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RESEARCH ARTICLE

Ankle dexterity remains intact in patients with incomplete spinal cord injury in contrast to stroke patients

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Abstract Patients with either incomplete spinal cord injury (iSCI) or stroke suffer from muscle weakness in the lower limb and impaired ambulation. The assessment of motor function in iSCI has so far focused on measures of muscle strength, while in stroke extensive research has been directed towards upper limb motor control. Slowness of movements was reported to be a common motor impairment of patients with lesions of the central nervous system (CNS). It may result from muscle weakness and deficits in dexterity, which is two aspects of motor control that are dependent on cortico-spinal tract (CST) integrity and are crucial to ambulation. Thus, this study investigated the impact of CST damage either at spinal (iSCI) or cortical level (stroke) on ankle dexterity and maximal movement velocity (MMV). Twelve iSCI, stroke and control subjects were tested. The patients were matched for gender, age and maximal voluntary contraction (MVC) in ankle dorsi- and plantar-flexion muscles. Dexterity and MMV were tested in the supine position. CST function was assessed by motor evoked potentials (MEPs). In both groups of patients, MMV and MEP latencies were comparably deteriorated. However, dexterity was preserved in iSCI, but impaired in the hemiparetic stroke leg. Therefore, iSCI patients showed a high dexterity within the preserved muscle strength, but suffered primarily from reduced MMV. In stroke patients, both dexterity and MMV were reduced. These differences might be considered in rehabilitation programs and regeneration therapies.

Keywords Dexterity · Motor evoked potential · Spinal cord injury · Stroke

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Introduction

Lesions of the central nervous system (CNS), such as those after stroke or spinal cord injury, are often associated with severe motor deficits. Particularly slowness of movement is a common motor impairment after CNS lesions (Miller and Claiborne 2005), which may result from muscle weakness and deficits in dexterity. Dexterity can be defined as the ability to coordinate muscle activity to meet environmental demands and is not restricted to manual tasks (Canning et al. 2004). Impairments in muscle strength and dexterity account for the majority of disability observed in stroke patients (Ada et al. 1996; Canning et al. 2000). In upper limb studies, reduced maximal torque, a decreased rate of torque development (Canning et al. 1999) and deficits in manual dexterity have been demonstrated (Ada et al. 1996), even in the limb ipsilateral to the brain lesion (Wetter et al. 2005). In lower limb studies of patients with an incomplete spinal cord injury (iSCI), muscle weakness and slowness in the development of voluntary torque was found (Jayaraman et al. 2006), while dexterity, assessed by accurate timing of ankle movements, was only slightly reduced (Wirth et al. 2008).

Aside from the aforementioned studies, little is known about the ability of iSCI and stroke patients to coordinate

muscle activity in the lower limb. In addition, studying motor control in patients with a lesion of the corticospinal tract (CST) at either the cortical or spinal level using the same paradigm might be useful for gaining deeper insight into the mechanisms underlying motor disability. Thus, the aim of the present study was to compare the impact of CST damage on dexterity and movement velocity of the lower limb between iSCI and stroke patients (hemiparetic and non-affected leg). The main research questions to be answered were (1) whether dexterity and movement velocity were similarly affected in the ankle after stroke and iSCI, (2) whether these deficits might be related to CST integrity as assessed by means of motor evoked potentials (MEPs) and (3) whether dexterity, movement velocity, strength and MEPs were also impaired in the ipsilateral limb to the brain lesion in stroke patients.

Methods

Design

In this experimental study, the outcome measures dexterity, movement velocity and CST integrity were compared between three groups, two patient groups (iSCI and stroke) and a control group, respectively. The parameters dexterity and movement velocity were determined during an ankle task. CST integrity was measured separately by transcranial magnetic stimulation. The assessor was not blinded to the subject's condition. The details of the experiments are described in the following paragraphs.

Subjects

All procedures were in accordance with the standards of the local ethics committee and with the Declaration of Helsinki. All subjects gave informed written consent to participate in the study. The stroke patients were recruited from the Neuro-rehabilitation hospitals of Valens and Wald, Switzerland, the iSCI patients from the spinal cord injury center of Balgrist, University Hospital, Zurich, Switzerland. The control subjects were recruited via the local university department for senior citizens in Zurich, Switzerland. Twelve stroke patients (six females; mean age = 65.75 years ± standard deviation 10.54), 12 iSCI patients, matched for gender and age (62.25 years ±8.25) and 12 control subjects, matched for gender and age (63.25 years ±10.71) were tested (Table 1).

In order to exclude that any differences in performance of the patient groups might simply be based on muscle strength, the stroke and the iSCI patients were also matched for maximal voluntary contraction (MVC) [normalized for body weight (Hsu et al. 2002)]. MVC of the ankle dorsiflexor and plantarflexor muscles was measured using a custom-built torque measuring device that prevented any movement at the ankle and any influence of the weight of the lower limb on the torque measurement (Diehl et al. 2006; van Hedel et al. 2007). As the axis of the measurement device was in line with the longitudinal axis of the leg, forces along the longitudinal axis of the lower leg did not result in torque. Strain-gauges, attached on both sides of the torque device, recorded bending of the aluminum bar exerted by isometric plantar or dorsal torque. The output of the strain gauges was recorded with a sampling rate of 50 Hz, amplified and converted from V into Nm. The subjects were asked to isometrically contract their ankle dorsiflexor muscles as forcefully as possible. The measurement was taken when they had been holding the torque constant for about 2 s. Finally, the torque data were normalized by dividing torque by body weight (Nm/kg) (Hsu et al. 2002). MVC in dorsiflexion was 0.23

Table 1 Characteristics of the stroke and iSCI patients

Characteristic	SCI	Stroke	Controls
Age (years) (mean ± SD)	62.25 (±8.25)	65.75 (±10.54)	63.25 (±10.71)
Gender (male:female)	6:6	6:6	6:6
Strength dorsiflexor muscles (Nm/kg) (mean ± SD)	0.26 (±0.15)	0.23 (±0.11)	0.48 (±0.10)
Strength plantarflexor muscles (Nm/kg) (mean ± SD)	0.22 (±0.15)	0.23 (±0.14)	0.35 (±0.16)
Time since lesion (months) (mean ± SD)	13.26 (±31.72)	14.43 (±23.59)	
Maximal gait speed (m/s) (mean ± SD)	0.85 (±0.67)	1.07 (±0.53)	2.29 (±0.36)
Preferred gait speed (m/s) (mean ± SD)	0.63 (±0.47)	0.78 (±0.29)	1.56 (±0.13)
Side of lesion (left:right hemisphere)		3:9	
Level of lesion			
C3–C8	5		
T1–T11	5		
T12–L5	2		

Nm/kg (±0.11) for the stroke patients, 0.26 Nm/kg (±0.15) for the iSCI subjects and 0.48 (±0.10) for the control subjects. In plantarflexion, normalized MVC was 0.23 Nm/kg (±0.14) for the stroke patients, 0.22 Nm/kg (±0.15) for the iSCI patients and 0.35 (±0.16) for the control subjects (Table 1).

Only stroke patients with no subcortical brain lesion were included in the study. Thus, the stroke patients suffered mainly from ischemic brain lesions (stroke of the middle cerebral artery area as assessed by computer tomography or magnetic resonance imaging) with the exception of one patient who had an intracranial hemorrhage. Furthermore, only patients with scores above 24 points in the Mini Mental State (Folstein and Folstein 1975) were included according to the standard cutoff score for the definition of cognitive impairment, which is either equal or below 24 points (Adunsky et al. 2002). In addition, patients with spasticity >2 on the modified Ashworth scale, or with ankle contractures, were excluded from the study (Canning et al. 1999). Thus, spasticity ranged from 0 to 2 scores on the modified Ashworth scale in the stroke group and from 0 to 1 in the iSCI group (no significant difference between the groups). The inclusion criteria to be fulfilled by the iSCI patients were some preserved motor function in the muscles of the ankle [ASIA category C or D (American Spinal Injury Association 2002)] and damage to the upper motor neuron. Additional pathologies to the lower motor neuron were excluded by reflex measurements in the clinical routine, but reflex integrity was not specifically assessed in the present study. The control subjects were included if they had no neurologic, cardiovascular and orthopedic diagnosis that might interfere with the aim of this study.

Time since the onset of injury was on average 14.43 months (±23.59) in the stroke group and 13.26 months (±31.72) in the iSCI group (Table 1). All stroke patients were ambulatory, while three iSCI patients were not able to walk. Maximal gait speed [assessed in a 10 Meter Walk Test (van Hedel et al. 2005, 2006)] was on average 1.07 m/s (±0.53) for the stroke patients, 0.85 m/s (±0.67) for the iSCI patients and 2.29 (±0.36) for the control subjects. Averaged preferred gait speed (10 Meter Walk Test) was 0.78 m/s (±0.29) in the stroke group, 0.63 m/s (±0.47) in the iSCI group and 1.56 m/s (±0.13) in the control group (Table 1).

Outcome measures

Dexterity and MMV

Computer-generated tones were presented to the subjects in blocks of different frequencies (0.8, 1.6 and 2.4 Hz). The subjects (in supine position) were instructed to follow the pacing tones with their foot (1) as accurately as possible and (2) with the largest range of motion (ROM) possible. For each frequency, the subjects had to perform 20 dorsi- and plantar-flexion repetitions. The subjects were able to see their feet, but were not explicitly instructed to visually control the foot movements. The movements were recorded by means of an electric goniometer (Biometrics Ltd., Gwent, UK) with the sampling rate set at 1,000 Hz. Data from the first five movement cycles were not included in the analysis, since a minimum of 3–5 signals are required for picking up the beat (Aschersleben and Prinz 1995). From the remaining 15 ankle dorsiflexions and 15 plantar-flexions, dexterity was defined as deviation from target frequency and was determined for each frequency by averaging the duration of movement cycles, converting the result to frequency and comparing it to the target frequency (Wirth et al. 2008). Maximal movement velocity (MMV) of the ankle was calculated by deriving the goniometer data and then averaging the maximal movement speed per cycle (degrees/s).

CST integrity

Transcranial magnetic stimulation (TMS) and electromyography (EMG) were performed analogous to previous studies (Diehl et al. 2006; van Hedel et al. 2007). Single pulses of 200 μs were delivered by means of a magnetic stimulator (MagPro, Denmark). For all measurements, a figure eight-shaped coil was used. Individual coil position and stimulation threshold were determined at the beginning of the recording. Threshold intensity was defined as the percentage of stimulator output that evoked a MEP amplitude of at least 50 μV in approximately 50% of 10 consecutive stimuli. Stimulation intensity was set at 1.2 × threshold intensity (Diehl et al. 2006; van Hedel et al. 2007). TMS was performed in all patients at 20% MVC using the above described torque measuring device (Diehl et al. 2006; van Hedel et al. 2007), while visual feedback about the contraction level was provided. Excitability and facilitation of MEP was studied during a dynamic contraction condition of the tibialis anterior muscle (Diehl et al. 2006). The subjects executed an isometric, but continuously increasing contraction of the tibialis anterior muscle with a relative increase of 20% MVC per second, with visual feedback. When 20% of MVC was reached, a stimulus was delivered automatically. Data were recorded with a sampling rate of 2,000 Hz and the average of five measurements per condition was analyzed (Diehl et al. 2006; van Hedel et al. 2007).

The EMG electrodes were placed on the middle of the tibialis anterior muscle belly (inter-electrode distance 2 cm). The level of background muscle activity was calculated

by the root mean square of the tibialis anterior muscle during 20 ms before the stimulus. MEP amplitude was determined by calculating the root mean square over a time window of 20 ms from the onset of the MEP and by subtracting the background activity from the total MEP (van Hedel et al. 2007). MEP latency was defined as the time between TMS trigger and the MEP response using the cumulative sum method, which allows for a reliable determination of MEP latency and amplitude (King et al. 2006). Lastly, the MEP latency values were normalized by dividing latency by body height (ms/m) (van Hedel et al. 2007). MEP could be evoked in all subjects apart from one iSCI subject. For the statistical analysis, MEP amplitude of this subject was regarded as non-existent (=0 mV). In addition, since non-parametrical tests were used and the absolute values are thus not considered for the statistical analysis, a value higher than the most prolonged value in the iSCI group was taken for the MEP latency. However, these MEP values were discarded from those analyses that required absolute values.

Data analysis

All data analyses were performed using Soleasy software version 6.1 (ALEA solutions GmbH, Zurich, Switzerland).

The hemiparetic leg of the stroke patients was compared to the more affected leg of the iSCI subjects (defined by MVC of the dorsiflexor muscles, which were the focus of the present study) and to the non-dominant leg of the control subjects. In addition, the non-affected leg of the stroke patients was compared to the non-dominant leg of the control subjects. The dominant foot was self-reported as the foot used for kicking a ball, as this is regarded the predominant test (Gabbard and Hart 1996).

All statistical analyses were performed using SPSS software version 14.0 (Chicago, USA). To determine dexterity, the deviation between target and performed frequency was statistically analyzed using one sample *t* tests. To compare the outcome parameters dexterity and MMV between the groups, the non-parametrical Kruskal–Wallis test and, post hoc, Mann–Whitney *U* tests were used. In addition, to investigate the relationship between the MEP parameters and the outcome parameters, linear regression analyses with standardized regression coefficients (backward method) were done. The MEP data (latency and amplitude) and the dexterity data were transformed (logarithmic transformation) for the analysis in order to obtain normal residual distribution (Sachs 1991). The significance level α was set at 0.05 for all analyses and was adjusted to 0.05/3 = 0.0167, where three comparisons were performed (post hoc tests and one sample *t* tests).

Results

Comparison of stroke (hemiparetic leg) and iSCI patients to control subjects

Dexterity and MMV

Compared to target frequency, dexterity was significantly reduced only in the hemiparetic leg of the stroke patients at 2.4 Hz ($df = 2$, $\chi^2 = 10.83$, $P = 0.004$) compared to the iSCI patients ($P = 0.01$) and the control subjects ($P = 0.001$) (Table 2). For visualization, individual results of performance in dexterity of a representative iSCI patient and a stroke patient are shown in Fig. 1a and b.

With a view to MMV, the three groups differed in MMV in dorsiflexion (0.8 Hz: $df = 2$, $\chi^2 = 17.66$, $P < 0.001$; 1.6 Hz: $df = 2$, $\chi^2 = 21.26$, $P < 0.001$; 2.4 Hz: $df = 2$, $\chi^2 = 16.86$, $P < 0.001$) and in MMV in plantarflexion (0.8 Hz: $df = 2$, $\chi^2 = 17.10$, $P < 0.001$; 1.6 Hz: $df = 2$, $\chi^2 = 20.16$, $P < 0.001$; 2.4 Hz: $df = 2$, $\chi^2 = 20.87$, $P < 0.001$). Post hoc analysis showed that the stroke and the iSCI patients were impaired in MMV in dorsi- and plantarflexion compared to the control subjects ($P \leq 0.001$). Comparing the stroke and the iSCI group, no significant difference was found (Table 2).

CST integrity

MEP latency was different between the groups ($df = 2$, $\chi^2 = 13.48$; $P = 0.001$). Post hoc analysis showed that the stroke and the iSCI patients had prolonged latencies compared to the control subjects (stroke: $P < 0.001$; iSCI: $P = 0.008$) (Table 2). In addition, no significant difference in MEP latency was found between the stroke and the iSCI patients. As for the MEP amplitude, the three groups did not significantly differ ($df = 2$, $\chi^2 = 5.82$; $P = 0.05$) (Table 2).

Relation between dexterity, MMV and CST integrity

The linear regression analyses showed that particularly the MEP latency, but to a lesser extent also the MEP amplitude contributed to explaining some variability in the parameters of the ankle task (Fig. 2). For the independent variable dexterity at 2.4 Hz, the standardized regression coefficients were 0.32 ($P = 0.05$) for the MEP latency (Fig. 2a) and −0.31 ($P = 0.06$) for the MEP amplitude (Fig. 2b) ($R^2 = 0.25$). With regard to MMV at 2.4 Hz as dependent variable, the standardized regression coefficients were −0.66 ($P < 0.001$) for MEP latency (Fig. 2c) and 0.35 ($P = 0.002$) for MEP amplitude (Fig. 2d) ($R^2 = 0.68$).

Table 2 Outcome parameters of ankle task and transcranial magnetic stimulation in all groups

Groups									Differences between groups			
	SCI $n = 12$		Stroke, hemiparetic leg ($n = 12$)		Stroke, unaffected leg ($n = 12$)		Controls ($n = 12$)		Controls minus SCI	Controls minus Stroke, hemiparetic leg	Controls minus Stroke, unaffected leg	SCI minus Stroke, hemiparetic leg
	Hz	%	Hz	%	Hz	%	Hz	%				
Dexterity												
0.8 Hz	0.01 (±SD 0.01)	0.9	0.02 (±0.04)	2.5	0.03 (±0.07)	3.8	0.00 (±0.00)	0.5	0.00 (95% CI −0.02 to 0.00)	−0.02 (−0.04 to 0.00)	−0.03 (−0.07 to 0.01)	−0.01 (−0.03 to 0.01)
1.6 Hz	0.07 (±0.19)	4	0.24 (±0.33)	15	0.07 (±0.09)	4	0.00 (±0.00)	1.9	−0.04 (−0.18 to 0.04)	−0.21 (−0.43 to −0.04)	−0.04 (−0.12 to −0.02)	0.00 1 (−0.40 to 0.06)
2.4 Hz	0.29 (±0.35)	12	0.66 (±0.53)	28	0.25 (±0.26)	10.4	0.18 (±0.16)	7.5	−0.11 (−0.34 to 0.12)	−0.48*** (−0.81 to −0.15)	−0.07 (−0.25 to 0.11)	−0.37* (−0.75 to 0.01)
MMV												
0.8 Hz (degrees/s)	134.06 (±69.07)		91.92 (±57.06)		207.51 (±62.21)		238.81 (±58.09)		104.75*** (50.71 to 158.78)	146.89*** (98.14 to 195.64)	31.30 (−19.66 to 82.26)	42.14 (−11.50 to 95.78)
1.6 Hz (degrees/s)	150.45 (±79.77)		94.97 (±64.95)		229.60 (±62.83)		263.31 (±52.60)		112.86*** (55.65 to 170.07)	168.34*** (118.30 to 218.38)	33.71 (−15.35 to 82.77)	55.48 (−6.11 to 117.07)
2.4 Hz (degrees/s)	144.73 (±71.88)		99.53 (±76.09)		211.54 (±58.62)		256.34 (±67.58)		111.61*** (52.54 to 170.68)	156.81*** (95.88 to 217.74)	44.80 (−8.76 to 98.36)	45.20 (−17.47 to 107.87)
CST integrity												
MEP latency (ms/m)	21.26 (±0.25)		24.08 (±0.39)		19.54 (±2.04)		18.94 (±0.17)		−2.32** (−2.50 to −2.14)	−5.14*** (−5.39 to −4.89)	−0.60 (−1.82 to 0.63)	−2.82 (−3.10 to −2.54)
MEP amplitude (mV)	0.13 (±0.12)		0.15 (±0.08)		0.23 (±0.08)		0.23 (±0.14)		0.10 (−0.01 to 0.21)	0.08 (−0.02 to 0.18)	0.00 (−0.10 to 0.10)	−0.02 (−0.11 to 0.07)

CI confidence interval, *CST* cortico-spinal tract, *MMV* maximal movement velocity, *MEP* motor evoked potential, *SD* standard deviation

* $0.01 < P \leq 0.05$

** $0.001 < P \leq 0.01$

*** $P \leq 0.001$

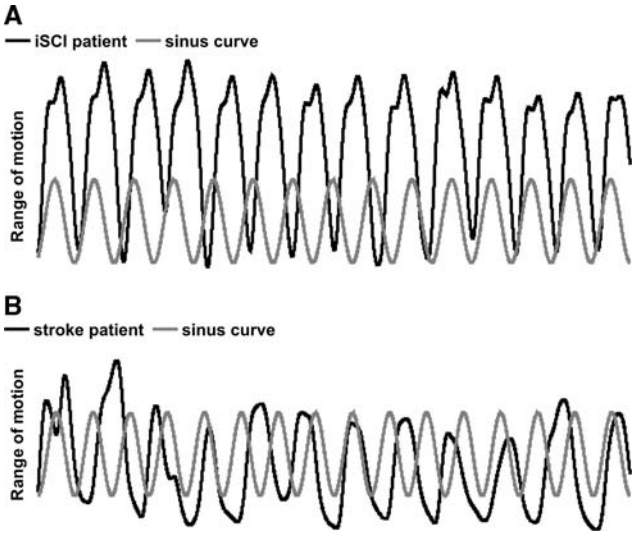

Fig. 1 Dexterity. **a** Representative example of dexterity at 2.4 Hz of an iSCI patient [female 57 years, normalized MVC in dorsiflexion 0.26 Nm/kg, deviation from target frequency: 0.17 Hz (average in iSCI group 0.29 Hz)] compared to the ideal sinus curve. **b** Individual results of a representative stroke patient (left hemisphere) [female 71 years, normalized MVC in dorsiflexion 0.22 Nm/kg, deviation from target frequency 0.53 Hz (average in stroke group 0.66 Hz)] compared to the ideal sinus curve

Non-affected leg of the stroke patients compared to the non-dominant leg of the control subjects

Dexterity in the non-affected leg of the stroke patients was not significantly reduced compared to the non-dominant leg of the control subjects (Table 2). MMV did not significantly differ in dorsiflexion (Table 2), but was reduced in plantarflexion at all frequencies ($P = 0.01$ at 0.8 Hz and 1.6 Hz; $P = 0.02$ at 2.4 Hz). MVC was reduced in dorsiflexion ($P = 0.006$) and was 0.36 Nm/kg (±0.07) in the stroke group and 0.48 Nm/kg (±0.10) in the control group. The MEP parameters were not altered in the non-affected leg of the stroke patients (Table 2).

Discussion

The impairment of lower limb control after iSCI and stroke is clinically summarized as upper motoneuron syndrome, which implies a similar impairment of motor function. However, similarities or divergences between iSCI (spinal lesion) and stroke (cortical lesion) patients in motor control of the lower limb have been less investigated so far. The purpose of the present study was to compare ankle dexterity and maximal ankle movement velocity between iSCI and stroke patients. To allow for comparison, the patients were matched not only for gender and age, but also for voluntary dorsi- and plantar-flexor strength. The study used a paradigm that requires minimal muscle strength to assess dexterity as an independent variable from muscle weakness. It could be shown that ankle dexterity was impaired only in the hemiparetic leg of the stroke patients, while the iSCI patients performed comparably to the control subjects. Although movement speed in the ankle task and the MEPs were significantly deteriorated in the patient groups, the spinal CST damage in iSCI had only limited influence on dexterity in this paradigm which accounted for muscle weakness. Therefore, the upper motor neuron syndrome of the lower limb in iSCI differs from stroke patients in respect to dexterity which should be considered for rehabilitation programs and interventional trials.

Ankle control in iSCI patients compared to the hemiparetic leg of the stroke patients

The results of reduced movement velocity and impaired dexterity in the ankle of stroke patients are analogous to previously reported upper limb studies, where it was found that loss of strength, and particularly slowness to develop peak torque, was a more significant contributor to disability than loss of dexterity (Canning et al. 1999, 2000, 2004). Also the results of the iSCI patients in terms of movement speed are consistent with previous results on this issue (Jayaraman et al. 2006; Wirth et al. 2008). The reduction of maximal movement speed of the ankle and gait speed in both cortical and spinal lesions of the CST confirms that slowness of movement is a predominant characteristic in upper motor neuron syndrome (Miller and Claiborne 2005). The finding of more affected dexterity in the hemiparetic compared to the non-affected leg and the iSCI patients indicates that the applied paradigm is sensitive to assess differences in ankle dexterity. Since the patient groups were matched for strength, the difference in dexterity cannot be accounted for by weakness. Also time since injury was on average very similar in both patient groups. Nevertheless, in the iSCI group, the majority of patients (7 out of 12) were 1 month post injury, while this applied for two stroke patients only. However, four stroke patients were 2 months post injury, which also made the proportion of patients who were in an early phase after injury very comparable between the two groups. In addition, in contrast to the iSCI patients, all stroke patients were ambulators, the majority even without any walking aids. Thus, dexterity might not have been substantially impacted by muscle disuse in the stroke group. Spasticity, in turn, could have influenced dexterity, since it was slightly (although not significantly) higher in the stroke group. To exclude that the finding of impaired dexterity was not simply based on spasticity, we analyzed the dexterity data excluding the three stroke patients with a modified Ashworth score equal to 2 (and the

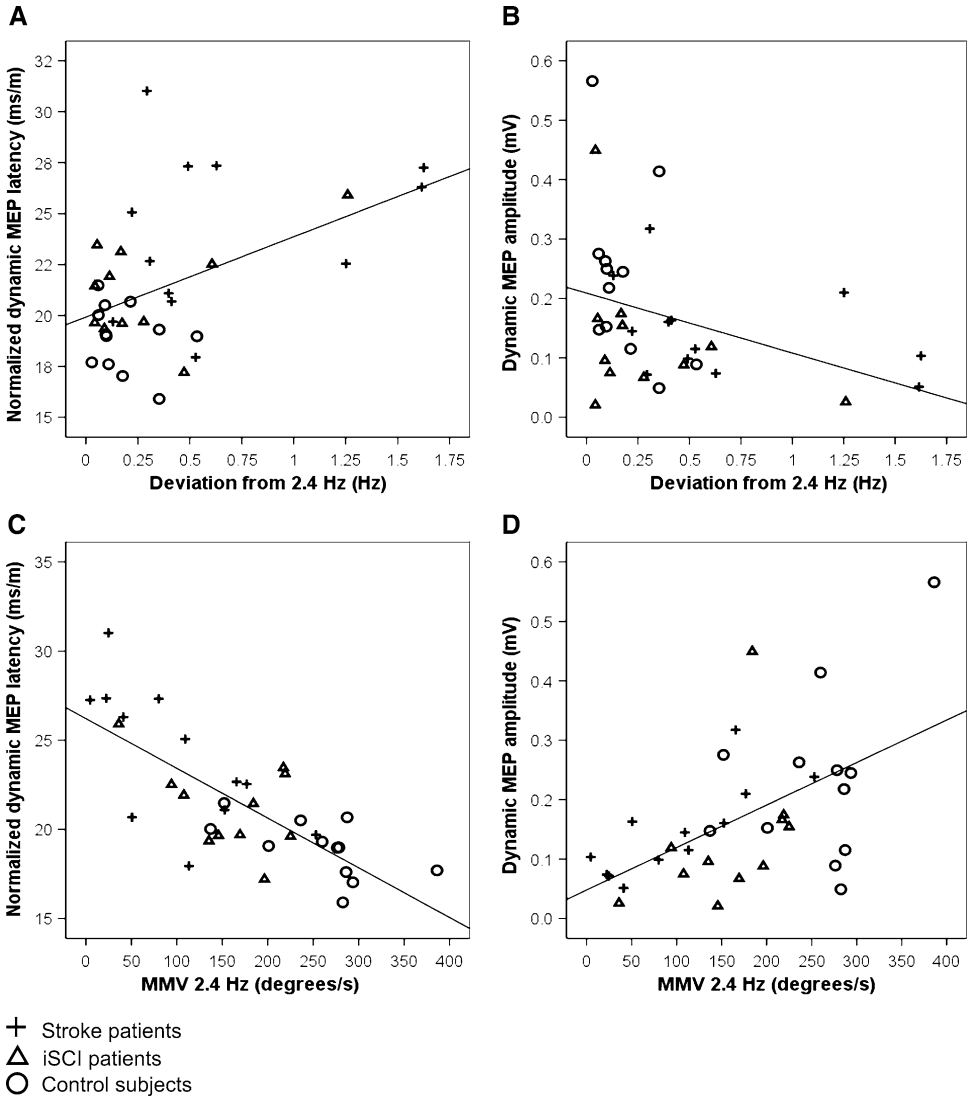

Fig. 2 Relationship of the MEP parameters (latency and amplitude) to dexterity and MMV in all groups. *Top* relationship between dexterity and **a** MEP latency [normalized for body height (ms/m)] and **b** MEP amplitude. Dexterity is presented as deviation between the target frequency 2.4 Hz and the frequency, which was performed by the subjects. *Bottom* relationship between MMV at 2.4 Hz and **c** MEP latency [normalized for body height (ms/m)] and **d** MEP amplitude

corresponding iSCI patients and control subjects) and came to the same result of impaired dexterity at high frequency compared to the control subjects and the iSCI patients. Furthermore, damage to other pathways than CST, particularly to proprioceptive afferents, might have influenced dexterity. However, visual control of foot position was allowed during the whole experiment and in addition, an earlier study had revealed that reduced proprioception did not negatively affect motor performance in the present experimental protocol (Wirth et al. 2008). Another factor underlying low dexterity could be slowness of movement (Ada et al. 1996). Indeed, at the same level of strength, maximal movement speed was somewhat slower in the stroke than in the iSCI group (but not significantly different). Yet in the present task, the patients were asked to first and foremost follow the target frequency accurately and secondly, to perform maximal range of motion, requiring that accuracy be favored at the cost of range of motion. Thus, reduced maximal movement speed did not necessarily have an impact on accuracy, but on range of motion. Higher-order motor planning deficits were reported to be present in stroke patients with subcortical CST lesion even without clinical evidence of apraxia. These deficits occurred primarily in patients with damage to the left brain hemisphere (Raghavan et al. 2006). In the present study, however, the right hemisphere was affected in the majority of patients. Furthermore, patients with subcortical lesion were excluded from participating. Thus, motor planning deficits were unlikely to be the factor limiting dexterity, which is in line with the preserved dexterity of the ipsilateral stroke leg. More probably and in line with the results of two stroke studies using EMG during distinct upper limb (Ada et al. 1996; Canning et al. 2000) and cyclic lower limb movements (Kautz and Brown 1998), timing abnormalities (inaccuracy in an elbow flexion-extension tracking task; prolonged and phase advanced muscle excitation during

pedaling) have been attributed to an impaired ability to fine tune muscle activation. It was hypothesized that a lack of precise modulation of the firing rate of motor units as well as their impaired synchronization, which has been reported in stroke patients (Farmer et al. 1993; Gemperline et al. 1995) might be the underlying factors (Canning et al. 2000). Also in iSCI patients, motor unit firing rate and synchronization were impaired (Smith et al. 1999; Zijdewind and Thomas 2003). However, no study directly compared these impairments in the two patient groups. A deficit in motor unit synchronization was hypothesized to reflect CST damage (Smith et al. 1999). Accordingly, the rate of transmission of information from the cortex to the spinal cord was proposed to limit dexterity (Darian-Smith et al. 1996). Prolonged MEP latencies of the hemiparetic limb in stroke have been reported elsewhere (Hendricks et al. 2003). However, in the present study, the MEP latencies were not significantly prolonged compared to the iSCI patients. Nevertheless, the relationship between dexterity and the MEP parameters showed a clear trend towards significancy ($P = 0.05$ for MEP latency, $P = 0.06$ for MEP amplitude). Thus, reduced CST conductivity might be one factor that contributed to the finding of impaired ankle dexterity in stroke patients.

Although muscle strength was reported to mainly determine gait speed in iSCI (Kim et al. 2004) and stroke patients (Hsu et al. 2003), rhythmic ankle re-education training with visual and auditory feedback during sitting, standing and walking was reported to lead to a significant increase in walking speed in stroke patients (Mandel et al. 1990). Thus, the present findings are supportive to previous studies where training of ankle dexterity has been considered to be of value for optimizing gait outcome in stroke.

Ankle control in the non-affected leg of the stroke patients

Abnormal muscle activity in terms of strength and dexterity in the non-affected leg after stroke has been reported in several studies (Bohannon and Williams 1995; Williams 2004). Abnormal descending signals (altered excitability of the opposite hemisphere) and changes in excitability of spinal reflexes are proposed to be underlying factors in this phenomenon (Yarosh et al. 2004). As for strength, impairments in various muscle actions have been observed in the non-affected leg after stroke (Bohannon and Williams 1995; Williams 2004). Ankle dorsiflexion was thereby the least impaired muscle action and the reported strength values ranged between 92% (Bohannon and Williams 1995) and 75% (Williams 2004) of normal, which is line with the present study, where the ankle dorsiflexor muscles of the stroke patients had 75% of strength of those of the control subjects. Also with a view to dexterity, the finding of no deficit in the non-affected leg in the present study is in line with previous results. Slowing and clumsiness after stroke were reported in the non-affected hand (Sunderland 2000), but these deficits were observed only in more complex tasks, such as the grooved pegboard and the maze coordination test (Haaland and Delaney 1981), while finger tapping and grip strength were normal (Haaland and Delaney 1981; Wetter et al. 2005).

Relevance for interventional treatments in iSCI

Recent therapeutic intervention studies in iSCI patients have mainly focused on task-specific locomotor training (Dobkin et al. 2006). However, a training strategy that aimed at increasing muscle strength by combining lower extremity resistance training with plyometric training (high-velocity contractions) was reported to lead not only to an increase in peak torque and the average rate of torque development, but also to enhanced gait speed in chronic iSCI patients (Gregory et al. 2007). Thus, task-specific rehabilitation strategies could be beneficially combined with interventions that aim at enhancing muscle strength.

In this context, also new cell based therapies that aim at neural repair after iSCI could be most effective by augmenting a patient's capacity to generate muscle strength. Furthermore, the preserved motor control, as evident by the intact dexterity, could be supportive to control recovered muscle activation where the increase of strength is accomplished by rather indirect and detoured neural regeneration (Bareyre et al. 2004).

Limitations

Reflex integrity to exclude additional lower motor neuron pathologies in iSCI patients with injury levels below Th11 and spinal shock symptoms in iSCI patients 1 month post injury was not specifically measured in the present study, but in the clinical routine. By means of these data, it could be ensured that none of the iSCI patients (data of one patient missing) had additional lower motor neuron lesion and that all but one patient were out of spinal shock. Thus, spinal shock might have affected the results, but there is multiple evidence that the spinal shock affects rather alpha-motoneuron and reflex excitability (Ditunno et al. 2004), while less is known about its influence on voluntary motor control.

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