JPn Neuroscience

The Journal of Physiology

J Physiol 590.10 (2012) pp 2443–2452

The motor cortex drives the muscles during walking in human subjects

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Key points

- It is often assumed that automatic movements such as walking require little conscious attention and it has therefore been argued that these movements require little cortical control.
- In humans, however, the gait function is often heavily impaired or completely lost following cortical lesions such as stroke.
- In this study we investigated synchrony between cortical signals recorded with electroencephalography (EEG) and electromyographic signals (EMG activity) recorded from the tibialis anterior muscle (TA) during walking.
- We found evidence of synchrony in the frequency domain (coherence) between the primary motor cortex and the TA muscle indicating a cortical involvement in human gait function.
- This finding underpins the importance of restoration of the activity and connectivity between the motor cortex and the spinal cord in the recovery of gait function in patients with damage of the central nervous system.

Abstract Indirect evidence that the motor cortex and the corticospinal tract contribute to the control of walking in human subjects has been provided in previous studies. In the present study we used coherence analysis of the coupling between EEG and EMG from active leg muscles during human walking to address if activity arising in the motor cortex contributes to the muscle activity during gait. Nine healthy human subjects walked on a treadmill at a speed of 3.5–4 km h^{-1}. Seven of the subjects in addition walked at a speed of 1 km h^{-1}. Significant coupling between EEG recordings over the leg motor area and EMG from the anterior tibial muscle was found in the frequency band 24–40 Hz prior to heel strike during the swing phase of walking. This signifies that rhythmic cortical activity in the 24–40 Hz frequency band is transmitted via the corticospinal tract to the active muscles during walking. These findings demonstrate that the motor cortex and corticospinal tract contribute directly to the muscle activity observed in steady-state treadmill walking.

(Received 3 January 2012; accepted after revision 27 February 2012; first published online 5 March 2012)

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Abbreviations EEG, electroencephalography; EMG, electromyography; MEG, magnetoencephalography; NIRS, near-infrared spectroscopy; SPECT, single-photon emission tomography; TA, tibialis anterior; TMS, transcranial magnetic stimulation.

DOI: 10.1113/jphysiol.2012.227397

Introduction

It is often assumed that cortical activity during a movement implies deliberate conscious control, whereas subcortical and spinal networks are responsible for automatic movements that require little conscious attention. From this point of view, undemanding steady-state walking would be expected to involve little cortical activity and this is indeed also what has been seen in cats (Armstrong, 1988). Significant cortical activity is only observed when the cat walks in a challenging environment or when forced to step over obstacles (Armstrong, 1988; Armstrong & Marple-Horvat, 1996; Drew *et al.* 2004, 2008). The motor cortex in the cat and other animals has therefore been suggested to play only a facultative role during walking (Armstrong, 1988)

However, an increasing number of electrophysiological and imaging studies have provided evidence that the motor cortex may play a more significant role during undemanding steady-state walking in humans. Using imaging techniques such as single-photon emission tomography (SPECT) and near-infrared spectroscopy (NIRS) significant activation is thus observed in the sensorimotor cortex during both real and imagined walking (Fukuyama *et al.* 1997; Miyai *et al.* 2001). Experiments using transcranial magnetic stimulation (TMS) have also demonstrated that the corticospinal tract is easily excited throughout the gait cycle (Schubert *et al.* 1997; Petersen *et al.* 1998, 2001; Capaday *et al.* 1999). Petersen *et al.* (2001) also demonstrated that weak TMS may depress the EMG activity from the active muscles during walking and argued that this depression was caused by removal of the corticospinal contribution to the ongoing EMG activity.

All of this evidence is indirect and/or confounded by the necessity of applying external perturbing stimuli. More conclusive evidence would require the application of methodology similar to that used in animal experiments, where functional connectivity between recordings of individual or populations of corticospinal cells and motor output can be demonstrated during the performance of motor behaviours via techniques such as spike-triggered averaging. EMG averages constructed from the discharges of corticospinal neurones in behaving animals not only reveal the presence of anatomical projections, but can also illustrate the extent to which the corticospinal input contributes to the generation of the motor behaviour being studied (Fetz & Cheney, 1987; Lemon, 1993). This approach is evidently not possible in humans, but time (cross-correlation) and frequency (coherence) domain techniques for the detection of coupling between signals provides a convenient analytical framework from which functional coupling between localised cortical activity (measured by MEG or EEG) and motor output (EMG) can be identified in human subjects (Halliday *et al.* 1995). This approach has been successfully used to reveal coherent activity between localised recordings from the motor cortex and the EMG generated during maintained voluntary motor tasks (Conway *et al.* 1995; Mima *et al.* 2000; Hansen *et al.* 2002; Mendez-Balbuena *et al.* 2011; Omlor *et al.* 2011)

In the present study we have applied these techniques during human treadmill walking. We report that EEG and EMG from the active leg muscles during treadmill walking are coupled, suggesting that the corticospinal tract contributes to the ongoing muscle activity during periods of steady-state treadmill walking in human subjects.

Methods

Nine healthy human subjects (mean age, 23.4 ± 4.1 years; 4 men and 5 women) participated in the study. The local ethics committee approved all experimental protocols (J. No. KF 01-055/98) and all subjects provided informed written consent prior to participation.

Experimental methods

Paired bipolar surface EMG recordings (Ag–AgCl electrodes; 1 cm^2 recording area, 2 cm between poles) were made from the belly of the right anterior tibial (TA) muscle. The EMG signals were amplified (5–10 k) and filtered (high pass: 1 Hz; low-pass: 1000 Hz). Concurrent monopolar EEG recordings were made from Ag–AgCl electrodes placed on the scalp using a 28-electrode cap (Quick-Cap, Neuromedical Supplies) which positioned electrodes in accordance with the 10-20 international standard. Combined earlobes were used as reference. The ground electrode was situated in the EEG cap between the Fz and FCz electrode. The EEG signals were amplified (Neuroscan, Synamp II) and filtered (1–500 Hz). To obtain the time of heel strike during treadmill walking a heel switch was attached to the leg that the TA muscle EMG signals were recorded from. All the subjects were experienced treadmill users and walked at their preferred walking speed between 3.5 and 4 km h^{-1} (normal walking). In seven of the subjects additional experiments with a walking speed of 1 km h^{-1} (slow walking) and a static dorsiflexion were performed. Five minute epochs of continuous treadmill walking and 2 min of static contraction were sampled at 2000 Hz and stored for off-line analysis. To assist in the collection of artifact-free EEG, subjects were requested to relax face and jaw muscles and to minimize eye blinks and swallows during data capture periods. The subjects were also requested not to swing the arms actively during walking, but let them hang as relaxed as possible.

Data pre-processing and artefact rejection

In order to speed up computations the data were down-sampled off-line to a sampling rate of 500 Hz. Channels with high impedance, significant drift or excessive 50 Hz noise were removed using visual inspection of the EEG signals. This was performed in the EEG-LAB scroll viewer (Delorme & Makeig, 2004). The remaining EEG channels were re-referenced to a common average reference. A common average reference in the post-processing steps has been shown to increase the magnitude of EEG–EMG, but also to alter the scalp distribution of this so that more frontal areas may show significant coupling with the EMG activity possibly due to volume conduction from the sensory motor area (Mima & Hallett, 1999). For the latter aspect the present result for the ∼30 Hz coherence scalp maps showed a localization corresponding well to the sensorimotor cortex. Indeed, in some cases for coherence in the lower frequency band (8–12 Hz) some frontal coherence was observed. However, in most cases the Cz or CPz electrode showed the largest peak values for this frequency band and hence do not alter the results of the present experiments.

Independent component analysis (ICA) was performed on the complete time series using the runica algoritm provided in EEG-LAB (see Fig. 1). The complete dataset was then split into segments of interest of 800 ms prior to and 200 ms after heel strike, and components that showed spatial and temporal characteristics of eye blinks and facial muscles were projected out of the data (Jung *et al.* 2000). Two hundred and fifty epochs were used for each subject for normal walking and 150 epochs for slow walking. In the case of static muscle activation a complete time series of 2 min was used.

Previously removed bad channels were added back into the data by means of spherical interpolation on the time series of interest (epochs). None of the files contained more than two bad EEG channels.

Data analysis

Spectral analysis methods were used to investigate coupling between EEG and EMG signals in the frequency domain. The signals, after full wave rectification in the case of the EMGs, are assumed to be realisations of stationary zero mean time series, which we denote by x and y. Power spectra are estimated using a periodogram approach, where the discrete Fourier transforms are constructed from sections of data taken at a fixed offset time with respect to a trigger point (heel strike) in each step cycle. Estimates of the spectra are constructed by averaging periodograms across all step cycles. We use $f_{xx}(\lambda)$ and $f_{yy}(\lambda)$ to represent the Fourier transforms of processes x, in this case the TA EMG and y, in this case the EEG signal, at frequency λ. The cross spectrum between x and y is denoted by $f_{xy}(\lambda)$, and is estimated in a similar manner. Coherency, which is complex number (eqn (1)), is an estimate of the linear relationship of the two signals at a given frequency λ and defined as the normalised cross-spectrum

$$R_{xy}(\lambda) = \frac{f_{xy}(\lambda)}{\sqrt{f_{xx}(\lambda) f_{yy}(\lambda)}} \tag{1}$$

Coherence can then be defined as the squared modulus of the coherency (eqn (2))

$$|R_{xy}(\lambda)|^2 = \frac{|f_{xy}(\lambda)|^2}{f_{xx}(\lambda) f_{yy}(\lambda)} \tag{2}$$

Furthermore, we are interested in the statistics of the coherency estimates. For the Z-transformed magnitude of coherency ($\arctan\mathrm{h}(|R_{xy}(\lambda)|)$ the variance can be estimated using $1/2L$ where L denotes the number of segments (Halliday *et al.* 1995). In the case of the complex coherency estimate, variances for the real (Re) and imaginary (Im) parts can be estimated separately in the following way as described in details by Nolte *et al.* (2004)

$$\mathrm{Var}(\mathrm{Im}(R_{xy}(\lambda))) = \frac{1}{2L}(g(R_{xy}(\lambda))\sin^2(\Phi) + \cos^2(\Phi)) \tag{3}$$

$$\mathrm{Var}(\mathrm{Re}(R_{xy}(\lambda))) = \frac{1}{2L}(g(R_{xy}(\lambda))\cos^2(\Phi) + \sin^2(\Phi)) \tag{4}$$

where L is the number of segments, Φ is the phase and g is given by

$$g(x) = (1 - |x|^2)\frac{\arctan\mathrm{h}^2(|x|)}{|x|^2} \tag{5}$$

Finally, a P value can be calculated from the number of standard deviations the estimate differs from zero. A significance level of $\alpha < 0.05$ was chosen.

Since signals obtained from the TA muscle were compared with respect to all scalp electrodes, a Bonferroni correction was applied to correct for multiple comparisons. In this case α was set to $0.05/n$, where n was the number of comparisons made. An average coherency across subject was taken for the Z-transformed estimates and then transformed back with the inverse transform.

The time–frequency plots were computed using a sliding window of 250 ms with an increment of 50 ms through the entire section of interest. The offset values given in the Results represent the beginning of each window.

For the head plots, a window length of 500 ms was used corresponding to the period of EMG activity prior to the heel strike (see Fig. 1).

Recent studies (Hansen *et al.* 2002; Hansen & Nielsen, 2004; Petersen *et al.* 2010) have reported that common

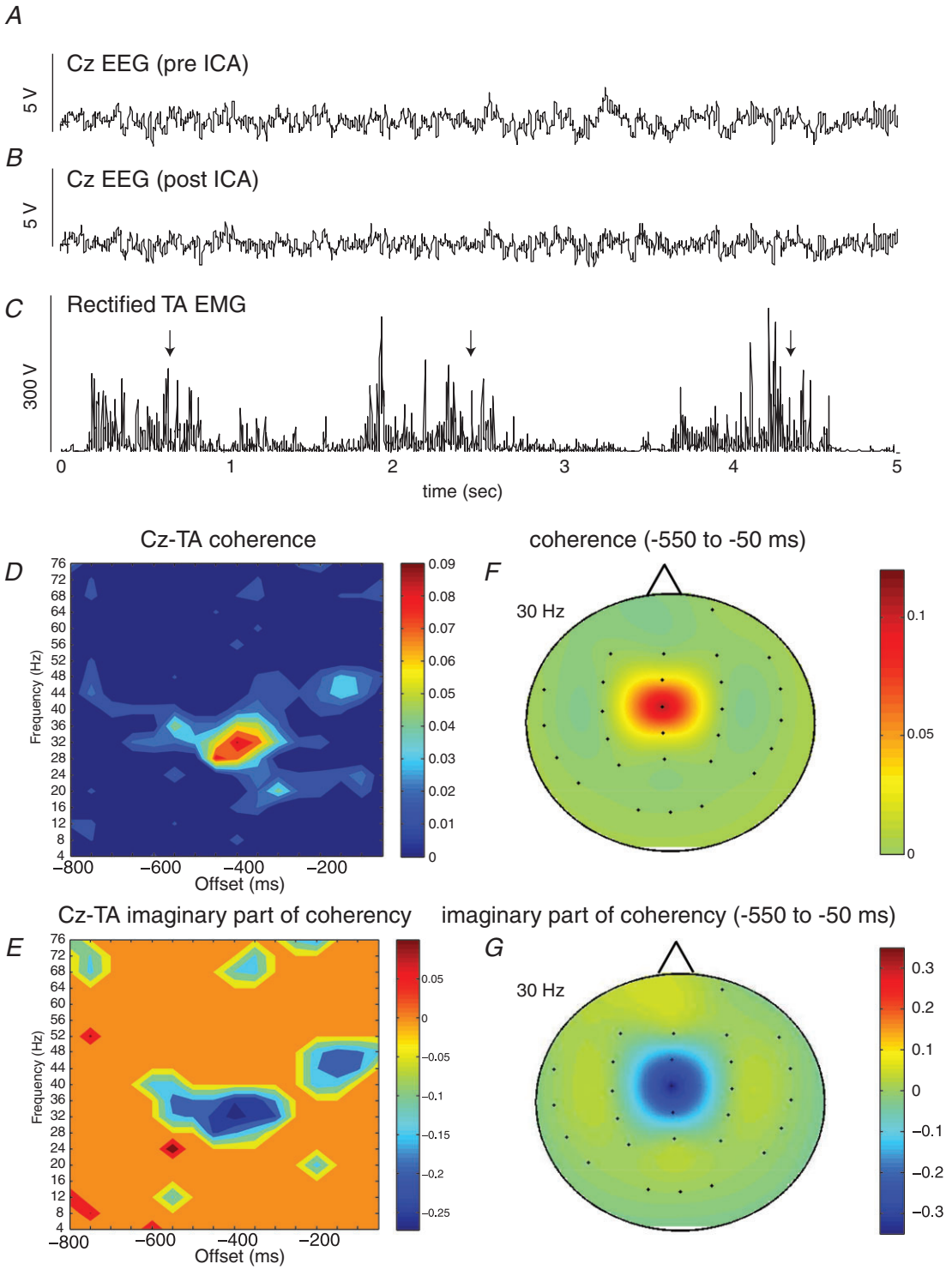

Figure 1. Data from a single subject during 5 min of slow treadmill walking at 1 km h^{-1}
Cz EEG before (*A*) and after removal of unwanted components (*B*) and raw EMG (*C*) activity from three steps. The black arrows indicate the time of heel strike. The EMG activity had an onset around 600 ms prior to the heel strike and lasted until around 200 ms after the heel strike. Significant estimates of coherence (*D*) and the imaginary part of coherency (*E*) between the Cz EEG and TA muscle EMG were observed in the ∼24–40 Hz frequency band for offset values between –600 and –100 ms with a peak at 30 Hz between –500 and –300 ms. Coherence (*F*) and the imaginary part of coherency (*G*) between all 28 EEG electrodes and the TA EMG at 30 Hz taken from segments of data from –550 to –50 ms. Significant coupling between the TA muscle EMG and the leg area of the motor cortex was observed. ICA, independent component analysis.

input (EMG–EMG coherence) to TA motor units is present during human walking. Compared with EEG signals, the EMG signals are less noisy and the source of activity is known. For the present dataset, we chose to display both coherence and the imaginary part of coherency. The latter estimate essentially represents the phase between the two signals, in this particular case between the EEG and the EMG signals and we included this measure to confirm that the observed coupling between cortex EEG and the TA EMG was real and not due to volume conduction of noise that would spread to EEG and EMG electrodes simultaneously. We hypothesised that the imaginary part of the coherency estimate would be present due to the conduction time from the cortex to the muscle. It has been argued that this approach may not be suitable for estimation of true interactions between different EEG electrodes (Stam *et al.* 2007) since the phase difference between signals may be very small due to short conduction distances; however, the delay introduced by transmission through the corticospinal pathways should be larger and hence would lead to imaginary parts of the complex coherency estimate.

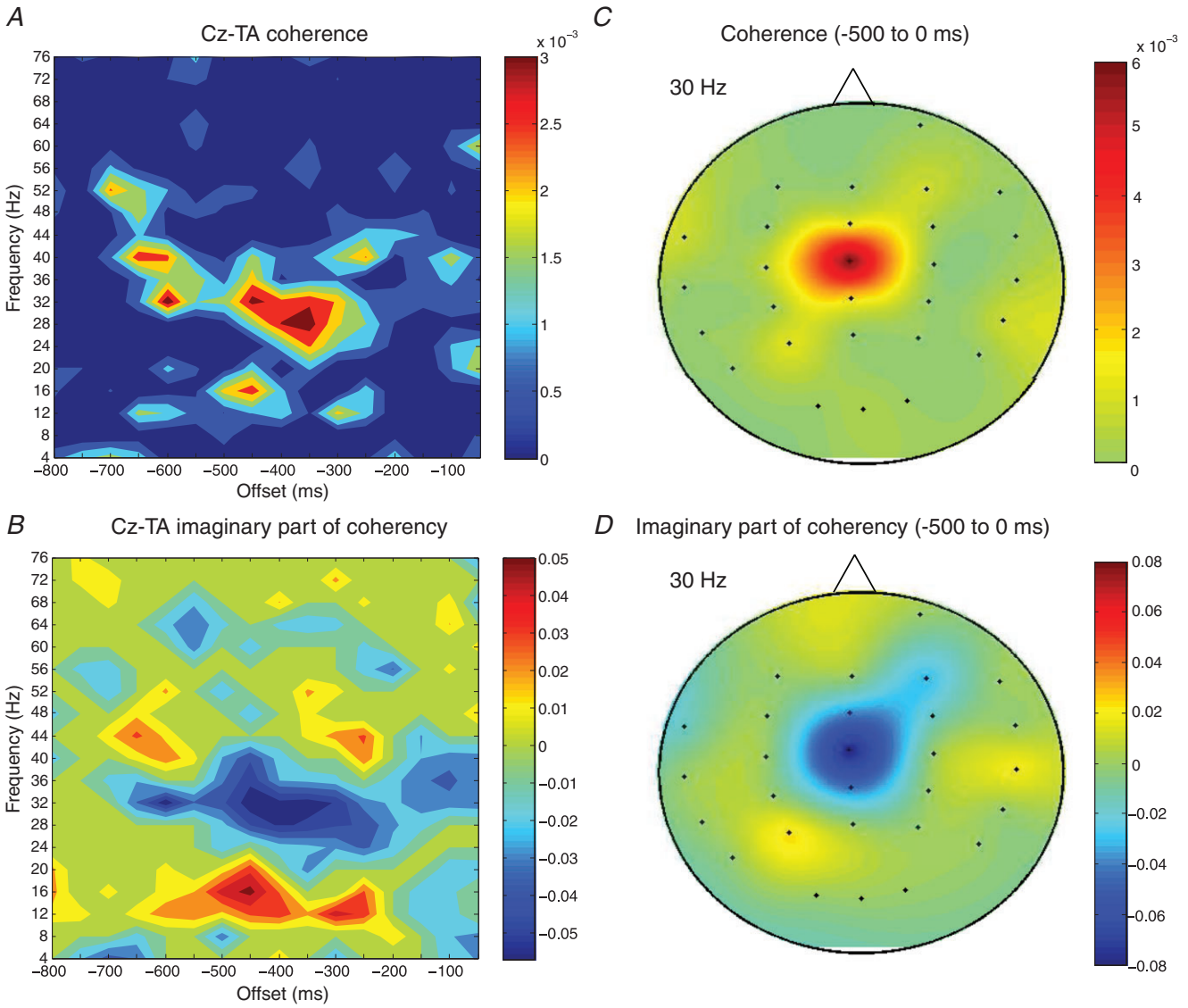

Figure 2. Pooled data from nine subjects during normal walking (3.5–4 km h^{-1})
Time–frequency analysis of averaged coherence (*A*) and imaginary part of coherency between the Cz EEG and the TA muscle EMG (*B*). Significant coupling was observed in the ∼24–40 Hz frequency domain for offsets between –700 ms and –200 ms prior to the heel strike. Average coherence (*C*) and the imaginary part of coherency (*D*) at 30 Hz between all 28 EEG electrodes and the TA EMG taken from segments of data from –500 and 0 ms where peak values of coupling were observed.

Table 1. Individual magnitudes of coherence and the electrode displaying maximal coupling with the TA EMG in the 8–12 Hz and 24–40 Hz frequency band for normal and slow walking and in the 15–30 Hz frequency band for static muscle activation. (NS=not siginificant)

	Normal walking		Slow walking		Static contraction
Subject	8–12 Hz peak frequency and coherence (electrode)	24–40 Hz peak frequency and coherence (electrode)	8–12 Hz peak frequency and coherence (electrode)	24–40 Hz peak frequency and coherence (electrode)	15–30 Hz peak frequency and coherence (electrode)
1	8 Hz, 0.026 (C3)	40 Hz, 0.038 (Cz)	12 Hz, 0.016 (CPz)	32 Hz, 0.018 (Cz)	20 Hz, 0.10 (Cz)
2	8 Hz, 0.036 (Cz)	40 Hz, 0.082 (Cz)	8 Hz, 0.018 (Cz)	40 Hz, 0.060 (Cz)	29 Hz, 0.35 (Cz)
3	12 Hz, 0.020 (Cz)	32 Hz, 0.103 (Cz)	8 Hz, 0.020 (Cpz)	32 Hz, 0.095 (Cz)	19 Hz, 0.25 (Cz)
4	12 Hz, 0.054 (Cz)	36 Hz, 0.019 (Cz)	12 Hz, 0.020 (Cz)	30 Hz, 0.040 (Fcz)	15 Hz, 0.02 (Cz)
5	8 Hz, 0.130 (CPz)	32 Hz, 0.028 (FCz)	12 Hz, 0.023 (Cz)	36 Hz, 0.060 (Cz)	19 Hz, 0.18 (Cz)
6	8 Hz, 0.024 (CPz)	28 Hz, 0.054 (Cz)	8 Hz, 0.026 (Cz)	28 Hz, 0.020 (Cz)	25 Hz, 0.10 (Cz)
7	NS	32 Hz, 0.042 (Cz)	NS	34 Hz, 0.040 (Cz)	25 Hz, 0.05 (Cz)
8	NS	24 Hz, 0.033 (Cz)	—	—	—
9	12 Hz, 0.050 (CPz)	36 Hz, 0.084 (CPz)	—	—	—

Results

Figure 1 shows data from a single subject during 5 s of steady-state treadmill walking at 1 km h^{-1}. Cz EEG from one subject during walking is shown before (Fig. 1*A*) and after (Fig. 1*B*) removal of unwanted components (independent component analysis, ICA). Rectified EMG collected from the TA muscle is shown in Fig. 1*C*.

The EMG activity in the TA muscle had an onset of approximately 600 ms prior to heel strike and lasted until approximately 200 ms after heel strike (arrows). Time–frequency plots of coherence and the imaginary part of coherency are given in Fig. 1*D* and *E*. Significant coherence (Fig. 1*D*) between the TA muscle EMG and the Cz EEG electrode in the ∼24–40 Hz frequency band was observed for offsets between –600 ms and –100 ms prior to the heel strike, peaking between –500 ms and –300 ms. The imaginary part of coherency showed a similar result (Fig. 1*E*). In Fig. 1*F* and *G* the coherence and the imaginary part of coherency at 30 Hz between the TA muscle EMG and all 28 EEG electrodes were plotted. This was done for the entire segment of –550 ms to –50 ms prior to the heel strike. This corresponded to the interval before the heel strike with the largest coherence estimates and maximal EMG activity. Significant coupling was observed between the TA EMG and the leg area of the motor cortex.

Figure 2 shows pooled data from the nine subjects walking at their own preferred walking speed between 3.5 and 4.0 km h^{-1}. Individual peak coherence magnitudes and the electrode that showed the largest magnitude of coherence are given in Table 1. The pooled data were in line with the results from the single subject data. Significant coherence (Fig. 2*A*) and imaginary part of coherency (Fig. 2*B*) could be observed in the 24–40 Hz frequency band for offsets between –700 ms and –200 ms prior to the heel strike. Furthermore, significant coupling was also observed in a lower frequency band from 8 to 12 Hz with distinct peaks in early, mid and late swing phase. In Fig. 2*C* and *D* the coherence and imaginary part of coherency at 30 Hz between the TA muscle EMG and all 28 EEG electrodes is plotted. Maximal coupling was observed in the interval from –500 to 0 ms and this interval was therefore used for this plot. The results confirm those from the single subject that coupling between the TA EMG and EEG was confined to recordings above the leg motor area. The ∼10 Hz coherence (not shown) showed a similar localization with additional spread toward more frontal areas.

In Fig. 3 the results from walking at 1 km h^{-1} are shown. Peak coherence magnitudes can be found in Table 1. In line with the previous results, we found significant coupling between the TA muscle EMG and the Cz EEG in the ∼24–40 Hz frequency band with a peak in the segments from –500 to –400 ms prior to heel strike. Note that the colour bar scaling is different in *A*–*D* in this figure compared with Fig. 2*A*–*D*. In the 8–12 Hz frequency band small, but significant peaks were observed in early and late swing phase.

In Fig. 4 shows the pooled results from 2 min of static dorsi-flexion. Figure 4*A* shows the significant coherence between the Cz EEG and the TA EMG. At 20 Hz a clear peak with a magnitude of 0.04 is seen. Individual values of peak coherence values and frequency are given in Table 1. In Fig. 4*B* and *C* the coherence magnitude and the imaginary part of the coherence estimate is plotted for all electrodes at 20 Hz. Both estimates were again confined to the leg motor area with some minor spread towards the left frontal areas.

Discussion

We have demonstrated in this study that EEG recorded over the leg area of the motor cortex is coupled with EMG activity recorded from the TA muscle in the swing phase during treadmill walking. The coupling characteristics resemble closely those previously reported for cortico-muscular coherence during finger or precision grip tasks

in sitting subjects (Conway *et al.* 1995; Mima *et al.* 2000; Hansen *et al.* 2002). Although the coherence observed in the present study was mainly above 25 Hz, it is within the frequency band of 15–35 Hz reported previously for corticomuscular coherence (Conway *et al.* 1995). During walking it has previously been shown that populations of TA motor units show coupled oscillatory activity during walking in a frequency band (5–50 Hz) that includes the 25–35 Hz frequency band in which we found significant corticomuscular coupling here (Halliday *et al.* 2003; Hansen *et al.* 2005; Nielsen *et al.* 2008; Barthelemy *et al.* 2010). The observation that the TA intramuscular coherence in the frequency band from 15 to 50 Hz is greatly reduced following spinal cord injury and stroke (Hansen *et al.* 2005; Nielsen *et al.* 2008; Barthelemy *et al.* 2010) is consistent with our finding that at least the oscillatory drive in the 25–35 Hz frequency band originates from the corticospinal tract. A contribution of motor cortical activity to TA EMG activity during walking has been implicated before from indirect experiments using transcranial magnetic stimulation (Schubert *et al.* 1997; Petersen *et al.* 1998, 2001; Capaday *et al.* 1999). Most relevant to the present study, Petersen *et al.* (2001) demonstrated that sub-threshold TMS in early swing

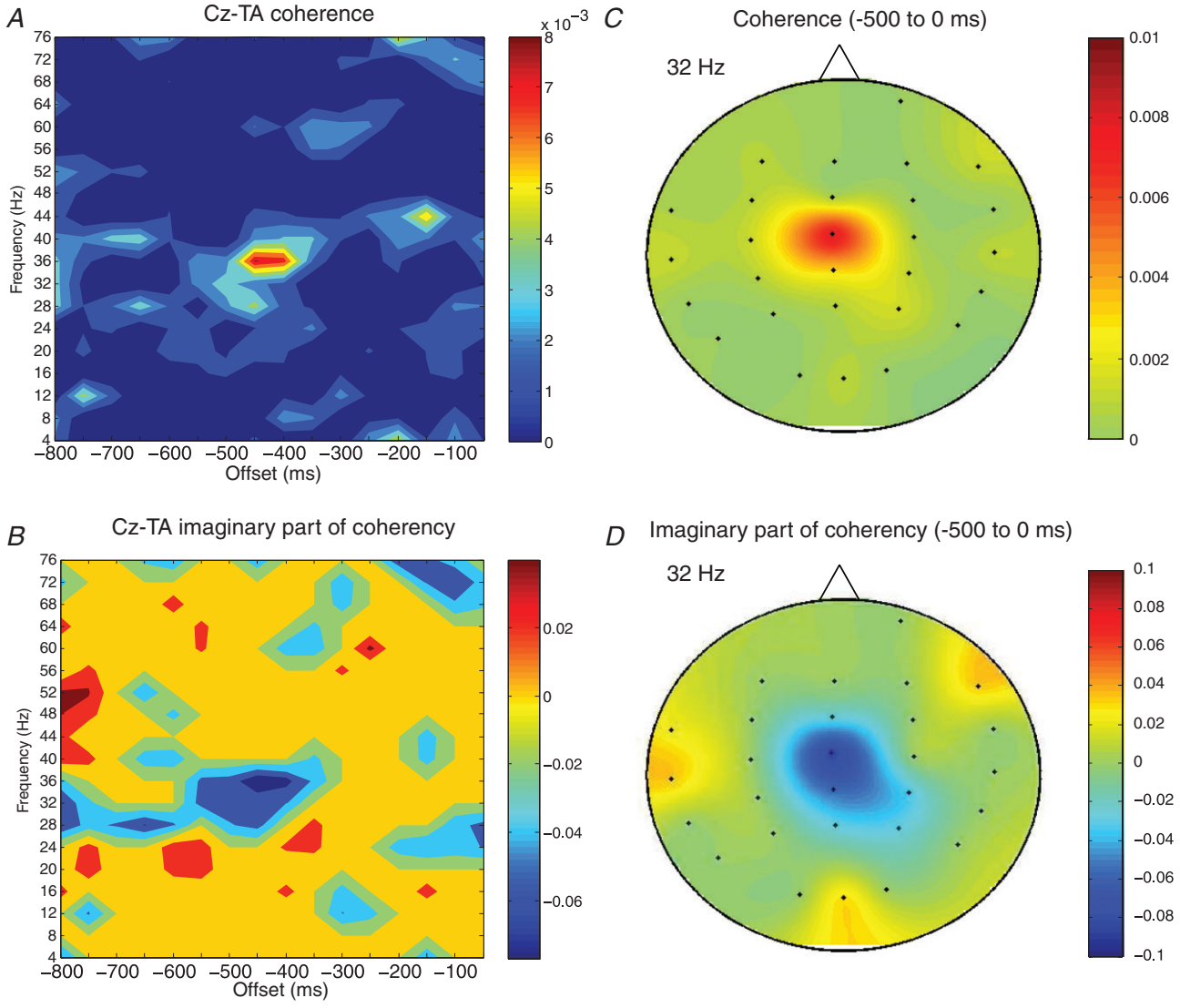

Figure 3. Pooled data from seven subjects during slow walking at 1 km h^{-1}
Time–frequency analysis of averaged coherence (*A*) and imaginary part of coherency between the Cz EEG and the TA muscle EMG (*B*). As for normal walking, significant coupling was observed in the ∼24–40 Hz frequency domain for offsets between –700 ms and –200 ms prior to the heel strike. Average coherence (*C*) and the imaginary part of coherency (*D*) at 32 Hz between all 28 EEG electrodes and the TA EMG taken from segments of data from –500 and 0 ms where peak values of coupling were observed. Note the different colour bar scaling compared with Fig. 2.

resulted in a drop in the TA EMG activity, which was interpreted as removal of corticospinal drive to the muscle activity. Our observation of significant corticomuscular coherence that also covered this time of the gait cycle is consistent with this.

The significant imaginary parts of the complex coherency estimate indicated that the coupling in the present study was not due to simple volume conduction of activity or non-physiological noise. The negative sign of the imaginary part of the coherency estimates at ∼30 Hz suggested that the cortical activity was leading the muscle activity (Nolte *et al.* 2004). For the data from the single subject displayed in Fig. 1*B* and *C*, this was consistent with a time lag of around 27 ms (subject was around 160 cm tall), which would correspond to the cortico-spinal conduction time. Previous studies have similarly reported time lags estimated from EEG/MEG–EMG coherence that correspond reasonably to the corticospinal conduction time for the upper limb in the monkey (Baker *et al.* 1997) and in human subjects (Gross *et al.* 2000). However, it should be noted that delays consistent with cortico-spinal transmission have not always been detected in MEG or EEG–EMG coherence studies (Conway *et al.* 1995; Halliday *et al.* 1998)

Coherence in the 8–12 Hz frequency domain was present in the normal walking condition during the swing phase with peaks in early, mid and late swing, and in addition some minor peaks were identified for the slow walking experiment. The coherence magnitudes (see Table 1) were generally lower than for the 24–40 Hz frequency band. Interestingly, the imaginary part of coherency in the ∼10 Hz frequency bands showed opposite signs, but subsequent analysis showed that this coupling was located at the same spot as the ∼30 Hz coupling. Peripheral nerve stimulation performed during static muscle contraction has been shown to induce ∼10 Hz coherence between the EEG and TA EMG (Hansen & Nielsen, 2004), possibly through a cerebellar–thalamic–cortical network (Marsden *et al.* 2000). Therefore the present finding may reflect inflow of afferent information to the cortical network.

Strikingly, the peak coherence frequency was always higher in the walking experiments compared with static contraction experiments. Furthermore, the peak coherence in the normal walking shifted towards higher frequencies in the early and late swing phase where the EMG is dynamically changing compared with mid swing. This is in line with previous results on EEG–EMG coherence where a shift from the beta band (∼15–30 Hz) towards the gamma frequency range was observed when subjects switched from a static to a dynamic task (Omlor *et al.* 2007).

This has been argued to reflect binding of sensory information between the periphery and the cortex. Sensory feedback from the active muscle, its antagonists and the joint it acts on must be assumed to be largest around the beginning of the swing phase, where the TA is activated and the angular velocity of ankle dorsiflexion is at its greatest, and just after heel strike where the ankle joint is moved in plantar flexion direction. More sophisticated experimental protocols, with manipulation of sensory feedback during walking could address this issue further

The magnitude of peak coherence in the 24–40 Hz frequency domain varied between subjects and was generally highest in the static contraction experiment (see Table 1). In the case of the pooled data, subjects showed peak coherence at slightly different frequencies and offsets, which affected the overall pooled data. A recent study has reported large inter-subject differences in the magnitude of beta band coherence between EEG and TA EMG during a static motor task (Ushiyama *et al.* 2011). Several factors such as scalp thickness and individual differences in the tendency of neurones to fire in synchrony could influence the magnitude of coherence. In addition, recent studies have reported that the magnitude

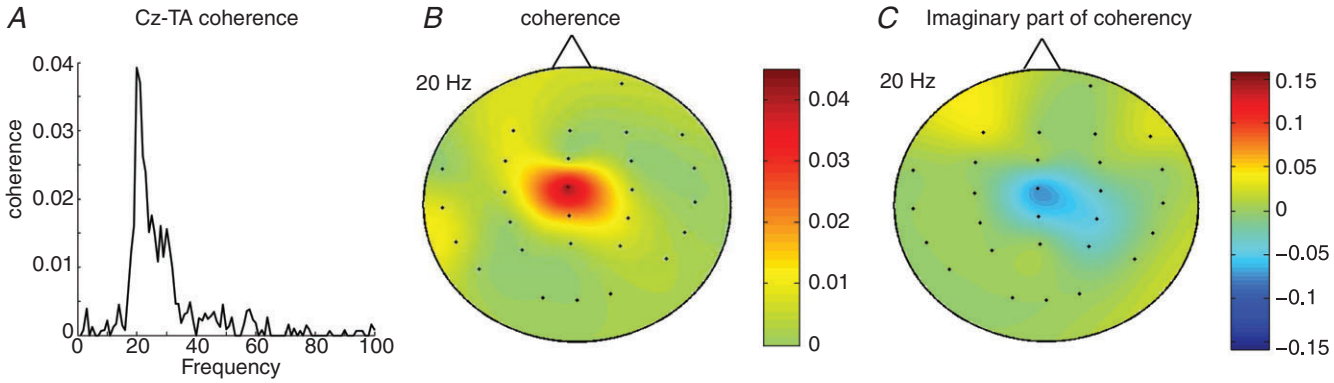

Figure 4. Pooled data from seven subjects during static muscle contraction
Average coherence between the Cz EEG and the TA EMG (*A*). A clear peak was detected around 20 Hz. Average coherence (*B*) and the imaginary part of coherency (*C*) at 20 Hz between all 28 EEG electrodes and the TA EMG.

of coherence in the beta band may also depend on learning of a novel motor task (Perez *et al.* 2006). Furthermore, it has recently been shown that subjects, who show no initial significant EEG–EMG coupling, developed coupling in the 20–30 Hz frequency band after learning of a static motor task and coupling at ∼35 Hz after learning of a dynamic motor task (Mendez-Balbuena *et al.* 2011). In the present experiment all subjects were experienced treadmill walkers and learning effects must be assumed to be minimal.

Corticomuscular coherence in the 15–35 Hz frequency band is furthermore depressed during the dynamic ramp phase of a contraction, but is re-established some time into the maintained contraction (Feige *et al.* 2000; Kilner *et al.* 2000). Consistent with this we found maximal values of coherence during static contraction and in mid swing where the EMG level is stable, and also generally larger values of coherence in the slow walking compared with the faster walking (compare Figs 2*C* and 3*C*), which may also be due to the more static nature of the EMG in mid swing during slow walking. During walking, the sensory feedback evoked by the ongoing rhythmic movements may very well modulate the levels of corticomuscular coupling. Sensory feedback evoked by either stimulation of peripheral nerves or muscle stretch has been shown to desynchronize cortical activity and depress cortico-muscular coherence in the 15–35 Hz frequency band very effectively for a period of up to 500–800 ms (Hansen & Nielsen, 2004). Furthermore, a reduction in the magnitude of corticomuscular coherence in this frequency band has been observed during ischaemic nerve block where sensory feedback was removed (Pohja & Salenius, 2003).

It should be emphasized that the analysis in the present study only allows that an oscillatory corticospinal drive is revealed, whereas any non-oscillatory drive would go undetected. It should also be noted that the data cannot be used to make any conclusions regarding how important the cortical drive is for the activation of the muscles and what percentage of the motoneuronal drive originates from the cortex. It is interesting to note that oscillatory activity in the nervous system has been speculated to provide an energy-efficient mechanism to control spinal motoneurones (Baker *et al.* 1999) and such efficiencies may be beneficial in highly repetitive behaviours such as stepping. Nevertheless, the observation described here that cortical activity does directly contribute to the muscle activity driving uncomplicated treadmill walking emphasises the important role of corticospinal function in human walking, and the importance that restoration of this activity and connectivity will have in promoting gait recovery for subjects with stroke or spinal cord lesions. The presence of specific cortical events that can be measured in the EEG during gait also opens up for interesting perspectives in relation to the emerging field of brain–machine interfaces for use in rehabilitation of gait.

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Author contributions

T.H.P., B.A.C. and J.B.N. designed the experiment. T.H.P., B.A.C., M.W.O. and J.B.N. performed the experiments. T.H.P. and J.B.N. analysed and interpreted the data. T.H.P., B.A.C. and J.B.N. drafted the manuscript and all authors approved the final version. All experiments were performed at the University of Copenhagen.

Acknowledgements

We are grateful to Guido Nolte for providing scripts to analyse and plot the imaginary part of coherency. The study was funded by The Royal Society, The Danish Health Research Council, The Danish Society of Multiple Sclerosis, the Desiree and Niels Yde's Foundation and The Wellcome Trust.