RESEARCH ARTICLE

Bilateral motor unit synchronization is functionally organized

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Abstract To elucidate the neural interactions underlying bimanual coordination, we investigated in 11 participants the bilateral coupling of homologous muscles in an isometric force production task involving fatiguing elbow flexion and extension. We focused on changes in motor unit (MU) synchronization as evident in EMG recordings of relevant muscles. In contrast to a related study on leg muscles, the arm muscles did not exhibit MU synchronization around 16 Hz, consistent with our hypothesis that 16 Hz MU synchronization is linked to balance maintenance. As expected, bilateral MU synchronization was apparent between 8 and 12 Hz and increased with fatigue and more strongly so for extensor than for flexor muscles. MU synchronization in that frequency band is interpreted in terms of common bilateral input and substantiates the idea that common input is functionally organized. Since these findings are consistent with the literature on mirror movements, they suggest that both phenomena may be related.

Keywords EMG coherence ·
Common drive · Motor overflow ·
Muscle fatigue · Physiological tremor

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Introduction

During bilateral movements the participating limbs do not move independently but influence each other (e.g., Treffner and Turvey 1996). Such mutual influences have been extensively investigated in bimanual rhythmic tasks involving finger oscillations and manual circle drawing (see, e.g., Kelso 1995 for an overview). Inter-limb coordination is governed by interactions between limbs arising from various constraints residing at different levels of the motor system (Carson and Kelso 2004) and several studies have been carried out to pinpoint the neural structures involved (see Carson 2005, for a review). Recently, evidence has been presented that bilateral coupling primarily stems from shared efferent information (Ridderikhoff et al. 2005a; Spencer et al. 2005) and that afferent processes only play an ancillary role. For instance, muscles that are normally coactivated may share a common drive arising from branched presynaptic fibers or from presynaptic synchronization of last-order inputs (Carr et al. 1994). Similarly, bilateral coupling may arise from cortical neural crosstalk between bilateral motor areas through the corpus callosum (e.g., Franz et al. 1996; de Oliveira et al. 2001; Ridderikhoff et al. 2005b).

To examine the interactions and processes underlying bimanual coordination, bilateral coupling has been probed by manipulating various parameters, including movement tempo and amplitude (Peper et al. 1995; Post et al. 2000a, b), amount of torque applied (Peper and Carson 1999), handedness (Treffner and Turvey 1996), attention (Swinnen et al. 1996; Amazeen et al. 1997), and more. Remarkably, muscle fatigue has hardly been investigated in this context, even though it appears a particularly expedient vehicle to gain insight

into the interactions governing interlimb coordination. The reason is that during unilateral fatiguing contractions there is an increase of contralateral coactivation of homologous muscles termed motor irradiation of motor overflow (Zijdewind and Kernell 2001; Aranyi and Rosler 2002), indicating increased bilateral coupling during fatigue and/or high effort contractions. In line with those findings, we observed in a previous study an increase in 6–11 Hz motor unit (MU) synchronization between homologous quadriceps muscles during fatiguing contractions (Boonstra et al. 2006). Based on this result, we hypothesized that increased bilateral MU synchronization and increased motor overflow are different manifestations of the same underlying increase in bilateral coupling during fatiguing contractions.

MU synchronization at a certain frequency band implies a common, rhythmic input (McAuley et al. 1997) that can be quantified via conventional coherence analyses between surface EMGs (cf. Miller and Sigvardt 1998). MU synchronization as indexed by coherence analyses differs from short-term synchronization as indexed by cross-correlation analyses, which typically result in a narrow central peak in the cross-correlation histogram signifying synchronous discharge times of two MUs caused by common presynaptic input from branched axons (Sears and Stagg 1976; Kirkwood and Sears 1978). That is, while cross-correlation analyses estimate the strength of the common input to two MUs, coherence analyses reveal details of the frequency of the common input (Farmer et al. 1993; Semmler 2002). In the latter case, MU synchronization reflects periodicities in the firing of common presynaptic input to MUs that modulates the EMG amplitude and is likely caused by presynaptic synchronization (Kirkwood et al. 1982). Although rhythmically synchronized input to MUs may arise from various levels of the nervous system (see McAuley and Marsden 2000, for a review), most researchers seem to agree that it arises at a supraspinal level (Farmer et al. 1993; McAuley et al. 1997; Grosse et al. 2002; Semmler 2002). As such, MU synchronization might be functionally organized and may act as a binding mechanism integrating spatially distributed, neural activity. In short, it may serve as a neural control mechanism (Farmer 1998; Singer 1999; Varela et al. 2001).

To investigate the functional organization of bilateral coupling between homologous muscles, we investigated the difference in increase of MU synchronization during fatiguing elbow flexion and extension. In a recent study on mirror movements, bilateral coupling was stronger between extensor muscles than between flexor muscles (Ridderikhoff et al. 2005b). Similarly,

we expected MU synchronization to increase more strongly for extensor than for flexor muscles. We further sought to determine whether earlier results regarding the synchronization between leg muscles (Boonstra et al. 2006) also hold for arm muscles. In that previous study we did not only find bilateral MU synchronization around 10 Hz, but also around 16 Hz, which was not affected by fatigue. We hypothesized that the latter MU synchronization was related to balance maintenance (cf. Sharott et al. 2003), and thus did not expect to find it in homologous arm muscles. As in the leg muscles, however, we expected to find an increase in fatigue-related 10 Hz interlimb synchronization in the arm muscles. As mentioned before, in light of the study of Ridderikhoff et al. (2005b), a stronger increase in 10 Hz synchronization was expected for extensor than for flexor muscles. Such distinct effects on MU synchronization could indeed support the alleged link between fatigue-related MU synchronization and mirror movements, as both forms of bilateral coupling are affected by fatigue and differ for extensor and flexor muscles.

Methods and materials

Participants

Eleven healthy students (nine males and two females) from the Vrije Universiteit Amsterdam participated in the experiment (mean age 23 years; range 22–27 years). All participants signed an informed consent after having been informed about the nature of the experiment. The Ethical Committee of the Faculty of Human Movement Sciences of the Vrije Universiteit had approved the experimental protocol before the experiment was conducted.

Procedure

Participants were seated in an adjustable chair in upright position and securely strapped in place by two belts to minimize the number of mechanical degrees of freedom that could be recruited in performing the experimental task, which was an isometric force production task (Fig. 1). For every participant the chair was adjusted such that both hip and knee joints were flexed at approximately 90°. Participants could exert isometric force, either by elbow flexion or extension, against a metal bar that was placed in front of the participants such that the upper arm was in vertical position and the lower arm in horizontal position with an elbow angle of approximately 90°. At both ends of the
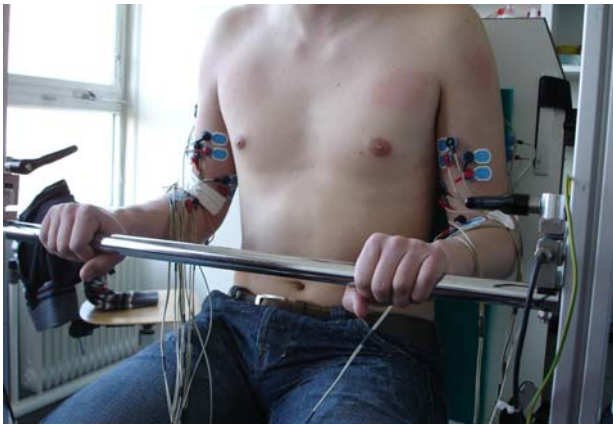
Fig. 1 Experimental setup. Participants were sitting in an adjustable chair with a metal bar in front them with force sensors on both ends for recording the exerted force

metal bar, a force transducer (STS Metric, capacity 200 kg, accuracy class C1) was attached to record the exerted force. Note that the use of the solid bar might have caused "cross-talk" between the two force sensors potentially yielding finite synchronization values between the force signals. In the present study, however, force signals were only used to control the level of force exerted and synchronization was based solely on EMG recordings.

Because the metal bar had to be adjusted for flexion and extension force production individually, the experiment was divided into two trial sets (flexion and extension), the order of which was counterbalanced across participants. Prior to the experimental trials, the maximum voluntary contraction (MVC) was determined based on two consecutive 10 s recordings, during which participants were instructed to gradually build up force and verbally reinforced to exert maximal force. The maximum force of both recordings was used as MVC reference. Subsequently, participants were asked to produce forces at 20 or 40% MVC to induce different levels of fatigue. Both the required and produced forces were displayed as horizontal lines on a computer screen placed in front of the participants, who were instructed to tune their force output to the desired force level. In order to encourage and help participants to perform elbow flexions or extensions of both arms as a functional unit, the exerted force of both force sensors were summed and displayed as a single line.

Each participant conducted 2 (flexion/extension) × 2 (force level) × 3 (repetitions) = 12 trials lasting 90 s each. All participants were able to maintain the required force level for the duration of the trial and participants rested for 3 min between trials. The order of force levels and trials was randomized across participants. By performing isometric contractions at different

force levels, the effect of fatigue on MU synchronization could be examined when comparing the change of MU synchronization over time in the different force level conditions.

Data acquisition

Surface EMG was recorded from m. biceps brachii caput longum (BL), m. biceps brachii caput breve (BB), m. brachioradialis (BR), m. brachialis (B), m. triceps brachii caput longum (TLo), m. triceps brachii caput laterale (TLa), and m. triceps brachii caput mediale (TM) of both arms. For the purpose of the present study only data from BL, BB, TLo, TLa and TM were analyzed. The electrodes (Ambu® Blue Sensor N) were placed in a bipolar montage with an interelectrode distance of about 2 cm with locations following SENIAM guidelines (Hermens et al. 2000). Data were amplified, band-pass filtered, digitized, and stored on disk (5–1,000 Hz, 22-bit sampling depth, 2 kHz sampling rate, Porti 5–16/ASD, TMS International, Enschede, The Netherlands).

Data analysis

EMG data were filtered off-line using a second-order bidirectional Butterworth high-pass filter (cutoff frequency 20 Hz) to eliminate movement artifacts. EMG signals were further full-wave rectified using the Hilbert transform (cf. Myers et al. 2003) and normalized relative to the MVC as determined at the beginning of the experiment. Each trial was divided into six consecutive intervals (I, II, ..., VI) of 15 s each. Per interval, mean power and median frequency of the EMGs were computed. The median frequency was calculated from the non-rectified EMG using Welch's periodogram method with Hamming windows of 1,024 samples length and overlapping 768 samples. The spectral power estimate per interval (I, II, ..., VI) was thus based on 114 overlapping data segments with a frequency resolution of 0.98 Hz.

We used identical settings when estimating the power spectral densities and coherence spectra of the EMG. In contrast to the calculation of the median frequency, these two analyses were based on the rectified EMG (the dc-value of each 1,024 samples data window was removed before computing the Fourier transform). The resulting power spectral densities were log-transformed (Halliday et al. 1995) and Fisher's transform was applied to the coherence spectra before conducting statistical tests (Amjad et al. 1997). Both rescaling procedures 'stabilize' the variances of the underlying distributions and were used before testing spectral

estimates for statistical differences (Rosenberg et al. 1989; Farmer et al. 1993; Gerloff et al. 1998).

Statistical analysis

Power and coherence spectra were submitted to a principal component analysis (PCA) to identify spectral components common across participants, conditions, and muscles. PCA was exploited in its capacity to reduce the dimensionality of the data by extracting major frequency components or modes and to quantify the strength of these frequency components in different conditions and muscles. The quality of the data reduction can be evaluated via the eigenvalues representing the contribution of the components extracted by the PCA to the total variance, while conditions can be compared in terms of by the corresponding coefficients of the eigenvector (see Boonstra et al. 2005; Boonstra et al. 2006, for similar applications). The advantage of such a combined analysis is that the effects of fatigue that are present in this high-dimensional signal vector can be examined in a concise and readily interpretable manner (even if the effects are small or the data are noisy). Hence, the spectra of all participants and conditions were combined into a single signal vector yielding three different high-dimensional signal vectors: a vector for the power spectra of the EMGs with 11 (participants) $\times 2$ (tasks) $\times 2$ (force levels) $\times 6$ (intervals) $\times 10$ (muscles) = 2,640 signals, a vector for interlimb coherence during flexion trials with $11 \times 2 \times 2 \times 6 \times 2$ ($BL_{left}-BL_{right}$, $BB_{left}-BB_{right}$) = 528 signals, and a vector for the interlimb coherence during extension trials with $11 \times 2 \times 2 \times 6 \times 3$ ($TLo_{left}-TLo_{right}$, $TLa_{left}-TLa_{right}$, $TM_{left}-TM_{right}$) = 792 signals.

To allow comparison with more conventional statistical approaches, we also analyzed the effects of fatigue on the amplitude, median frequency, and coherence by performing a four-way ANOVA ($2 \times 2 \times 2 \times 2$; task $\times$ muscle $\times$ force level $\times$ interval) with repeated measures on the first and last intervals per trial (cf. Boonstra et al. 2006). This design was used to evaluate whether changes in amplitude, median frequency, or coherence were different between tasks (flexion, extension) or muscle groups (biceps, triceps). To keep the design compact, however, we averaged over the various biceps and triceps muscles (or combinations in the case of coherence) so that the factor 'muscle' had two levels (biceps and triceps); note that such averaging was not necessary for the PCA. The design was completely balanced with equal record lengths for all conditions. Coherence was evaluated in 8–12 Hz frequency bands; this choice was based on the PCA results (see Results). To facilitate the interpretation of significant

results obtained with the ANOVAs, effect sizes (f) were calculated in terms of partial η^2 (Cohen 1988).

Results

The maximal combined force exerted on both force sensors was 396 ± 112 N for elbow flexion and 341 ± 80 N for elbow extension ('$\pm$' refers to between-subject standard deviation). EMG_{max} during elbow flexion was 8.57 ± 2.89 mV and 0.80 ± 0.18 mV for the biceps and triceps muscles, respectively, and EMG_{max} during elbow extension was 0.84 ± 0.31 mV (biceps) and 6.91 ± 3.43 mV (triceps). Coactivation was $10.3 \pm 3.9\%$ during flexion MVC and $13.9 \pm 7.1\%$ during extension MVC.

Both EMG amplitude and median frequency showed marked effects of muscle fatigue (Fig. 2). The EMG amplitude of all muscles increased significantly in time ($F(1,10) = 68.4$; $P < 0.001$; $f = 0.87$). The increase of EMG amplitude was significantly greater at a 40% MVC force level compared to the 20% force condition as revealed by a significant force by interval interaction ($F(1,10) = 32.4$; $P < 0.001$; $f = 0.76$). There was no significant difference between the EMG amplitude of the biceps and triceps muscles, whereas the amplitude was slightly larger during flexion than during extension ($F(1,10) = 9.40$; $P = 0.012$; $f = 0.48$). As could be expected, the EMG amplitudes were larger in the 40% force condition. Interestingly, the effects of fatigue were similar for the agonist and antagonist muscles. Median frequencies of the EMGs of all muscles decreased significantly in time ($F(1,10) = 90.6$; $P < 0.001$; $f = 0.90$) and this decrease was greater in the 40% force condition ($F(1,10) = 62.5$; $P < 0.001$; $f = 0.86$). The EMGs' median frequency showed significant main effects of muscle ($F(1,10) = 9.60$; $P = 0.011$; $f = 0.49$), task ($F(1,10) = 28.1$; $P < 0.001$; $f = 0.74$), and force level ($F(1,10) = 12.2$; $P = 0.006$; $f = 0.55$) indicating that the median frequency was higher for the biceps, during extension and at the low force condition, respectively. Again, the effect of fatigue on the EMGs' median frequency was similar for agonist and antagonist muscles (Fig. 2). The decrease was even greater for the antagonist than for the agonist muscles ($F(1,10) = 35.8$; $P < 0.001$; $f = 0.78$; decrease in median frequency: biceps flexion, 7.9 Hz; biceps extension, 13.1 Hz; triceps flexion, 11.3 Hz; triceps extension, 9.1 Hz).

The power spectra of both force sensors revealed that the low-frequency range around 2 Hz contained most power with a second, smaller peak around 10 Hz. The mean normalized power averaged over both force
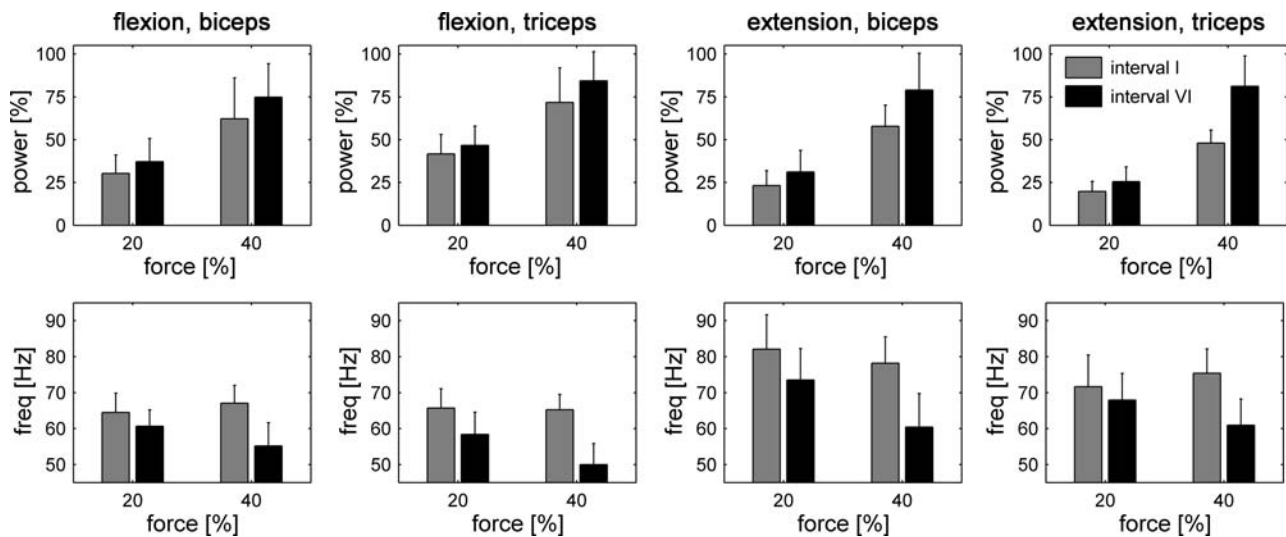
Fig. 2 *Top panels*: power as percentage MVC in the first and last 15 s interval for both tasks (flexion and extension) and averaged over different heads of the biceps and triceps; *lower panels*: idem, but for median frequency of the unrectified EMG

sensors in the 8–12 Hz frequency band was larger in the high force condition and increased in time (flexion: interval I low force, $1.23 \pm 0.88\%$; interval VI low force, $3.43 \pm 2.45\%$, interval I high force, $3.27 \pm 2.53\%$, interval VI high force, $4.43 \pm 2.65\%$; and for extension: interval I low force, $1.45 \pm 0.60\%$; interval VI low force, $2.77 \pm 1.49\%$, interval I high force, $2.34 \pm 1.41\%$, interval VI high force, $5.25 \pm 2.81\%$). The power of the rectified EMGs revealed a peak at a slightly higher frequency, i.e., between 10 and 20 Hz (Fig. 3). The first mode of the PCA of the power spectra of the EMG explained 83% (biceps) and 81% (triceps) of the total variance and represented the spectral distribution common to all muscles irrespective of condition. It displayed a fairly broad spectral peak that was maximal around 15 Hz. The second mode (about 8% of the total variance) had a peak around 18 Hz and its strength decreased in time, particularly in the high force condition, as revealed by the coefficients of the corresponding eigenvector.[1] The third mode displayed a modulation of the peak frequency of the first mode: adding the third mode to the first caused a shift of the 15 Hz peak towards lower frequencies (subtracting it thus yielded a shift towards higher frequencies). For both biceps and triceps muscles, the strength of the third mode increased in time implying that the peak of the first mode shifted towards lower frequencies in the course of a trial. Although PCA did not extract the 8–12 Hz band as a separate frequency component,

8–12 Hz EMG power was higher in the high force condition and increased in time during each trial (Fig. 5).

Coherence between EMGs of both arms displayed a clear peak around 10 Hz, particularly in the high force condition, and appeared stronger for the triceps muscles (Fig. 3). PCA of the interlimb coherence spectra yielded a first mode that covered 13% (biceps) and 23% (triceps) of the total variance (Fig. 4). For both muscle combinations, the first mode revealed a peak around 10 Hz that was indeed stronger for the triceps than for the biceps.[2] The interlimb coherence between the triceps was strongest during extension at high force and, in this condition, it increased over time. The structure of the eigenvector coefficients of the first mode of the interlimb coherence between the biceps was less clear-cut but coherence seemed to increase over time both during the high force flexion and extension contractions. The second mode represented a modulation of the frequency of the peak of the first mode similar to the third on the first mode of the power spectra (i.e., adding the second mode to the first caused a shift of the 10 Hz peak towards lower frequencies, whereas subtracting it yielded a shift towards higher frequencies). Again, the structure of eigenvector coefficients was most obvious for the triceps: during extension at high force, the strength of the mode increased considerably in time. This indicates that the peak frequency of the 10 Hz coherence decreased in frequency over time during extension at high force.

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[1] Recall that PCA modes yield eigenvector coefficients that signify the strength of these frequency components in the original spectra of different condition and thus allow for a comparison between conditions.

[2] The original coherence spectra can be reconstructed by multiplying the projection of each mode with the coefficients of the eigenvector.
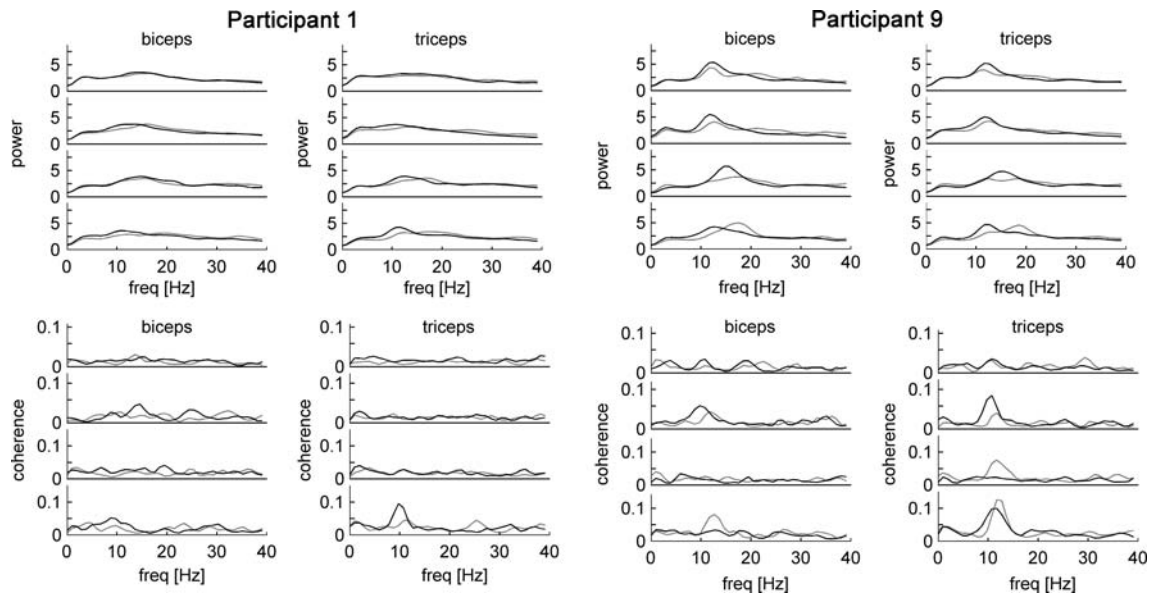
Fig. 3 Power and coherence spectra for two participants. *Top panels*: the normalized power spectra of the rectified EMG for biceps and triceps (averaged over different heads of both arms) in four different conditions (from top to bottom: flexion low force, flexion high force, extension low force, extension high force) for the first (*gray line*) and last interval (*black line*). *Lower panels*: idem, but now for the interlimb coherence spectra
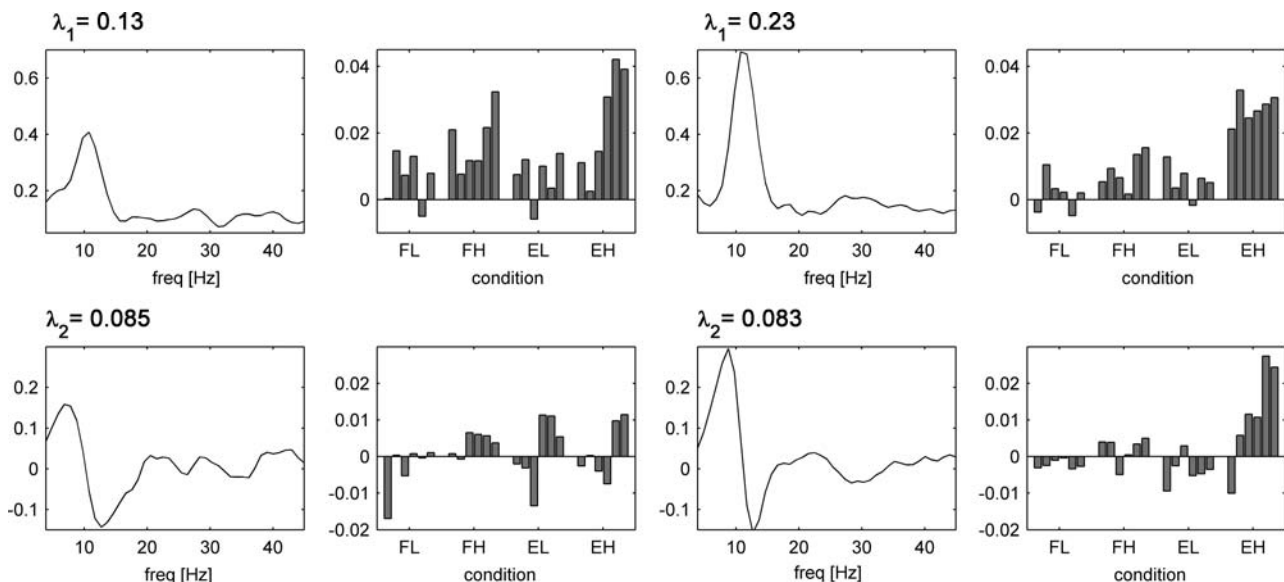

Fig. 4 First two PCA modes of interlimb coherence between biceps (*left two columns*) and triceps muscles (*right two columns*). *First column*: first two principal coherence spectra (eigenvalues 13%, 9%) of the biceps muscles; *second column*: mean coefficients for corresponding eigenvectors for flexion low force (*FL*), flexion high force (*FH*), extension low force (*EL*), and extension high force (*EH*) at the six time intervals; *columns 3 and 4*: idem, but now for the PCA of the interlimb coherence between the triceps muscles

By and large, the results of the ANOVA on the coherence spectra were in agreement with the PCA results. The 8–12 Hz intralimb coherence was significantly higher during the high force condition ($F(1,10) = 8.90$, $P = 0.014$, $f = 0.47$; mean coherence: 20% MVC, 0.018 ± 0.004; 40% MVC, 0.025 ± 0.010) and increased in time ($F(1,10) = 5.55$, $P = 0.040$, $f = 0.36$; mean coherence: interval I, 0.020 ± 0.006; interval VI, $0.023 \pm$

0.008). The force by interval interaction was almost significant ($F(1,10) = 4.73$, $P = 0.055$, $f = 0.32$), reflecting a stronger increase in coherence in the high force condition (Fig. 5). Further, there was a main effect of task ($F(1,10) = 5.43$, $P = 0.042$, $f = 0.35$; mean coherence: flexion, 0.020 ± 0.006; extension, 0.023 ± 0.007), but no main effect of muscle ($P > 0.05$), indicating that coherence was similar for biceps and triceps. Finally, there
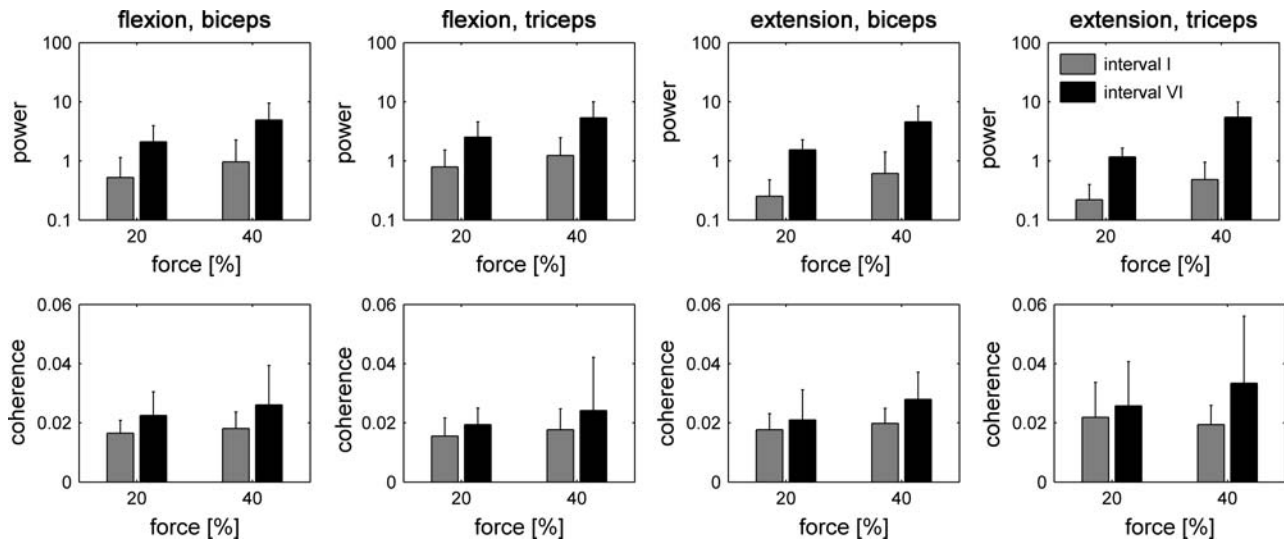
Fig. 5 Mean power and interlimb coherence in the 8–12 Hz frequency band. *Upper panels*: the subject average of the EMG power in the 8–12 Hz frequency band of the first and last interval in the four different conditions. Data are plotted on a log scale and the error bars indicate the between-subjects standard deviation. *Lower panels*: idem, but now for the interlimb coherence in the 8–12 Hz frequency band

was a significant task by muscle interaction ($F(1,10)$ = 4.97, $P = 0.050$, $f = 0.33$), which revealed that coherence between triceps muscles was higher during extension.

Discussion

PCA revealed fatigue-related 8–12 Hz interlimb coherence for both the biceps and triceps muscles. As expected, 8–12 Hz synchronization was stronger during the high force condition and increased significantly over time. The increase in interlimb synchronization was particularly strong between the triceps muscles during high force extension. Interlimb synchronization was associated with fatigue in that EMG amplitude increased and median frequency decreased as fatigue increased and these changes were stronger in the high force condition. However, although there were significant differences in interlimb coherence, absolute coherence levels were rather low and PCA converged only slowly, indicating that bilateral MU activity remained largely uncoupled (i.e., MU synchronization was weak). The 8–12 Hz interlimb coherence was accompanied by a similar peak in the power spectra of the force sensor showing tremor activity at 8–12 Hz. The 10 Hz peak in the coherence spectra shifted towards lower frequencies over time, particularly during flexion at high force. This change-over was accompanied by a similar shift towards lower frequencies in the power spectra of the rectified EMG. Given that participants were holding the bar with both hands, the increase in 8–12 Hz interlimb coherence with fatigue might have

been related to an increase in 8–12 Hz power in the force exerted on the bar via a common afferent feedback that, in principle, could have simultaneously influenced MU discharges in both limbs. The increase in 8–12 Hz synchronization, however, agreed with the results of an earlier study (Boonstra et al. 2006) in which bilateral force was exerting against mechanically independent force sensors. That is, it seemed unlikely that mechanical cross-talk (in combination with afferent feedback) caused the here-observed coherence. In contrast to our previous study, no synchronized activity was found around 16 Hz, which is consistent with our hypothesis that 16 Hz activity is related to balance maintenance (cf. Sharott et al. 2003).

We quantified muscle fatigue in terms of changes in EMG amplitude and median frequency. In all likelihood, fatigue-related increase in EMG amplitude is caused by the recruitment of additional MUs and the increase in firing frequency that are invoked to compensate for reduced muscle contractility (Lippold et al. 1960; Viitasalo and Komi 1977; Bigland-Ritchie et al. 1986). A decreasing median frequency, on the other hand, is thought to be related to the progressive slowing of the conduction velocity of action potentials along the muscle fiber resulting from an increase in extracellular metabolites (Mortimer et al. 1970; Lindstrom et al. 1977). However, because the relative decrease of the median frequency often exceeds the relative decrease of conduction velocity, other researchers have pointed at additional factors such as synchronization of MU firing (Bigland-Ritchie et al. 1981; Krogh-Lund and Jorgensen 1993). Noteworthy in

this regard is the here reported increase in EMG amplitude and decrease in median frequency of the antagonist muscles matching the change of the agonist muscles. It is unlikely that this decrease in median frequency of the antagonist muscles was caused by increased extracellular metabolites because the cocontraction level was only about 10% of the agonist activity and peripheral fatigue was therefore not to be expected. Indeed, several studies have shown that the force generating capacity of the antagonist muscles remained equal after fatiguing contractions of the agonist muscles (e.g., Levenez et al. 2005). Although the effects on the EMG amplitude and median frequency of the antagonist muscle could, in principle, result from cross-talk, it is typically interpreted as an effect of a common drive to the agonist-antagonist couple (Psek and Cafarelli 1993; Ebenbichler et al. 1998; Mullany et al. 2002; Levenez et al. 2005). In support of this interpretation, an increase in amplitude and a decrease in median frequency have been found in the contralateral homologous muscle after unilateral muscle fatigue (Morrison et al. 2005). Note that common drive refers to a homogeneous input to human MU pools (De Luca et al. 1982; De Luca and Erim 1994) and that antagonist cocontraction could be instrumental in maintaining joint stability (Solomonow et al. 1988).

Similar to the 6–11 Hz interlimb synchronization between quadriceps muscles found in our previous study (Boonstra et al. 2006), the increase in 8–12 Hz interlimb coherence found in the present experiment seems related to physiological tremor. Most obviously, the 8–12 Hz MU rhythm resembles physiological tremor in its frequency content (see McAuley and Marsden 2000, for an overview). The relation between bilateral 8–12 Hz MU synchronization and physiological tremor is supported further by a contralateral increase in physiological tremor after unilateral muscle fatigue (Morrison et al. 2005). Similarly, the decrease in peak frequency of the 8–12 Hz interlimb coherence over the course of a trial resembles the decrease in peak frequency of the acceleration in tremor activity during fatiguing contractions (Vaillancourt and Newell 2000). As described in the introduction, MU synchronization at a certain frequency points at a common rhythmic input (cf. McAuley et al. 1997). That is, rhythmic MU synchronization, e.g., underlying tremor activity, reflects in-phase MU rhythms that are additional to those at the MU intrinsic firing rates (cf. Christakos et al. 2006). Put differently, there is not necessarily an entrapment of the firing rates of individual MUs into the 8–12 Hz rhythm, but MU synchronization, as measured by surface EMG, reflects rhythmic modulation of the total EMG amplitude and suggests rhythmic input.

Note, however, that the amount of common bilateral input remained fairly small throughout the experiment in agreement with previous findings of bilateral independent tremor activity (Marsden et al. 1969; Lauk et al. 1999; Marsden et al. 1999; Hurtado et al. 2000). There, MU synchronization was examined primarily between bilateral hand muscles, whereas in the present experiment the increase in 8–12 Hz bilateral synchronization was found between upper-arm muscles. As such, our results support a distal-axial difference in bilateral synchronization, revealing synchronization in different frequency bands for different muscle groups (Marsden et al. 1999).

The stronger increase in bilateral 8–12 Hz synchronization between extensor muscles compared to flexor muscles agrees with the stronger bilateral coupling found in a recent study on mirror movements from our research group (Ridderikhoff et al. 2005b). It thus strengthens the alleged link between 8–12 Hz interlimb coherence and fatigue-related increase in motor overflow (Boonstra et al. 2006). That is, there seems to be a fatigue-related increase in common bilateral input, which may be reflected either in increased 8–12 Hz MU synchronization or increased mirror movements (or both). As described in the Introduction, there are several neural pathways via which the fatigue-related increase in bilateral coupling could come about, but several findings point at a supraspinal origin (cf. Carson 2005). For instance, afferent fibers have no direct, crossed effect on MUs of homologous muscle but only modulate the excitability of interneurons (Harrison and Zytnicki 1984; Arya et al. 1991; McCrea 2001). Furthermore, behavioral data showed that afferent information plays only an ancillary role in bilateral coupling (Ridderikhoff et al. 2005a; Spencer et al. 2005). It was shown for both maximal and submaximal contractions that there is a common drive to homologous muscles that might be instigated by interneuronal connectivity between both motor areas (Oda 1997). Interestingly, 8–12 Hz MU synchronization as well as mirror movements were stronger for extensor muscles than for flexors muscles, consistent with a functional organization of common drive. Put differently, most precisely controlled unimanual movements are made by using flexor muscles and it is therefore to be expected that flexor muscles are controlled more independently than extensor muscles (cf. Carson and Riek 2001). Interestingly, this line of argument supports the idea that movements are, in principle, controlled bimanually and in order to achieve unimanual movements the contralateral side is inhibited (cf. Daffertshofer et al. 2005). Such a control structure would imply that contralateral inhibition is less developed for extensors than for flexors as extensors

appear to be hardly used in isolation. A remarkable decrease in the functional coupling between cortical motor areas was found during bimanual skill acquisition (Andres et al. 1999) and, in view of the present results, it would be rather interesting to investigate whether such changes in bilateral coupling differ between flexor and extensor movements.

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