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RESEARCH ARTICLE

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# Coupling of antagonistic ankle muscles during co-contraction in humans

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**Abstract** In 35 healthy human subjects coupling of EMGs recorded from the tibialis anterior (TA) and soleus (Sol) muscles during voluntary co-contraction was analysed in the time and frequency domains. Two patterns were observed in different subjects or in the same subject on different occasions. One pattern consisted of central peaks in the cumulant density function of the two signals, which was often accompanied by coherence in the 15–35 Hz frequency band. The other pattern consisted of a central trough in the cumulant density function, which was mostly accompanied by coherence around 10 Hz. When this was the case oscillations were usually observed in the cumulant density function with time lags of 100 ms. Both patterns could be observed in the same subject, but usually not at the same time. Coherence around 10 Hz associated with a central trough in the cumulant density function was less common during weak than during strong co-contraction. The central peak with coherence in the 15–35 Hz frequency band in contrast tended to be most common during weak contraction. There was a tendency for the 10-Hz coherence with central trough to occur when the contractions had been maintained for some time. Both patterns could be observed when sensory feedback in large diameter afferents was blocked by ischaemia. When a central peak with coherence in the 15–35 Hz frequency band was observed for paired TA and Sol EMG recordings (10 out of 19 subjects), a coupling in the same frequency band was also observed between the EMG activities from the two muscles and the EEG activity recorded from the leg area of the motor cortex. When the central trough and the coherence around 10 Hz was observed for the EMG recordings (8 out of 19 subjects), no significant coherence was observed between EEG and EMG in 7 of the 8 subjects. In the last subject coherence around 10 Hz was observed. It is suggested that these findings signify the existence of two different central input systems to antagonistic ankle motoneurones: one input activates one muscle while depressing the antagonist and the other coactivates antagonistic motoneurones. The data suggest that at least the latter input depends on motor cortical activity.

**Keywords** Coherence · Co-contraction · Motor unit synchronization

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## Introduction

It is possible to obtain insight into the organization of the synaptic input to spinal motoneurones from the correlation and coherence between active motor units during voluntary movement in human subjects (Halliday et al. 1995; Farmer et al. 1997). A central peak is usually seen in the cross-correlogram constructed from synergistic motor units with distinct coherence peaks in the 7–12 and 15–35 Hz frequency bands (Farmer et al. 1993a). Coupling between localized cortical activity recorded by magnetoencephalography (MEG) or electroencephalography (EEG) and muscle activity recorded by electromyography (EMG) may also be observed in the 15–35 Hz frequency band (Conway et al. 1995; Salenius et al. 1997; Halliday et al. 1998). This suggests that the 15–35 Hz coherence of motor unit activity reflects the discharge frequency of corticospinal cells (see also Baker et al. 1997). The 15–35 Hz coherence is associated with short-term synchronization of the motor unit activity (Farmer et al. 1993a; Halliday et al. 1999). The origin of coherence in the 7–12 Hz frequency band is not resolved. Findings supporting a peripheral mechanism involving the spinal component of the stretch reflex arch have been reported, as have findings supporting a central origin (Lippold 1970; Joyce and Rack 1974; Elble and Randall 1976; Marsden 1978; Llinas and Pare 1995; McAuley et al. 1997; Halliday et al. 1999).

Whereas motor units from the same and synergistic muscles thus have a tendency to discharge synchronously, this is not always the case in motor units recorded from antagonistic muscles. Gibbs et al. (1994) and Farmer et al. (1998) thus found a central trough in the cross-correlograms constructed from the multiunit EMG recordings from antagonistic ankle and wrist motor units. A similar trough was also reported by Nielsen and Kagamihara (1994), who recorded single motor unit activity from antagonistic ankle muscles. In that study some antagonistic motor unit pairs were also reported to show short-term synchrony. As discussed by Nielsen and Kagamihara (1994), these findings suggest that at least two different input systems are involved in the activation of co-contracted antagonistic muscles.

This hypothesis is further explored in the present study by analysing the coupling of the tibialis anterior and soleus muscles during voluntary co-contraction. Part of this material has been published in abstract form (Nielsen et al. 1999).

## Materials and methods

The experiments were performed on 35 healthy subjects, aged 21–38 years, 11 women and 24 men. The experiments were performed in accordance with the Helsinki Declaration. All subjects gave informed written consent to the experiments, which were approved by the local ethics committee (J. No. KF 01–055/98).

### Experimental protocol

The subjects were seated in an armchair with the examined leg semiflexed in the hip (120°), the knee flexed to 160° and the ankle in 110° plantarflexion. The foot was attached to a foot plate. In the beginning of each experiment the maximal voluntary dorsi- or plantarflexion torque which the subject could keep for 5 s was recorded. The subjects received visual feedback of the torque exerted on the foot pedal as well as the rectified and integrated electromyographic (EMG) activity from the tibialis anterior (TA) and soleus (Sol) muscles (see below). The subjects were asked to perform tonic isometric dorsiflexion, plantarflexion as well as co-contraction of both plantarflexors and dorsiflexors. In the latter case the subjects first performed a dorsi- or plantarflexion to a certain level and then, while maintaining the same level of rectified and integrated EMG activity in the dorsi- or plantarflexors, adjusted the torque level to zero by co-contraction of the antagonistic muscles.

The subjects performed three different levels of dorsiflexion and co-contraction corresponding to weak (below 5% of maximal voluntary effort), medium (around 10–15% of maximal voluntary effort) and strong contraction (around 25–30% of maximal voluntary effort). It was ensured that the same level of EMG activity was recorded from the TA muscle during co-contraction and dorsiflexion.

In general each recording lasted 2 min, but in some experiments the subjects were asked to maintain a medium level of co-contraction for as long as possible.

### Surface EMG recordings

Surface EMG was recorded from the TA and Sol muscles in 23 subjects. Bipolar surface Ag-AgCl electrodes (1 $cm^2$ recording area, 2 cm between poles) were placed over the respective muscles. For the TA recordings, the electrode pairs were placed over the muscle at a separation of at least 10 cm. The signals were amplified (×5,000–10,000), filtered (1–1,000 Hz) and stored as waveforms on a computer for later analysis. Raw data without filtering were also stored for coherence analysis (see below).

### Needle EMG recordings

In 12 of the subjects recordings were also obtained from monopolar needle electrodes (Dantec type 13R05, 0.3×20 mm) inserted into the muscles. In general two electrodes were inserted into the TA muscle and one electrode was inserted into the Sol muscle. The two electrodes in the TA muscle were inserted at a separation of at least 10 cm. The signals were treated in the same way as the surface EMG recordings.

### EEG recordings

In 19 subjects electroencephalographic (EEG) activity was recorded simultaneously with EMG recordings from the TA and Sol muscles. Bipolar EEG recording was done from two EEG needle electrodes (0.1×5 mm) inserted into the scalp at the vertex and 2 cm in front of the vertex. The ground electrode was placed on the right arm. The signals were amplified (×50,000) and filtered (high pass: 1 Hz; low pass: 1,000 Hz) before being stored on a computer together with the EMG signals.

### Coherence analysis

Coherence of EMG signals has been described in detail in previous publications (Farmer et al. 1993a; Halliday et al. 1995) and will be described only briefly here.

The coherence function between the two rectified EMG signals is defined at frequency $\lambda$ as:

$$\left|R_{xy}(\lambda)\right|^2 = \frac{\left|f_{xy}(\lambda)\right|^2}{f_{xx}(\lambda) f_{yy}(\lambda)} \quad (1)$$

Coherence functions provide normative measures of linear association on a scale from 0 to 1. For the present data, the coherence provides a measure, at each Fourier frequency $\lambda$, of the fraction of the activity in one surface EMG signal which can be predicted by the activity in the second surface EMG signal. In this way, the coherence is used to quantify the strength and frequency of rhythmic synaptic inputs, which are distributed across the motoneurone pool (Farmer et al. 1993a).

For coherence between the TA and Sol muscles the spike trains generated by triggering on the largest voltage discharges in the surface or needle EMG recordings were used in order to minimize the influence of cross-talk between the electrodes due to volume conduction. For coherence between EEG and EMG recordings the raw analog signals were used. Because of the distance between these recording electrodes, cross-talk due to volume conduction was unlikely. Possible cross-talk between amplifiers was checked for and could be discarded.

### Cumulant density function

The largest spikes in the multiunit (surface or needle) EMG recorded from the electrodes placed over or in the TA and Sol muscles were selected by way of a manually set voltage discriminator window. This was done in order to minimize the influence of cross-talk between the recording electrodes. The times of spike occurrence were stored and used for analysis of the cumulant density estimate of the two signals.

Estimates of the cumulant density function are used to characterize the correlation between the two rectified surface

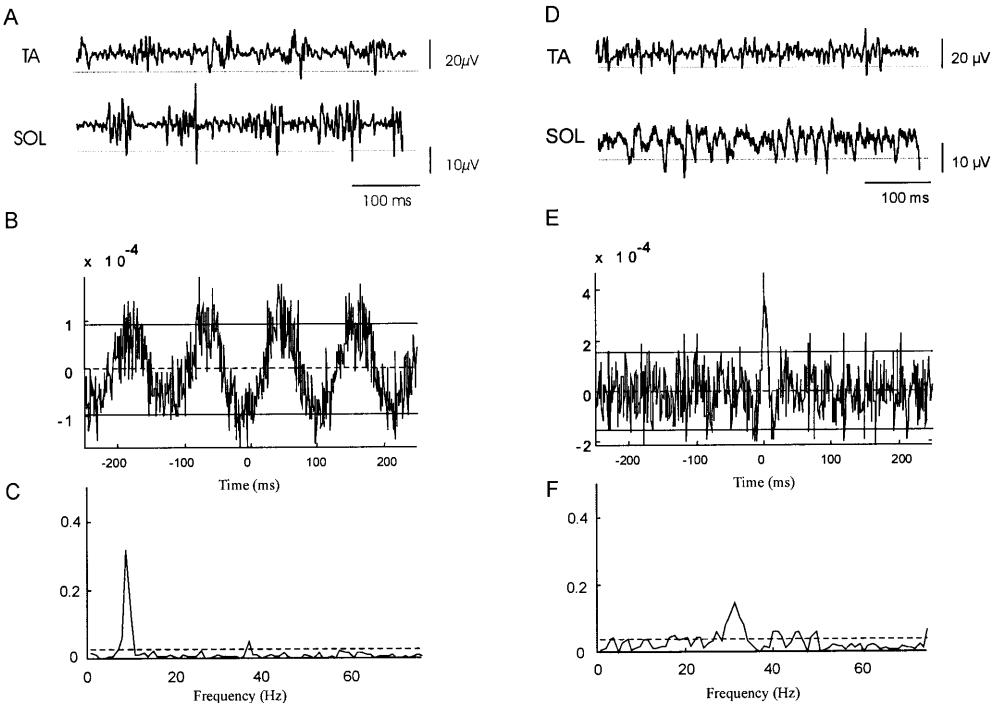


**Fig. 1** Coupling between antagonistic ankle muscles; the data are from two different subjects (**A–C** and **D–F**, respectively). **A** and **D** show the surface EMG activity recorded from the tibialis anterior (*TA*) and Sol (*TA*) muscles during co-contraction of the two antagonistic muscles. The *dashed horizontal lines* mark the trigger levels. The total recording period was 2 min. **B** and **E** show cross-correlation histograms constructed from spike trains generated by triggering on the largest EMG amplitudes in each of the EMG recordings. The histograms are based on 2,115 and 2,367 trigger events, respectively. **C** and **F** show the coherence for the same spike trains as in **B** and **E**. The *horizontal lines* in **B–F** are 95% confidence limits

EMG signals. The cumulant density function, denoted by $q_{xy}(u)$, is defined as the inverse Fourier transform of the cross spectrum:

$$q_{xy}(u) = \int_{-\pi}^{\pi} f_{xy}(\lambda) e^{i\lambda u} d\lambda \qquad (2)$$

For two uncorrelated signals the cumulant has an expected value of zero; deviations from this indicate a correlation between the two EMG signals at a particular time lag, $u$.

### Changes in coherence and cumulant density estimate during ischaemia

In experiments on three subjects the effect of blocking transmission in large diameter afferents in the lower limb was investigated. The subjects were asked to perform a medium level co-contraction of ankle dorsi- and plantarflexors for periods of 2 min before and during ischaemia. Ischaemia was induced by inflating a blood pressure cuff placed around the thigh just above the knee joint. The pressure in the cuff was maintained at 240 mmHg. Transmission in large diameter afferents was tested by stimulating the tibial nerve in the popliteal fossa below the cuff and recording the Sol H-reflex. The stimuli were 1-ms rectangular pulses and the stimulating electrode was a ball placed in the popliteal fossa. The reference electrode was a plate placed above the patella. When it was no longer possible to evoke an H-reflex in the Sol muscle, it was assumed that transmission in large diameter afferents had been blocked. This happened in general from 20 to 25 min after inflation of the cuff. The subject was then immediately asked to perform the medium level co-contraction for 2 min, after which the cuff was released.

## Results

### Synchronization and coherence of motor unit activity recorded from the TA and Sol muscles

Figure 1 illustrates data from two different subjects. Both were instructed to perform a medium level co-contraction (corresponding to around 10–15% of maximial voluntary effort) for a period of 2 min. In the subject used for the illustration in Fig. 1A–C, the EMG recorded from the TA and Sol muscles showed distinct bursts of activity at intervals of around 100 ms (Fig. 1A). The bursts of activity in the two muscles did not occur at the same time, but were usually out of phase with each other. This is also reflected in the cumulant density function for the two EMG recordings (Fig. 1B), which shows a clear central trough with several peaks and troughs on either side at intervals of 100 ms. As expected from the interval between the bursts of EMG activity and the lag between the peaks (and troughs) in the cumulant density function, coherence was found around 10 Hz (Fig. 1C).

In the subject illustrated in Fig. 1D–F, similar bursts of EMG activity were not observed (Fig. 1D). *The cumulant density function* constructed from the two EMG recordings showed a narrow central peak of synchronization (Fig. 1E). This is similar to previous observations of short-term synchronization of TA motor unit activity (Datta et al. 1991; Farmer et al. 1993a; Nielsen and Kagamihara 1994). In common with these previous studies, this form of synchronization was associated with coherence in the 15–35 Hz range (Fig. 1F).

Figure 2 illustrates that very similar observations could be obtained when the surface recordings were replaced by

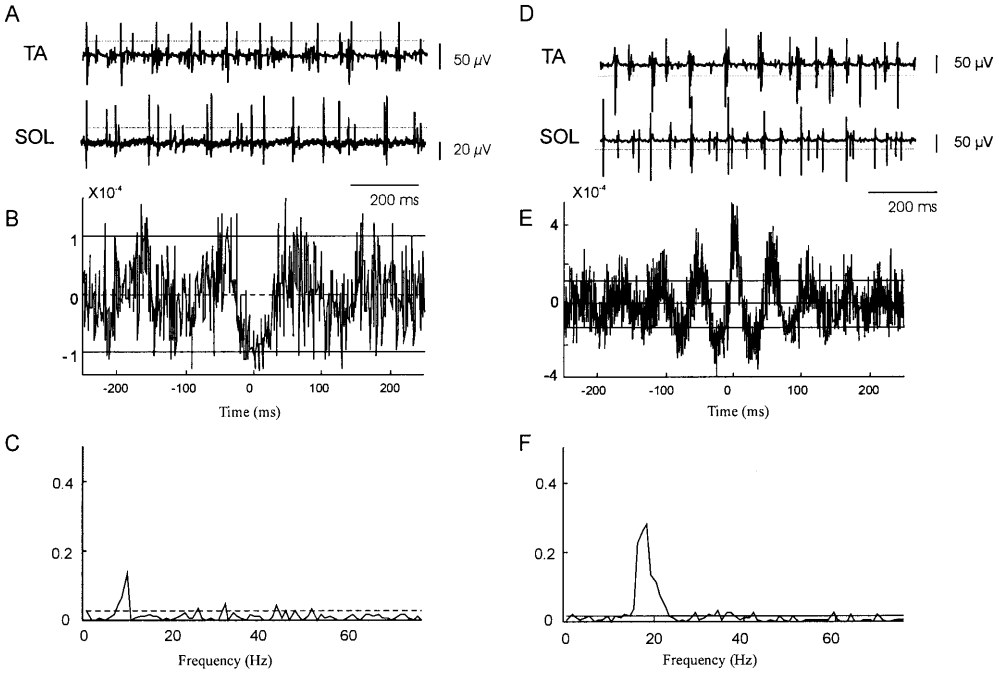


**Fig. 2A–F** Examples of coupling between antagonistic leg muscles; the data are from two different subjects. The organization of the figure is the same as in Fig. 1. The only difference is that the EMG was recorded by needle electrodes inserted into the muscles. The cumulant density function in **B** and **E** and the coherence plots in **C** and **F** are based on 1,978 and 2,134 trigger events

**Table 1** Summary of data from all investigated subjects

| | 7–12 Hz coherence | 18–35 Hz coherence | No coherence | Total |
|---|---|---|---|---|
| Surface recordings | | | | |
| Peak in correlogram | 0 (0%) | 10 (30%) | 6 (18%) | 16 (48%) |
| Trough in correlogram | 6 (18%) | 0 (0%) | 3 (9%) | 9 (27%) |
| No peak and no trough | 1 (3%) | 3 (9%) | 4 (12%) | 8 (24%) |
| Total | 7 (21%) | 13 (39%) | 13 (39%) | 33 |
| Needle recordings | | | | |
| Peak in correlogram | 0 (0%) | 4 (24%) | 2 (12%) | 6 (35%) |
| Trough in correlogram | 3 (18%) | 0 (0%) | 1 (6%) | 4 (24%) |
| No peak and no trough | 4 (24%) | 0 (0%) | 3 (18%) | 7 (41%) |
| Total | 7 (41%) | 4 (24%) | 6 (35%) | 17 |

needle electrodes inserted into the respective muscles. The data are from two subjects different from those illustrated in Fig. 1. As seen from the EMG recordings in Fig. 2A, D, it was possible to identify the different motor units contributing to the EMG signals when using the needle electrodes. Approximately three to four motor units were recorded by each of the needle electrodes. As for the surface recordings burst-like out of phase activity (Fig. 2A) as well as tonic synchronized activity (Fig. 2D) was recorded by the needle electrodes, resulting in either a central trough in the cumulant density function (Fig. 2B) associated with 7–12 Hz coherence (Fig. 2C) or a central peak (Fig. 2E) associated with 15–35 Hz coherence (Fig. 2F).

Data from all investigated subjects are summarized in Table 1. Thirty-three surface recordings were taken from 21 subjects. In 16 of the 33 recordings a central peak of synchronization was found in the cumulant density function constructed from the TA and Sol motor unit activity. The average duration of this peak was 11.3±0.6 ms (average ± SEM). This is very similar to what has been reported previously for short-term synchronization of pairs of single TA and Sol motor units (Nielsen and Kagamihara 1994). In 10 of the 16 recordings the central peak was associated with significant coherence in the 15–35 Hz frequency band, whereas no coherence was observed in the remaining 6 recordings. In 9 of the total 33 recordings a trough was observed in the cumulant density function. In six of these nine recordings the trough was associated with significant coherence in the 7–12 Hz frequency band. In these cases there were clear oscillations in the cumulant density function as shown in Fig. 1B.

In 17 experiments on 12 subjects, recordings were made from needle electrodes inserted into the TA and Sol muscles. In six of these recordings a peak was observed in the cross-correlogram, which was associated with coherence in the 15–35 Hz frequency band in four of the recordings. A trough was observed in four recordings. In three of the recordings this trough was associated with

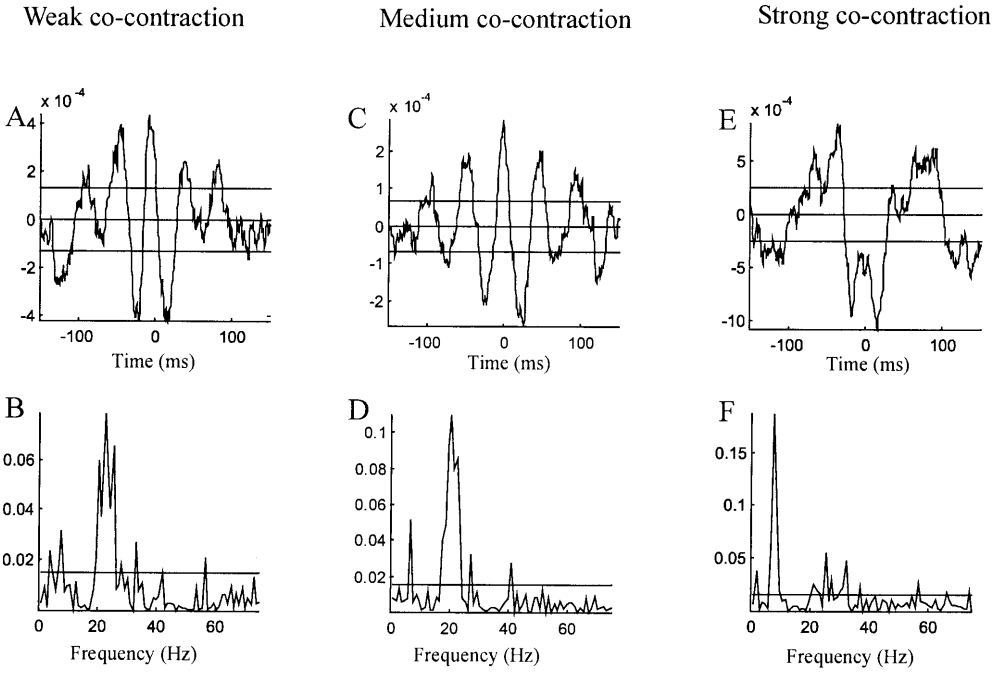


**Fig. 3** Coupling between antagonistic muscles at different levels of co-contraction; cumulant density function **(A, C, E)** and coherence **(B, D, F)** of TA and Sol EMG signals recorded during weak **(A, B)**, medium **(C, D)** and strong co-contraction **(E, F)** in a single subject. Otherwise the legend is the same as for Fig. 1

coherence in the 7–12 Hz frequency band and in these cases there were clear (out of phase) oscillations present in the cumulant density functions. The more selective needle recordings thus reduced the occurrence of significant peaks and troughs to the same extent without any significant changes in the relative proportion.

Although the needle electrodes record selectively from the respective muscles, with very little risk of cross-talk, we asked two subjects, in whom clear peaks of synchronization were observed between the antagonistic muscles, to coactivate a single TA motor unit and a single Sol motor unit. In both subjects clear central peaks and coherence around 20 Hz were observed for the antagonistic motor unit pairs.

## Influence of strength of contraction

In 28 subjects cross-correlation and coherence were estimated for three different levels of co-contraction (weak, medium and strong corresponding to <5%, 10–15% and 25–30% of maximal voluntary effort, respectively). Recordings were made from both surface and needle electrodes, but since essentially similar findings were made only the analysis of the surface EMG recordings is reported in the following. The general trend was that the stronger the level of contraction the more frequent was the occurrence of coherence in the 7–12 Hz frequency band and the central trough in the cumulant density function. Thus, during weak co-contraction only 3 out of the 28 subjects showed this pattern, whereas it was observed in 8 and 13 of the 28 subjects during medium and strong co-contraction, respectively. A tendency for the opposite relationship was observed for the 15–35 Hz coherence associated with a central synchronization peak. This pattern was observed in 11 of the 28 subjects during weak and medium co-contraction, but only in 8 during strong co-contraction. For the remaining subjects no significant peaks or troughs were observed. Figure 3 shows data from a subject in whom a central peak associated with 15–35 Hz coherence during weak and medium co-contraction switched to a central trough with 7–12 Hz coherence for strong co-contraction.

## Influence of duration of contraction

Five subjects were asked to maintain a medium to strong co-contraction for as long as possible. Cumulant density functions and coherence were then calculated in 2-min segments from the beginning to the end of the contraction. Data from one such experiment are illustrated in Fig. 4. For the first 2-min segment the pattern of a central peak with coherence in the 15–35 Hz frequency band was observed (Fig. 4A, B). However, the recording from 4–6 min after the onset of contraction showed a central trough with coherence around 10 Hz (Fig. 4C, D). Two minutes later a central peak was seen again, although not associated with any significant coherence (Fig. 4E, F) and finally shortly before the subject had to stop the contraction, the central trough with coherence around 10 Hz was seen once more. The burst-like EMG activity at around 10 Hz was visible as a clear tremor or oscillation of the foot. The subject at the same time reported that he was having difficulties maintaining a stable co-contraction. When the pattern reverted into synchronized activity the subject reported that he was once again 'in control' of the contraction.

Similar changes from central peak with 15–35 Hz coherence into central trough with 10 Hz coherence were

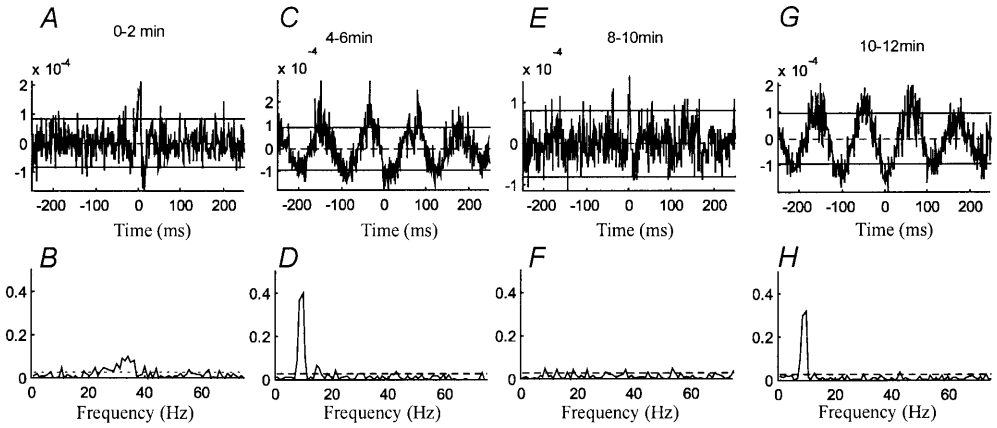


**Fig. 4** Coupling between antagonistic ankle muscles at different times during voluntary co-contraction; the subject was asked to maintain a steady co-contraction corresponding to around 20% of the maximal voluntary effort for a period of 15 min, and cross-correlation histograms **(A, C, E, G)** and coherence **(B, D, F, H)** were calculated for spike trains generated by triggering on the largest EMG activity in the surface EMG recordings from the TA and Sol muscles. Cross-correlation and coherence were calculated for periods of 2 min (**A, B** 0–2 min after onset of contraction; **C, D** 4–6 min; **E, F** 8–10 min; **G, H** 10–12 min). The calculations were based on 1,697, 1,852, 1,831 and 2,131 trigger events in **A– B**, **C– D**, **E– F** and **G– H**, respectively

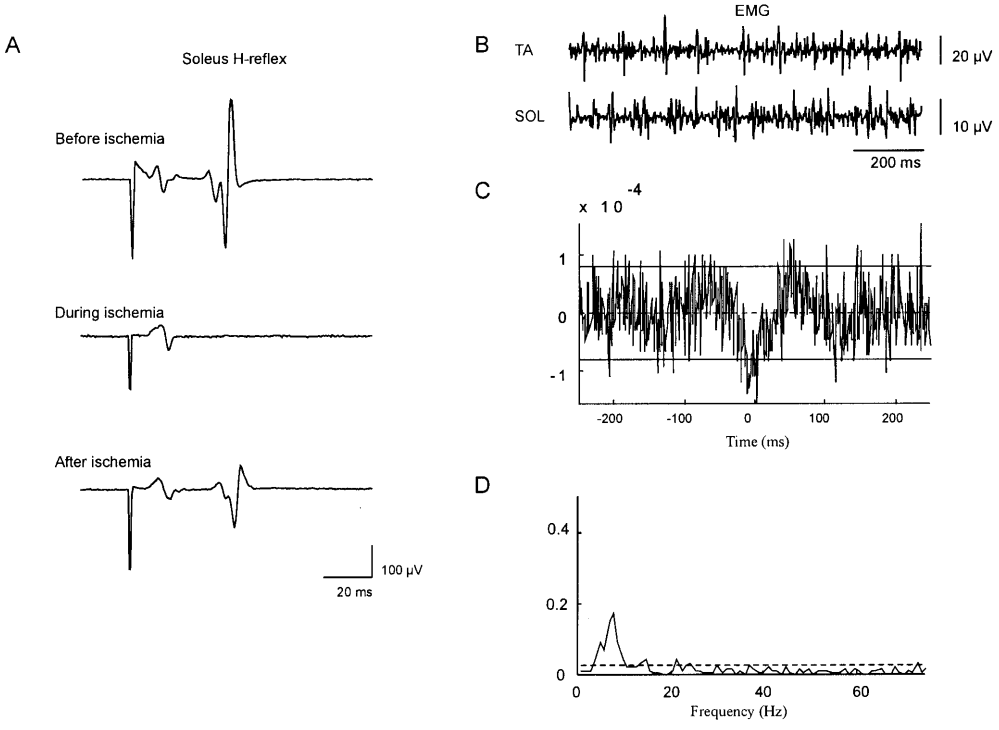


**Fig. 5** Coupling between antagonistic ankle muscles during ischaemia of the leg; **A** shows the Sol H-reflex evoked by stimulation of the tibial nerve below the cuff before (*upper trace*), 20 min after induction of ischaemia (*middle trace*) and after the cuff was released (*lower trace*). **B** shows the TA and Sol EMG activity recorded 20 min after induction of ischaemia, whereas **C** and **D** show the cross-correlation histogram and coherence plot constructed from spike trains generated from the EMG activity (1,646 trigger events). The *horizontal lines* in **C** are 95% confidence limits

observed in the other four subjects. The periods with central trough and 10 Hz coherence were not seen in the very beginning of the recordings, but became more frequent the longer the duration of the contraction. However, there was no clear association with fatigue since short-term synchronization and 15–35 Hz coherence was often observed just prior to the time that the subjects had to stop the contraction due to fatigue.

## Synchronization and coherence after block of transmission in large diameter afferents

To investigate whether the central trough associated with 10 Hz coherence could be caused by Ia afferent activity, ischaemia was induced in the leg by inflating a cuff placed around the thigh (Fig. 5). Twenty minutes after inflation of the cuff, transmission in large diameter afferents was effectively blocked, as shown by the disappearance of the Sol H-reflex evoked by stimulation of the tibial nerve distal to the cuff (Fig. 5A). However,

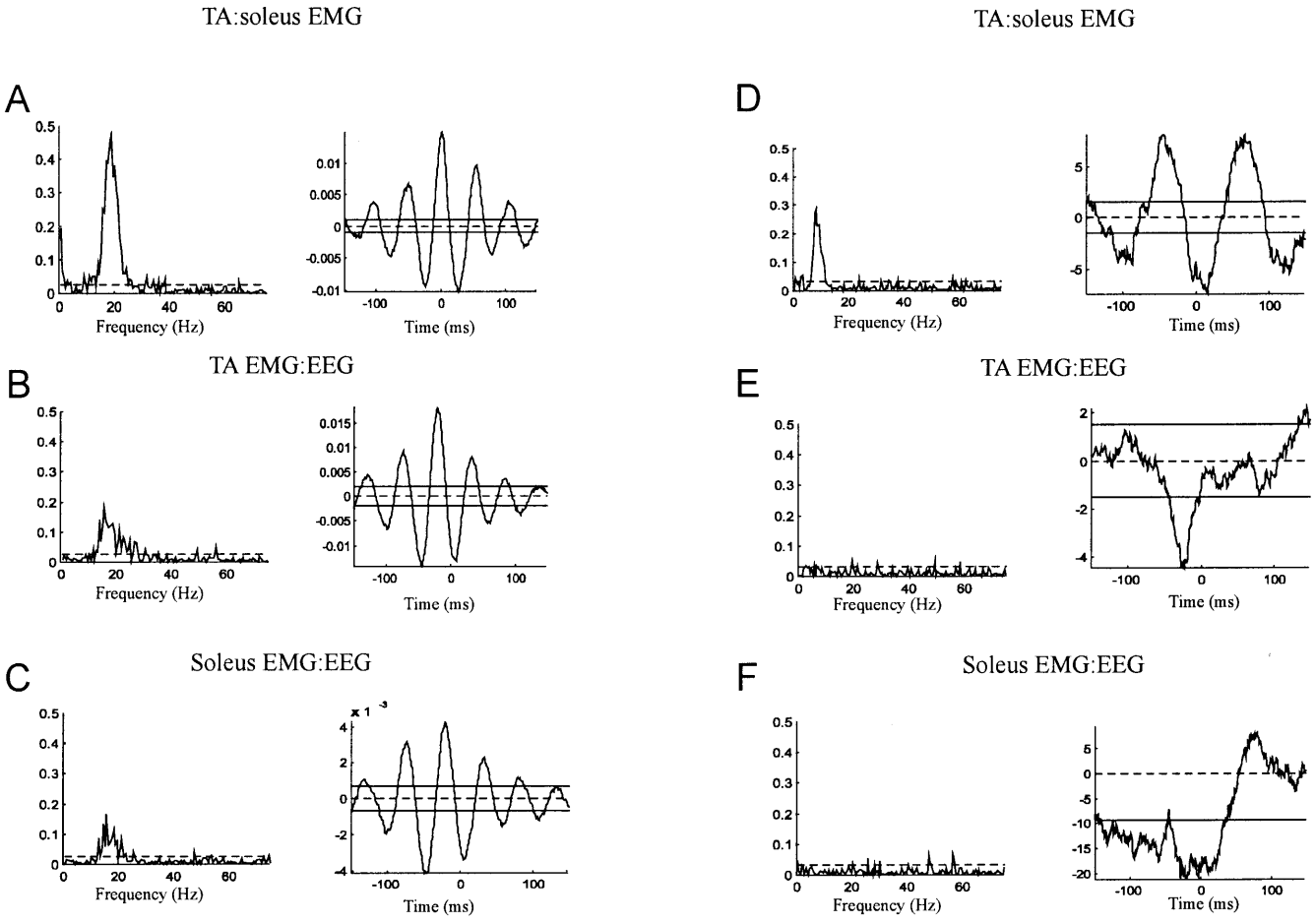


**Fig. 6** Corticomuscular coupling during antagonist co-contraction; data from two different subjects (**A–C** and **D–F**, respectively). In each of **A–F** the *left graphs* show the coherence plot whereas the *right graphs* show the cumulant density function constructed for the TA and Sol EMGs **(A, D)**, TA EMG and EEG **(B, E)** and Sol EMG and EEG **(C, F)**. The *horizontal lines* are 95% confidence limits

transmission in motor axons was still intact, since the subject was still able to activate the TA and Sol muscles (Fig. 5B). Despite the block of large diameter afferents the central trough associated with 7–12 Hz coherence was still observed (Fig. 5C, D). Similar observations were made in two other subjects. In two other subjects it was observed that coherence in the 15–35 Hz frequency band was also present during ischaemia.

### Coupling between cortex and muscle during co-contraction of TA and Sol

In 19 subjects coherence between the EEG recorded over the leg area of the motor cortex and the TA and/or Sol EMG activity was investigated. Data from two different subjects are shown in Fig. 6. The subject illustrated in Fig. 6A–C showed short-term synchronization of TA and Sol EMG activity with coherence in the 15–35 Hz frequency band (Fig. 6C). In contrast, the other illustrated subject showed a central trough with 10 Hz coherence (Fig. 6F). In the first subject coherence in the 15–35 Hz frequency band was also observed between the EEG recording and the TA EMG (Fig. 6A) and between the EEG and the Sol EMG (Fig. 6B). However, for the other subject no coherence between the EEG and either of the muscles was observed (Fig. 6D, E).

In 10 of the 19 subjects coherence in the 15–35 Hz frequency band was found between the TA and Sol motor unit activities. In all of these subjects EEG-EMG coherence was found at the same frequency. In eight subjects coherence around 10 Hz was found between the TA and Sol EMG activities. In four of the subjects no EEG-EMG coherence was found, whereas EEG-EMG coherence in the 15–35 Hz frequency band was found in three subjects. In only a single subject was coherence between the EEG and EMG in the 7–12 Hz frequency band found.

## Discussion

The main finding in the present study is that antagonistic ankle muscles may be coupled either in phase or out of phase during tonic isometric co-contraction. In-phase coupling (central peak in the cumulant density function)

tended to be associated with coherence in the 15–35 Hz frequency band, whereas out-of-phase coupling (central trough in the cumulant density function with secondary peaks at 100-ms lags) was associated with 7–12 Hz coherence.

## Is the synchronization of the antagonistic muscles explained by cross-talk?

One concern in this kind of experiment is that the short-term synchronization may be caused by cross-talk between the recording electrodes. This is especially a worry since other studies have failed to demonstrate any synchronization between antagonistic muscles (Gibbs et al. 1994; Farmer et al. 1997; see, however, Nielsen and Kagamihara 1994). We attempted to reduce the likelihood of cross-talk by setting a triggering threshold, which ensured that the spike trains from which the cumulant density functions were generated, only reflected the largest amplitude EMG activity in each of the two recordings. We also confirmed that similar findings were obtained when recording the EMG activity by needle electrodes inserted into the muscles. This more selective recording of the EMG activities did reduce the incidence of short-term synchronization between the EMG activities, but the incidence of central troughs was reduced to the same extent. This suggests that the lower incidence of significant synchronization (and central troughs) mainly reflected the lower number of motor units contributing to the recorded EMG activities and hence the lower sensitivity in detecting physiological coupling between the signals. The observation of distinct peaks of coherence also speaks against a significant contribution of cross-talk. If cross-talk had played a significant role, coherence over a broad range of frequencies would have been observed. The observation that the pattern of coupling (from central peak with 15–35 Hz coherence to trough with 7–12 Hz coherence) could change within the same experimental session (cf. Figs. 3, 4) also suggests that cross-talk was not responsible for our observations. Furthermore, if cross-talk had been responsible for the observed short-term synchronization, much narrower and larger peaks than what we observed would have been expected. Finally, in two subjects recordings were made from pairs of single motor units recorded from the two antagonistic muscles (as in the study by Nielsen and Kagamihara 1994). In both subjects coherence around 20 Hz and clear short-term synchrony was observed for these pairs of motor units. Since the discharge of each motor unit was checked throughout each of the experiments and it was ensured that the motor units were different and belonged to different muscles, cross-talk could not explain the coupling at least in these cases.

## When are central peaks and central troughs seen?

Central peaks and central troughs could be observed in the same subject on different occasions and even within the same recording sessions only a few minutes apart. In some trials we observed that the pattern changed from synchronization with 15–35 Hz coherence to central trough with 7–12 Hz coherence within the same 2-min recording. Switching from one pattern to the other and back again in some cases may have happened several times within the same 2-min period. In some recordings we did have the impression of a central trough with a superimposed central peak (cf. Fig. 3). It is thus possible that the two patterns may coexist, but our data would rather suggest that one or the other dominates at different times and that the switch from one pattern to the other may take place within a rather short time span. Nielsen and Kagamihara (1994) observed that different pairs of motor units would show short-term synchronization, whereas other pairs showed central troughs. In that study it was suggested that this reflected a difference in the synaptic input to the different motor unit pairs. However, since the motor unit pairs with the different coupling patterns were not recorded at the same time in the study by Nielsen and Kagamihara (1994), we would suggest, based on the present experiments, that their observations were rather explained by changes in the coupling pattern at different times of the recordings.

We made several attempts in the present study to reveal whether one or the other pattern was seen more often under certain conditions. We did see a tendency for the out-of-phase activity associated with 7–12 Hz coherence to be more common for long-lasting contractions, but this was not consistent and even when the contractions had lasted more than 15 min and the subjects had developed significant muscle fatigue, short-term synchronization with 15–35 Hz coherence could still be observed on some occasions. Another factor, which may influence the level of synchrony and coherence, is the attention of the subject (Schmied et al. 2000, which probably varies considerably during longer lasting contractions. However, we had no way of evaluating the level of attention in the present study.

Central troughs with 7–12 Hz coherence also tended to be more common for strong than for weak contractions, but this was also rather inconsistent. The subjects also were not able to produce any of the two patterns voluntarily. When asked to make fast rhythmic alternating contractions of the ankle extensors and flexors, broad (trough lasting 200 ms) out-of-phase coupling (secondary peaks at lags of more than 200 ms) with coherence at no more than 5 Hz was observed.

Some subjects reported that they could perceive some kind of change at the time when the subsequent analysis revealed a switch between the two patterns. Generally, the subjects reported that they found it easy to control the level of co-contraction when short-term synchronization was observed, whereas they felt that they were 'losing control of the contraction' when the anti-synchronization

pattern was observed. We have no idea what this signifies in terms of the central control of the muscle activity during co-contraction when the two patterns were observed, but since it was reported spontaneously by the subjects and seemed consistent, we believe that it may give a clue for future experiments addressing this question.

## Which mechanisms are responsible for 15–35 Hz coherence and synchronization of antagonistic muscles?

Short-term synchronization of synergistic and homonymous motor unit activity similar to that observed in the present study has been suggested to be caused by common synaptic input from branches of last order neurones to the spinal motoneurones (Datta and Stephens 1990; Datta et al. 1991), although a contribution from synchronized discharges of separate last order inputs cannot be fully disregarded (Vaughan and Kirkwood 1997). Indeed, in the present study the central peaks observed in the cumulant density function were often of a similar size to the secondary peaks observed at 50 ms lag from time zero and we therefore believe that they were generated mainly by the synchronized discharges of separate inputs to the antagonistic motoneurones. If significant synchronization caused by common inputs from last order neurones had contributed, larger central peaks would have been expected.

Short-term synchronization has been observed in deafferented patients (Farmer et al. 1993a), but is reportedly absent or greatly reduced in patients with lesions of the corticospinal tract (Farmer et al. 1993a, 1993b). Furthermore, short-term synchronization is generally associated with coherence in the 15–35 Hz frequency band as also observed in the present study (see also Farmer et al. 1993a; Halliday et al. 1999), and coupling between cortical EEG (or MEG) activity and EMG activity in this frequency band has now been reported in a number of studies (Conway et al. 1995; Salenius et al. 1997; Marsden et al. 2000). When short-term synchronization with 15–35 Hz coherence was observed in the present study between the antagonistic EMG recordings, we also observed coherence between the EEG and EMG activities in a similar frequency band. When there was no coherence in this frequency band between EMG recordings, it was also not observed between the EEG and EMG recordings. This suggests that the short-term synchronization with 15–35 Hz coherence is caused by rhythmic discharges in the corticospinal neurones projecting to the spinal motoneurones, as already suggested in several previous studies (see Farmer et al. 1997 for review).

In the study by Farmer et al. (1998) synchronization of antagonistic wrist muscles was only observed in patients with myoclonus, but not in healthy subjects. It is unclear whether our data from leg muscles can be compared to their data from arm muscles, but our observation of synchronization of antagonistic muscles in a significant number of healthy subjects does suggest that this is not only an abnormal phenomenon. It seems likely that such synchronization is more frequent in myoclonic patients as a consequence of accentuated activity in pathways also present in healthy subjects rather than the existence of pathways, which are only present in the patients.

## Which mechanisms are responsible for the 7–12 Hz coherence and the anti-synchronization of antagonistic muscles?

It seems likely that the central trough with 7–12 Hz coherence is related to physiological tremor, which has been shown to consist of a peripheral mechanical oscillation at the limb's resonance frequency as well as a 'central component' in the 6–15 Hz frequency band (Raethjen et al. 2002). A coherence of 7–12 Hz is also observed for discharges of homonymous and synergistic motor unit discharges, but is generally thought not to be associated with short-term synchrony of these motor units (Farmer et al. 1993a). We find it likely that the input system, which is responsible for the 7–12 Hz coherence of homonymous muscles, is also responsible for the 7–12 Hz coherence and the troughs in the cumulant density function for the antagonistic muscle pairs. We consider that the system responsible for this coherence is not sufficiently strong or does not branch sufficiently in the homonymous and synergistic motoneuronal pools in order to contribute significantly to the short-term synchrony observed for such muscles, but, nevertheless, presumably via spinal inhibitory interneurones diminish the tendency of antagonistic motor units to discharge at exactly the same time, but rather out of phase (cf. the oscillations at 100-ms lags in Fig. 1B). One input system which fulfills this criterion is the Ia afferents. They have been shown likely not to discharge very synchronously in human subjects (Gandevia et al. 1986) and they activate spinal inhibitory interneurones, which project to antagonists. Furthermore, Ia afferents often discharge around 10 Hz during similar tonic isometric contractions to those investigated in the present study (Vallbo 1970; Nielsen et al. 1994). However, although a contribution from Ia afferents cannot be ruled out, other mechanisms must be involved, since we observed the central troughs with 7–12 Hz coherence when transmission in Ia afferents was completely blocked by ischaemia. Other studies have similarly suggested that Ia afferents are not involved in the generation of 7–12 Hz coherence of synergistic and homonymous motor unit activity (Vallbo and Wessberg 1993; Wessberg and Vallbo 1995, 1996; McAuley et al. 1997).

The central component of physiological tremor has mainly been attributed to subcortical circuits and in particular the inferior olive has been thought to play a role (Allum et al. 1978; Llinas and Pare 1995). Recently, corticomuscular coherence at frequencies around 10 Hz has also been found in subjects with physiological and essential tremor and it has therefore been suggested that

the motor cortex may contribute to the generation of this rhythmicity (Hellwig et al. 2001; Raethjen et al. 2002; see, however, Halliday et al. 2000). In the present study we did observe coherence between EEG and EMG in a single subject in the 7–12 Hz frequency band, but this was rare as compared to the much more consistent coherence in the 15–35 Hz frequency band. The reason for this may be that the motor cortex is not as intimately involved in the generation of the 7–12 Hz activity as it is in the 15–35 Hz activity. If subcortical structures such as the inferior olive (Llinas and Pare 1995) are primarily responsible for the generation of the rhythm and only part of the rhythmic activity is transmitted by the corticospinal tract to the muscles, it may be difficult to detect in the coherence between EEG and EMG in healthy subjects. However, when the rhythmicity is enhanced as in essential tremor (Hellwig et al. 2001) or when recording the cortical activity by the more sensitive electrocorticogram technique (Raethjen et al. 2002), it may be demonstrated.

### Functional considerations

Our observations suggest that at least two different systems/pathways are responsible for the activation of antagonistic muscles during co-contraction. The system responsible for the in-phase coupling of the muscles would have the advantage that the two muscles may be coactivated without any opposing inhibition. This may be an advantage in tasks where co-contraction of antagonistic muscles is necessary in order to increase the stiffness of the ankle joint. The system responsible for the troughs would seem more adequate for the control of extension/flexion movements, where it is essential that the antagonists are relaxed during agonist contraction. The reason why both systems, nevertheless, seem to contribute to the activation of the muscles during co-contraction is unclear. The subjects were required to co-contract the antagonist muscles, while sitting down with their foot attached to a plate. This is a somewhat artificial situation very unlike the situations where co-contraction around the ankle joint would normally occur (balancing on a platform, walking on a beam). In this artificial situation it may be that it is not possible to maintain the most adequate system switched on continuously. Alternatively, and probably more likely, it may be that co-contraction as well as extension/flexion movements are generated by a balance between the two activation systems. From biomechanical considerations such an organization has previously been suggested to exist (Levin et al. 1992).

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