MOVEMENT-PROVOKED MUSCLE TORQUE AND EMG ACTIVITY IN LONGSTANDING MOTOR COMPLETE SPINAL CORD INJURED INDIVIDUALS

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Muscle resistance was evaluated by measurement of movement-provoked torque performed during simultaneous thigh muscle EMG recordings in individuals with a motor complete spinal cord injury (SCI). Fifteen men with a motor complete (ASIA grade A or B) cervical injury participated in the study. The activity started at an average of 0.11 seconds after the start of the provoking movement as evidenced by EMG recordings. However, no activity at all was found before the end of the movement provocation for 0.3 seconds in >60% of the test situations, whereas muscle torque was recorded in all cases. Significantly higher resistive muscle torque (p = 0.049) was provoked during extension movement compared with that of flexion. On the contrary, the maximum muscle activity was significantly higher (p = 0.009) during flexion movement compared with that during extension, with no differences between muscle groups. The resistive muscle torque seems to measure the passive viscoelastic component rather then the active spastic component of the movement-provoked muscle resistance in our group of motor complete SCI individuals.

Key words: SCI, spasticity, viscoelasticity, involuntary movements, resistive torque.

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INTRODUCTION

The basis for this study was to evaluate active and passive components of movement-provoked muscle resistance. In individuals with motor complete tetraplegic SCI, knee moments were biomechanically induced during simultaneous EMG recordings.

Two recent studies (1, 2) have evaluated involuntary dynamic muscle torque as a measure to distinguish between individuals with spasticity and non-injured by summation of 4 or 5 consecutive torque peaks. However, none of these studies have evaluated simultaneous EMG activity and therefore it has not been possible to judge whether resistive torque is induced by muscle activity and/or passive resistive components. Spasticity has been defined to include the following active muscle components; increased muscles tone (tonic stretch reflex), increased tendon reflexes (phasic stretch reflex), increased exteroceptive reflexes (flexion reflex) and pathological radiation (clonus) (3–5). Each of these variables may include coactivation. This definition has been used in our former studies as the operational definition of spasticity (6–8).

Several authors have emphasized that comparisons of muscle resistance are possible only when identical test positions are used since e.g. muscle stiffness and spasticity will vary with the current degree of muscle elongation (1, 9). Also, the velocity of elongation has been shown to affect the resistive muscle torque differently for individuals with spasticity and non-injured, the former reacting to higher velocity by increased torque values (1). Recent studies (1, 2, 9, 10) have used the same variation of velocities and Perell et al. (1) recommended 120°/second to demonstrate differences in resistive muscle torque between groups with spastic and normal muscles, respectively. Maximum velocity was used in the present study for resemblance with the clinical situation using the Modified Ashworth Scale (MAS) ratings for spasticity evaluation. In a former study the velocity of MAS ratings was estimated to be approximately 350°/second (6).

We recently showed movement-provoked MAS ratings of spasticity to correlate significantly with simultaneously recorded EMG activity (peak, duration) of thigh muscles in motor complete SCI individuals (6).

The aim of this study was to measure movement-provoked resistive muscle torque during simultaneous thigh muscle EMG recordings in individuals with a motor complete spinal cord injury (SCI). The torque evaluation was assumed to represent either muscle activity (i.e. spasticity) only, passive resistive components (i.e. viscoelasticity) only or a combination thereof.

MATERIAL AND METHODS

Subjects

Out of the 353 individuals with SCI, constituting the near-total prevalence group in the greater Stockholm area, 15 males with a motor complete (ASIA grade A or B) cervical injury were randomly selected to participate in the study. All subjects had a spastic paresis. The mean age was 33 years (range 21 to 48 years) with a mean time since injury of 9 years (range 1 to 21 years). Two of the subjects used Baclofen in doses of 20 to 60 mg/day orally. The mean MAS rated spasticity for knee flexion movement was 1.3 for the right side and 1.0 for the left (range 0-4) and for knee extension movement 1.0 for the right side and 1.1 for the left



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.

(range 0–4). All individuals had to be able to lay on their back, with hips and knees straight, to be included in the study.

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

Test procedure

Movements, ranging from 10° to 90° and vice versa, of the knee were performed with a velocity of 250°/second by a Kin-Com dynamometer (125E Plus computer, CHATTECX Corp, Chattanooga Group, Hixon, TN 37343) using the executive software to move the resistive lever at the determined speed. Data were further transferred to the Microsoft Excel 5.0 program for continued calculations. The subject's right knee was always tested first. One flexion of the knee was performed and thereafter one extension. The subjects were tested seated inclined with a backrest giving a hip flexion angle of 40° during the test procedure (Fig. 1). Each subject was strapped with belts both around the pelvis and the distal thighs. Measurements of extensor and flexor resistive torque, respectively, during simultaneous EMG recordings were performed using the Kin-Com dynamometer. Positive torque values represented pushing against the lever (resistive force in antagonists) and negative torque values represented pulling away from the lever (agonistic force).

Disposable EMG Blue Sensor N-00-5 surface electrodes (Medicotest A/S, Sävedalen, Sweden) were placed with an inter-center-electrode distance of 30 mm bilaterally over the bulge of the rectus femoris and the

	Table I. Resistive torque	peak (Nm) values at na	o, less or more than 50% duration o	f EMG muscle activity (uV
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	Side	Movement	n ^b	Torque peak mean	EMG max		EMG actual ^c	
EMG					Antag. mean	Agonist mean	Antag. mean	Agonist mean
No ^a	Right	Flexion	8	17.4				
	C C	Extension	11	20.8				
	Left	Flexion	7	24.0				
		Extension	10	29.3				
Yes <50%	Right	Flexion	5	12.7	196.2	36.3	72.2	7.3
	Ū.	Extension	3	13.5	62.7	79.8	28.7	13.6
	Left	Flexion	0	0.0	0.0	0.0	0.0	0.0
		Extension	2	23.0	27.0	30.0	2.0	30.0
Yes >50%	Right	Flexion	1	1.8	534.0	213.0	231.0	46.0
	e	Extension	0	0.0	0.0	0.0	0.0	0.0
	Left	Flexion	7	20.7	222.7	164.8	34.1	164.8
		Extension	2	17.7	25.0	51.0	5.0	51.0

^a No defined as duration of EMG activity less than 0.02 second.

^b n = total number of tests (56 = 14 subjects \times 2 sides \times 2 movements).

^c Actual EMG at torque peak value.



Fig. 2. Movement-provoked resistive torque (Nm) and EMG activity (uV) during a flexion movement of the left knee in one SCI subject. Positive torque peak values indicate resistance to the flexion movement and negative torque peak values add to the flexion movement.

lateral biceps femoris muscles. The EMG setup was part of the Kin-Com dynamometer training and testing system. The EMG signals were amplified $(1000\times)$ in alternating current miniature preamplifiers and fed to an EMG processing system. The signals were fullwave rectified and bandpass filtered, using a three-pole active Paynter filter (20–1000 Hz) and sampled at 100 Hz by the 12-bit analogue/digital converter of the Kin-Com system.

At the same test occasion, presented elsewhere (6), clinical MAS ratings of thigh muscle spasticity during knee flexion and extension and simultaneous EMG recordings were performed before the Kin-Com dynamometer testing of the right knee joint and after the Kin-Com dynamometer testing of the left knee joint (6). The whole test procedure took 30–45 minutes.

Figure 1 describes the time course and temporal relationships of the measures performed: biomechanical torque and EMG activity (present study), MAS rating and EMG activity (presented elsewhere, see ref. 6), respectively.

For KinCom measurements of resistive muscle torque and EMG recordings, one of the 15 subjects was not included in the calculations due to incomplete data. For 7 subjects the tests were performed on two different occasions 6 months apart, thus enabling analysis over time. Half of the group (8 subjects) was not evaluated for differences between

test occasions, due to functional electrical stimulation (FES) training. The effect of FES training on spasticity has been evaluated elsewhere.

Data processing

The EMG baseline for the 0.3-second recording period during each movement was defined as the mean of the first 10 point estimates (0.02 second) plus two standard deviations (11) for each subject, side, and muscle group, respectively. Duration of EMG activity was defined as a minimum of 10 consecutive point estimates (0.02 second) above baseline. Maximum EMG activity was defined as the highest voltage sampled during the whole recording period. Peak torque was defined as

Data analyses and statistics

Both the movement-provoke d resistive torque and the recorded EMG activity were evaluated for differences between test occasions and between sides, movement directions and muscle groups using one-way ANOVA. Pearson's coefficients were calculated for correlations between resistive torque values and EMG activity values. General Linear Model (MANOVA) for repeated measures were used to evaluate

Table II. Correlations between movement-provoked resistive muscle torque peak and EMG max or actual EMG activity at peak torque (n = 14)

			EMG max		EMG actual	
Side	Movement		Antagonist	Agonist	Antagonist	Agonist
Right	Flexion Extension	Torque peak Torque peak	-0.27 -0.16	$-0.40 \\ -0.66$	-0.41 0.18	$-0.50 \\ -0.38$
Left	Flexion Extension	Torque peak Torque peak	-0.14 -0.09	$-0.20 \\ -0.12$	-0.17 -0.03	$-0.28 \\ -0.28$

differences between test occasions within subjects for both resistive torque values and EMG muscle activity values, separately. The Bonferoni procedure, being a post hoc test, was used to identify changes in individual mean values between test occasions not shown for group data values. Descriptive statistics were used to describe resistive torque and EMG activity values of different durations.

RESULTS

The start of the EMG activity occurred at an average of 0.11 second after the start of the provoking movement. However, in >60% of the test situations no EMG activity at all was found before the end of the provoking movement as performed by the Kin-Com dynamometer, whereas resistive torque was found in all cases. The mean resistive torque peak varied between 17.4 Nm and 29.3 Nm when no EMG activity was shown and between 1.8 Nm and 23.0 Nm when EMG activity was shown (Table I).

In Figure 2 the simultaneously recorded resistive torque and EMG activity of one flexing movement of the left knee joint of one subject is shown. A lower torque value was seen when EMG muscle activity was traced while a higher torque value was seen when the EMG activity was silent.

Significantly higher torque peak resistance was seen after movement provocation of the left side (n = 15, p < 0.001), compared with and tested after the right side and during extension movement compared with that of flexion (n = 15, p = 0.049). Contrary, the maximum EMG thigh muscle activity was significantly higher during flexion movement compared with that during extension (n = 15, p = 0.009), with no differences between muscle groups. For the 7 subjects not undergoing intervention (FES training) no differences were seen between the two test occasions for torque and EMG values, respectively.

The resistive torque curve during the movement showed the same pattern at both occasions in 50% of the tests (n = 7) and in the rest of the tests an opposite pattern at the second occasion compared with that at the first occasion. Significant differences between test occasions for movement-provoked resistive torque peaks (n = 7, p < 0.001) and for maximum EMG activity (n = 7, p = 0.038) was found within each subject. No differences were seen between actual EMG activity values at peak torque within each subject.

Correlations were calculated between movement-provoked resistive torque and simultaneous EMG activity values. Peak torque and maximum EMG activity, as well as actual EMG activity at peak torque were compared and showed no significant correlations (Table II).

DISCUSSION

This study measured the movement-provoked resistive muscle torque during simultaneous recordings of EMG activity in individuals with SCI. The subjects were homogeneous with regard to involuntary muscle activity (ASIA-grade; A, B). The relationship between this muscle activity (i.e. spasticity) and passive muscle components (i.e. viscoelasticity) of the resistive torque was analysed.

The most important finding of the present study was that a movement provocation velocity of 250°/second did not elicit any EMG activity in more than 60% of the tests. Resistive muscle torque, on the other hand, was seen in 100% of the tested movement provocations. In earlier studies (1, 2, 9, 10) the velocity used never exceeded 120°/second, which was probably not fast enough to elicit spasticity. Lamontagne et al. (12) evaluated the reflex and non-reflex muscle resistance provoked by either a hand-held or an isokinetic dynamometer. The more prevalent EMG activity was related to higher velocities. Isokinetic movement provocation at 180°/second showed EMG activity in 3 of the 9 subjects but in all subjects except one when the hand-held dynamometer (187-394°/second) was used. We have shown that EMG activity was elicited in all 15 subjects when manually applied movement provocation was performed at approximately 350°/second using the MAS for spasticity rating (6).

One major difference in the present study from our earlier study (6) is the time of EMG recording. Even though the velocity during the movement-provoked MAS rating was faster, the EMG recording continued for 10 seconds in our former study (6). In the present study the EMG recording lasted as long as the movement provocation. During this biomechanical movement provocation the EMG activity either started and peaked or just peaked after the end of the recording period of 0.3 second, since 60% of the provocations showed no EMG activity at all before the end of that recording period. In comparison, during movement-provoked MAS-rating, EMG activity was seen in all cases.

The present study showed that movement provoked resistive muscle torque values were not increased during EMG verified muscle activity (i.e. spasticity). This is compatible with reports by both Perell et al. (1) and Douglas et al. (13) who showed muscle stiffness to be lower in spastic subjects than in noninjured individuals, as evidenced by lower resistive muscle torque after isokinetic movement provocation in the spastic subjects in the former study and as significantly higher peak velocity in the second study.

Knutsson et al. (14) showed decreased voluntary muscle torque with increasing velocity, both in healthy individuals and in patients with spastic paraparesis. Broberg & Grimby (15) showed, in a heterogeneous group of subjects with spasticity in the lower extremities, increased resistive muscle torque with increasing velocity by passive movement provocation.

The effect of test position on spasticity and viscoelastic muscle components has been discussed with higher resistive muscle torque values for extension movement when SCI subjects were seated and for flexion movement in supine (1, 2). Our subjects were seated inclined with a backrest giving a hip flexion angle of 40° . Still, the resistive muscle torque values were significantly higher during extension movement. This higher resistive muscle torque during extension movement and inversely the significantly higher EMG activity during flexion movement may indicate that knee flexors were mainly stiff (passive viscoelastic muscle resistance) and knee extensors were mainly spastic (active involuntary muscle resistance) in our group of motor complete SCI subjects.

The order between tests and thus the number of repetitions performed on one side before testing the other side, is known to influence neuromuscular activity (i.e. spasticity) but to our knowledge not the viscoelastic muscle components (6, 9, 13). The present study showed significantly lower movementprovoked resistive muscle torque values for the right side (tested first) with no differences in EMG activity between sides. The rather small population may explain differences in torque values seen between sides in the present study where possible individual differences, inherent to the injury and not possible to detect with the ASIA classification, may have a comparably large impact. Franzoi et al. (9) found no differences in torque values between sides while they used both legs individually for evaluation.

In the present study, no correlations were found between passive movement-provoked resistive peak torque and maximum EMG or actual EMG activity at peak torque. This is at variance with Franzoi et al. (9) and Broberg & Grimby (15) expecting a positive correlation between EMG activity and resistive torque, since both studies showed higher peak torque values (Nm) with more severe spasticity (as assessed with Ashworth). Most of the correlation coefficients in the present study were negative indicating only a modest, if any, contribution to the movement provoked muscle torque by the contraction associated EMG activity, irrespective of muscle group. Although standardized and validated (11), EMG recordings in the present study obviously only represent a fraction of the total electrical activity of the anatomical muscle recorded. This fact may weaken the correlation between torque and EMG even if there would be causal relationship between the two.

In conclusion the resistive muscle torque seems to measure the passive viscoelastic component rather then the active spastic component of the movement-provoked muscle resistance in our group of motor complete SCI individuals.

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