

REPEATED MAXIMUM RECIPROCAL KNEE MOVEMENTS IN PATIENTS WITH MINIMAL OVERT SYMPTOMS AFTER ISCHAEMIC STROKE: AN EVALUATION OF MECHANICAL PERFORMANCE AND EMG

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ABSTRACT. The ability to perform reciprocal knee flexions and knee extensions was investigated in patients with minimal or no overt motor symptoms after stroke. Ten patients and 22 controls performed 10 maximal reciprocal knee extensions and knee flexions without intervening rest period using an isokinetic dynamometer (Cybex II). Peak torque (PT; Nm), signal amplitude (RMS) and mean frequency (f_{mean}) of the electromyography were registered for each extension and flexion separately and as the ratio extension/flexion. The patients exhibited pronounced motor deficit despite no or minimal overt clinical symptoms. The reduced motor capacity in the knee muscles was shown as a decrease in the PT and in a different electromyographic pattern compared with the controls. A bilateral affection was found with the formerly hemiparetic side mostly affected. Repeated reciprocal contractions influenced the motor performance, shown as an increase of the PT ratio in the patients, which was especially pronounced with an increasing number of contractions.

Key words: dynamic movements, electromyography, hemiplegia, muscle strength, physical therapy, reciprocal, repeated.

INTRODUCTION

Normal reciprocal voluntary movements are regulated by central inhibition in the antagonists in parallel with excitation in the agonists, already described early in this century (33). In humans it has been demonstrated that supraspinal mechanisms simultaneously regulate the activity in Ia inhibitory interneurons and the motoneurons during extension-flexion movements (7). When there is an upper motor neuron syndrome this regulation can be disordered, which might be clinically manifested as increased co-contraction (17). However, the mechanisms behind impairment when performing reciprocal movements are poorly understood and, also, a decreased recruitment of the agonists has been reported (4, 11, 30, 31). Moreover, it

has been suggested that peripheral rather than central origins change the ability to perform elbow movements in patients with hemiparesis (39).

When voluntary strength is investigated in hemiparetic patients, divergent opinions exist on which muscle groups are affected most. Colebatch & Gandevia (5) and Gandevia (13) reported, consistent with the description that the course of recovery is regularity in sequence (37), that the distal muscles were more severely affected than the proximal muscles. In contrast, Adams et al. (1) were not able to demonstrate any significant differences between muscles of the lower limb. Colebatch et al. (6) found that the elbow flexors were relatively more weakened than the elbow extensors, contrary to conventional clinical teaching.

Thus, the results concerning the muscular (and electromyographic—EMG) output in patients with upper motor neuron syndrome are inconsistent. The diverse results could at least partly be explained by the fact that patients with different motor capacity have been compared and that different study designs have been used. Hence, studies have been performed either during static or during dynamic conditions, or during unidirectional or reciprocal movements.

Another problem is that patients who, according to clinically used scales, achieve complete or nearly complete restoration of motor function after stroke very often report fatigue in the former paretic side.

The aim of the present study was to investigate the capacity of repetitive reciprocal maximal voluntary dynamic knee movements in patients with minimal or no overt motor symptoms after ischaemic stroke and in healthy controls.

MATERIAL AND METHODS

Subjects

Patients from the departments of Neurology and Physical Medicine and Rehabilitation, University Hospital of Umeå participated in this study. The inclusion criteria was a cerebral

infarction with initial hemiparesis with clinically complete or nearly complete restoration of motor function. The motor capacity according to the scale of Fugl-Meyer et al. (12) was 95 (± 4) out of 100 points (range 89–100). The corresponding score for the lower limbs separately was 33 (± 1) out of 34 (range 30–34). The motor capacity according to the scale of Fugl-Meyer was evaluated simultaneously with the other investigations of muscle performance. Six had initially a right-sided hemiparesis and four a left-sided one. The time since damage was 9 (± 5) months, range 3–18 months. The patients investigated were 7 males and 3 females, age 40 (± 7) years, range 27–52. Twenty-two healthy voluntary subjects participated as controls, 14 males and 8 females, 28 (± 6) years, range 21–44. Before the start of the study, each individual was informed of the testing procedures and thereafter gave written consent. The study was approved by the Research Committee at the Medical Faculty, Umeå University.

Isokinetic test

The tests were performed using an isokinetic dynamometer (Cybex II Lumex, New York). The test consisted of 10 repeated reciprocal maximal contractions at the angular velocity of 90°s^{-1} ($1.52\text{ rad}\cdot\text{s}^{-1}$), i.e. each contraction cycle consisted of a maximal knee extension immediately followed by a maximal flexion without any intervening rest. The angular velocity was chosen according to recommendations given by Dvir (9) and has been used in several other studies at our laboratory. Before the test, at submaximal levels, the subjects were instructed on how to achieve the standardized range of motion during the contraction cycle. During the experiment, subjects were frequently encouraged verbally to perform maximally. Both limbs were tested with an intervening 2 hour rest. For the patient group the unaffected limb, which was defined as the limb with no former symptoms, was first tested. For the control group the left and right limb were randomly selected. The subjects lay in a supine position with the hip of the investigated thigh flexed at 20 degrees. The hip and the investigated thigh were fixed with belts. Great care was taken to align the flexion-extension axis of the knee joint with the movement axis of the dynamometer and to ensure, with the help of the belts, that the position was maintained throughout the experiment.

Electromyographic signals, using silver-silver-chloride surface electrodes (centre-to-centre distance: 20 mm), were obtained from the rectus femoris, vastus lateralis, semitendinosus and biceps femoris muscles. The electrodes were placed over the most prominent part of the muscles and on similar sites on the different limbs. Before the electrodes were attached, the skin area was dry shaved and rubbed with alcohol and ether (4:1) and ECG-electrode paste was attached to the electrodes (Medicotest, Ølstykke, Denmark). The rest EMG and the true weight of the leg were measured before the actual test, in order to compensate for background noise (i.e. reduction) and gravity, respectively.

A specially designed data acquisition system for analysis (Myoelectric Signal Acquisition System—MYSAS) of the EMG signals and related mechanical parameters was used for registration and analysis (for details concerning registration and analysis of EMG see 19, 23). In this study two EMG parameters and one mechanical parameter were calculated, i.e. root mean square (RMS) and mean frequency of the power density spectrum (f_{mean}) together with peak torque (PT).

Analysis and statistics

Throughout the 10 reciprocal knee movements, the PT of the extension contractions and the flexion contractions were analysed both separately and as ratios. The two PT ratios calculated were:

1. the ratio between extension and flexion for each contraction cycle; and
2. the ratio between the two limbs (in control group: non-dominant / dominant; and in patient group: affected / unaffected for each phase of the contraction cycle).

The two EMG parameters (RMS and f_{mean}) were determined separately for the extension and flexion phases. All the RMS values were expressed as percentages of the initial contraction. Changes during the 10 contractions were determined by comparing the initial values, i.e. mean of contraction nos. 1–3, and the final values, i.e. mean of contraction nos. 8–10. Results in the text, tables and figures are generally given as mean values \pm one standard deviation (± 1 SD). Wilcoxon signed-rank test and Kruskal-Wallis one-way analysis of variance tests were used to test intra-individual differences and differences between groups, respectively. All statistical

Table I. The peak torque (PT) values for the knee extension phase and for the knee flexion phase of reciprocal movements.

Comparison between dominant (D) and non-dominant (ND) limbs of the controls and between unaffected (UA) and affected (A) limbs of the patients clinically recovered from hemiplegia with no or minimal overt motor symptoms after brain infarction. Means ± 1 SD are shown

Contractions	Peak torque (PT, Nm)					
	Controls			Patients		
	D (n = 22)	ND (n = 22)	p	UA (n = 10)	A (n = 10)	p
<i>Extension</i>						
Mean (1–10)	166	164		142	113	0.009
SD	± 50	± 47	0.390	± 40	± 47	
<i>Flexion</i>						
Mean (1–10)	86	84		67	46	0.005
SD	± 29	± 27	0.550	± 23	± 18	

p refers to level of significance.

Table II. The peak torque (PT) values of knee extension phase and knee flexion phase.

Comparison between the initial (mean of contractions 1–3) and the final (mean of contractions 8–10), for dominant (D) and non-dominant (ND) limbs of the controls and for unaffected (UA) and affected (A) limbs of the patients clinically recovered from hemiplegia with no or minimal overt motor symptoms after brain infarction. Means \pm 1 SD are shown

	Peak torque (PT, Nm)					
	Contraction 1–3	Contraction 8–10	<i>p</i>	Contraction 1–3	Contraction 8–10	<i>p</i>
Controls	<u>D</u>	<u>D</u>		<u>ND</u>	<u>ND</u>	
Extension						
Mean	172	160		168	159	
SD	\pm 53	\pm 49	0.007	\pm 47	\pm 47	0.005
Flexion						
Mean	89	84		86	81	
SD	\pm 29	\pm 29	0.017	\pm 27	\pm 28	0.007
Patients	<u>UA</u>	<u>UA</u>		<u>A</u>	<u>A</u>	
Extension						
Mean	146	137		115	112	
SD	\pm 38	\pm 42	0.013	\pm 45	\pm 50	0.575
Flexion						
Mean	70	51		63	43	
SD	\pm 24	\pm 18	0.007	\pm 23	\pm 19	0.013

p refers to level of significance.

tests were performed at the 5% significance level ($p \leq 0.05$, two-tailed) using the statistical package SYSTAT[®] for the Macintosh (version 5.2, Systat Inc., Evanston, IL).

RESULTS

Peak torque (PT)

Peak torque (PT) in the patients was generally significantly lower on the affected side compared to the unaffected side (Table I). A significant decrease of PT was found when the initial three contractions (contraction nos. 1–3) were compared with the final contractions (contraction nos. 8–

10), during both the extension phase and the flexion phase in each limb of the controls (Table II). A significant decrease in PT was found for extension and flexion on the unaffected side, and for flexion but not for extension on the affected side (Table II).

The relative level (the mean of PT for the 10 contractions, affected vs affected side) of the flexors ($70 \pm 4\%$), was significantly lower ($p = 0.005$) than for the extensors ($79 \pm 3\%$) in the patient group. In Fig.1 the corresponding ratios are depicted for both the patients and the controls.

The mean of the 10 extension/flexion ratios for the

Table III. The knee extension/flexion peak torque (PT) ratios of each limb.

Comparison between dominant (D) and non-dominant (ND) limbs of the controls and between unaffected (UA) and affected (A) limbs of the patients clinically recovered from hemiplegia with no or minimal overt motor symptoms after brain infarction. Means \pm 1 SD are shown

Contractions	Controls			Patients		
	D (<i>n</i> = 22)	ND (<i>n</i> = 22)	<i>p</i>	UA (<i>n</i> = 10)	A (<i>n</i> = 10)	<i>p</i>
Mean (1–10)	1.98	2.00		2.23	2.60	
SD	\pm 0.34	\pm 0.31	0.720	\pm 0.57	\pm 0.57	0.059
Mean (1–3)	2.00	2.02		2.19	2.29	
SD	\pm 0.36	\pm 0.35	0.638	\pm 0.49	\pm 0.52	0.333
Mean (8–10)	1.98	2.01		2.33	2.83	
SD	\pm 0.33	\pm 0.35	0.626	\pm 0.52	\pm 0.69	0.074

p refers to level of significance.

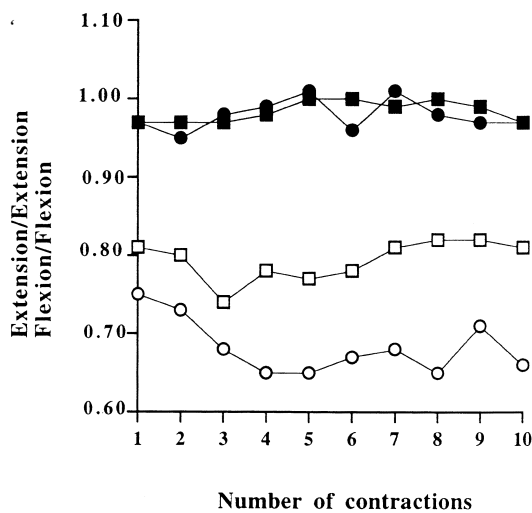


Fig. 1. The ratios peak torque (PT) between the limbs for knee extensions and knee flexions in the controls and in the patients throughout the 10 contraction cycles. The ratios of the control group: extension non-dominant (ND) limb/extension dominant (D) limb (■) and flexion non-dominant (ND) limb/flexion dominant (D) limb (●), and the ratios of the patient group: extension affected (A) limb/extension unaffected (UA) limb (□) and flexion affected (A) limb/flexion unaffected (UA) limbs (○).

affected limbs was significantly higher for the patients as compared to the controls (vs dominant limb of the control, $p = 0.001$; vs non-dominant limb of the control, $p = 0.002$) (Table III). While in the controls no significant difference was found between the limbs, in patients there was a tendency towards a difference in the ratio, although this was not statistically significant ($p = 0.059$) (Table III).

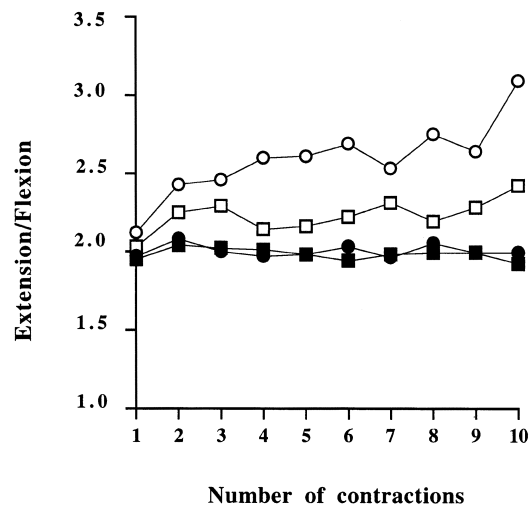


Fig. 2. The PT ratios knee extension/knee flexion for each limb of the control group, dominant (■) and non-dominant (●) and of the patient group, unaffected (□) and affected limbs (○).

For the affected limb a significant increase of the extension/flexion ratio occurred throughout the successive contractions (contraction nos. 1–3 vs nos. 8–10) ($p = 0.047$) (Fig. 2).

f_{mean} of EMG

Significant decreases of the f_{mean} occurred for all muscles under investigation in both limbs of the controls and in the unaffected limb in the patient group, throughout the 10 contractions (Tables IV and V). By contrast, no significant decrease for any muscle of the affected limb existed in the

Table IV. *f_{mean} of the EMG for the controls in the two knee extensor muscles and the two knee flexor muscles.* Comparisons between the initial (mean of contractions 1–3) and final (mean of contractions 8–10), for the dominant (D) and non-dominant limbs (ND). Means ± 1 SD are shown

	<i>f_{mean} controls</i>					
	D Contraction 1–3	D Contraction 8–10	<i>p</i>	ND Contraction 1–3	ND Contraction 8–10	<i>p</i>
<i>Rectus femoris</i>						
Mean (Hz)	91	80		92	81	
SD	± 16	± 15	<0.001	± 19	± 17	<0.001
<i>Vastus lateralis</i>						
Mean (Hz)	82	79		86	80	
SD	± 12	± 12	0.048	± 13	± 12	0.003
<i>Semitendinosus</i>						
Mean (Hz)	120	107		114	100	
SD	± 20	± 21	0.009	± 24	± 22	<0.001
<i>Biceps femoris</i>						
Mean (Hz)	128	109		124	106	
SD	± 22	± 17	<0.001	± 22	± 22	<0.001

p refers to level of significance.

Table V. f_{mean} of the EMG in the two knee extensor muscles and the two knee flexor muscles.

Comparisons between the initial (mean of contractions 1–3) and final (mean of contractions 8–10), in the unaffected (UA) and the affected (A) limbs in patients clinically recovered from hemiplegia with no or minimal overt motor symptoms after brain infarction. Means ± 1 SD are shown

	f_{mean} patients					
	UA Contraction 1–3	UA Contraction 8–10	p	A Contraction 1–3	A Contraction 8–10	p
<i>Rectus femoris</i>						
Mean (Hz)	101	84		92	88	
SD	± 17	± 12	0.007	± 17	± 14	0.169
<i>Vastus lateralis</i>						
Mean (Hz)	91	78		83	80	
SD	± 16	± 11	0.005	± 8	± 12	0.721
<i>Semitendinosus</i>						
Mean (Hz)	111	98		123	121	
SD	± 8	± 10	0.007	± 23	± 20	0.333
<i>Biceps femoris</i>						
Mean (Hz)	129	119		124	116	
SD	± 22	± 29	0.037	± 28	± 22	0.139

p refers to level of significance.

patients (Table V). No significant difference existed in f_{mean} between the affected and the unaffected limb for any of the muscles during the initial part of the test. For the final part (contraction nos. 8–10), a significant difference between the unaffected and affected limb existed only for the semitendinosus ($p = 0.007$).

RMS of EMG

In general the RMS throughout the 10 contractions had greater variability in the patients than in the controls (Tables VI and VII). Significant increases throughout the

10 contraction cycles were found for all muscles of the controls, except for the semitendinosus. No significant changes were found for any of the investigated muscles of the patients.

DISCUSSION

The PT is the resultant value of agonist and antagonist torques (3) and in patients with upper motor lesions and pronounced motor deficits the PT is mainly affected during

Table VI. The RMS of the EMG (determined as percentage of contraction 1) throughout the 10 contractions in dominant (D) and non-dominant (ND) limbs of the controls.

Comparisons between initial (mean of contractions 1–3) and final (mean of contractions 8–10). Means ± 1 SD are shown

	RMS controls					
	D Contraction 1–3	D Contraction 8–10	p	ND Contraction 1–3	ND Contraction 8–10	p
<i>Rectus femoris</i>						
Mean (%)	109	130		107	134	
SD	± 10	± 24	<0.001	± 6	± 25	<0.001
<i>Vastus lateralis</i>						
Mean (%)	107	126		107	122	
SD	± 10	± 23	0.001	± 11	± 30	0.005
<i>Semitendinosus</i>						
Mean (%)	106	110		108	117	
SD	± 10	± 10	0.179	± 10	± 23	0.055
<i>Biceps femoris</i>						
Mean (%)	106	116		106	121	
SD	± 8	± 19	0.014	± 7	± 17	0.001

p refers to level of significance.

Table VII. The RMS of the EMG (determined as percentage of contraction 1) throughout the 10 contractions for the unaffected (UA) and affected (A) limbs of the patients clinically recovered from hemiplegia with no or minimal overt motor symptoms after brain infarction.

Comparisons between initial (mean of contractions 1–3) and final (mean of contractions 8–10). Means \pm 1 SD are shown

	RMS patients					
	UA Contraction 1–3	UA Contraction 8–10	<i>p</i>	A Contraction 1–3	A Contraction 8–10	<i>p</i>
<i>Rectus femoris</i>						
Mean (%)	134	135		118	120	
SD	\pm 94	\pm 67	0.374	\pm 18	\pm 47	0.859
<i>Vastus lateralis</i>						
Mean (%)	166	170		106	107	
SD	\pm 191	\pm 160	0.110	\pm 21	\pm 27	0.760
<i>Semitendinosus</i>						
Mean (%)	97	93		96	96	
SD	\pm 10	\pm 17	0.646	\pm 16	\pm 23	0.859
<i>Biceps femoris</i>						
Mean (%)	99	97		99	106	
SD	\pm 11	\pm 14	0.610	\pm 11	\pm 22	0.241

p refers to level of significance.

reciprocal movements (2, 7, 8, 31). Due to the higher fatigability of the flexors in the present patient group, an increase of PT extension/flexion ratios occurred throughout the test. Hence, based on the present results, it is reasonable to conclude that also patients with minimal overt symptoms are impaired when performing repeated reciprocal contractions without intervening rest.

A reduced force output of both the extensors and flexors has been found in several other studies, but in contrast to our investigation those measurements were performed unidirectionally and/or with single maximal contractions (3). The definition of fatigue is a failure to maintain mechanical output (10). It has been shown that muscle groups which act as opposing muscles exhibit different patterns of fatigue (32). In healthy subjects the extensors exhibited relatively more fatigability than the flexors during 100 repeated maximal reciprocal contractions (22). In the present patients deficits were found in both the extensors and flexors, although the flexors showed greater reduction, which is in accordance with the findings of Watkins et al. (38). However, the present results differed from the results of Adams et al. (1), who found that no muscle groups were especially affected in the hemiparetic lower limb.

The pattern of muscle fatigue is generally dependent on the composition and recruitment of muscle fibre types in healthy subjects. An increase in the recruitment of high threshold type-II fibres has been related to an increase in force output and increased fatigability in healthy subjects

(36). However, the divergent fatigue patterns between the flexors and the extensors can probably not be explained by differences in recruitment of type-II motor units.

The decreased ability to execute reciprocal movements could, according to the literature, either be due to abnormal increased restraint of the antagonists (20) and/or decreased recruitment of agonists (31). Eventually a decreased reflex threshold might contribute to co-activation of the extensors (20). However, the reflex-mediated changes alone might not explain the differences in fatigability between the extensors and flexors in the patients. Non-reflex-mediated responses (i.e. increases in the passive stiffness of tendons and joints or muscles and in the intrinsic stiffness of the contracting muscle fibres) might also lead to an increased resistance to stretch (34). This could be of importance when explaining the higher fatigability of the flexors of the patients since stiffness is related to the muscle volume (i.e. number of engaged crossbridges) (34) and the extensors are considerably greater in volume than the flexors (29).

In the unfatigued state RMS will reflect both recruitment of motor units and increase in firing frequencies of active motor units. In agreement with other studies of maximal dynamic contractions (23), an increase of RMS occurred in all muscle groups in the controls. In the patients no increase occurred and a great variability of the RMS values in both the unaffected and affected limb was found. As patients with upper motor lesions exhibit an inability to maintain constant short isometric contractions (30), it was likely that a high degree of variability in the RMS would also be found

in dynamic contractions. Both the high degree of variability of RMS and the lack of an increase in RMS during fatigue may be interpreted as an impairment in the regulation of the central drive. That the same pattern concerning RMS was seen in both limbs of the patients might reflect the affection of the "non-paretic" limb, which has been reported in patients with hemiparesis (1, 18). Hence, the real reduction of PT in the affected limb of our patients is unknown, as the unaffected limb cannot be used as a healthy reference. Theoretically it could anticipate that a compensatory utilization of the "unaffected limb" would increase the mechanical performance on that side, even beyond the pre-disease level (thus enhancing the difference between the limbs). Against such an interpretation that the "unaffected" limb is affected, it could be argued that f_{mean} showed a pattern in the "unaffected" limb similar to the controls.

The existing model of EMG in the frequency domain favours the idea that the fibre diameters are the major factor behind the waveform of the motor unit action potential trains and the conduction velocity of the muscle fibre membranes; the latter is linearly related to f_{mean} and median frequency (25). In contrast to this model, a significant positive correlation has been found between f_{mean} and proportion of type-II muscle fibres (14) and some studies only support the model for the type-II muscle fibres (16, 21). No definite conclusion regarding the effect of the force level upon f_{mean} in the *unfatigued* state in clinically healthy subjects can be drawn from the literature (15, 16). No obvious difference existed between the patients and the controls in f_{mean} during the initial part of the test. This finding is contrary to our study of patients with multiple sclerosis (35) who had higher initial f_{mean} than the controls and to Moglia et al. (27), who reported a significantly lower f_{mean} in the affected limb.

The controls of the present study exhibited a pattern with decrease in PT and f_{mean} with simultaneous RMS increase throughout the test, which is consistent with isokinetic studies of clinically healthy subjects (23). In agreement with our study of patients with multiple sclerosis (35) no decreases of f_{mean} of the affected limb were found throughout the test. Bourbonnais Vanden Noven (4) reviewing the literature, concluded that type-II muscle fibre atrophy and hypertrophy of type-I muscle fibres occur in the hemiparetic muscle. However, the literature is somewhat heterogenous, which could be due to differences in duration and/or degree of paresis in the patient groups investigated. There are conflicting opinions regarding the mechanism behind the reported decrease in f_{mean} during fatigue. Decrease of the conduction velocity of the muscle fibre membranes has often been favoured as the major

cause (24), but also other mechanisms such as increasing synchronization of the firing frequency, decrease in active motor units and changes in intrinsic muscle properties have been suggested (15, 16). Recent studies have shown that the f_{mean} shift during fatigue is positively correlated with the type-II muscle fibres (14, 16, 21). A reduced central drive will reasonably mean decreased ability in recruiting high threshold type-II fibres. A decreased ability in recruiting high threshold type-II fibres will be associated with a lower absolute mechanical output (PT), together with small or no change in the two EMG parameters as found in this study for both the extensors and the flexors (26, 40). In agreement with this interpretation, recruitment failure has been found in the quadriceps femoris in hemiparetic patients (28) where force output during voluntary activation was lower than during superimposed electrical activation.

In conclusion, the patients with minimal overt clinical symptoms showed motor deficits when compared to the clinically healthy controls. To enhance good rehabilitation in this patient group with minimal overt clinical symptoms it might be of importance to determine the degree of actual recovering using repetitive reciprocal movements.

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REFERENCES

1. Adams, R. W., Gandevia, S. C. & Skuse, N. F.: The distribution of muscle weakness in upper motoneuron lesions affecting the lower limb. *Brain* 113: 1459-1476, 1990.
2. Bohannon, R. W.: Correlation of knee extension force and torque with gait speed in patients with stroke. *Phys Theory Pract* 7: 185-190, 1991.
3. Bohannon, R. W.: Muscle strength in patients with brain lesions: measurements and implications. *In* Muscle Strength (ed. K. Harms-Ringdahl), pp. 187-225. Churchill Livingstone, London, 1993.
4. Bourbonnais, D. & Vanden Noven, S.: Weakness in patients with hemiparesis. *Am J Occ Ther* 43: 313-319, 1989.
5. Colebatch, J. G. & Gandevia, S. C.: The distribution of muscular weakness in upper motor neuron lesions affecting the arm. *Brain* 112: 749-763, 1989.
6. Colebatch, J. G., Gandevia, S. C. & Spira, P. J.: Voluntary muscle strength in hemiparesis: distribution of weakness at the elbow. *J Neurol Neurosurg Psych* 49: 1019-1024, 1986.
7. Crone, C. & Nielsen, J.: Central control of disynaptic reciprocal inhibition in humans. *Acta Phys Scand* 152: 351-363, 1994.
8. Duncan, P.: The effect of a prior quadriceps contraction on

- knee flexor torque in normal subjects and multiple sclerosis patient with spastic paraparesis. *Phys Pract* 3: 11–17, 1987.
9. Dvir, Z.: Isokinetics—muscle testing, interpretation and clinical applications. Churchill Livingstone, London, 1995.
 10. Edwards, R. H. T.: Human muscle function and fatigue. In *Human Muscle Fatigue: Physiological Mechanisms*, CIBA Foundation Symposium No. 82. (eds. R. Porter & J. Whelan), pp. 1–18. Pitman Medical, London, 1981.
 11. Fellows, S. J., Kaus, C. & Thilman, A. F.: Voluntary movement at the elbow in spastic hemiparesis. *Ann Neurol* 36: 397–407, 1994.
 12. Fugl-Meyer, A. R., Jääskö, L., Leyman, I., Olsson, S. & Steglind, S.: The post stroke patient hemiplegic patient. *Scand J Rehab Med* 7: 13–31, 1975.
 13. Gandevia, S. C.: Strength changes in hemiparesis: measurements and mechanisms. In *Spasticity: Mechanisms and Management* (eds. A. F. Thilman, D. J. Burke & W. A. Rymer), pp. 111–122. Springer, Berlin, Heidelberg, 1993.
 14. Gerdle, B., Wretling, M.-L. & Henriksson-Larsén, K.: Do the fibre-type proportion and the angular velocity influence the mean power frequency of the electromyogram? *Acta Phys Scand* 134: 341–346, 1988.
 15. Gerdle, B. & Karlsson, S.: The mean frequency of the EMG of the knee extensors is torque dependent both in the unfatigued and the fatigued states. *Clin Phys* 14: 419–432, 1994.
 16. Gerdle, B., Karlsson, S., Crenshaw, A.G. & Fridén, J.: The relationships between EMG and muscle morphology throughout fatiguing static knee extension at two force levels in the unfatigued and the fatigued states. *Acta Phys Scand*, in press.
 17. Hammond, M. C., Kraft, G. H., Nutter, P. B., Trotter, M. J. & Robinson, L. M.: Co-contraction in the hemiparetic forearm: quantitative EMG evaluation. *Arch Phys Med Rehab* 69: 348–351, 1988.
 18. Jones, R. D., Donaldsson, I. M. & Parkin, P. J.: Impairment and recovery of ipsilateral sensory-motor function following unilateral cerebral infarction. *Brain* 112: 113–132, 1989.
 19. Karlsson, S., Erlandsson, B. E. & Gerdle, B.: A personal computer-based system for real-time analysis of surface EMG signals during static and dynamic contractions. *J Electro Kinesiol* 4: 170–180, 1994.
 20. Knutsson, E. & Mårtensson, A.: Dynamic motor capacity in spastic paresis and its relation to prime mover dysfunction. Spastic reflexes and antagonist co-activation. *Scand J Rehab Med* 12: 93–106, 1980.
 21. Kupa, E. J., Roy, S. H., Kandarian, S. C. & De Luca, C. J.: Effects of muscle fiber type and size on EMG median frequency and conduction velocity. *J Appl Phys* 79: 23–32, 1995.
 22. Lindström, B. & Gerdle, B.: The interrelationships between EMG, peak torque and perceived fatigue during repeated maximum isokinetic knee flexion with and without active knee extension. *Phys Theory Pract* 10: 17–25, 1994.
 23. Lindström, B., Karlsson, S. & Gerdle, B.: Knee extensor performance of dominant and non-dominant limb throughout repeated isokinetic contractions, with special reference to peak torque and mean frequency. *Clin Phys* 15: 275–286, 1995.
 24. Lindström, L., Magnusson, R. & Petersén, I.: Muscular fatigue and action potential conduction velocity changes studies with frequency analysis of EMG signals. *Electro* 4: 341–357, 1970.
 25. Lindström, L. & Magnusson, R.: Interpretation of myoelectric power spectra: a model and its implications. *Proc IEEE* 65: 653–662, 1977.
 26. Linssen, W., Stegeman, D., Joosten, E., Binkhorst, R., Merks, M., Ter-Laak, H. & Notermans, S.: Fatigue in type I fiber predominance: a muscle force and surface EMG study on the relative role of type I and type II muscle fibers. *Muscle Nerve* 14: 829–837, 1991.
 27. Moglia, A., Alfonsi, E., Zandrini, C., Ciano, C., Rascaroli, M., Toffola, E. D. & Arrigo, A.: Surface-EMG analysis of rectus femoris in patients with spastic hemiparesis undergoing rehabilitation treatment. *Electro Clin Neurophys* 31: 123–128, 1991.
 28. Newham, D. J., Mayston & Davis J. M.: Quadriceps isometric force, voluntary activation and relaxation speed in stroke. *Muscle Nerve Suppl* S53, 1996.
 29. Overend, T. J., Cunningham, D. A., Kramer, J. F., Lefcoe, M.S. & Paterson, D. H.: Knee extensor and knee flexor strength: cross sectional area ratios in young and elderly men. *J Gerontol* 47: M204–M210, 1992.
 30. Rosenfalck, A. L. & Andreassen, S.: Impaired regulation of force and firing pattern of single motor units in patients with spasticity. *J Neurol Neurosurg Psych* 43: 907–916, 1980.
 31. Sahrmann, S. A. & Norton, B. J.: The relationship of voluntary movement to spasticity in the upper motor neuron syndrome. *Ann Neurol* 2: 460–465, 1977.
 32. Shahidi, A. V. & Matieu, P. A.: Endurance time characteristics of human ankle dorsiflexors and plantarflexors. *Eur Appl Phys* 71: 124–130, 1995.
 33. Sherrington, C. S.: Reciprocal inhibition as a factor in the co-ordination of movements and posture. *Quart J Exp Phys* 6: 251–310, 1913.
 34. Sinkjaer, T. & Magnussen, I.: Passive, intrinsic and reflex-mediated stiffness in the ankle extensors of hemiparetic patients. *Brain* 117: 355–363, 1994.
 35. Svensson, B., Gerdle, B. & Elert, J.: Endurance training in patients with multiple sclerosis. Five case studies. *Phys Ther* 74: 1017–1026, 1994.
 36. Thorstensson, A. & Karlsson, J.: Fatigability and fibre composition of human skeletal muscle. *Acta Phys Scand* 98: 318–322, 1976.
 37. Twichell, T. E.: The restoration of motor function following hemiplegia in man. *Brain* 74: 443, 1951.
 38. Watkins, M. P., Harris, B. A. & Kozolowski, B. A.: Isokinetic testing in patients with hemiplegia—a pilot study. *Phys Ther* 64: 184–189, 1984.
 39. Wolf, S. L., Catlin, P. A., Blanton, S., Edelman, J., Lehrer, N. & Schroeder, D.: Overcoming limitations in elbow movement in the presence of antagonist hyperactivity. *Phys Ther* 74: 826–835, 1994.
 40. Young, A.: The relative isometric strength of type I and type II muscle fibers in the human quadriceps. *Clin Phys* 4: 23–32, 1984.

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