Effect of sensory feedback from the proximal upper limb on voluntary isometric finger flexion and extension in hemiparetic stroke subjects

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This study investigated the potential influence of proximal sensory feedback on voluntary distal motor activity in the paretic upper limb of hemiparetic stroke survivors, and the potential effect of voluntary distal motor activity on proximal muscle activity. Ten stroke subjects and ten neurologically intact control subjects performed maximum voluntary isometric flexion and extension, respectively, at the metacarpophalangeal (MCP) joints of the fingers, in two static arm postures and under three conditions of electrical stimulation of the arm. The tasks were quantified in terms of maximum MCP torque (MCP\textsubscript{flex} or MCP\textsubscript{ext}) and activity of targeted (flexor digitorum superficialis or extensor digitorum communis) and non-targeted upper limb muscles. From a previous study on the MCP stretch reflex post-stroke, we expected stroke subjects to exhibit a modulation of voluntary MCP torque production by arm posture and electrical stimulation, and increased non-targeted muscle activity. Posture 1 (flexed elbow, neutral shoulder) led to greater MCP\textsubscript{flex} in stroke subjects than posture 2 (extended elbow, flexed shoulder). Electrical stimulation did not influence MCP\textsubscript{flex} or MCP\textsubscript{ext} in either subject group. In stroke subjects, posture 1 led to greater non-targeted upper limb flexor activity during MCP flexion and to greater elbow flexor and extensor activity during MCP extension. Stroke subjects exhibited greater elbow flexor activity during MCP flexion and greater elbow flexor and extensor activity during MCP extension than control subjects. The results suggest that static arm posture can modulate voluntary distal motor activity and accompanying muscle activity in the paretic upper limb post-stroke.
STROKE survivers frequently experience upper limb hemiparesis, consisting of impaired motor control of the upper limb contralateral to the site of the stroke. Hand function in general, and finger extension in particular, are strongly affected (Trombly 1989; Trombly et al. 1986). While local impairment mechanisms, such as hand muscle weakness (Kamper et al. 2006; Kamper et al. 2003; Kamper and Rymer 2001) and excessive agonist-antagonist co-activation (Kamper et al. 2003; Kamper and Rymer 2001), have been described, other, non-local, mechanisms may also be involved in the impairment of hand function after stroke. Indeed, reflex coupling exists between muscles of the proximal and the distal segments of the upper limb (Alexander and Harrison 2003; Cavallari and Katz 1989; Cavallari et al. 1992; Gracies et al. 1991; Kasai et al. 1994; 1992; McClelland et al. 2001). This heteronymous coupling could influence the activation of muscles throughout the upper limb during voluntary motor activity, and abnormal manifestations of this coupling may play a substantial role in distal motor impairment post-stroke. Specifically, sensory feedback from the arm may impact hand function.

In a recently conducted study, we found that static arm posture and surface electrical stimulation of the arm modulated the magnitude of the stretch reflex response of spastic finger flexor muscles in hemiparetic stroke survivors (Hoffmann et al. 2009). The magnitude was greatest in an arm posture in which the elbow was flexed and the shoulder was in a neutral posture, and increased when biceps brachii was stimulated. These results suggest that proximal sensory feedback can modulate distal reflex activity in the hand post-stroke. A similar modulating effect of proximal sensory feedback may exist for voluntary motor activity in the hand post-stroke, but, to our knowledge, this has not yet been investigated. In neurologically intact individuals, voluntary distal upper limb motor activity has been shown to be modulated by static arm posture (Dominici et al. 2005; Ginanneschi et al. 2005; Ginanneschi et al. 2006).
Heteronymous coupling within the upper limb further suggests that distal motor activity may influence the activity of proximal muscles. In that respect, imposed stretch of the spastic finger flexors elicits activity of non-stretched muscles throughout the relaxed upper limb of hemiparetic stroke survivors (Hoffmann et al. 2009), and during voluntary motor activity, abnormal coupling of muscle activities between upper limb joints is commonly observed after stroke, notably between the elbow and the shoulder (Beer et al. 1999; Dewald and Beer 2001; Dewald et al. 1995; Sangani et al. 2009).

The aim of the present study was to investigate whether, in hemiparetic stroke subjects, sensory feedback from the proximal upper limb influences voluntary distal upper limb motor activity, specifically maximum voluntary isometric force production in the hand. Subjects were asked to generate maximum voluntary isometric flexion and extension torque about the metacarpophalangeal (MCP) joints of the four fingers. Different conditions of proximal sensory feedback were compared by testing two static arm postures (i.e., combinations of static shoulder and elbow angles) and by applying surface electrical stimulation to either biceps brachii (BB) or triceps brachii (TB). Torque about the MCP joints and patterns of muscle activities throughout the upper limb were investigated. Specific interest was given to co-activation between a primary agonist (flexor digitorum superficialis (FDS) for MCP flexion, extensor digitorum communis (EDC) for MCP extension) and other muscles. Based on results from our previous study, we expected static arm posture and electrical stimulation of the arm to influence voluntary MCP torque production about the MCP joints in stroke subjects. Specifically, we hypothesized that MCP flexion torque would be greater in an arm posture involving a flexed elbow and in the presence of BB stimulation in stroke subjects. Furthermore, we hypothesized that voluntary MCP flexion and MCP extension would be accompanied by abnormal activity of muscles throughout the upper limb in stroke subjects.
Subjects

Ten hemiparetic stroke survivors (six men and four women) exhibiting chronic unilateral motor deficits volunteered to participate in the present study (see Table 1 for clinical data). Stroke subjects were aged between 48 and 75 years (mean, 60.2 years), and all of them were at least 1 year post-incident (range, 13 to 144 months). Function of the paretic upper limb was evaluated using the Fugl-Meyer Assessment of Sensorimotor Recovery After Stroke (Fugl-Meyer et al. 1975): upper extremity motor scores ranged from 26 to 62 out of a maximum score of 66. Six of the ten stroke subjects had right hemiparesis, four of them had left hemiparesis. Ten neurologically intact individuals (six women and four men) participated in the study as control subjects. Control subjects were aged between 26 and 67 years (mean, 42.1 years). We did not match stroke subjects and control subjects in terms of age, because we did not expect changes in the potential influence of sensory feedback from the proximal upper limb with age. In stroke subjects, the paretic upper limb was studied; in control subjects, the dominant upper limb was studied. The paretic upper limb was the dominant upper limb prior to the stroke in six of the ten stroke subjects. All subjects gave informed consent in accordance with the Helsinki Declaration, and the experimental protocol was approved by the Institutional Review Board of Northwestern University.

Protocol

The potential influence of sensory feedback from the proximal upper limb on distal voluntary motor activity was investigated through the performance of maximum voluntary isometric finger flexion and extension at the MCP joints. The subjects were seated next to an experimental table, and their four fingers were coupled to the shaft of a servomotor (1.4 HP, Kollmorgen Corporation, Radford, VA) fit into the table, as previously described (Hoffmann et al. 2009). A fiberglass cast placed around the subject's forearm and wrist maintained the
wrist in a posture of neutral flexion/extension and neutral abduction/adduction with respect to the forearm and kept the thumb extended and abducted from the palm. The cast was clamped within a jig in order to prevent arm translation, as well as to ensure that the hand was supported and stabilized without requiring voluntary motor activity by the subjects. The positions of the cast and the jig were adjusted such that the MCP joints were aligned along a vertical line extending from the shaft of the motor. The subject’s forearm was maintained in a posture of neutral pronation/supination.

Experimental trials consisted of producing either maximum voluntary isometric flexion or maximum voluntary isometric extension at the MCP joints, with the servomotor maintaining the MCP joints at 20° of flexion. The subjects produced a single maximum voluntary isometric contraction (MCP flexion or MCP extension) per trial, and were instructed to maintain the maximum contraction for two to three seconds. Between two successive trials, the motor slowly rotated the MCP joints from 20° of flexion to 10° of extension, where they were held for a few seconds before being slowly rotated back to 20° of flexion; this was done in order to minimize any wind-up effects of finger flexor muscle activity with repeated trials in stroke subjects (Kamper et al. 2003).

In order to investigate the potential effect of static proprioceptive feedback from the proximal upper limb, experimental trials were performed in two different static arm postures, which corresponded to two different combinations of shoulder and elbow angles. For posture 1, the goal posture consisted of 90° of elbow flexion, 0° of shoulder flexion, and 0° of shoulder abduction; for posture 2, the goal posture consisted of full elbow extension (0° of elbow flexion), 90° of shoulder flexion, and 0° of horizontal shoulder abduction (Fig. 1). The actual mean values of the shoulder and elbow angles across the ten stroke subjects were: for posture 1, 74° of elbow flexion, 21° of shoulder flexion, and 30° of shoulder abduction; for posture 2, 19° of elbow flexion, 71° of shoulder flexion, and 34° of horizontal shoulder abduction. Across the five control subjects, the actual mean shoulder and elbow angles were: 74° of elbow flexion, 23° of shoulder flexion, and 35° of shoulder abduction for posture 1, and 16° of elbow flexion, 73° of shoulder flexion, and 27° of horizontal shoulder abduction.
for posture 2. The two arm postures used in the present study had been previously shown to exhibit differences in the magnitude of the stretch reflex response of spastic finger flexor muscles in hemiparetic stroke subjects (Hoffmann et al. 2009). In both arm postures, the subject's arm rested on a cushioned support placed between the elbow and the experimental table. This ensured that the arm was supported without requiring voluntary motor activity by the subjects. Care was taken to make certain that the subjects did not feel any discomfort in either of the two arm postures at any point throughout the experiment.

The potential effect of sensory feedback from the proximal upper limb was further investigated through electrical stimulation of either BB or TB. Three stimulation conditions, namely "No stimulation", "BB stimulation" and "TB stimulation", were tested in each of the two static arm postures. For the "BB stimulation" and "TB stimulation" conditions, electrical stimulation was delivered by means of a neuromuscular stimulator (300PV, Empi, St. Paul, MN) and a pair of surface stimulating electrodes (American Imex, Irvine, CA) placed over the long head of BB or the long head of TB, respectively. Stimulation intensity was set to 120% of motor threshold; motor threshold was identified by palpation and visual observation. The duration of the stimulation pulse was 300 μs, and stimulation frequency was 35 to 40 Hz depending on comfort. Stimulation was turned on before the beginning of the trial and was maintained until after the end of the maximum voluntary isometric contraction produced by the subject. Electrical stimulation of BB or TB was intended to activate Ia afferents from that muscle, but undoubtedly also produced activation of cutaneous receptors. All subjects perceived the stimulation levels as non-noxious.

Each subject performed three maximum voluntary isometric MCP flexion contractions and three maximum voluntary isometric MCP extension contractions in both arm postures under all three stimulation conditions. Thus, a total of 36 experimental trials ((3 MCP flexion trials + 3 MCP extension trials) X 3 stimulation conditions X 2 arm postures) were performed by each subject. The subjects successively performed all of the 18 trials in a given arm posture and were then moved to the other arm posture. The order in which the two arm postures were tested was not controlled. In effect, all the subjects but two stroke subjects
were tested in posture 1 first. In a given arm posture, the subjects successively performed
three trials of a given contraction (MCP flexion or MCP extension) under a given stimulation
condition, and the testing order of contractions and stimulation conditions varied randomly
across subjects. There was a short rest period of approximately 30 to 60 s between two
successive trials. An auditory cue signaled the beginning of each trial.

Data collection

Throughout the experimental trials, torque generated about the MCP joints was
measured by means of a torque transducer (Transducer Techniques, Temecula, CA). The
EMG signals from nine upper limb muscles were recorded by means of pairs of active
surface recording electrodes with differential amplification (Delsys Inc., Boston, MA).
Recording electrodes were lightly coated with conductive gel and positioned above the
muscle belly of the following nine muscles: FDS, EDC, flexor carpi ulnaris (FCU),
brachioradialis (B), BB, TB, pectoralis major (PM), latissimus dorsi (LD), deltoideus medius
(DM). EMG signals were amplified (X 1,000 to X 10,000) and band-pass filtered between 20
and 450 Hz (two Bagnoli 8-channel EMG systems, Delsys Inc., Boston, MA). At the
beginning of the experimental session, the subjects were instructed to perform maximum
voluntary contractions (MVCs) for each of the nine muscles; these MVCs were performed for
the purpose of normalizing the EMG signals obtained during the experimental trials (cf.
Analysis). The recorded EMG signals from the nine muscles were simultaneously displayed
on a computer screen, allowing for on-line visual inspection of the signals. In particular, if
crosstalk was detected, placement of the corresponding recording electrode(s) was changed
until the perceived crosstalk was eliminated.

The MCP torque and EMG signals were low-pass filtered at 225 Hz and then
sampled at 500 Hz for off-line analysis.

Analysis
The MCP torque data were used to quantify the maximum isometric torque that the subjects produced during the MCP flexion and MCP extension trials. For each trial, the sampled MCP torque signal was smoothed using a 100-ms sliding window to compute a moving average. The maximum value of the smoothed signal during the trial (maximum MCP flexion torque or maximum MCP extension torque, respectively) was then located. In order to account for differences in strength between subjects, the maximum MCP torque value determined for each trial was then normalized according to the following method: for each MCP flexion trial, the maximum MCP flexion torque value for that trial was divided by the maximum MCP flexion torque value across all MCP flexion trials from the same subject, yielding $MCP_{flex}$; for each MCP extension trial, the maximum MCP extension torque value for that trial was divided by the maximum MCP extension torque value across all MCP extension trials from the same subject, thereby yielding $MCP_{ext}$. In addition, the instant at which the maximum MCP torque value occurred ($t_{flex}$ or $t_{ext}$, respectively) was determined for each trial.

The EMG data were used to quantify the patterns of upper limb muscle activities accompanying the production of the maximum isometric MCP torque. Each recorded EMG signal was first notch filtered at 60, 120 and 180 Hz. The signal was subsequently squared and passed through a low-pass filter (10 Hz cut-off frequency), before the square root was taken. This signal was then normalized by the maximum EMG activity value measured for the corresponding muscle across the entire experimental session, i.e., the maximum value recorded across the MVCs performed at the beginning of the experimental session and the experimental trials. This normalized signal ($EMG_{normalized}$) was subsequently used to quantify EMG activity of each of the nine upper limb muscles during the MCP flexion and MCP extension trials. Specifically, the "net EMG activity" ($EMG_{net}$) was computed for each muscle. First, a trapezoidal integration of $EMG_{normalized}$ was performed over a time window defined from 200 ms before $t_{flex}$ or $t_{ext}$ to 100 ms after $t_{flex}$ or $t_{ext}$. This integration yielded the "total EMG activity" ($EMG_{total}$). Baseline EMG activity ($EMG_{baseline}$) for each muscle was quantified.
by integrating $EMG_{normalized}$ over a baseline time window of 200 ms before the onset of voluntary MCP flexion or MCP extension. $EMG_{baseline}$ was multiplied by 1.5 to account for the difference in duration of the time window used to quantify $EMG_{total}$ (300 ms) and the baseline time window (200 ms). Two different durations of time windows were employed as 200 ms proved to be the best choice for quantifying baseline EMG activity without including contaminating artifacts in the baseline time window while 300 ms was preferable for describing muscle activation. After this multiplication, $EMG_{baseline}$ was subtracted from $EMG_{total}$, and the resulting value was divided by the duration of the time window used to quantify $EMG_{total}$ (300 ms), thereby yielding $EMG_{net}$.

Additional variables were computed for each experimental trial in order to investigate co-activation between a primary agonist of the respective contraction ("targeted muscle": FDS for MCP flexion, EDC for MCP extension) and the remaining, "non-targeted" muscles, using the quantified $EMG_{net}$. Specifically, co-activation between the targeted muscle and each non-targeted muscle X was respectively quantified by "$FDS_{andX} = \frac{X_{net}}{FDS_{net} + X_{net}}$" (for the MCP flexion trials) or "$EDC_{andX} = \frac{X_{net}}{EDC_{net} + X_{net}}$" (for the MCP extension trials).

The recorded EMG signals were sometimes contaminated by electrocardiogram (ECG) artifacts. If such contamination occurred, the ECG artifacts were removed before the EMG signal was used for analysis. The spikes in the EMG signal that were due to ECG activity were first used to compute a mean ECG spike template, which was then subtracted from the EMG signal at each location where an ECG spike occurred. Furthermore, since proximal electrical stimulation interfered with the recording of the EMG signals, EMG data from the "BB stimulation" and "TB stimulation" conditions were not used for analysis. Finally, some EMG data from the "No stimulation" condition were excluded from the analysis because of contamination by other artifacts.

Statistical analysis
Statistical analyses were performed using SPSS software (SPSS Inc., Chicago, IL).

Three multivariate analyses of variance (MANOVAs) were performed. A first MANOVA investigated the maximum isometric torque that the subjects produced during the MCP flexion and MCP extension trials, using "arm posture" (two levels: "posture 1" and "posture 2"), "stimulation condition" (three levels: "No stimulation", "BB stimulation", "TB stimulation"), and "subject group" (two levels: "stroke subjects" and "control subjects") as fixed factors and MCP_{flex} and MCP_{ext} as dependent variables. A second MANOVA investigated the net EMG activities accompanying the production of the maximum isometric MCP torque, using arm posture, "contraction" (two levels: "MCP flexion" and "MCP extension"), and subject group as fixed factors and the nine EMG_{net} as dependent variables. A third MANOVA investigated the co-activation between the targeted muscle and the non-targeted muscles accompanying the production of the maximum isometric MCP torque, using arm posture and subject group as fixed factors and the eight FDS_{andX} and the eight EDC_{andX} as dependent variables. When a fixed factor proved significant in a MANOVA, post-hoc univariate repeated measures analyses of variance (ANOVAs) or t-tests were performed on the corresponding dependent variables. To account for multiple statistical tests, a Bonferroni correction was employed such that the significance level was set to $\alpha = \frac{0.05}{3} = 0.017$ for each MANOVA and each post-hoc univariate repeated measures ANOVA and t-test.

RESULTS

Effects of arm posture and proximal electrical stimulation on MCP torque

Arm posture influenced maximum voluntary isometric torque production about the MCP joints, with differences between stroke subjects and control subjects. The MANOVA performed on MCP_{flex} and MCP_{ext} showed a statistically significant dependence upon arm posture ($p < 0.017$), subject group ($p < 0.001$), and the interaction between arm posture and
subject group (p < 0.017), but not upon stimulation condition (p = 0.993) or the remaining
interactions (arm posture and stimulation condition: p = 0.966; stimulation condition and
subject group: p = 0.794; arm posture, stimulation condition, and subject group: p = 0.577).

Post-hoc univariate repeated measures ANOVAs using arm posture as the within-
subject factor and subject group as the between-subjects factor were subsequently
performed on MCP_{flex} and on MCP_{ext}, respectively. Mean maximum normalized MCP flexion
torque (MCP_{flex}) exhibited significant effects of arm posture (p < 0.001) and subject group (p
< 0.017), and a significant interaction between arm posture and subject group (p < 0.001).
Mean MCP_{flex} was 0.86 ± 0.04 (mean ± 95% confidence interval) in stroke survivors and 0.85
± 0.03 in control subjects in posture 1, and 0.72 ± 0.06 in stroke survivors and 0.85 ± 0.04 in
control subjects in posture 2 (FIG. 2A). Separate paired-samples t-tests performed for stroke
subjects and control subjects, respectively, indicated a significant difference in mean MCP_{flex}
between posture 1 and posture 2 in stroke subjects (p < 0.001, 2-tailed), but not in control
subjects (p = 0.886). As compared to control subjects, the normalized MCP flexion torque in
stroke subjects exhibited a 15.3% deficit in posture 2, but none in posture 1. Mean maximum
normalized MCP extension torque (MCP_{ext}) exhibited a significant effect of subject group (p
< 0.001), but the effect of arm posture did not reach significance (p = 0.039), and there was
no significant interaction between arm posture and subject group (p = 0.808). In posture 1,
mean MCP_{ext} was 0.74 ± 0.06 in stroke subjects and 0.93 ± 0.02 in control subjects, and in
posture 2, it was 0.70 ± 0.05 in stroke subjects and 0.87 ± 0.03 in control subjects (FIG. 2B).
The mean difference in MCP_{ext} between posture 1 and posture 2 was similar for the two
subject groups (5.7% in stroke subjects, and 6.9% in control subjects). The normalized MCP
extension torque was greatly reduced in both arm postures in stroke subjects as compared
to control subjects (20.4% in posture 1, and 19.5% in posture 2). Thus, stroke subjects had
difficulty repeatedly producing and sustaining maximum MCP extension. For the majority of
subjects, the maximum MCP torque value used to normalize the MCP torque data was
observed in posture 1, for both the MCP flexion trials (eight of the ten stroke subjects, seven
of the ten control subjects) and the MCP extension trials (seven of the ten stroke subjects, nine of the ten control subjects).

In order to investigate a potential relationship between maximum normalized MCP torque (MCP<sub>flex</sub> and MCP<sub>ext</sub>, respectively) and the impairment level of stroke subjects, correlation analyses were performed. Correlation analyses for the MCP flexion trials indicated no statistically significant correlation between MCP<sub>flex</sub> and Fugl-Meyer score (Pearson correlation coefficient R = 0.131, p = 0.317, 2-tailed), while for the MCP extension trials there was a statistically significant positive correlation between MCP<sub>ext</sub> and Fugl-Meyer score (R = 0.316, p < 0.05). Conversely, the stroke subjects' Fugl-Meyer scores were significantly negatively correlated with the difference between posture 1 and posture 2 in MCP<sub>flex</sub> (R = -0.385, p < 0.05), but not with the difference between posture 1 and posture 2 in MCP<sub>ext</sub> (R = -0.032, p = 0.867).

**Effect of arm posture on upper limb muscle activities**

In the MANOVA performed on the nine EMG<sub>net</sub>, the effect of arm posture and the interactions between arm posture and contraction or/and subject group were not statistically significant (arm posture: p = 0.361; arm posture and contraction: p = 0.257; arm posture and subject group: p = 0.197; arm posture, contraction, and subject group: p = 0.162). Likewise, in the MANOVA performed on the eight FDSandX and the eight EDCandX, the effect of arm posture (p = 0.690) and the interaction between arm posture and subject group (p = 0.580) were not statistically significant. Based on these results, we investigated potential trends with respect to arm posture for the EMG<sub>net</sub> and the FDSandX and the EDCandX. FIG. 3 shows, for both stroke subjects and control subjects and for both the MCP flexion trials and the MCP extension trials, the mean net EMG activity (EMG<sub>net</sub>) of each of the nine upper limb muscles in posture 1 and posture 2. Although the effect of arm posture was not significant, the EMG data appeared to suggest a trend for upper limb muscle activities to be influenced by arm posture during maximum voluntary isometric contraction at the MCP joints.
During the MCP flexion trials, a trend for the mean net EMG activity of the targeted muscle FDS (FDS$_{net}$) to be greater in posture 1 was observed in control subjects (FIG. 3C), but not in stroke subjects (FIG. 3A). The activity of non-targeted upper limb muscles during the MCP flexion trials also appeared to be differentially influenced by arm posture in the two subject groups. In particular, a trend towards greater upper limb flexor activity (mean FCU$_{net}$ and mean BB$_{net}$) in posture 1 was observed in stroke subjects (FIG. 3A), whereas control subjects exhibited a trend towards greater elbow flexor activity in posture 2, in terms of both mean BB$_{net}$ (FIG. 3C) and the mean co-activation between FDS and BB (FDSandBB: 0.26 ± 0.08 (mean ± 95% confidence interval) in posture 1 vs. 0.41 ± 0.09 in posture 2).

During the MCP extension trials, the mean net EMG activity of the targeted muscle EDC (EDC$_{net}$) appeared not to be different between arm postures in either subject group (FIG. 3B and FIG. 3D). Similar to the MCP flexion trials, arm posture appeared to influence the activity of non-targeted upper limb muscles during the MCP extension trials with differences between the two subject groups. A trend towards greater elbow flexor activity (mean BB$_{net}$) was observed in posture 1 in stroke subjects (FIG. 3B), as was the case during the MCP flexion trials. In addition, elbow extensor activity (mean TB$_{net}$) tended to be greater in posture 1 in stroke subjects (FIG. 3B). Similar trends were observed for the mean co-activation between EDC and BB and between EDC and TB, respectively (EDCandBB: 0.28 ± 0.10 vs. 0.17 ± 0.07; EDCandTB: 0.45 ± 0.08 vs. 0.30 ± 0.09), in stroke subjects. Control subjects appeared not to exhibit differences in elbow flexor or elbow extensor activity.

Correlation analyses were performed in order to investigate a potential relationship between the EMG data and the impairment level of stroke subjects. For the MCP flexion trials, there was no significant correlation between the stroke subjects' Fugl-Meyer scores and any of the nine EMG$_{net}$ or any of the eight FDSandX. For the MCP extension trials, FDS$_{net}$, B$_{net}$, BB$_{net}$, and EDCandBB all exhibited a significant positive correlation with the stroke subjects' Fugl-Meyer scores (FDS$_{net}$: R = 0.488, p < 0.05; B$_{net}$: R = 0.506, p < 0.05, BB$_{net}$: R = 0.481, p < 0.05, EDCandBB: R = 0.500, p < 0.05). No significant correlation was
observed between the stroke subjects' Fugl-Meyer scores and the difference between posture 1 and posture 2 in any of the nine EMG$_{\text{net}}$ or any of the eight FDSandX, for the MCP flexion trials, or the difference between posture 1 and posture 2 in any of the nine EMG$_{\text{net}}$ or any of the eight EDCandX, for the MCP extension trials. Note that in these correlation analyses for the EMG data, only data from the "No stimulation" condition could be used, in contrast to the correlation analyses for the MCP torque data, in which data from all three stimulation conditions were used.

**Effects of contraction and subject group on upper limb muscle activities**

In the MANOVA performed on the nine EMG$_{\text{net}}$, the effects of contraction (p < 0.001) and subject group (p < 0.017) and the interaction between contraction and subject group (p < 0.001) were statistically significant. In the MANOVA performed on the eight FDSandX and the eight EDCandX, the effect of subject group (p = 0.150) was not statistically significant. Based on these results, we performed post-hoc t-tests in order to investigate potential statistically significant differences between contractions and between subject groups for the EMG$_{\text{net}}$, and we investigated potential trends with respect to subject group for the FDSandX and the EDCandX. Paired-samples t-tests were performed to compare each of the nine EMG$_{\text{net}}$ between the MCP flexion trials and the MCP extension trials, separately for each of the two subject groups. Independent-samples t-tests were performed to compare each of the nine EMG$_{\text{net}}$ between stroke subjects and control subjects, separately for the MCP flexion trials and for the MCP extension trials. Table 2 shows, for both stroke subjects and control subjects, the mean net EMG activity (EMG$_{\text{net}}$) of each of the nine upper limb muscles for the MCP flexion trials and the MCP extension trials, as well as the mean difference between the MCP flexion trials and the MCP extension trials in EMG$_{\text{net}}$ ($\Delta$EMG$_{\text{net}}$). Independent-samples t-tests were performed to compare each of the nine $\Delta$EMG$_{\text{net}}$ between stroke subjects and control subjects.
Stroke subjects exhibited reduced task specificity in terms of the upper limb muscle activities accompanying maximum voluntary isometric MCP flexion or extension, respectively. Reduced task specificity in activity was apparent for the targeted muscles FDS and EDC, as the mean difference between the MCP flexion trials and the MCP extension trials in both FDS$_\text{net}$ ($\Delta$FDS$_\text{net}$) and EDC$_\text{net}$ ($\Delta$EDC$_\text{net}$) was significantly smaller in stroke subjects than in control subjects (Table 2). Reduced task specificity in activity was furthermore observed for some non-targeted muscles, such as FCU (Table 2). On the other hand, stroke subjects appeared to exhibit a task-specific difference in activity for the non-targeted muscle TB, as the MCP extension trials were accompanied by significantly greater mean TB$_\text{net}$ than the MCP flexion trials in stroke subjects, while there was no significant difference in control subjects (Table 2). Accordingly, mean $\Delta$TB$_\text{net}$ was significantly greater in stroke subjects than in control subjects (Table 2).

The EMG data exhibited further differences in upper limb muscle activities between stroke subjects and control subjects, for both the MCP flexion trials and the MCP extension trials. During the MCP flexion trials, stroke subjects exhibited a deficit in activating the targeted muscle, as mean FDS$_\text{net}$ was significantly smaller in stroke subjects (0.37 ± 0.07) than in control subjects (0.47 ± 0.04) ($p < 0.017$, 2-tailed) (FIG. 4A). In addition to the significantly smaller mean activity of the targeted muscle, the MCP flexion trials were characterized by significantly greater mean activity of its direct antagonist (EDC$_\text{net}$) in stroke subjects as compared to control subjects (0.23 ± 0.07 vs. 0.11 ± 0.03, $p < 0.01$) (FIG. 4A). A similar trend was observed for mean FDS and EDC (0.36 ± 0.10 vs. 0.19 ± 0.04) (FIG. 4C). Moreover, stroke subjects overall exhibited greater activity of non-targeted upper limb muscles. Notably, greater elbow flexor activity was observed, as mean B$_\text{net}$ was significantly greater in stroke subjects (FIG. 4A) and a similar trend existed for mean FDS and BB (FIG. 4C). During the MCP extension trials, a deficit in activating the targeted muscle was again observed in stroke subjects, as mean EDC$_\text{net}$ was reduced in stroke subjects as compared to control subjects, although the difference did not reach significance (0.37 ± 0.09 vs. 0.46 ± 0.07, $p = 0.078$) (FIG. 4B). Similar to the MCP flexion...
trials, the MCP extension trials were also characterized by greater activity of the direct
antagonist of the targeted muscle in stroke subjects as compared to control subjects.
Indeed, mean FDS\textsubscript{net} was significantly greater in stroke subjects than in control subjects
(0.15 ± 0.07 vs. 0.06 ± 0.02, p < 0.05) (FIG. 4B), and a similar trend was observed for mean
EDC\textsubscript{and}FDS (0.28 ± 0.13 vs. 0.14 ± 0.06) (FIG. 4D). Again similar to the MCP flexion trials,
the MCP extension trials furthermore exhibited greater activity of non-targeted upper limb
muscles overall in stroke subjects. In particular, both greater elbow flexor activity and greater
elbow extensor activity were observed, as mean TB\textsubscript{net} was significantly greater in stroke
subjects (FIG. 4B) and mean BB\textsubscript{net} (FIG. 4B) and mean EDC\textsubscript{and}B and mean EDC\textsubscript{and}TB
(FIG. 4D) exhibited a similar trend. In contrast to the MCP flexion trials, mean FCU\textsubscript{net} was
significantly greater in stroke subjects (FIG. 4B), and a similar trend existed for mean
EDC\textsubscript{and}FCU (FIG. 4D).

DISCUSSION

Effect of arm posture on voluntary MCP flexion and extension

The production of maximum voluntary isometric torque about the MCP joints was
influenced by static arm posture in stroke subjects, but only in the direction of flexion, and
appeared not to be influenced in control subjects. Stroke subjects produced significantly
greater mean maximum normalized MCP flexion torque (MCP\textsubscript{flex}) when the elbow was flexed
and the shoulder was in a neutral posture (posture 1) than when the elbow was extended
and the shoulder was flexed (posture 2). Arm posture did not have an effect on MCP\textsubscript{flex} in
control subjects, and did not have an effect on mean maximum normalized MCP extension
torque (MCP\textsubscript{ext}) in either subject group. Compared to control subjects, mean maximum
normalized MCP torque in stroke subjects was reduced in posture 2 for MCP flexion and in
both arm postures for MCP extension.
Several studies have investigated the effect of static arm posture on force or strength in the hand or fingers in neurologically intact subjects, with various and contradictory results (Balogun et al. 1991; Desrosiers et al. 1995; Kuzala and Vargo 1992; Mathiowetz et al. 1985; Oxford 2000; Roman-Liu 2003; Stegink Jansen et al. 2003; Su et al. 1994; 1993). Notably, while some investigators have documented greater grip strength in an extended elbow posture (Kuzala and Vargo 1992; Oxford 2000; Su et al. 1994; 1993), others have found it to be greater in a flexed elbow posture (Mathiowetz et al. 1985) or to be unaffected by elbow posture (Desrosiers et al. 1995). Our results suggest no significant effect of static arm posture on either voluntary MCP flexion torque or voluntary MCP extension torque in neurologically intact subjects, although voluntary MCP extension torque exhibited a trend to be greater in posture 1, by a relatively modest amount (6.9% increase with respect to posture 2) (FIG. 2B). In stroke subjects, on the other hand, we observed significantly greater voluntary MCP flexion torque in posture 1 (19.4% increase with respect to posture 2). This suggests a fundamental change in the effect of static proximal upper limb posture on distal voluntary motor activity after stroke.

We propose that the observed effects of static arm posture cannot be attributed merely to the biomechanics of the finger muscles. Both FDS and EDC cross the elbow: the humeroulnar head of FDS originates from the medial epicondyle of the humerus, and EDC originates from the lateral epicondyle of the humerus. As a consequence, changes in elbow angle could potentially influence the length of FDS or/and EDC, respectively, and thus influence the force and the torque that the muscle(s) can generate. In a previous paper (Hoffmann et al. 2009), however, we have argued that the variation in FDS length with elbow angle is minimal, based on an estimation using a musculoskeletal model developed with the SIMM software (MusculoGraphics, Santa Rosa, CA). We obtained similar results for EDC, as the model estimated the difference in EDC musculotendon length between 0 and 90° of elbow flexion to be on the order of 1% of the minimum estimated EDC musculotendon length. From these estimations, we propose that the differences between posture 1 and posture 2 in the present study cannot be attributed merely to differences in FDS length or
EDC length between the two arm postures. The differences between arm postures could furthermore potentially be attributed to fatigue of the subjects, given that all the subjects but two stroke subjects and one control subject were tested in posture 1 first. However, if fatigue occurred between posture 1 and posture 2, one would expect it to affect both the MCP flexion trials and the MCP extension trials, whereas this was not observed (in stroke subjects, only mean MCP flex was affected by arm posture). Furthermore, the two stroke subjects who were tested in posture 2 first exhibited greater mean MCP flex in posture 1 than in posture 2, contrary to what would be expected if fatigue occurred.

Rather, the results of the present study suggest a modulation of distal motor output by static posture of the proximal upper limb in hemiparetic stroke subjects. In neurologically intact subjects, it has been shown that the corticospinal activation of distal upper limb muscles in response to transcranial magnetic stimulation under resting conditions can be modulated by static arm posture (Dominici et al. 2005; Ginanneschi et al. 2005; Ginanneschi et al. 2006). A similar modulating influence of static arm posture on distal motor output was observed in response to voluntary muscle activation, suggesting that static arm posture can influence the accessibility and recruitment of the corticospinal pathways during voluntary activation (Dominici et al. 2005). Weakness, which in stroke subjects can affect both finger flexors and extensors (Cruz et al. 2005; Kamper et al. 2006), likely results from a direct reduction in the corticospinal drive from the affected hemisphere. It is possible that the significantly greater mean MCP flex observed in posture 1 in stroke subjects during the MCP flexion trials in the present study reflects a greater ability to voluntarily activate finger flexor muscles when the arm is placed in posture 1 as compared to posture 2, or in other words a greater impairment in voluntary finger flexion in posture 2. Indeed, the mean value of MCP flex in stroke subjects was similar to the one in control subjects in posture 1, whereas it was smaller than the one in control subjects in posture 2. However, arm posture did not appear to affect the mean net EMG activity of the targeted muscle FDS (FDS net) in stroke subjects. Other, non-recorded muscles, such as flexor digitorum profundus and dorsal and palmar interossei, may be involved.
Static arm posture may also modulate the activity of spinal circuits and thus indirectly modulate the motor output of a muscle or muscle groups in response to descending drive. In the studies by Dominici et al. (2005) and Ginanneschi et al. (2005) mentioned above, static arm posture had the same effect on the motor output of a distal upper limb muscle in response to transcranial magnetic stimulation and on the excitability of the H-reflex response of that muscle. The results observed for the MCP flexion trials in stroke subjects of the present study are comparable to those of a recent study (Hoffmann et al. 2009) which showed that the magnitude of the stretch reflex response of the spastic finger flexors in relaxed stroke subjects was greater in posture 1, both in terms of reflex MCP flexion torque and in terms of reflex FDS activity. Taken together, our two studies suggest that the spinal excitability of finger flexors post-stroke is increased in posture 1. Descending pathways influence the activity of spinal circuits, and an alteration in tonic descending synaptic input to motoneuron pools, potentially due to baseline changes in cortical excitation or inhibition after stroke, is thought to be involved in spasticity after stroke (Katz and Rymer 1989; Powers et al. 1988). Possibly altered descending influence on spinal activity could be involved in the modulation of both reflex activity and voluntary motor activity of finger flexors post-stroke by static arm posture.

Stroke subjects and control subjects exhibited a similar mean difference in MCP\textsubscript{ext} between posture 1 and posture 2, although a trend for mean MCP\textsubscript{ext} to be greater in posture 1 was observed that was more pronounced in control subjects than in stroke subjects. However, with respect to control subjects, mean MCP\textsubscript{ext} was reduced in stroke subjects in both arm postures, as opposed to the posture-dependent reduction observed for MCP\textsubscript{flex}. Increased co-activation between finger extensors and finger flexors may have limited MCP extension torque in stroke subjects (Kamper et al. 2006; Kamper and Rymer 2001). In that respect, the mean activity of the direct antagonist FDS (FDS\textsubscript{net}) of the targeted muscle EDC and the mean co-activation between EDC and FDS (EDCandFDS) were greater in stroke subjects than in control subjects and were not influenced by arm posture during the MCP extension trials in stroke subjects in the present study (FDS\textsubscript{net}: 0.17 ± 0.13 in posture 1 vs.
0.13 ± 0.09 in posture 2; EDCandFDS: 0.29 ± 0.21 vs. 0.27 ± 0.20), suggesting generalized exaggerated co-activation between finger extensors and finger flexors, i.e. independent of arm posture. MCP\textsubscript{ext} was significantly positively correlated with the Fugl-Meyer scores of stroke subjects, indicating greater ability to voluntarily extend the fingers for less severely impaired stroke survivors. The observation of a trend, in control subjects as compared to stroke subjects, for mean MCP\textsubscript{ext} to be greater in posture 1 may suggest a more limited modulating influence of arm posture on voluntary MCP extension than on voluntary MCP flexion, and a reduction of this influence after stroke. This reduction may be due to an intrinsic limit in the residual ability of stroke subjects to voluntarily activate finger extensor muscles. The observation that contrary to MCP\textsubscript{ext}, MCP\textsubscript{flex} was not significantly positively correlated with the stroke subjects' Fugl-Meyer scores further suggests that the ability to voluntarily extend the fingers may be more dependent on impairment level than the ability to voluntarily flex the fingers, in accordance with previous studies reporting preferential impairment of voluntary finger extension (Cruz et al. 2005; Kamper et al. 2006). Taken together with the significantly smaller mean MCP\textsubscript{flex} observed in posture 2 as compared to posture 1 in stroke subjects, the significant negative correlation between Fugl-Meyer score and the difference between posture 1 and posture 2 in MCP\textsubscript{flex} suggests that more severely impaired stroke survivors may exhibit a posture-dependent impairment in voluntary finger flexion, namely reduced voluntary finger flexion with the elbow extended and the shoulder flexed. Less severely impaired individuals may tend towards being able to generate the same amount of voluntary finger flexion regardless of elbow and shoulder posture, as appears to be the case in neurologically intact individuals.

Coupled activities of upper limb muscles

Differences existed between stroke subjects and control subjects in terms of the patterns of upper limb muscle activities that accompanied the MCP flexion trials and the MCP extension trials. Stroke subjects appeared to exhibit excessive co-activation between
proximal and distal upper limb muscles, during both voluntary finger flexion and voluntary finger extension. This excessive proximal-distal co-activation appeared to be at least partly modulated by arm posture. In particular, posture 1 appeared to elicit elbow flexor activity in stroke subjects. Furthermore, reduced task specificity appeared to exist in stroke subjects, both for the targeted muscles (FDS and EDC, respectively) and for non-targeted muscles. The results of the present study suggest an alteration in the effect of descending drive associated with distal voluntary motor activity on upper limb muscle activity and in the modulation of voluntary upper limb motor activity by static arm posture in stroke subjects.

The patterns of upper limb muscle activities that accompanied the MCP flexion trials and the MCP extension trials in stroke subjects in the present study could be associated with the abnormal coupling of the activities of specific upper limb muscle groups during voluntary motor activity often observed after stroke, in particular between the shoulder and the elbow (Beer et al. 1999; Dewald and Beer 2001; Dewald et al. 1995; Sangani et al. 2009). Stereotypical muscle activation patterns of "flexor synergy", characterized notably by shoulder abduction and external rotation and elbow flexion, or "extensor synergy", characterized notably by shoulder adduction and internal rotation and elbow extension (Brunnstrom 1970), could be involved in the greater activity of non-targeted upper limb muscles during voluntary motor activity at the MCP joints in stroke subjects in the present study. The differences in patterns of upper limb muscle activities observed between arm postures could then reflect a modulation of abnormal coupling by static arm posture. Such a modulation has been reported previously between the shoulder and the elbow (Ellis et al. 2007). The MCP extension trials exhibited a significant positive correlation between the stroke subjects' Fugl-Meyer scores and the activity of upper limb flexors (FDS, B, and BB). It is possible that stroke survivors with a higher Fugl-Meyer score are more able to voluntarily extend their fingers, but that this greater ability comes at the cost of an increase in unwanted activation of muscles throughout the upper limb, and of upper limb flexors in particular, possibly as an inability to "move out of synergy" and individuate muscle activations.
While the present study did not directly investigate neural pathways, it is informative to consider prior studies which may be relevant to our findings regarding the coupled activities of upper limb muscles in stroke subjects. Stroke may result in alterations in regulatory mechanisms at the cortical level, leading to abnormal coupling of muscle activities (Gerachshenko et al. 2008; Lum et al. 2003). For instance, it has been suggested that disruption of pre-contraction suppression of antagonist activity is involved in abnormal BB activity during voluntary forearm pronation post-stroke (Gerachshenko et al. 2008). As a consequence of the loss of corticospinal pathways, voluntary motor activity in stroke subjects may involve increased reliance on alternative, residual descending pathways. Increased reliance on brainstem pathways has been suggested to underlie the emergence of abnormal coupling between upper limb muscles or muscle groups after stroke (Schwerin et al. 2008). Ellis and co-workers (Ellis et al. 2007) observed a modulating effect of static shoulder posture on the abnormal coupling between shoulder adduction and elbow extension after stroke and suggested that static arm posture can modulate the balance between descending influence from reticulospinal pathways, potentially favoring upper limb flexion, and from vestibulospinal pathways, potentially favoring upper limb extension. In the macaque monkey, the reticulospinal tract has been shown to facilitate ipsilateral flexor muscles of the shoulder, the elbow, and the wrist (Davidson and Buford 2006; 2004) and to make excitatory ipsilateral connections to motoneurons projecting to distal upper limb muscles including hand muscles (Riddle et al. 2009). Increased use of reticulospinal pathways after stroke, and modulation of their descending influence by static arm posture, could potentially be involved in some of the observations of the present study, and specifically the greater activity of non-targeted elbow flexors, and possibly the greater mean MCP\textsubscript{flex} in posture 1 in stroke subjects. There is evidence that, in parallel with its transmission via the monosynaptic corticospinal pathways, the descending corticospinal drive to upper limb motoneurons in humans is in part transmitted via a system of propriospinal interneurons located at the cervical level of the spinal cord (Pierrot-Deseilligny 2002; 1996). These interneurons are thought to have divergent projections onto motoneurons of multiple upper limb muscles (Mazevet and
Pierrot-Deseilligny 1994) and may therefore be involved in coupling of muscles throughout the upper limb. The part of the corticospinal drive that is supposed to be transmitted via this propriospinal system has been shown to be increased after stroke (Mazevet et al. 2003; Pierrot-Deseilligny 1996; Stinear and Byblow 2004), which could result in increased coupling of upper limb muscle activities (Mazevet et al. 2003; Pierrot-Deseilligny 2002).

Absence of effect of proximal electrical stimulation on voluntary MCP flexion and extension

In the present study, proximal electrical stimulation had no effect on the production of maximum voluntary isometric torque about the MCP joints in stroke subjects or in control subjects, neither for MCP flexion nor for MCP extension.

Conversely, in a previous study (Hoffmann et al. 2009), proximal electrical stimulation modulated the magnitude of the stretch reflex response of the spastic finger flexors in relaxed hemiparetic stroke survivors. Specifically, fast imposed extension of the MCP joints elicited greater reflex MCP flexion torque during stimulation of BB than when no stimulation was applied or during stimulation of TB. No effect of proximal electrical stimulation was observed for neurologically intact control subjects pre-activating their finger flexors (unpublished observation). The combined results of the previous study and the present one suggest that in the upper limb post-stroke, proximal electrical stimulation can influence distal reflex activity but may not influence distal voluntary motor activity. One potential explanation for this discrepancy may be a difference in finger flexor motoneuron recruitment. In the previous study, stroke subjects were relaxed, such that motoneurons were presumably not recruited before the onset of the imposed MCP extension. It is possible that in stroke subjects, BB stimulation increases the excitability of motoneurons at rest by lowering their recruitment threshold, and that this results in additional recruitment of motoneurons in response to imposed MCP extension. In the present study, subjects produced maximum voluntary isometric contraction, and it is possible that BB stimulation increased the excitability of motoneurons already being voluntarily recruited by stroke subjects without BB
stimulation and thus did not increase muscle activation. The motoneurons involved could be 
motoneurons with lower recruitment threshold (Calancie and Bawa 1984). In control 
subjects, BB stimulation may not influence the excitability of motoneurons, or the influence 
may exist but have no effect because the motoneurons that are influenced are already 
voltarily recruited, both in the situation of pre-activation in the previous study and in the 
situation of maximum voluntary isometric contraction in the present study. An alternative 
explanation for the discrepancy between the two studies is based on the evidence that 
peripheral afferents do not exert presynaptic inhibition on descending motor pathways 
(Berardelli et al. 1987; Jackson et al. 2006; Nielsen and Petersen 1994) and that the 
influence of peripheral afferent input on spinal motor circuits is reduced during voluntary 
motor activity (Seki et al. 2003). This may prevent an influence of proximal electrical 
stimulation on voluntary distal upper limb motor activity in the present study.

Conclusion

The present study provides evidence for a modulating effect of static arm posture on 
voluntary distal upper limb motor activity in hemiparetic stroke subjects. Static arm posture 
also modulated the activities of upper limb muscles that accompanied voluntary distal upper 
limb motor activity, with differences between stroke subjects and neurologically intact control 
subjects in both the coupling patterns of muscle activities and the effect of arm posture on 
these patterns. The results of the present study could potentially open possibilities for upper 
limb rehabilitation strategies after stroke involving manipulation of static posture of upper 
limb joints. In that respect, further study is warranted in order to investigate how effects such 
as the ones observed in the present study may impact the ability of hemiparetic stroke 
survivors to perform functional movements of the fingers, the hand, and the arm.
ACKNOWLEDGEMENTS

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Jennifer Kahn is currently affiliated with the Department of Physical Therapy and Human Movement Sciences, Feinberg School of Medicine, Northwestern University, Chicago, Illinois.

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REFERENCES


TABLE 1. Demographic and clinical data for the stroke subjects participating in the study

The subject's age is indicated in years ("y"). The time at which the experiment was conducted with respect to the occurrence of the subject's stroke ("Time after stroke") is indicated in months ("m"). "Side" indicates whether the subject had right ("R") or left ("L") hemiparesis and thus which upper limb was studied. "Clinical score" indicates the subject's Fugl-Meyer upper extremity motor score (out of a maximum score of 66). "Handedness" indicates whether the subject was right-handed or left-handed prior to her/his stroke.

FIG. 1. Schematic representation of the two arm postures used in the study. The thick black vertical line symbolizes where the subjects' fingers were coupled to the shaft of the servomotor, the thick black horizontal line symbolizes the surface of the experimental table, and the small grey rectangle symbolizes the cushioned support used to support the subjects' arm.

FIG. 2. Effect of arm posture on maximum normalized MCP flexion (A) and MCP extension (B) torque in stroke subjects and control subjects. For each subject group, each box represents the mean value of MCP$^{\text{flex}}$ or MCP$^{\text{ext}}$, respectively, for the corresponding arm posture (dark grey: posture 1, light grey: posture 2). Bars represent 95% confidence intervals. Asterisks indicate a statistically significant difference between posture 1 and posture 2 (***, $p < 0.001$).

FIG. 3. Differences between arm postures in net EMG activity of the nine upper limb muscles during the MCP flexion (A and C) and MCP extension (B and D) trials in stroke subjects (A and B) and control subjects (C and D). For each subject group, each box represents the mean value of EMG$^{\text{net}}$ for the corresponding muscle and the corresponding arm posture (dark grey: posture 1, light grey: posture 2). Bars represent 95% confidence intervals.
FIG. 4. Differences between stroke subjects and control subjects in net EMG activity (A and B) and co-activation between the targeted muscle and non-targeted muscles (C and D) during the MCP flexion (A and C) and MCP extension (B and D) trials. Each box represents the mean value of EMG$_{net}$ (A and B) for the corresponding muscle, or of FDSandX (C) or EDCandX (D), respectively, for the corresponding pair of muscles, and the corresponding subject group (dark grey: stroke subjects, light grey: control subjects). Bars represent 95% confidence intervals. In A and B, asterisks indicate a statistically significant difference between stroke subjects and control subjects (* p < 0.017, ** p < 0.01).

TABLE 2. Differences between MCP flexion trials and MCP extension trials in net EMG activity of the nine upper limb muscles in stroke subjects (top) and control subjects (bottom)

For each subject group, "EMG$_{net}$, Flexion" and "EMG$_{net}$, Extension" show the mean value and the 95% confidence interval for EMG$_{net}$ for the corresponding muscle and the corresponding contraction, and "ΔEMG$_{net}$" shows the mean value and the 95% confidence interval for the difference between the MCP flexion trials and the MCP extension trials in EMG$_{net}$ for the corresponding muscle. $p_{contraction}$ values (2-tailed) refer to differences between MCP flexion trials and MCP extension trials in EMG$_{net}$. $p_{group}$ values (2-tailed) refer to differences between stroke subjects and control subjects in ΔEMG$_{net}$. 
TABLE 1. **Demographic and clinical data for the stroke subjects participating in the study**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age (y)</th>
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<th>Clinical score</th>
<th>Handedness</th>
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<tbody>
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The subject's age is indicated in years ("y"). The time at which the experiment was conducted with respect to the occurrence of the subject's stroke ("Time after stroke") is indicated in months ("m"). "Side" indicates whether the subject had right ("R") or left ("L") hemiparesis and thus which upper limb was studied. "Clinical score" indicates the subject's Fugl-Meyer upper extremity motor score (out of a maximum score of 66). "Handedness" indicates whether the subject was right-handed or left-handed prior to her/his stroke.
TABLE 2. Differences between MCP flexion trials and MCP extension trials in net EMG activity of the nine upper limb muscles in stroke subjects (top) and control subjects (bottom)

<table>
<thead>
<tr>
<th>Muscle</th>
<th>EMG&lt;sub&gt;net&lt;/sub&gt;, Flexion</th>
<th>EMG&lt;sub&gt;net&lt;/sub&gt;, Extension</th>
<th>p&lt;sub&gt;contraction&lt;/sub&gt;</th>
<th>∆EMG&lt;sub&gt;net&lt;/sub&gt;</th>
<th>p&lt;sub&gt;group&lt;/sub&gt;</th>
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<td></td>
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<tr>
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<td>0.001</td>
<td>0.21 ± 0.11</td>
<td>0.001</td>
</tr>
<tr>
<td>EDC</td>
<td>0.23 ± 0.07</td>
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<td>0.033</td>
<td>-0.15 ± 0.14</td>
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<td>0.28 ± 0.07</td>
<td>0.000</td>
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<tr>
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<td>0.000</td>
<td>0.28 ± 0.10</td>
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</tr>
<tr>
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<td>0.000</td>
<td>0.22 ± 0.09</td>
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<td>0.003</td>
<td>-0.07 ± 0.04</td>
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</table>

For each subject group, "EMG<sub>net</sub>, Flexion" and "EMG<sub>net</sub>, Extension" show the mean value and the 95% confidence interval for EMG<sub>net</sub> for the corresponding muscle and the corresponding contraction, and "∆EMG<sub>net</sub>" shows the mean value and the 95% confidence interval for the difference between the MCP flexion trials and the MCP extension trials in EMG<sub>net</sub> for the corresponding muscle. p<sub>contraction</sub> values (2-tailed) refer to differences between MCP flexion trials and MCP extension trials in EMG<sub>net</sub>. p<sub>group</sub> values (2-tailed) refer to differences between stroke subjects and control subjects in ∆EMG<sub>net</sub>. "

866 TABLE 2. Differences between MCP flexion trials and MCP extension trials in net EMG activity of the nine upper limb muscles in stroke subjects (top) and control subjects (bottom)

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<tr>
<th>Subject</th>
<th>Sex</th>
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<td>p&lt;sub&gt;contraction&lt;/sub&gt;</td>
<td>ΔEMG&lt;sub&gt;net&lt;/sub&gt;</td>
<td>p&lt;sub&gt;group&lt;/sub&gt;</td>
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<td>0.21 ± 0.11</td>
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<td>-0.15 ± 0.14</td>
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<td>0.12 ± 0.05</td>
<td>0.000</td>
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<td>0.14 ± 0.06</td>
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<td>0.11 ± 0.05</td>
<td>0.000</td>
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<td>0.26 ± 0.07</td>
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<td>0.06 ± 0.03</td>
<td>0.000</td>
<td>0.20 ± 0.09</td>
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<td>-0.03 ± 0.07</td>
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</table>
A  Stroke, Flexion

B  Stroke, Extension

C  Control, Flexion

D  Control, Extension