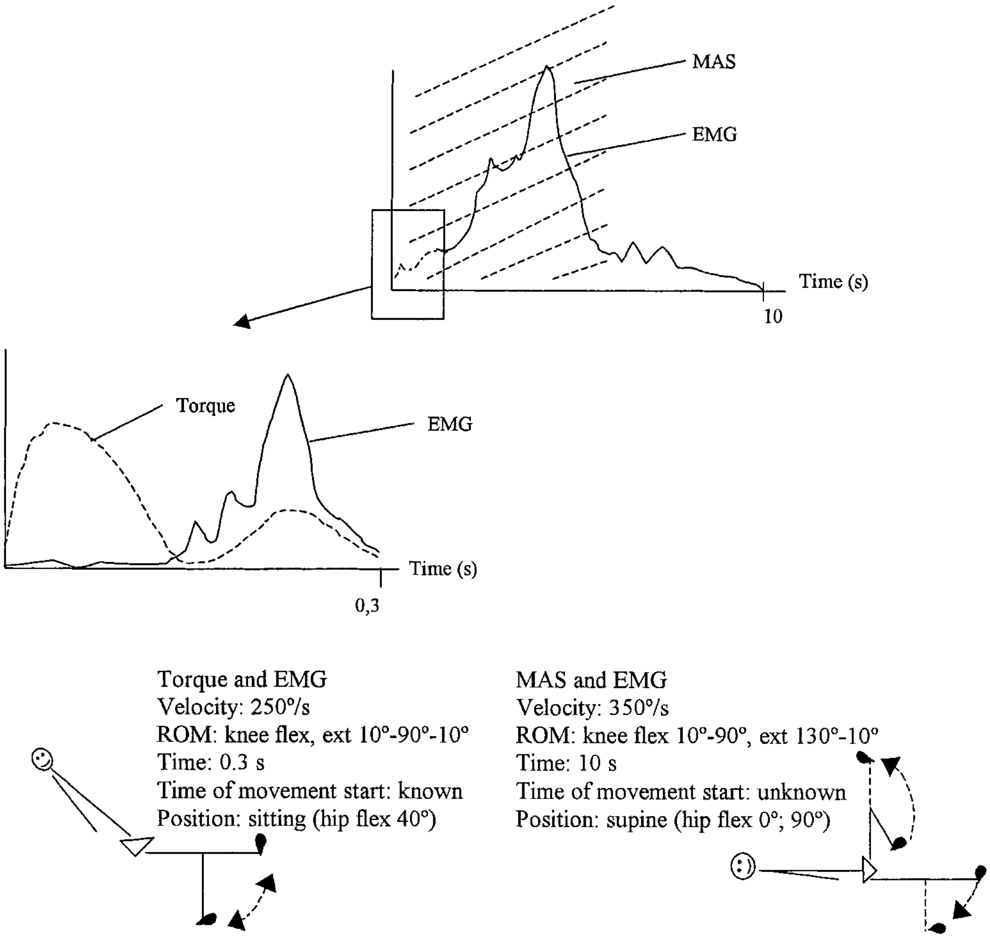

Fig. 1. Description of time course and temporal relationship between recordings. The hatched area in the upper panel indicates the approximate time interval when the investigator makes the Modified Ashworth Scale (MAS) estimate.

of velocity was used to maintain smooth motion and a constant cranking frequency of 50 revolutions per minute. Stimulation was automatically stopped at fatigue, i.e. when the number of revolutions per minute decreased below 35, in spite of maximal stimulation intensity. A maximum of five sets were performed to reach 30 min of exercise at each training session. Load was added by 1/8 kilopound (kp) (~6.1 Watts) each time the subject was able to perform 30 min of continuous exercise for three consecutive sessions. The study group undertook FES cycling in 30-minute sessions 3 times weekly for 6 months.

Statistics

Spearman correlations were calculated between MAS ratings of spasticity and EMG activity recordings. Pearson product moment correlations were calculated between resistive torque and EMG activity values, and among muscle volumes, resistive torque and EMG activity. Separate univariate analyses were performed for MAS, EMG, torque and muscle mass to examine main effects for "group" and "time". If a significant main effect for time was observed, a post hoc test using simple effects was performed to identify the time point of change. For all variables (MAS, EMG, torque and muscle mass) differences between groups and time (before, after and after deconditioning), as well as within subject, were calculated using General Linear Model repeated measures. In all cases the criterion for significance was set at $p < 0.05$.

RESULTS

FES

The group assigned to FES exercise cycled an average of 2.6 (range 2.3–3.1) times weekly for 6 months. The mean load was 18.3 Watts (W) (range 12.2–24.4 W). All subjects cycled at 0 W resistance for an average of 8 sessions (range 3–19), at 6.1 W resistance for an average of 7 sessions (range 4–18) and at 12.2 W resistance for an average of 20 sessions (range 6–30). Five subjects reached 18.3 W resistance and cycled an average of 35 sessions at that level (range 20–53), and one subject reached 24.4 W resistance and cycled for 25 sessions. A mean of 1.9 (range 1.6–2.4) sets was performed each 30-min training session throughout the whole period.

Body composition

There were no significant changes in body weight during the study period, while muscle tissue volume was increased by about 10% in the training group (mean 1307, SD 511 cm^3, $p < 0.001$). AT volumes were neither significantly changed in the abdominal compartment nor in the LE. Moreover, caloric intake was not restricted in this study, resulting in no significant changes in BW. As shown in this study, exercise of specific muscle groups of for example LE results in an increase of LE muscle volume. However, a reduction of adipose tissue in a specific region during training was not achieved in this study.

No correlation was observed between the increased LE muscle volumes and movement-provoked resistive muscle torque or EMG activity values after 6 months of FES training.

Spasticity

No effect of training or deconditioning on spasticity was seen in the training group as compared to the control group for MAS ratings of movement-provoked spasticity or EMG peak and duration of activity. Nor was any change seen in the training group as compared to the control group for isokinetic peak muscle torque, EMG maximum, or actual EMG activity at peak torque during Kin-Com provoked knee movements. As representatives of this lack of training effect seen in all parameters of spasticity evaluation, three parameters – the movement-provoked resistive muscle torque peak (Table I), the MAS rating of spasticity (Table II) and the EMG peak activity (Table III) – are shown.

Significant correlations (Spearman) were found between individual EMG activity recordings (peak, duration) and movement-provoked MAS ratings in 26% of the test situations, irrespective of group and time (Table IV). For duration of EMG activity, lower correlation coefficients were seen after 6 months of FES training, only to increase again after 6 months of deconditioning (Table IV). Correlations calculated between resistive torque values and EMG values after isokinetic movement provocation showed no significant relationships, irrespective of time and group.

During Kin-Com movement provocation no EMG activity was observed before the end of the movement in 68% of the test situations for the intervention group and in 55% of the test situations for the control group. In the cases with no EMG activity during movement, resistive torque was shown in all cases, with a mean torque peak of 24 Nm for the intervention group and 21 Nm for the controls. For the 32% of test situations showing EMG activity in the FES group during movement, resistive mean torque peak values varied between 18 and 26 Nm and average EMG maximum activity between 53 and 118 µV. For the 45% of test situations showing EMG activity in the control group during the movement, resistive mean torque peak values varied between 8 and 21 Nm and mean EMG maximum activity between 76 and 278 µV. A significantly shorter EMG duration measured during isokinetic movement provocation was seen after 6 months for both groups ($p < 0.001$).

Self-ratings of spasticity (VAS) performed before the start of and after FES intervention showed no change for either group. When each subject was analysed individually, some subjects showed increased, some decreased, others no change in VAS-rated spasticity, regardless of group (Fig. 2a–d).

DISCUSSION

In the present study, we evaluated the effect of FES training on body composition by CT and on spasticity by both objective and subjective measures.

Mohr et al. (21) published the only study to date reporting progression in training duration and load during the course of FES sessions, and those results are therefore compared with the present study. Subjects in the latter study completed an average of 2.6 training sessions each week for the 6-month training period. The SCI subjects in the study by Mohr and colleagues accomplished 2.3 sessions each week while training for a year. Two of our subjects took a comparably longer time to reach 30 min of continuous pedalling without resistance, 15 and 16 sessions, respectively. Load was added and tolerated in all subjects. Mohr et al. reported two slowly adapting subjects, one of whom took 32 sessions to perform 30 min of continuous exercise with load, and another who reached 30 min of continuous training only after more than 6 months (~55 sessions), tolerating minimal added load. In our study the slowest adapting subject had a spinal cyst operated 1 month

Table I. *Mean resistive torque for right and left sides and for flexion and extension movement in both groups, respectively, before and after the 6-month FES training period and after 6 months of deconditioning*

			Torque (Nm)								
			Before			After			Deconditioning		
Group	Side	Movement	N	Mean	SD	N	Mean	SD	N	Mean	SD
FES	Right	Flexion	7	19.3	9.5	7	16.1	11.2	7	17.2	18.1
		Extension	7	23.5	4.0	7	17.7	10.3	7	13.6	19.2
	Left	Flexion	7	22.5	12.1	7	23.4	9.6	7	25.7	15.7
		Extension	7	29.1	12.1	7	25.2	7.2	7	28.5	15.6
Control	Right	Flexion	7	9.9	9.9	7	17.1	12.9	3	5.2	28.5
		Extension	7	15.0	14.2	7	16.9	13.5	3	−0.8	19.7
	Left	Flexion	7	22.1	7.9	7	12.1	16.0	3	25.3	13.3
		Extension	7	24.3	9.5	7	13.2	17.9	3	25.1	8.8

Table II. *Mean MAS rating for right and left sides and for flexion and extension movement in both groups, respectively, before and after the 6-month FES training period and after 6 months of deconditioning*

			MAS (0–5)								
			Before			After			Deconditioning		
Group	Side	Movement	N	Mean	SD	N	Mean	SD	N	Mean	SD
FES	Right	Flexion	6	0.7	1.2	6	0.7	1.2	6	1.0	0.6
		Extension	6	1.0	1.3	6	0.3	0.5	6	1.0	0.9
	Left	Flexion	4	0.8	1.0	4	1.0	1.4	4	1.3	1.3
		Extension	5	0.6	0.9	5	0.4	0.5	5	1.0	0.7
Control	Right	Flexion	7	1.7	1.6	7	1.3	1.3	4	1.0	0.8
		Extension	7	1.0	1.5	7	0.6	0.5	4	1.5	1.0
	Left	Flexion	6	1.7	1.5	6	1.5	1.4	4	1.3	1.5
		Extension	6	1.5	1.6	6	0.5	0.5	4	1.0	0.8

before the start of exercise, and another subject was just 1 year after injury compared to 19 and 20 years in the report by Mohr et al. (21). The two slowly adapting subjects in the present study and a third subject, also 1 year after injury, reached a resistance of 12.2 W. Of the 5 remaining subjects, 4 reached 18.3 W and 1 reached 24.4 W. Our study does not support long time inactivity or more pronounced atrophy as explanations of slow adaptation.

Our report showed an effect on LE muscle volume, but no other effect on body composition. The Hjeltnes et al. (18) group was the only one to show decreased body fat (1.9%) after FES training. Their subjects cycled for a comparably short and intense period of time (7 times/5 days/week for 8 weeks). Tetraplegics, although often rather slim, have been shown to accumulate truncal fat as opposed to persons without SCI (29, 30). Hjeltnes et al. (18) showed significantly decreased body fat in the LE and of the whole body of SCI subjects after 8 weeks of FES intervention, although not in the trunk when evaluated separately. No relation was found between increased LE muscle volume and movement-provoked resistive torque in the training group. Sunnerhagen et al. (31) showed that voluntary muscle torque correlated with increased muscle volume in the affected side of patients after a stroke. Since the subjects in our study had no voluntary muscle control, the resulting 10% increased LE muscle volume may have been insufficient to result in increased resistive torque or increased MAS ratings.

It should be stressed that the increase we observed in LE muscles during treatment could in part be due to both protein and water restitution. Neither CT nor DXA can separate between these two constituents of lean tissues. Fat-free mass (FFM)

Table III. *Mean EMG peak activity for right and left sides and for flexion and extension movement in both groups, respectively, before and after the 6-month FES training period and after 6 months of deconditioning*

				EMG peak (uV)								
				Before			After			Deconditioning		
Group	Side	Movement	Muscle group	N	Mean	SD	N	Mean	SD	N	Mean	SD
FES	Right	Flexion	Antagonist	6	50.7	69.9	6	106.2	86.1	6	103.3	63.8
			Agonist	6	38.3	46.0	6	69.8	55.8	6	49.8	37.7
		Extension	Antagonist	6	85.7	65.2	6	96.3	67.1	6	44.0	48.2
			Agonist	6	41.3	32.3	6	37.0	29.9	6	66.0	27.3
	Left	Flexion	Antagonist	4	50.5	44.6	4	110.0	177.7	4	93.5	81.2
			Agonist	4	29.0	28.0	4	89.5	147.0	4	58.8	54.8
		Extension	Antagonist	5	97.0	75.5	5	156.4	77.3	5	39.8	18.6
			Agonist	5	37.2	18.4	5	46.6	24.2	5	115.8	37.1
Control	Right	Flexion	Antagonist	7	231.9	203.2	7	245.3	170.3	4	144.0	110.4
			Agonist	7	91.0	117.4	7	79.4	40.1	4	63.3	47.3
		Extension	Antagonist	7	121.7	69.2	7	263.7	148.1	4	76.0	47.1
			Agonist	7	46.1	20.0	7	102.1	70.6	4	211.0	129.7
	Left	Flexion	Antagonist	6	251.5	358.6	6	327.0	341.4	4	157.5	142.4
			Agonist	6	74.3	100.5	6	95.3	89.8	4	78.5	46.1
		Extension	Antagonist	6	136.3	59.0	6	176.0	104.2	4	61.5	30.4
			Agonist	6	50.5	25.7	6	57.3	25.8	4	186.8	70.3

Table IV. *Correlations (Spearman) between movement-provoked MAS rating and EMG activity values before and after FES intervention and after 6 months of deconditioning (N total = 15, 8 training subjects and 7 control subjects)*

				Before						After						Deconditioning					
				EMG peak			EMG duration			EMG peak			EMG duration			EMG peak			EMG duration		
Group	Side	Movement		N	Antag	Agonist	N	Antag	Agonist	N	Antag	Agonist	N	Antag	Agonist	N	Antag	Agonist	N	Antag	Agonist
FES	Right	Flexion	**MAS**	6	0.86*	0.68	6	0.86*	0.50	6	0.78	0.54	6	−0.10	−0.34	6	0.68	0.34	6	0.68	0.51
		Extension	**MAS**	6	0.88*	0.52	6	0.88*	0.94**	6	0.62	0.63	6	0.83*	0.21	6	0.84*	0.48	6	0.42	0.48
	Left	Flexion	**MAS**	4	0.89	0.74	4	0.89	0.74	4	0.95	0.33	4	0.50	0.32	4	0.32	0.95	4	−0.50	0.50
		Extension	**MAS**	5	0.78	0.45	5	0.89*	0.45	5	−0.29	−0.58	5	0.58	0.44	5	0.89*	0.67	5	0.45	0.89*
Control	Right	Flexion	**MAS**	7	0.90**	0.53	7	0.92**	0.97**	7	0.95**	0.76*	7	0.85*	0.72	4	0.95	0.95	4	0.83	0.50
		Extension	**MAS**	7	0.79*	0.75	7	0.79*	0.79*	7	0.87*	0.72	7	0.29	0.29	4	0.78	0.78	4	0.82	0.78
	Left	Flexion	**MAS**	6	0.63**	0.77	6	0.93**	0.93**	6	0.77	0.47	6	0.77	0.97**	4	0.95	0.63	4	0.63	0.74
		Extension	**MAS**	6	0.27	0.02	6	0.79	0.93**	6	−0.10	0.29	6	0.68	0.29	4	0.63	0.63	4	0.63	0.95

The number of subjects varies for sides, movements and test occasion (before, after, deconditioning) due to either incomplete data or missing individuals, as for deconditioning in the control group.

includes the extracellular fluid and stromal vascular cells of adipose tissue and also cell membranes, intracellular fluid and all cytoplasmic organelles of the adipocytes themselves. Therefore, it is not possible to mix information from DXA and CT examinations.

The distribution of fibre types in skeletal muscles is known to shift towards fast twitch and fast fatiguable within the first 2 years after spinal cord injury. The study by Mohr et al. (21) showed a partial normalization by a shift towards more fatigue-resistant contractile proteins after 1 year of FES training. Owing to the invasive character of this method, biopsies were not performed in the present study.

We have earlier shown that 80% of individual EMG recordings during movement-provoked spasticity correlate significantly with the subjective assessment using the MAS in a group of 15 SCI individuals (26). In the present study, when the same correlations were calculated per group ($n = 8$ and 7, respectively) and over time, only 26% of the individual EMG recordings correlated significantly with the MAS. The lower number of individuals in each group, half the number compared to our earlier study, may cause correlations not to reach statistical significance. In the training group, correlation coefficients between MAS rating and EMG duration of activity decreased after intervention and increased after 6 months of deconditioning. The lower correlation seen at test situations after the FES training may be explained by shorter duration of EMG activity without any corresponding decrease in MAS ratings. No significant correlations were found in the present study between movement-provoked resistive muscle torque (Nm) and simultaneously recorded EMG activity, irrespective of time.

It is unclear whether only the phasic, only the tonic, or both stretch reflexes were elicited when movement-provoked MAS ratings were performed. The provoking movement velocity (~350°/s) recorded when the investigator performed the MAS rating elicited EMG activity in all cases. Conversely, when biomechanical movement provocation of resistive torque was performed, the velocity was 250°/s, which elicited EMG activity in 30–45% of cases depending on group. Resistive torque (Nm) was seen in all cases, indicating muscle stiffness irrespective of spasticity (EMG activity). Both Daly et al. (32) and Yarkony et al. (12) discussed the possibility that FES may decrease tonic spasticity but increase phasic spasticity in both hemiplegic and SCI subjects. Stefanovska et al. (33) showed decreased duration of EMG activity in subjects with hemiplegia, indicating a shift from mainly tonic spasticity, with a longer duration of EMG activity, to mainly phasic spasticity, with a shorter duration of EMG activity. In the present study, shorter EMG duration during isokinetic movement provocation was seen after the 6-month period, although irrespective of group.

Several studies have shown immediately decreased spasticity after FES intervention in SCI individuals (13, 32, 36). Both Daly et al. (32) and Robinson et al. (36) argued that the described reduction in spasticity shown immediately after electrical stimulation might simply reflect muscle fatigue, which lasts

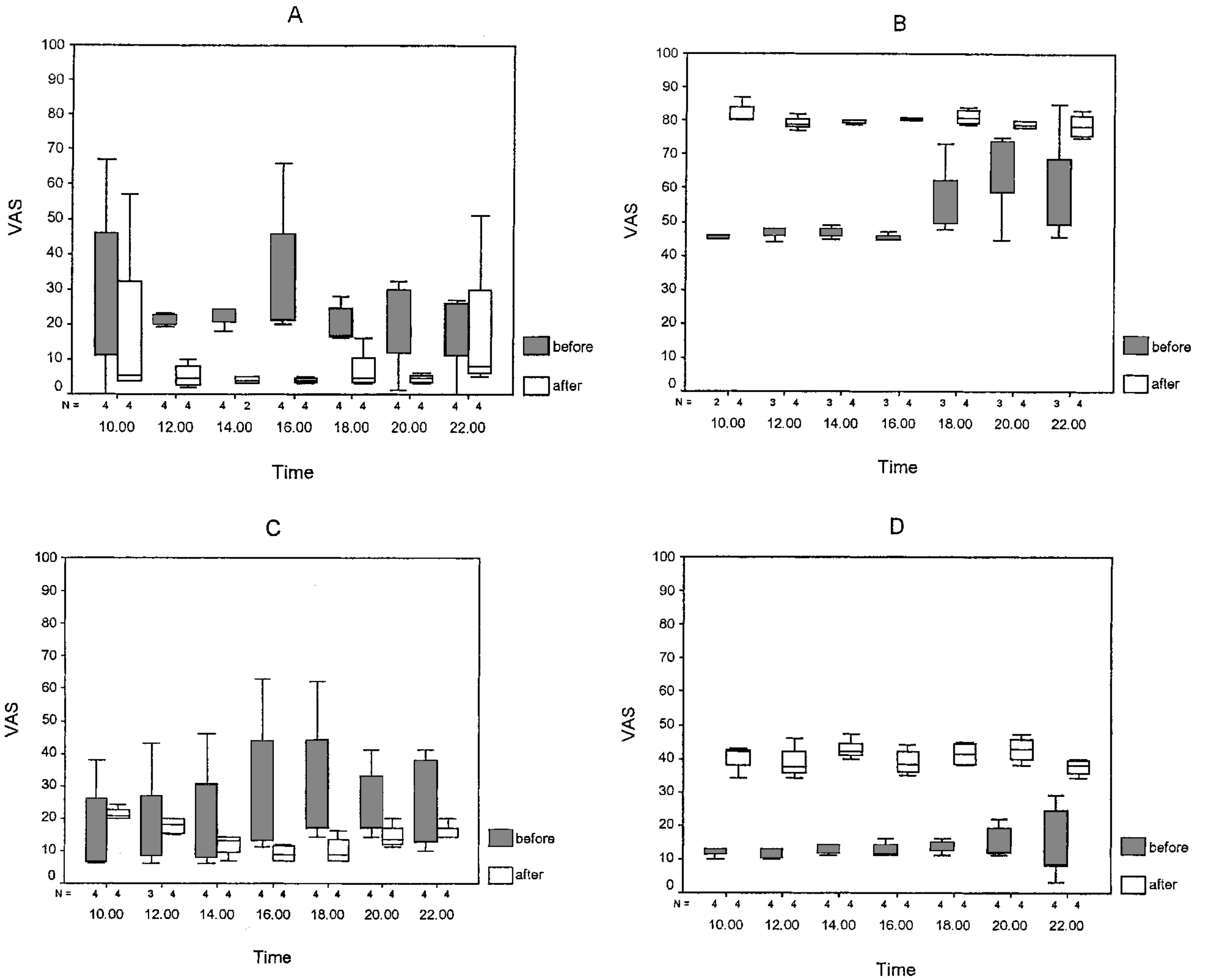

Fig. 2. Four individuals with spinal cord injury; A and B are FES subjects, C and D control subjects. Subjects A and C show decreased self-rated spasticity while subjects B and D show increased self-rated spasticity. (N = number of days).

for about 24 hours after electrical stimulation. Our evaluation of resistive torque, EMG activity and MAS rating after FES training was performed in the week after the last training session and showed no significant changes. A study by Robinson et al. (15) evaluated the long-term effects of electrical stimulation in subjects with SCI and showed a tendency toward increasing spasticity.

The present study showed a large variation in EMG and torque values between subjects in this rather small population, which was a problem when the effect of spasticity intervention was evaluated. Conversely, this variation was beneficial when different methods of spasticity evaluation were compared for correlation.

We have earlier investigated self-rated spasticity in subjects with SCI (28), and showed significant fluctuations in spasticity during the day in subjects with cervical injuries compared to that of individuals with thoracic injuries. No significant differences were shown between consecutive days within subjects (28). The results of self-ratings in the present study showed changed spasticity in some and unchanged in others after FES intervention.

In conclusion, leg muscle volume increased 10% as a result of 6 months of FES training in motor-complete tetraplegic SCI individuals. The objective and subjective evaluation of movement-provoked passive viscoelastic and active muscle resistance showed no change as a result of the FES intervention.

ACKNOWLEDGEMENTS

Supported by grants from the Swedish Medical Research Council (no. 19x-05720, 14x-06555), the Norrbacka Eugenia Foundation, the Karolinska Institute Committee for the Caring Sciences, the Swedish Foundation for Healthcare Sciences and Allergy Research, Cancer- och Trafikskadades Riksförbund (CTRF), Riksförbundet för Trafik- och Polioskadade (RTP), the Ebba and Gustav Ljungren Foundation and the Spinalis Foundation. We thank Professor Jan Ekholm for generously providing technical and laboratory facilities. The excellent technical and statistical assistance of Ms Lisbeth Broman is gratefully acknowledged.

REFERENCES

1. Kandel ER, Schwartz JH, Jessell TM, editors. The principles of neural science (third edition). New York: Elsevier; 1991.
2. Sköld C, Levi R, Seiger Å. Spasticity after traumatic spinal cord injury: nature, severity, and location. Arch Phys Med Rehabil 1999; 80: 1548–1557.
3. Lance JW. The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture. Neurology 1980; 30: 1303–1313.
4. Katz RT, Rovai GP, Brait C, Rymer WZ. Objective quantification of spastic hypertonia: correlation with clinical findings. Arch Phys Med Rehabil 1992; 73: 339–347.
5. O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. Brain 1996; 119: 1737–1749.
6. Walker JB. Modulation of spasticity: prolonged suppression of a spinal reflex by electrical stimulation. Science 1982; 216: 203–204.
7. Han JS, Chen XH, Yuan Y, Yan SC. Transcutaneous electrical nerve stimulation for treatment of spinal spasticity. Chin Med J 1994; 107: 6–11.
8. Seib TP, Price R, Reyes MR, Lehmann JF. The quantitative measurement of spasticity: effect of cutaneous electrical stimulation. Arch Phys Med Rehabil 1994; 75: 746–750.
9. Odeen I, Knutsson E. Evaluation of the effects of muscle stretch and weight load in patients with spastic paraplegia. Scand J Rehabil Med 1981; 13: 117–121.
10. Bohannon RW. Tilt table standing for reducing spasticity after spinal cord injury. Arch Phys Med Rehabil 1993; 74: 1121–1122.
11. Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurement. Arch Phys Med Rehabil 1989; 70: 144–155.
12. Yarkony GM, Roth EJ, Cybulski GR, Jaeger RJ. Neuromuscular stimulation in spinal cord injury. II: Prevention of secondary complications. Arch Phys Med Rehabil 1992; 73: 195–200.
13. Granat MH, Ferguson AC, Andrews BJ, Delargy M. The role of functional electrical stimulation in the rehabilitation of patients with incomplete spinal cord injury – observed benefits during gait studies. Paraplegia 1993; 31: 207–215.
14. Douglas AJ, Walsh EG, Wright GW, Creasey GH, Edmond P. The effects of neuromuscular stimulation on muscle tone at the knee in paraplegia. Exp Physiol 1991; 76: 357–367.
15. Robinson CJ, Kett NA, Bolam JM. Spasticity in spinal cord injured patients. 2. Initial measures and long-term effects of surface electrical stimulation. Arch Phys Med Rehabil 1988; 69: 862–868.
16. Spungen AM, Bauman WA, Wang J, Pierson RN, Jr. Measurement of body fat in individuals with tetraplegia: a comparison of eight clinical methods. Paraplegia 1995; 33: 402–408.
17. Lönn L, Johansson G, Sjöström L, Kvist H, Odén A, Bengtsson BA. Body composition and tissue distributions in growth hormone deficient adults before and after growth hormone treatment. Obes Res 1996; 4: 45–54.
18. Hjeltnes N, Aksnes AK, Birkeland KI, Johansen J, Lannem A, Wallberg-Henriksson H. Improved body composition after 8 wk of electrically stimulated leg cycling in tetraplegic patients. Am J Physiol 1997; 273: R1072–1079.
19. Block JE, Steinbach LS, Friedlander AL, Steiger P, Ellis W, Morris JM, Genant HK. Electrically-stimulated muscle hypertrophy in paraplegia: assessment by quantitative CT. J Comput Assist Tomogr 1989; 13: 852–854.
20. Sloan KE, Bremner LA, Byrne J, Day RE, Scull ER. Musculoskeletal effects of an electrical stimulation induced cycling programme in the spinal injured. Paraplegia 1994; 32: 407–415.
21. Mohr T, Andersen JL, Biering-Sorensen F, Galbo H, Bangsbo J, Wagner A, Kjaer M. Long-term adaptation to electrically induced cycle training in severe spinal cord injured individuals. Spinal Cord 1997; 35: 1–16.
22. Kvist H, Sjöström L, Tylén U. Adipose tissue volume determinations in women by computed tomography: technical considerations. Int J Obes 1986; 10: 53–67.
23. Chowdhury B, Sjöström L, Alpsten M, Kostanty J, Kvist H, Löfgren R. A multicompartment body composition technique based on computerized tomography. Int J Obes Relat Metab Disord 1994; 18: 219–234.
24. Wang ZM, Pierson RN, Heymsfield SB. The five-level model: a new approach to organizing body-composition research. Am J Clin Nutr 1992; 56: 19–28.
25. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 1987; 67: 206–207.
26. Sköld C, Harms-Ringdahl K, Hultling C, Levi R, Seiger Å. Simultaneous Ashworth measurements and electromyographic recordings in tetraplegic patients. Arch Phys Med Rehabil 1998; 79: 959–965.
27. DeLuca CJ. The use of surface electromyography in biomechanics. J Appl Biomech 1997; 13: 135–163.
28. Sköld C. Spasticity in spinal cord injury: self- and clinically rated intrinsic fluctuations and intervention-induced changes. Arch Phys Med Rehabil 2000; 81: 144–149.
29. Bauman WA, Spungen AM, Raza M, Rothstein J, Zhang RL, Zhong YG, et al. Coronary artery disease: metabolic risk factors and latent disease in individuals with paraplegia. Mt Sinai J Med 1992; 59: 163–168.
30. Bauman WA, Spungen AM, Zhong YG, Mobbs CV. Plasma leptin is directly related to body adiposity in subjects with spinal cord injury. Horm Metab Res 1996; 28: 732–736.
31. Sunnerhagen KS, Svantesson U, Lönn L, Krotkiewski M, Grimby G. Upper motor neuron lesions: their effect on muscle performance and appearance in stroke patients with minor motor impairment. Arch Phys Med Rehabil 1999; 80: 155–161.
32. Daly JJ, Marsolais EB, Mendell LM, Rymer WZ, Stefanovska A, Wolpaw JR, Kantor C. Therapeutic neural effects of electrical stimulation. IEEE Trans Rehabil Eng 1996; 4: 218–230.
33. Stefanovska A, Vodovnik L, Gros N, Rebersek S, Acimovic-Janezic R. FES and spasticity. IEEE Trans Biomed Eng 1989; 36: 738–745.
34. Perell K, Scremin A, Scremin O, Kunkel C. Quantifying muscle tone in spinal cord injury patients using isokinetic dynamometric techniques. Paraplegia 1996; 34: 46–53.
35. Douglas AJ, Walsh EG, Wright GW, Edmond P. Muscle tone around the human knee in paraplegia. Q J Exp Physiol 1989; 74: 897–905.
36. Robinson CJ, Kett NA, Bolam JM. Spasticity in spinal cord injured patients: 1. Short-term effects of surface electrical stimulation. Arch Phys Med Rehabil 1988; 69: 598–604.