Brain (2000), **123,** 677–686

Frequency analysis of EMG activity in patients with idiopathic torticollis

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Summary

The pathophysiology of idiopathic dystonic torticollis is unclear and there is no simple test that confirms the diagnosis and excludes a psychogenic or voluntary torticollis in individual patients. We recorded EMG activity in the sternocleidomastoid (SCM) and splenius capitis (SPL) muscles of eight patients with rotational torticollis and eight age-matched controls, and analysed the signals in the frequency and time domains. All control subjects but one showed a significant peak in the autospectrum of the SPL EMG at 10–12 Hz, which was absent in all patients with torticollis. Conversely, patients with torticollis had evidence of a 4–7 Hz drive to the SPL and SCM that was absent in coherence spectra from controls. The pooled cumulant density estimates revealed a peak in both groups, and within the patient group there was a second narrow subpeak with a width of 13 ms. The activity in the SCM and SPL was in phase in the patients but not in the controls. The lack of any phase difference and the suggestion of short-term synchronization between SCM and SPL are consistent with an abnormal corticoreticular and corticospinal drive in dystonic torticollis. Clinically, the pattern of SPL EMG autospectra and of SCM–SPL coherence may provide a sensitive and specific feature distinguishing dystonic from psychogenic torticollis.

Keywords: torticollis; dystonia; frequency analysis; time domain analysis

Abbreviations: SCM = sternocleidomastoid muscle; SPL = splenius capitis muscle; TTL = transistor–transistor logic

Introduction

Torticollis (cervical dystonia, spasmodic torticollis) is a syndrome characterized by sustained involuntary muscle contraction, resulting in abnormal posture and twisting movements of the neck. In simple rotational torticollis the sternocleidomastoid (SCM) and splenius capitis (SPL) muscles contralateral and ipsilateral to the direction of head-turning are principally involved (Dauer *et al.*, 1998). The vast majority of cases are idiopathic (Berardelli *et al.*, 1998; Rutledge *et al.*, 1988).

The pathophysiology of cranial dystonia is still unclear. No consistent pathological or structural abnormality has been demonstrated (Dauer *et al.*, 1998), although, as in other types of dystonia, functional imaging has implicated the basal ganglia (Leenders *et al.*, 1993; Hierholzer *et al.*, 1994; Galardi *et al.*, 1996; Magyar-Lehmann *et al.*, 1997). EMG studies reveal that cervical dystonic movements are characterized by excessive and overlapping activity in agonist and antagonist muscle pairs (Podivinsky, 1968; Thompson *et al.*, 1990). Corticocortical inhibition of the motor cortical area projecting to the SCM is reduced (Hanajima *et al.*, 1998), and studies of somatosensory evoked potentials support the possibility of a shift in favour of excitation in the precentral cortex contralateral to the direction of head rotation (Kanovsky *et al.*, 1998). On the other hand, vestibular abnormalities are common in torticollis but may be secondary to the chronic, abnormal head posture (Bronstein and Rudge, 1988; Stell *et al.*, 1989; Lekhel *et al.*, 1997; Dauer *et al.*, 1998). Abnormalities in brainstem (Tolosa *et al.*, 1988; Nakashima *et al.*, 1989) and spinal (Panizza *et al.*, 1990; Deuschl *et al.*, 1992) inhibition have been found in dystonic torticollis, but they cannot alone be responsible for the abnormal movement pattern as they may be seen outside the area that is involved clinically and may not be limited to patients with dystonia (Berardelli *et al.*, 1998). The general conclusion of these studies was that the control of motor activities by the basal ganglia was disturbed, particularly at the level of the cortex, and resulted in reduced inhibition leading to excessive muscle activity and overflow to

uninvolved muscles (Berardelli *et al.*, 1998). However, so far no single abnormality has been found that reliably distinguishes individual patients with torticollis from subjects mimicking the abnormal posture.

In the present study we used frequency domain (coherence) and time domain (cumulant density) analyses of EMG activity in patients with cervical dystonia. These techniques can disclose oscillatory drives common to different motor units. The character of these rhythmic drives can provide clues about which motor structures are involved in a given activity, as recently demonstrated by Farmer and colleagues in dystonia of the upper limb (Farmer *et al.*, 1998). Coherence and cumulant density estimates have advantages over established cross-correlation techniques in that they are more sensitive to oscillatory influences and the confidence limits (CL) are readily calculated (Halliday *et al.*, 1995). Hitherto, investigations using these techniques have indicated the presence of four kinds of common drive, at around 1–2, 10, 20 and 40 Hz, during sustained voluntary activity in the distal upper limb (De Luca *et al.*, 1982; Farmer *et al.*, 1993*a*; Conway *et al.*, 1995; McAuley *et al.*, 1997). The drives at 20 and 40 Hz arise in the contralateral motor cortex (Farmer *et al.*, 1993*b*; Conway *et al.*, 1995; Salenius *et al.*, 1997; Brown *et al.*, 1998) and can be exaggerated in cortical myoclonus, where frequency analysis may prove to be of diagnostic use (Brown *et al.*, 1999). Here we investigate the pattern of rhythmic drive to the muscles of the neck in idiopathic cervical dystonia and compare it with that seen in healthy subjects.

Subjects and methods

Subjects

Informed consent was obtained from all subjects according to the declaration of Helsinki and with the approval of the local ethics committee. Eight patients (seven females and one male) aged between 35 and 69 years (mean ± SD, 56.3 ±11.7 years) and eight healthy controls (three females and five males) aged between 35 and 69 years (mean ± SD, 55.5 ± 12.5 years) participated. All patients had rotational torticollis (four left-sided and four right-sided). The position of the head varied from 10° to 40° from the straight-ahead position (mean ± SD, 25.6 ± 12.1°). Five of the patients had a slight laterocollis (5–10°) and three had a clinically mild, predominantly dystonic tremor of the head in the horizontal plane (yaw direction). The frequency of this head tremor was estimated from the EMG power spectrum and varied between 0.2 and 0.5 Hz. During testing, the control group matched the head position of the patients, so that their head position ranged from 10° to 40° from the straight-ahead position (mean ± SD, 25.6 ± 12.9°), four to the left and four to the right side. In addition, five healthy subjects imitated tremulous torticollis by making rotational yaw head movements for 120 s at 0.2 and 0.5 Hz. This moving control group matched the whole patient group and the subgroup of tremulous patients, with no significant difference in power in the autospectra of head acceleration in the band 0.12–1.1 Hz between the groups. The tonic control group, however, had significantly less power in the autospectrum of acceleration than the patients ($P > 0.001$). The patients did not have any other neurological disease. All patients were receiving regular treatment with botulinum toxin injections. They were tested either before the injections had become effective ($n = 3$, tested a mean of 9 days after the last injection) or after the effect of the last injection had worn off ($n = 5$, tested a mean of 140 days after the last injection). Case 1 was on benzhexol (daily dose 8 mg).

Methods

EMG activity was recorded in the SCM and SPL contralateral and ipsilateral to the direction of head turning. Patients and controls were seated in a chair with the chin in a chin-rest, while fixating a target straight ahead. Surface EMG electrodes (Ag–AgCl, 9 mm) were placed 1.5 cm apart over the middle of the SCM. Three 10 s periods of maximal voluntary contraction were recorded from the SCM. Concentric needle electrodes were then placed in the middle of the SCM and in the SPL. Patients were asked to keep their head in a neutral position if possible or, failing this, in a dystonic position. Healthy controls matched these positions and were asked to produce a weak contraction of the SCM and the contralateral SPL by turning their head against the chin-rest. Three periods of 120 s of dystonic and weak voluntary contraction were recorded in patients and controls, respectively. The interval between recordings was 1 min. Five healthy subjects also imitated tremulous torticollis by making rotational head movements for 120 s at 0.2 and 0.5 Hz (moving controls).

The surface and needle EMGs were amplified and filtered between 53 and 3000 Hz. The time constant was chosen to limit contamination of the EMGs by movement artefact. The ratio of the mean amplitude of the rectified surface EMG during dystonic or imitated sustained contraction to the mean amplitude during maximal voluntary contraction was calculated, to give an estimate of the percentage of maximal voluntary contraction which subjects produced during the experiment. In the patient group, the mean contraction of SCM during the dystonic movements was 51.6 ± 28.3% of the maximal voluntary contraction. The control group made voluntary contractions of 53.5 ± 24.5% of the maximal voluntary contraction.

The needle EMG was displayed on an oscilloscope. Motor unit potentials falling within an adjustable window were registered as 1 ms wide transistor–transistor logic (TTL) pulses using two spike processors (model D130, Digitimer, Welwyn Garden City, UK). The trigger level was always >50 μV. These TTL pulses, derived from multi-unit EMG signals, will henceforth be called SCM EMG and SPL EMG, according to the muscle sampled. An accelerometer was attached to the forehead to detect head tremor about the yaw

axis. Horizontal eye movements were recorded with surface electrodes over the outer canthus of each eye, and were used to confirm fixation of the target. Acceleration and extra-ocular muscle EMG were amplified and filtered (DC-300 Hz and 5s-300 Hz, respectively). All data were recorded on-line on a personal computer using an analogue-to-digital converter (CED 1401-plus, CED, Cambridge, UK). Signals were digitized with 12-bit resolution. Surface EMG and needle EMG were sampled at 5 kHz. Eye movements and head acceleration and TTL pulses were sampled at 1 kHz.

Analysis

The coherence between SCM and SPL multi-unit EMG and between SPL EMG and acceleration were analysed off-line on a PC using Spike 3 software and programs written by D. M. Halliday (Halliday *et al.*, 1995; Amjad *et al.*, 1997). A Fourier transform up to a frequency of 100 Hz was performed on separate segments of data of equal length (2048 data points unless stated otherwise). The data from each of the segments were then averaged and the autospectra, cross-spectra, coherence and associated phase were calculated. Coherence is a normalized, unitless value which ranges from 0 (linear independence) to 1. The frequency resolution was 0.48 Hz in coherence and phase spectra and the cumulant density function had a bin width of 1 ms. Finally, the data were Hanned using a moving average filter.

Frequency domain analysis

The coherence between SCM and SPL EMGs was pooled using an average of the individual coherence data weighted according to the length of the recording. To calculate the difference in coherence between the groups, the square root of the coherence was transformed using a variance-stabilizing transform (Fisher transform) to give data whose variance was given by $1/2L$, where L is the number of segments used to calculate the individual coherence. The data were then weighted by multiplying them by the inverse of their variance. The transformed and weighted data for each of the two groups were compared using a repeated measures general linear model across the frequency ranges indicated. *Post hoc* testing at each frequency over the frequency bands 3.9–6.8 and 10.2–14.6 Hz was performed using one-way ANOVA (analysis of variance). Data were considered to be significant if $P < 0.05$. The incidence of coherence between the SCM and the SPL above the 99% confidence level over the frequency band 4–7 Hz was also calculated for the pooled data of each individual patient and control subject. CL for coherence and autospectra were calculated as described previously (Halliday *et al.*, 1995).

The latency difference between the SCM and SPL EMGs was calculated from pooled phase data over frequency bands if at least four contiguous data points exceeded the 95% CL in the corresponding coherence spectra. Delays were calculated from the slope of the line fitted by linear regression according to the following formula: latency $= \Delta\phi/2\pi\Delta f$, where ϕ is the phase and f is the frequency.

To determine whether the torticollis patients and control subjects made the same amount of movement, acceleration was analysed with a segment length of 8192 data points to give power spectra with a frequency resolution of 0.12 Hz. Patients and controls were then compared at each frequency from 0.12 to 1.1 Hz using one-way ANOVA.

Time domain analysis

To obtain a measure of association in the time domain, the inverse Fourier transform of the cross-spectrum was calculated to determine the cumulant density. The pooled cumulant density was also calculated using a weighted average of the contributing data. The 95% confidence limits were calculated as described previously (Amjad *et al.*, 1989). The width of any peak in the cumulant density estimate was defined as the interval between crossings of the 95% confidence limits (sustained for at least five consecutive points), with the exception of the secondary peak in Fig. 2, which was estimated by eye using screen cursors. The latency of any peak was defined as the timing of the bin with the largest value. The area of a peak was the area bounded by the curve and the 95% confidence limits, with the exception of the secondary peak in Fig. 2, which was given by the area bounded by the curve and a level of 2 arbitrary units (the point at which the curve changed gradient).

Results

The examples of raw EMG and head acceleration records shown in Fig. 1 are from a healthy subject mimicking torticollis (A), a patient with a simple dystonic torticollis (B) and a patient with a tremulous dystonic torticollis (C). Four principal measures were derived by application of the Fourier methods: autospectra, coherence spectra, cumulant density functions and phase.

EMG autospectra

The pooled autospectra of the SCM EMG and SPL EMG during sustained contraction in the patient and control groups are shown in Figs 2A, 2B, 3A and 3B. The SPL showed a significantly higher peak at ~12 Hz in controls than in patients. In the torticollis group none of the individual patients showed a significant peak in the SPL autospectrum above 10 Hz, while seven out of eight control subjects did so (Table 1). In the moving controls (those mimicking head tremor), all five subjects showed a significant peak at ~12 Hz. (A similar feature was only seen in the SCM during movement among controls, when it was again absent in patients.)

EMG–EMG coherence

In the patient group, pooled spectra of the coherence between the SCM and SPL during involuntary contraction revealed

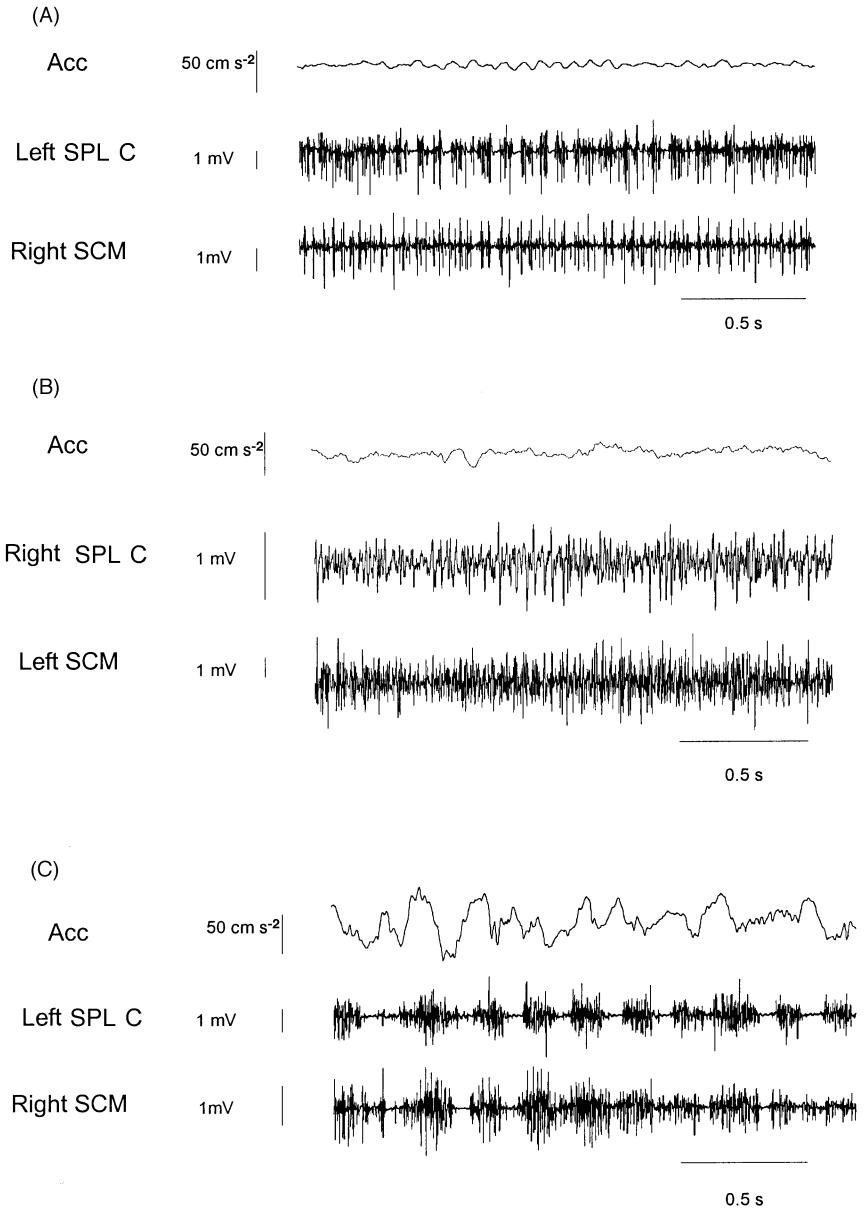

Fig. 1 Representative examples of raw unrectified EMG and head acceleration (Acc) records. (**A**) Control subject during voluntary rotation of the neck 35° to the left. (**B**) Patient with simple dystonic torticollis, in whom the neck was involuntarily rotated 30° to the right. (**C**) Patient with a tremulous dystonic torticollis in whom the neck was involuntarily rotated 35° to the left.

significant coherence in a band from 0.5 to 14.2 Hz, with a large peak below 7 Hz (Fig. 2C). There was also a small peak at ~25 Hz due to activity in case 5, which disappeared when this patient was omitted from the analysis. During voluntary sustained contraction in the control group, coherence was significant in the bands 0.5–1.5 Hz and 8.1–15.1 Hz (Fig. 3C). There was no significant difference between earlier and later trials. Thus, there was no increase in coherence due to possible fatigue.

Coherence differed significantly between the two groups from 3.9 to 6.8 Hz ($P = 0.008$). This low-frequency band was seen in seven out of eight patients and in only one of the eight control subjects (Table 1). In the moving control group, none of the five subjects showed significant coherence in the individual spectra from 4 to 7 Hz. In patients with torticollis the coherence at 10–14 Hz was low. Six out of eight of the spectra from individual patients showed small but significant coherence at this frequency. In contrast, a peak in coherence at 10–14 Hz was found in all the healthy subjects (together with a corresponding peak in the spectra of head acceleration). However, the difference between the groups in coherence at 10–14 Hz was not significant ($P = 0.056$).

To investigate whether the low-frequency drive found in the patient group was related to head tremor, the three patients with a slightly tremulous torticollis were compared with the

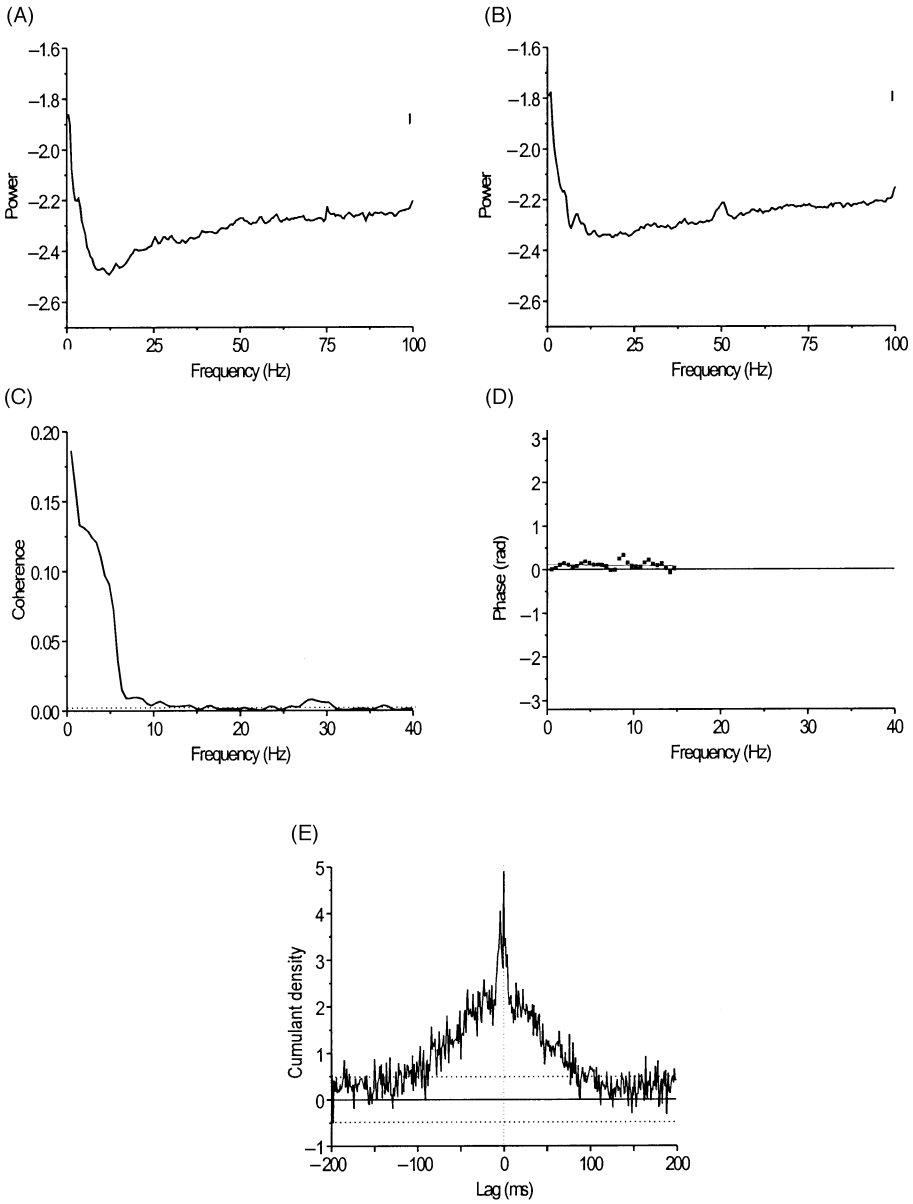

Fig. 2 Pooled results in all eight patients. (**A**) Autospectrum of SCM EMG. (**B**) Autospectrum of contralateral SPL EMG. (**C**) Coherence spectrum of SCM–SPL. (**D**) Phase spectrum. (**E**) Cumulant density estimate. The SCM and SPL show strong coherence up to 7 Hz and the activities are in phase (simultaneous). The central peak reflects the latter in the cumulant density. This central peak consists of a broad base and a superimposed narrower peak. In this and the following figures, power is plotted on a log scale and power and cumulant density are in arbitrary units. Vertical bars give the magnitude of the upper and lower 95% confidence limits in power spectra, the horizontal dotted lines are 95% confidence limits in coherence spectra and cumulant density estimates, and the short thin line in the phase spectrum is the linear regression line.

five patients without a tremor. The patients with the tremulous torticollis showed a pooled coherence spectrum from 0 to 7 Hz similar to that of the five patients without tremor.

The three patients with a slightly tremulous torticollis were also compared with five healthy subjects imitating head tremor about the yaw axis (Fig. 4). Coherence still differed significantly between the two groups from 3.9 to 6.8 Hz ($P = 0.026$).

Phase

The EMG–EMG phase results for the patients, controls and moving controls are shown in Figs 2–4. In the patient group, the SPL and SCM were in phase over the frequency band 0.5–14.2 Hz, the latency being –0.1 (± 7.2, 95% CL) ms. In the control group, the SCM led the SPL by 13.5 ± 1.0 ms over the frequency band 8.3–15.1 Hz. Similar results were found in tremulous patients and moving controls.

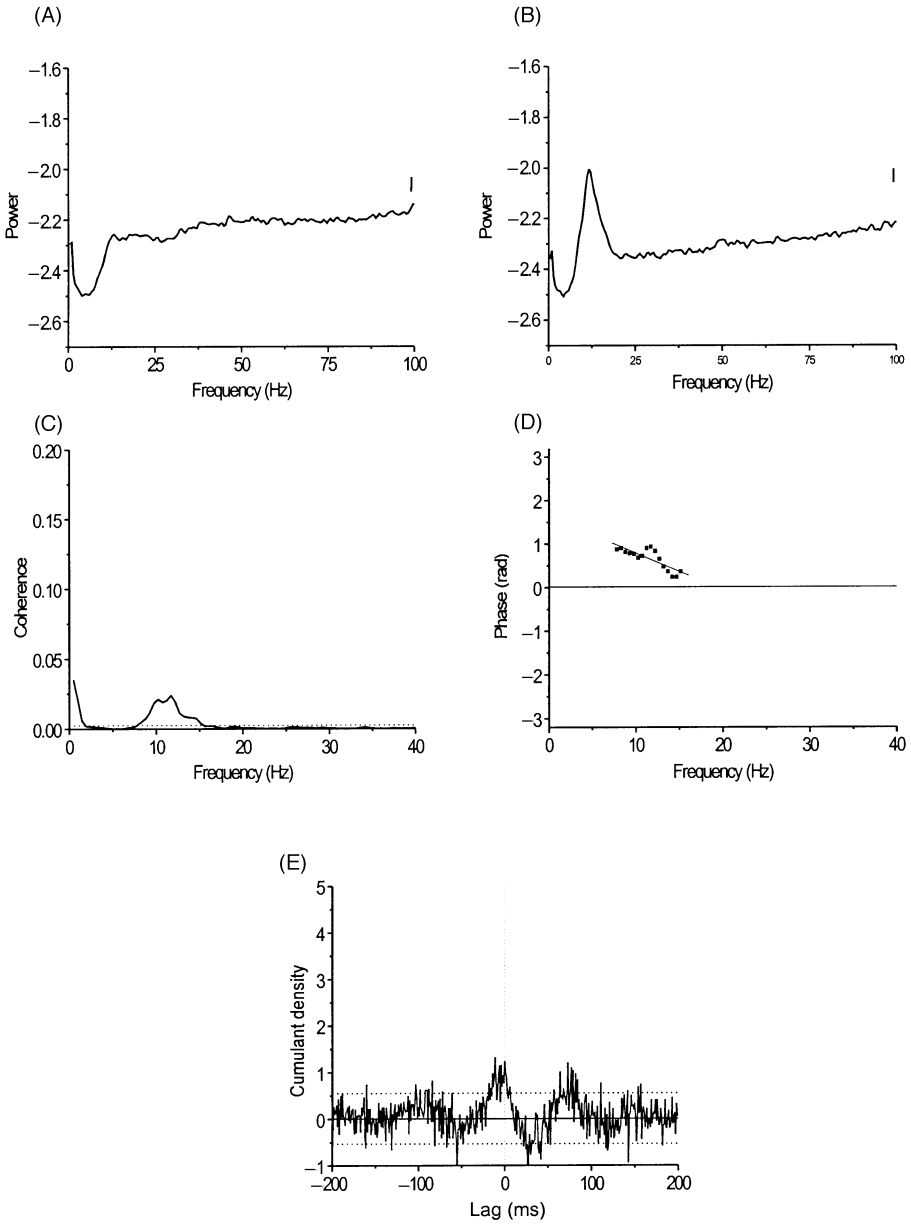

Fig. 3 Pooled results in eight healthy, age-matched controls making sustained contractions of the SCM and the contralateral SPL. (**A**) Autospectrum of SCM EMG. (**B**) Autospectrum of contralateral SPL EMG. (**C**) Coherence spectrum of SCM–SPL EMG. (**D**) Phase spectrum. (**E**) Cumulant density estimate. Note that there is a clear peak in the autospectrum of the SPL at ~12 Hz, which was absent in the patients (Fig. 1B). The SCM and SPL showed no significant coherence beyond 2 Hz, except for a peak at ~12 Hz, which was absent in the patients (Fig. 1C). The SCM phase-led the SPL by 13.5 ± 1.0 ms (95% confidence level). The phase difference is reflected by the offset in the peak in the cumulant density (width 17 ms, peak at ~–11 ms). Figs 1 and 2 are plotted at the same scale.

Table 1 *Factors discriminating between torticollis patients and controls*

	Significant peak >10 Hz in autospectra of SPL EMG* (%)	Significant coherence from 4 to 7 Hz between SCM EMG and SPL EMG† (%)
Sensitivity	88	89
Specificity	100	89

Power and coherence spectra were calculated with a resolution of 0.48 Hz and Hanned. The results are from all eight patients and the static controls. Tremor or movement did not lower the specificity or sensitivity. *>95% confidence level; †>99% confidence level.

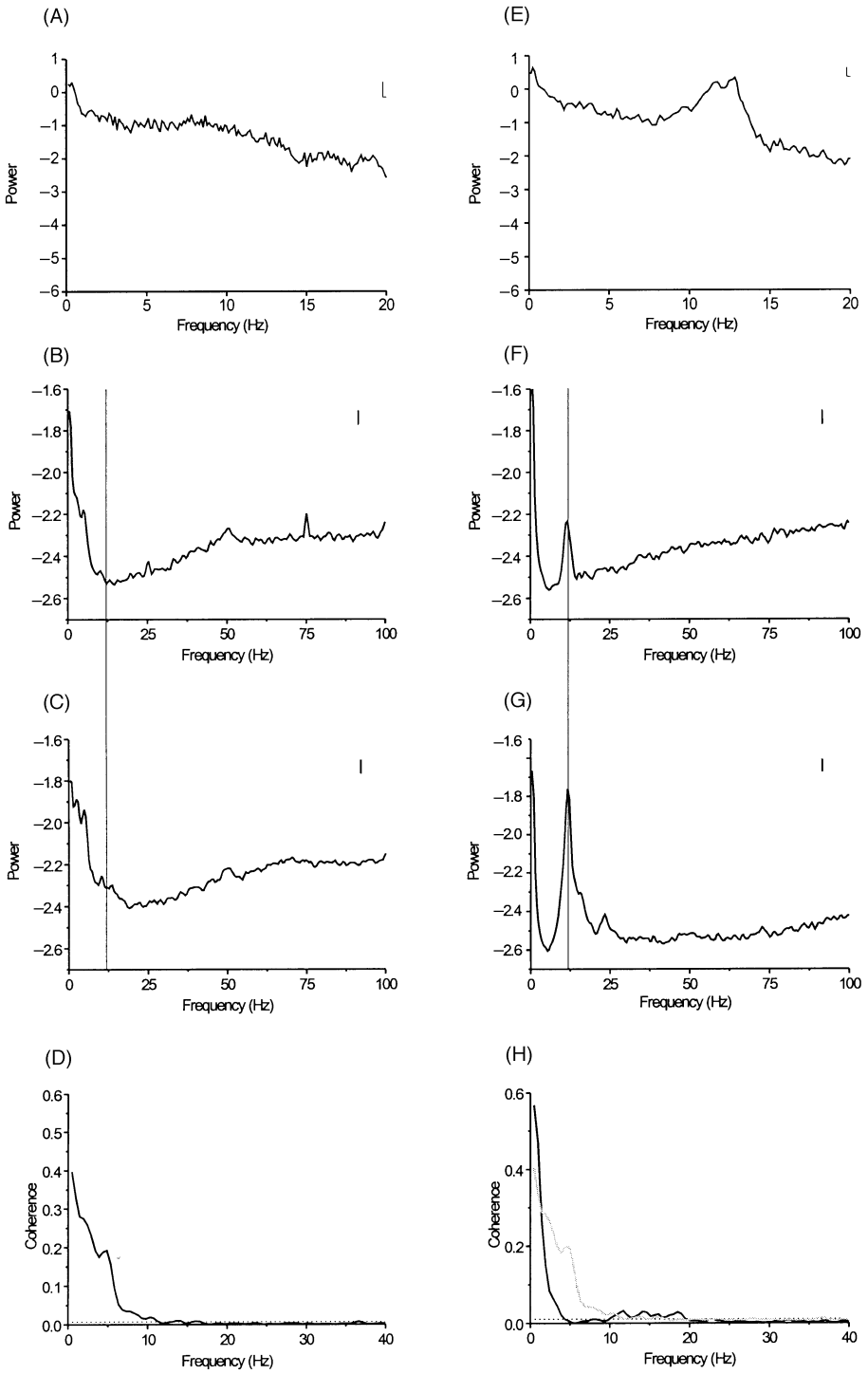

Fig. 4 Pooled results in three patients with tremulous torticollis (**A**–**D**) and five healthy control subjects (**E**–**H**) making phasic contractions of the SCM and the contralateral SPL at ~0.2 and ~0.5 Hz. Representative examples of autospectra of head acceleration from a patient (**A**) and control (**E**), showing that the two were well matched, with the exception that controls had a peak at ~12 Hz. Pooled autospectra of SCM EMG in patients (**B**) and controls (**F**). Pooled autospectra of SPL EMG. There was a clear peak at 12 Hz (thin vertical line) in controls (**G**), which was absent in patients (**C**). Coherence spectra of SCM–SPL, showing that patients had a broad band of high coherence extending out to ~7 Hz (**D**), in contrast to controls (**H**). In **H**, the coherence spectrum of the patients is shown in grey for ease of comparison.

Thus, EMG–EMG phase relationships tended to be different between controls and patients, as reflected in the peak offsets in the cumulant density estimates (see below). However, this was a group effect and could not be used to distinguish individual patients.

Cumulant density functions

Time domain representations (cumulant density estimates) were derived from the frequency domain measures by application of the inverse Fourier transform. The pooled cumulant density estimate in the patient group showed a significant central peak at 0 ms, with a width of 216 ms (Fig. 2E). A second central peak was seen on top of the first peak, with an estimated width of 13 ms. Case 5 showed significant pooled coherence at ~25 Hz. This drive is known to contribute to short-term synchronization (Farmer *et al*., 1993*b*). The pooled cumulant density of the patient group did not change when this patient was excluded from the analysis. In the control group the pooled cumulant density showed a significant but much smaller peak at –11 ms, with a width of 17 ms (Fig. 3E). The area of this peak was only 36% of that of the central subpeak in the patient group.

Principal factors discriminating between torticollis patients and controls

These are summarized in Table 1.

Discussion

Abnormal descending drive in dystonic torticollis

In the present study we have identified a number of important differences between patients with torticollis and healthy subjects in the way in which neck movements are organized. The most robust of these findings was the presence of a synchronizing drive at 4–7 Hz that was common to the SCM and the SPL during involuntary contraction in patients with rotational torticollis, which was not detected in healthy age-matched control subjects. It seems unlikely that this coherence at low frequency was entirely related to head tremor, as exclusion of the three patients with clinically tremulous torticollis did not change the pattern of coherence. Moreover, coherence between 4 and 7 Hz was significantly less in controls imitating head tremor than in patients with torticollis. Finally, in patients with more complicated forms of torticollis, a drive at 4–7 Hz may be found between different muscle pairs, in the absence of coherence at the lower frequencies (0.5–4 Hz) involved in head movement (personal observation).

Conversely, there was a tendency for reduced coherence at 10–14 Hz in the patients with torticollis compared with healthy controls, who showed a discrete peak in this band. A peak of similar frequency in the autospectrum of the voluntarily contracting SPL and of head tremor was related to this. These bands were absent in torticollis. The phase relationships between the SPL and the SCM were also different between patients and healthy controls, and they provide substantial evidence that the coherence at ~5 Hz in the patients was not simply due to the dropping of alternate beats of the normal oscillatory drive at 10–12 Hz.

The nature of the abnormal 4–7 Hz drive common to the SPL and SCM in torticollis was further defined by the time domain analyses. The pooled cumulant density estimates in the patients and controls were dominated by broad-peak motor unit synchronization with widths of 216 and 17 ms, respectively. These results suggest the synchronization of the presynaptic inputs to motor neurons in both patients and controls, as might be expected in synergistic muscles (Kirkwood *et al.*, 1982, 1984).

Alterations in the pattern of organization of involuntary dystonic movements have also been reported in patients with upper limb dystonia, all of whom had structural lesions or primary generalized torsion dystonia (Farmer *et al.*, 1998). In contrast to the agonist muscle pair SPL–SCM, investigated in the present study, limb dystonia was investigated in antagonist muscle pairs. Coherence was found in the 1–13 and 14–33 Hz bands, a picture normally restricted to closely related agonist muscles in the limb. The coherence found between 1 and 13 Hz would encompass the abnormal drive found between agonist muscles in cervical dystonia, but coherence in the 14–33 Hz band was absent in all but one of our patients. This might be due to differences in the underlying pathology. Alternatively, it might reflect the fact that the common presynaptic drive is at a lower frequency in the neck than in the hand during voluntary contraction, as suggested by our multi-unit results in controls and by single-unit studies of the SCM and SPL (J. F. Marsden and P. Brown, personal observations).

Diagnostic use of frequency analysis

The absence of a peak at 10–12 Hz in the autospectra of the SPL and the presence of significant coherence between the SCM and the SPL at 4–7 Hz are sensitive and specific features distinguishing dystonic torticollis from voluntary contraction, regardless of whether the latter is sustained or phasic. Thus, frequency domain analysis of multi-unit EMG spike activity may provide a diagnostic test for involuntary torticollis. The multi-unit recordings used here were simple to perform, well tolerated and could be completed within 30 min. Further study is necessary to determine if differences in the detailed pattern of common drive may also serve to separate idiopathic from secondary dystonias.

Pathophysiological implications

As summarized in the introduction, several lines of evidence point to an overexcitable motor cortex in dystonic torticollis. There are two features of the abnormal descending drive in

torticollis that are consistent with activity in the direct corticoreticular and corticospinal pathway.

First, ipsilateral SPL and contralateral SCM were in phase in the patients. The lack of any major difference in latency between these muscles is similar to the results obtained following magnetic stimulation of the motor cortex (Berardelli *et al.*, 1991), and is compatible with the more or less simultaneous activation of the SCM and the SPL via direct corticoreticular and corticospinal pathways. In contrast, in the controls the SCM led the SPL by ~13 ms during voluntary tonic contraction, which indicates that the motor system responsible for normal voluntary neck turning is, at least in part, distinct from the direct pathways that are synchronously activated by magnetic stimulation of the motor cortex.

Secondly, in the patients the pooled cumulant density estimate suggested that a narrow subpeak of ~13 ms in duration was superimposed upon the broad base due to the synchronization of presynaptic inputs to the motor neurons. Its presence raises the possibility of a common presynaptic drive to motor neurons from last-order axons branching to both the SCM and the SPL, given the dispersion due to variable central and peripheral conduction delays (for discussion, see Farmer *et al.*, 1991) and pooling across subjects. This finding requires confirmation as it implies that cervical dystonia may involve branched presynaptic inputs to motor neurons of the SCM and the SPL, in addition to the strong synchronization of presynaptic inputs at abnormally low frequencies. Such branching and short-term synchronization is usually associated with the corticospinal tract (Farmer *et al.*, 1990, 1991, 1993; Datta *et al.*, 1991), but it is interesting to note that time- and frequency-domain analyses have also implicated the direct corticospinal pathway in the co-activation of antagonist limb muscles seen in dystonia of the limb (Farmer *et al.*, 1998).

One further deviation from normality deserves comment. There was a tendency for the synchronization in the 10–12 Hz frequency band to be impaired, as seen in the coherence spectra and autospectra of the SPL, in patients with dystonic torticollis. It has been suggested that the olivary–cerebellar system may be responsible for rhythmic activity at this frequency (Llinas, 1991) and that such oscillations may have a role in sensorimotor integration (Nicolelis *et al.*, 1995). It has been widely hypothesized that deficiencies in sensorimotor integrations contribute to dystonia (Berardelli *et al.*, 1998), and the impairment in oscillatory activity at ~10 Hz, as reflected in the pattern of muscle discharge, may relate to difficulties in sensorimotor integration in this condition (Dauer *et al.*, 1998).

Conclusion

In summary, the lack of any phase difference and the suggestion of short-term synchronization between the SCM and the SPL would be consistent with an abnormal corticoreticular and corticospinal drive in dystonic torticollis. This, in turn, is concordant with the conclusions of comparable studies of antagonist muscle pairs in upper limb dystonia (Farmer *et al.*, 1998) and with studies suggesting increased excitability of the motor cortex in torticollis (Galardi *et al.*, 1996; Hanajima *et al.*, 1998; Kanovsky *et al.*, 1998). In addition, the pattern of SPL autospectra and of SCM–SPL coherence may provide a sensitive and specific feature distinguishing dystonic from psychogenic torticollis. It remains to be seen whether similar abnormalities serve to distinguish dystonia from psychogenic spasm in the limb.

Acknowledgements

We thank Dr K. Bhatia and Dr A. J. Lees for permission to study their patients, and Professor J. Rosenberg and Dr D. Halliday for loan of some of the analysis software. This project was supported by the TMR Programme of the Commission of the European Union (EU Grant no. BMH4-CT98-5112), the foundation Drie Lichten and Stichting Catharine van Tussenbroek, The Netherlands.

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Received August 6, 1999. Revised November 5, 1999.
Accepted November 8, 1999