Reduction of Common Synaptic Drive to Ankle Dorsiflexor Motoneurons During Walking in Patients With Spinal Cord Lesion

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INTRODUCTION

Impaired gait ability is one of the most important factors for decreased life quality in patients with lesions of the central motor pathways (Maxwell et al. 1999). To improve rehabilitation strategies in these patients, it is necessary to obtain a better understanding of the neuronal control mechanisms involved in gait regulation in healthy subjects and how this regulation is impaired in patients.

Cross-correlation and coherence analysis of motor-unit firing behavior have the potential of providing valuable information about the organization of the networks responsible for driving spinal motoneurons during movement (Farmer et al. 1993, 1997; Halliday et al. 1995). These measures allow a statistical analysis to be performed to characterize the time course (cross-correlation) and frequency content (coherence) of common presynaptic inputs that act to synchronize populations of motor units. This type of analysis was recently applied to human walking (Halliday et al. 2003; Hansen et al. 2001) where it was demonstrated that motor units from the same and synergistic muscles receive a common synaptic drive that is modulated during the gait cycle (Halliday et al. 2003; Hansen et al. 2001). In the present study, we have extended this type of analysis to patients with incomplete spinal cord lesion. We report that motor-unit coupling during walking is absent or weak in these patients, and we suggest that the appearance of motor unit synchrony during walking depends on an intact supraspinal drive to the spinal cord. Estimation of motor unit synchronization in patients may thus provide valuable physiological markers that can be used in the evaluation of gait restoration.

Participants

The experiments were performed in 20 patients with spinal cord lesion [SCL; 6 women and 14 men; average age: 42.6 ± 18.6 (SD) years]. The duration since the primary lesion varied between 1 and 28 yr. Eight patients had a lesion at cervical level, 10 patients had a lesion at thoracic level, and 2 patients a lesion at lumbar (L1-L2) level. The lesion was caused by trauma in 15 cases, A-V malformation in 3 cases, and syringomyelia in 2 cases. The ASIA impairment score varied between B and D. Fourteen of the SCL patients showed signs of spasticity (average score on the modified Ashworth scale of...
2.1) (see Bohannon and Smith 1987 for a reference on the modified Ashworth scale).

The maximal walking speed on a treadmill was tested in all patients. This was defined as the highest speed at which sustained walking was considered comfortable by the patient. In the SCL patients, three subjects were able to walk on the treadmill at speeds exceeding 4 km/h, which was considered consistent with normal ability. In the remaining 17 subjects, maximal speed varied from 0.2 to 2.7 km/h with an average of 0.84 km/h. Ten of these subjects were able to walk on the treadmill without any support, whereas the remaining 7 subjects required to use handrails for assistance. Control experiments were performed in 11 healthy subjects (6 women and 5 men; mean age: 31.5 ± 6 yr) none of whom had a record of neurological disease.

METHODS

Experimental methods

The local ethics committee approved all experimental protocols (J. No. KF 01-055/98), and all subjects provided informed written consent prior to participation. The experiments were performed according to the Helsinki declaration.

Paired bipolar surface EMG recordings (Ag-AgCl) electrodes; 1 cm² recording area, 2 cm between poles) were made from two sites over the tibialis anterior (TA) muscle. The recording sites were separated by a minimum distance of 10 cm during treadmill walking (see next section for a more detailed discussion of electrode placement and cross-talk). The EMG signals were amplified (5,000–10,000), filtered (high pass: 1 Hz; low-pass: 1,000 Hz) and stored as waveforms on a computer for later analysis. All subjects wore a heel switch under the sole of their shoe. This was used to provide the precise time of heel contact during each step cycle and acted as a trigger signal from which cumulant and coherence estimates during the swing phase could be determined. All data were sampled at 2,000 Hz. The patients walked at their maximal speed for 5 min. In five SCL patients, no consistent signal could be related to heel strike during walking sessions, and therefore the data could not be segmented into swing and stance phase and no phase specific coherence or cumulant estimates were obtained for these subjects (see Table 1 patients 16–20). Accordingly, coherence and cumulant density estimates were calculated from nonsegmented data; i.e., from the entire gait cycle. In other patients and in healthy subjects, coherence and cumulant density estimates were calculated from the swing phase only. Using data from the complete gait cycle in patients with no reliable heel contact allowed us to identify the presence or absence of significant coupling in these patients, but the data were not included in the calculation of pooled estimates of coherence and cumulant density (see following text) The healthy subjects walked at 1.0, 2.0, 3.0, and 4.0 km/h; 5 min of data was sampled in all cases.

Analytical methods

The analysis uses a Fourier transform based framework, which allows the correlation structure between the paired EMG signals to be characterized as a function of time and frequency (Halliday et al. 1995). Following standard practice, and the theoretical work of Myers et al. (2003), we preprocess the surface EMG using full-wave rectification. The rectified signals are then assumed to be realizations 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 transform is constructed from short 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. A segment length corresponding to the period of TA EMG activity during the swing phase was used for the present data. We use \( f_x(\lambda) \) and \( f_y(\lambda) \) to represent the power spectra of processes x and y, respectively. The cross spectrum between x and y is denoted by \( R_{xy}(\lambda) \) and is estimated in a similar manner to the auto spectra.

In the frequency domain, the correlation between the EMG signals is assessed through coherence functions (Brillinger 1981; Halliday et al. 1995). The coherence function between the two rectified EMG signals is defined at frequency \( \lambda \) as

\[
[R_{xy}(\lambda)]^2 = \frac{|f_x(\lambda)y(\lambda)|^2}{f_x(\lambda)f_y(\lambda)}
\]  

(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

### Table 1. Data recorded from spinal cord lesion patients and healthy subjects

<table>
<thead>
<tr>
<th>Patient</th>
<th>Gait Maximal Velocity, km/h</th>
<th>TMS Threshold (% of max Stimulator Output)</th>
<th>TMS Latency, ms</th>
<th>Coherence 10–20 Hz</th>
<th>Short-Term Synchrony</th>
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<tr>
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<tr>
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<td>35</td>
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<tr>
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<tr>
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<td>0.0002</td>
</tr>
<tr>
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<td>60</td>
<td>40</td>
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</table>

Maximal gait velocity for each of the 20 spinal cord lesion (SCL) patients, the threshold and latency of the tibialis anterior (TA) motor-evoked potential, the maximal peak of coherence in the 10- to 20-Hz frequency band for the two TA electromyographic recordings during walking and the size of the central peak of synchronization in the cumulant density function. A reliable signal from the heel trigger could not be obtained in these patients 16–20 (*). The cumulant and coherence estimates were therefore calculated for the whole gait cycle. This made it possible to disclose the presence or absence of significant synchronization features in the recordings from these patients, but a quantification of these features is not given because it would not be comparable to quantitative data obtained from the other patients and the healthy subjects. Below the data from the SCL patients, corresponding data from the 11 healthy subjects are shown; 4.0 km/h indicates normal gait ability.
strength and frequency of common rhythmic synaptic inputs, which are distributed across the motoneuron pool (Farmer et al. 1993). In the time domain, estimates of the cumulant density function are used to characterize the correlation between the two rectified surface EMG signals. The cumulant density function, denoted by \( q_{xy}(u) \), is defined as the inverse Fourier transform of the cross spectrum

\[
q_{xy}(u) = \int_{-\infty}^{\infty} f_{xy}(\lambda)e^{iu\lambda}d\lambda
\]

(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, \( u \). In the present experiments, which are exploring aspects of the common drive to motoneuron pools during locomotion, the dominant feature in cumulant density estimates is the central peak around zero lag, reflecting the presence of common synaptic input. Rhythmic inputs will induce symmetrical oscillatory components in the cumulant (Perkel et al. 1967), the frequency and strength of these components can be quantified from the corresponding coherence estimates. Cumulant density functions are analogous to cross-correlation functions often used to quantify spike train data and have a similar interpretation (Hilliday et al. 1995).

To summarize the correlation structure across subjects, estimates of pooled coherence and pooled cumulant density functions are used. Pooled coherence and cumulant functions provide a single measure, which summarizes the correlation structure across several data sets (Amjad et al. 1997). Pooled coherence estimates, like individual coherence estimates, provide a normative measure of measures of linear association on a scale from 0 to 1 (Hilliday and Rosenberg 2000). Pooled cumulant density estimates provide a measure of time domain correlation across subjects. The interpretation of pooled estimates is similar to those for individual records except any inferences relate to the population as a whole. In the pooled case, the time-dependent aspect of the analysis, using short segments at varying offset times relative to the heel trigger, provides a measure of how the correlation structure across subjects changes (in an average sense) during the step cycle.

**Electrode placement and cross-talk**

The pairs of recording electrodes were placed \( \pm 10 \) cm apart over TA muscle to minimize the risk of cross-talk between the two pairs of recording electrodes due to activity detected from overlapping motor units territories or from the passive electrototonic conduction of activity from nearby muscle tissue. This procedure was validated by placing pairs of electrodes at different distances along the muscle and calculating the coherence and cumulant density function between the different electrodes while the subject performed a voluntary tonic dorsiflexion. In the experiment illustrated in Fig. 1, the EMG coherence magnitude measured from electrodes placed near to one another (TA2 and TA3, Fig. 1A) varied between 0.6 and 0.8 at frequencies between 10 and 80 Hz (Fig. 1B), indicating that the variance in EMG TA2 can predict \( \sim 60–80\% \) of the variance in EMG TA3. Such large values are indicative of the presence of cross-talk between the two electrode recordings. Coherence \( >0.5 \) was found over a broad range of frequencies spanning the entire EMG spectral bandwidth. Also a very large and very narrow peak was observed in the cumulant density function for recordings taken from electrodes placed next to each other over the distal part of the TA muscle (Fig. 1C). In contrast, when recordings were made at a distance of \( >10 \) cm from each other, coherence was \( <0.05 \) and was significant only in distinct bands \( \sim 10 \) and 20 Hz. These values are in line with those obtained from experiments using needle recordings of pairs of individual motor units (Farmer et al. 1993). Furthermore, the central peak in the cumulant density function was much smaller and lacked the very narrow part observed for the recordings placed in close proximity. Similar observations were made in six subjects. These data provide strong evidence that the distant electrode recording sites are not influenced by cross-talk.

**Transcranial magnetic stimulation**

To investigate transmission in the corticospinal tract in the patient group transcranial magnetic stimulation (TMS) was applied in all.

**FIG. 1.** Minimal cross-talk between recordings at distances over 8–10 cm. Electromyographic (EMG) recordings were made by pairs of surface electrodes placed over the tibialis anterior (TA) muscle (A). The subject performed a tonic voluntary dorsiflexion for 200 s. Two of the electrode pairs were placed close to each other over the distal part of the muscle (TA2 and TA3), whereas a 3rd pair (TA1) was placed over the proximal part of the muscle at a distance of 10 cm from the most proximal of the 2 distally placed electrode pairs. B–D: the coherence and cumulant density function for the recordings from TA2 and TA3. E–G: the coherence and cumulant density function for the recordings from TA1 and TA3. Horizontal lines in C–E and G, 95% confidence limits. In B and F, the 95% confidence limit lies too close to the abscissa to be visualized.
subjects. The magnetic stimuli were applied by a figure-eight coil placed over the leg area of the motor cortex with the handle of the coil pointing backward. The stimulator was a Magstim 200 (Magstim, Dyfed, UK). MEPs were recorded from the TA muscle by the same surface EMG electrodes used for the coherence analysis. In these experiments, the subjects were seated in a reclining armchair with the examined foot attached to a plate that was connected to a torque meter. The subjects were asked to perform a voluntary dorsiflexion corresponding to 10% of their maximal dorsiflexion effort (measured as the maximal voluntary torque that the subjects could sustain for 10 s). The threshold of the MEP was determined as the stimulus intensity at which three of five stimuli evoked a response >100 μV. The latency of the MEP was determined as the first positive deflection exceeding this size in an average of five stimuli. All latency measurements were made for MEPs evoked at an intensity of 1.2 × MEP threshold. Threshold and latency measures in the patients were considered abnormal if their magnitude differed from the mean values in healthy subjects by two times the SD in the population of healthy subjects.

RESULTS

Coherence and synchronization of TA EMG activity during walking in spinal cord lesioned patients

Figure 2 illustrates data from a healthy subject (Fig. 2, A, C, and E) and an SCL patient (Fig. 2, B, D, and F). The healthy subject was instructed to walk at a speed of 1.0 km/h. The SCL patient walked at a speed of 1.2 km/h, which was the maximal speed he could sustain for the 5-min recording period. Comparison between EMG recordings in Fig. 2, A and B, illustrate that the SCL patient has a higher cadence (360 steps for the 5-min recording period) than the healthy subject (280 steps). The difference in cadence can be largely explained by the difference in the walking speed. However, it should be noted that despite this difference, the duration of the swing phase is similar in the two subjects (~600 ms). The cumulant density, which provides a measure of the temporal structure of the motor-unit synchronization existing between paired EMG recordings, is shown in Fig. 2, C and D, for the duration of the swing phase. The corresponding coherence measure, which provides an estimate of common frequencies associated with the ongoing locomotor behavior, is shown in Fig. 2, E and F. In the healthy subject, a narrow central peak of synchronization (lasting ~30 ms) with secondary peaks at lags of approximately ±55 ms was observed in the cumulant density estimate (Fig. 2C). The central peak is interpreted as resulting from the common central drive to the motoneurons, whereas the secondary features relate to the frequency of that drive (see also Farmer et al. 1993; Halliday et al. 2003; Hansen et al. 2001). In the SCL patient, neither the central peak nor the secondary features were present. In contrast, a broad low-amplitude peak with a duration of almost 200 ms was observed (Fig. 2D). In both the healthy subject and the SCL patient, a very high and significant coherence was observed at frequencies <10 Hz (Fig. 2, E and F). Coherence within this low-frequency range is assumed to be produced, in part, by the common envelope of the EMG activity during swing (Halliday et al. 2003). At frequencies around 15–20 Hz, an additional peak of coherence was observed in the healthy subject but not in the SCL patient. Coherence in this range of frequencies corresponds reasonably to the spacing of 55 ms between the secondary peaks observed in the cumulant density estimate of the healthy subject (Fig. 2C). Variable coherence of low amplitude was also observed in the healthy subject at frequencies >35 Hz. This feature of the coherence was absent in most healthy and SCL subjects and therefore will not be considered further in this study. Fig. 3

![Figure 2](http://jn.physiology.org/)

**FIG. 2.** Coherence and cumulant density function during walking in a single healthy subjects (A, C, and E) and a single SCL patient (B, D, and F). In A and B, the EMG activity recorded from the 2 pairs of electrodes placed over the TA muscle is shown. C and D: the cumulant density function for the 2 EMG recordings calculated for the periods with EMG activity in the TA muscle during the swing phase. E and F: the coherence calculated for the same periods. In C and D, the x axis indicates the time in milliseconds, whereas it indicates the frequency in hertz in E and F. The confidence intervals for the cumulant density and coherence estimates were very low and have been omitted from the figure because they could not be visually separated from the baseline.
shows pooled data from all 11 healthy subjects and 15 of the 20 SCL patients. The data were pooled for 600-ms segments prior to the time of heel strike in all subjects. The healthy subjects all walked at 1.0 km/h, whereas the average walking speed of the SCL patients was 1.5 km/h. The power spectra for the TA EMGs recorded from healthy (Fig. 3, A and B) and SCL (Fig. 3, C and D) subjects are very similar apart from the presence of a peak (marked by arrow) in the 10- to 20-Hz range in the healthy subjects. This supports the viewpoint that the overall muscle activity during swing is comparable in both groups of subjects. However, the presence of a 10- to 20-Hz peak in the healthy EMGs results in the emergence of clear differences in the TA EMG coherences seen in healthy and SCL subjects. The pooled coherence for the healthy subjects (Fig. 3 E) contains a distinct peak within the 10- to 20-Hz frequency range that is lacking from the equivalent pooled coherence from the SCL patients (Fig. 3 G). In fact, in the SCL patients coherence was restricted to frequencies <10 Hz, whereas significant coherence was observed in the healthy subjects up to frequencies ~50 Hz.

The pooled cumulant density function is shown over two lag ranges in Fig. 2, F (±100 ms) and I (±600 ms) for the healthy subjects and H (±100 ms) and J (±600 ms) for the SCL patients. In the healthy subjects, a central peak with a duration of 30–40 ms was observed on top of a broader period of synchrony lasting ~200 ms. In the SCL patients, only the broader period of synchrony was present.

In one respect, the results from healthy subjects and SCL patients are similar. Both exhibit broad-duration time domain synchrony and significant low-frequency coherence (<10 Hz). Hansen et al. (2001) and Halliday et al. (2003) described a similar broad-duration synchronization and argued that it was due to the EMG envelope during walking. At a gross level, the paired EMG recordings from healthy subjects and SCL patients have a similar appearance (Fig. 2, A and B), consisting of bursts of EMG activity. We suggest that these features are representative of the presynaptic mechanisms responsible for generating the rhythmic EMG bursts, which are present in healthy subjects, and preserved in the current group of SCL patients. Presynaptic synchronization of interneurons driving the spinal motoneurons (Datta et al. 1991; Farmer et al. 1993) may contribute to this broad term synchronization. These mechanisms operate over a different time scale and at a different frequency range from the mechanisms that generate the short-term synchronization and <10-Hz coherence in healthy subjects. It is this short term synchronization which is the focus of the present study.

Relation between walking speed and coherence/synchrony in healthy subjects

The average walking speed in the SCL patients in Fig. 3 was 1.5 km/h, whereas the healthy subjects walked at 1.0 km/h. Halliday et al. (2003) obtained qualitatively similar results as those shown here for healthy subjects, although the subjects in that study walked at a speed of 4.0 km/h. The small difference in the walking speed between healthy subjects and SCL patients in this study is thus unlikely to explain the differences in coherence and cumulant estimates. Nevertheless, we examined the influence of walking speed on the coherence and cumulant in healthy subjects by asking them to walk at several different speeds. Figure 4 shows data from a single healthy subject. With increased walking speed the duration of the TA EMG activity shortened and developed a more distinct burst-like pattern with
a clear peak in activity around the time of heel strike (Fig. 4, A–C). For the analysis of these data, the EMG records were segmented into periods reflecting the variations in burst duration with walking speed (e.g., 1 s for walking at 1.0 km/h, 800 ms for walking at 2.0 km/h, and 600 ms for walking at 4.0 km/h). For the subject illustrated in Fig. 4, a very clear peak was seen in the coherence between 10 and 20 Hz during walking at 1.0 km/h (Fig. 4D). This peak was smaller at 2.0 km/h and further reduced at 4 km/h (Fig. 4, E and F). This is also reflected in the cumulant density function where distinct rhythmic activity is seen at lags of 60 ms during walking at 1.0 and 2.0 km/h (Fig. 4, G and H) but not at 4.0 km/h (Fig. 4I). A short-lasting central peak was seen for all walking speeds. During walking at 4.0 km/h, the central peak is easily distinguished from the larger and broader period of synchrony. For the two slow walking speeds, this distinction is more difficult to make due to the fluctuations in the cumulant density due to the size of the secondary features just described.

Figure 5 shows pooled data from all 11 healthy subjects. The peak of coherence between 10 and 20 Hz was seen

![Figure 4](http://jn.physiology.org/)

**FIG. 4.** Changes in coherence and cummulant density functions with increasing walking speed in a single healthy subject. A–C: the EMG activity from the 2 surface EMG recordings while the subject was walking at 1, 2, and 4 km/h. Heel strike is marked by vertical arrows. D–F: the coherence between the 2 EMG recordings for the 3 walking speeds; and G–I: the cumulant density function. The abscissa in D–F is the frequency in hertz, whereas it is the time in milliseconds between events in the 2 recordings in G–I. The calculations were made for a 800-ms time period when the subject walked at 1 and 2 km/h and a 600-ms period when he walked at 4 km/h. This corresponded approximately to the time period in which EMG activity was observed in the two recordings during the swing phase prior to heel strike. The confidence intervals for the cumulant density and coherence estimates were very low and have been omitted from the figure because they could not be visually separated from the baseline.

![Figure 5](http://jn.physiology.org/)

**FIG. 5.** Pooled coherence (A–C) and cummulant density function (B–D) during walking at 1 km/h (A and B) and 4 km/h (C and D) in the 11 healthy subjects. The calculations were made for the 2 TA EMG recordings in time windows of 800 and 600 ms prior to heel strike for the slow and fast walking, respectively. The confidence intervals for the cumulant density and coherence estimates were very low and have been omitted from the figure because they could not be visually separated from the baseline.
during both slow (1.0 km/h) and fast (4.0 km/h) walking (Fig. 5, A–C). The central narrow peak in the cumulant was also clearly present during both slow and fast walking (Fig. 5, B–D). The SCL patients thus showed significantly less 10- to 20-Hz coherence and short-term synchrony than the healthy subjects, regardless of whether the healthy subjects walked at the same (slow) speed as the patients or at a higher speed that is more equivalent in terms of the effort experienced by the patients.

Relation between EMG coherence/synchrony and maximal walking speed

Within the patient group a considerable range of walking ability was evident based on the maximum walking speed attained by each patient (Table 1). Three of the patients were considered to show normal walking ability based on their maximal walking speed (Table 1; patients 7, 8, and 13), but only one of these (Table 1; patient 13) showed a cumulant density with a narrow central peak. This patient also showed coherence in the 10- to 20-Hz frequency band. A further five patients (Table 1; patients 1, 9, 12, 18, and 20) also presented with a narrow central peak in the cumulant, although their maximal walking speed was very low (ranging from 0.2 to 1.4 km/h). Two of these patients (Table 1; patients 12 and 20) also showed significant coherence at 10–20 Hz. We are therefore unable to establish a clear relationship between the presence and absence of features in the cumulant or coherence that correlate with maximum walking speed in the patient group.

TMS

In all subjects, transmission in the corticospinal tract was evaluated by TMS, while sitting down and performing a voluntary dorsiflexion corresponding to 10% of the maximal voluntary dorsiflexion effort. In the healthy subjects, the average latency of the motor-evoked potential (MEP) was 31.4 ± 1.7 (SE) ms. It was not possible to evoke any MEP in four of the patients (Table 1; patients 5, 9, 17, and 19). In the remaining patients, the average latency was 42.0 ± 7.0 ms, which was a statistically significant difference from the healthy subjects (ANOVA; \( P < 0.05 \)). Two of the three patients (Table 1; patients 8 and 13) with normal gait ability also had clearly delayed MEPs (44 and 52 ms), whereas the MEP in the third patient (Table 1; patient 7) was within the normal range (35 ms). The four patients in whom no MEP could be evoked (Table 1; patients 5, 9, 11, and 19) had maximal walking speeds of 0.7, 0.3, 0.3, and 1.2 km/h, and this places them in the low end of the range of walking speeds achieved in the population of patients. A very small central peak of synchrony was seen in only one of these patients (Table 1; patient 9) and a significant peak of coherence in the 10- to 20-Hz frequency band was not seen in any of the four patients. In Fig. 6 the maximal walking speed has been plotted for each individual patient as a function of the latency and threshold of the MEP (Fig. 6, ○). There was no significant relation between the latency of the MEP and the maximal walking speed (Spearman rank order). Similarly, no statistically significant relation could be established between MEP threshold (% of stimulator output)
and maximal walking speed in this patient population. Inclusion of the data from the healthy subjects (Fig. 6, ○) clearly highlights the differences between the two populations.

**Discussion**

In this study, we have demonstrated that two main characteristics of coupling between TA motor-unit activity in the swing phase of healthy human walking are absent or greatly reduced in SCL patients. Hansen et al. (2001) demonstrated that separate populations of TA motor units show synchronized discharges within a narrow time band (10–20 ms) during the swing phase of walking. Halliday et al. (2003) further demonstrated that this synchronization was associated with peaks of coherence around 10–20 Hz in the frequency domain. Both of these features were either strongly reduced or absent in the SCL patients.

Hansen et al. (2001) and Halliday et al. (2003) have already discussed in detail why cross-talk between the two surface electrode recordings is unlikely to explain the observed coupling in healthy subjects. Briefly, the distance between the pairs of EMG electrodes exceeds that of the overlap between individual motor unit territories (Roy et al. 1995; Smits et al. 1994). Also, signs of cross talk within the cumulant (large, narrow central peak) or coherence (large broad band coupling at minimal phase lags) estimates were not detected (Fig. 1). Furthermore, similar features to those reported here using surface EMG recordings are seen when selective intra-muscular wire recordings are used and where the coherence and cross-correlation estimates are based on events generated by triggering on the largest spikes in each of the recordings (Halliday et al. 2003; Hansen et al. 2001). Finally, it seems exceedingly unlikely that the recordings in the SCL patients were less contaminated by cross-talk than the recordings in the healthy subjects given that the duration and magnitude of TA EMG activity in healthy and SCL subjects walking at speeds ~1.0 km/h was similar. Less cross-talk in the patients also would not be seen as a reduction of a specific frequency component, such as the one at 10–20 Hz, but as a general decrease of coherence at all frequencies contained in the EMG signals.

The similar duration and magnitude of EMG in the SCL patients and healthy subjects also mitigates against the likelihood that the differences in synchrony observed are due to differences in the level of EMG activity. The central peak observed in the cumulant density estimates from the paired TA surface EMG recordings is similar to that observed in the cross-correlogram of pairs of single motor-unit recordings from the same and synergistic muscles during voluntary tonic contraction (Datta and Stephens 1990). It is generally accepted that common synaptic drive from collaterals of last order neurons is an important, but probably not exclusive, contributor to this phenomenon (Farmer et al. 1997 for discussion). During tonic voluntary contractions, the central peak seen in time domain estimates (cross-correlation or cumulant) of coupling between motor units is accompanied by symmetrical secondary peaks close to 50 ms that themselves are associated with coherence in the 15- to 35-Hz frequency band (Farmer et al. 1993). It is now generally accepted that this reflects the dominant discharge frequencies of the last order neurons responsible for the common synaptic drive. Halliday et al. (2003) argued that coherence in the 10- to 20-Hz frequency band during walking may similarly reflect the frequency of the synaptic input to the active motoneurons. This would fit well with the present observations. A central synchronization peak was present in only six SCL patients, and coherence in the 10- to 20-Hz frequency band was evident in three of these six patients. In no instance was coherence observed without the presence of a central synchronization peak in the cumulant. The two phenomena thus appeared to be reduced in parallel by the spinal lesion, suggesting that they are related to the same process within the CNS. However, in three subjects, who showed a central peak in the cumulant, there was a lack of the 10- to 20-Hz coherence component. This may indicate that while a branched common input exists to the TA motor pool, the pathway may lack activation within the normal range of frequencies during walking or that the output processes fail to preserve the timing precision necessary for coherence to be observed within the motor pool due to the effects of the spinal lesion on axonal conduction.

Short-term synchrony (Datta et al. 1991) and coherence in the 15- to 35-Hz frequency band (Farmer et al. 1993) observed during voluntary tonic contraction is reported to be reduced in patients with internal capsule lesions, suggesting that activity in the pyramidal tract is involved in their occurrence. The reduction of the central synchronization peak and 10- to 20-Hz coherence in the SCL patients included in the present study may similarly signify that pyramidal tract activity is of importance for their occurrence. That transmission in the pyramidal tract was affected in the patient group studied was documented by a significantly increased latency and threshold of MEPs elicited by TMS. Because only a few patients showed synchronization and coherence, it was not possible to test in the individual subjects whether the changes in synchronization were related to the changes in pyramidal tract transmission as revealed by TMS. Evidence that the pyramidal tract does play a role in the generation of EMG activity during human walking comes from the demonstration that the pyramidal tract cells are easily excited by TMS during human treadmill walking (Capaday et al. 1999; Petersen et al. 1998; Schubert et al. 1997) and seem to contribute significantly to the activation of the participating muscles (Petersen et al. 2001).

Although our findings are thus consistent with a loss of pyramidal tract contribution to the ongoing EMG activity it should be emphasized that other descending pathways as well as spinal networks are also affected in the SCL patients. This may contribute to our observations.

The observation that two patients showed normal gait ability yet lacked evidence of synchronization in time or frequency domain estimates council against a positive correlation existing between the presence of synchronization and maximal gait velocity. Similarly, given that some of the most disabled patients showed evidence of short-term synchronization during walking also suggest that no firm relationship between the presence of synchrony and maximal gait velocity can be promoted at present.

This is not too surprising given that the TA muscle is only one of many muscles involved in the step cycle and that the control or drive to this muscle may be poorly related to parameters that determine gait velocity. Thus the precise contribution of motor unit synchronization to gait performance in SCL subjects requires to be further evaluated. This could be
achieved by combining three-dimensional motion analysis with estimation of motor unit coupling to identify if a specific gait deficit in these patients can be associated with the loss of the synchronizing input. Correlating an identifiable component of neural drive with a feature of gait kinematics could have significance in relation to rehabilitation strategies aimed at improving locomotor performance.

One major feature of the observations made in this study is the ease with which the technique may be used to determine deficiencies in the central drive to the active motoneurons during walking. The technique only requires recording of EMG from the TA muscle by two pairs of surface EMG electrodes and appears to have a very high specificity and reasonable sensitivity: All healthy subjects investigated so far have shown the central synchronization peak, whereas this was the case in only 6 of the 20 SCL patients. In these six patients, the peak was much smaller than in healthy subjects. The technique is thus quick and easy to use and may provide valuable information about the state of the central control circuits that is unavailable with any other technique. It is also possible that the technique may be used to predict the extent of recovery in individual patients and have utility as a method for exploring regeneration within the human motor system. However, it should be pointed out that when using surface EMG the technique is limited to large muscles with relatively restricted motor unit territories such as the TA muscle.

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