

Essential tremor and cerebellar dysfunction: abnormal ballistic movements

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Background: Clinical characteristics reminiscent of cerebellar tremor occur in patients with advanced essential tremor. Ballistic movements are known to be abnormal in cerebellar disease. The hypothesis was proposed that ballistic movements are abnormal in essential tremor, reflecting cerebellar dysfunction.

Objective: To elucidate the role of the cerebellum in the pathophysiology of essential tremor.

Methods: Kinematic parameters and the triphasic electromyographic (EMG) components of ballistic flexion elbow movements were analysed in patients assigned to the following groups: healthy controls (n = 14), pure essential postural tremor (ET_{PT}; n = 17), and essential tremor with an additional intention tremor component (ET_{IT}; n = 15).

Results: The main findings were a delayed second agonist burst (AG₂) and a relatively shortened deceleration phase compared with acceleration in both the essential tremor groups. These abnormalities were most pronounced in the ET_{IT} group, which had additional prolongation of the first agonist burst (AG₁) and a delayed antagonist burst (ANT).

Conclusions: Abnormalities of the triphasic pattern and kinematic parameters are consistent with a disturbed cerebellar timing function in essential tremor. These abnormalities were most pronounced in the ET_{IT} group. The cerebellar dysfunction in essential tremor could indicate a basic pathophysiological mechanism underlying this disorder. ET_{PT} and ET_{IT} may represent two expressions within a continuous spectrum of cerebellar dysfunction in relation to the timing of muscle activation during voluntary movements.

Essential tremor is the most common movement disorder.^{1,2} Although often termed "benign essential tremor," it often causes severe disability of hand function.³ A reason for this may be an intention tremor component frequently adding to the classical postural tremor, especially in more advanced stages.^{4–7} The pathophysiology of essential tremor is still not completely understood. Increasing evidence from neurophysiological and positron emission tomography studies indicates that cerebellar dysfunction plays an important role in essential tremor.⁸ This hypothesis is supported clinically by tremor characteristics reminiscent of cerebellar disease and disturbed tandem walking in the advanced stages of this disorder.^{9–10}

Analysis of ballistic joint movements has proved a useful tool for the examination of cerebellar function.¹¹ In this paradigm, the characteristic triphasic electromyographic (EMG) burst pattern in the involved antagonistic muscle pair can be seen occurring in the following sequence: the first agonist burst (AG₁) initiates and accelerates movement, while the following antagonist burst (ANT) decelerates it; subsequently, in healthy persons the second agonist (AG₂) dampens the oscillations induced by this decelerating process.^{11,12} This triphasic pattern is not simply related to reflex mechanisms using peripheral feedback as a result of movement, but rather seems to be preprogrammed in the central nervous system, as it also occurs in deafferented subjects.^{13–15} In addition, the AG₂ burst is found still to be present when the motor efference to the antagonist muscle is blocked by lidocaine.¹⁶

Typical findings in patients with cerebellar disease include increased movement overshoot, delayed muscle activations within the triphasic EMG pattern, and an asymmetry of the velocity profile characterised by a shortened deceleration phase.^{17–19} Using this paradigm in patients with essential tremor, Britton *et al* found delayed AG₂ bursts.²⁰ The kinematic result was a shortened deceleration phase caused by an unop-

posed braking function of the antagonistic muscle. The movements were followed by some oscillations, the periods of which correlated with the delay of AG₂.

A previous clinical study showed that almost half the patients with essential tremor have intention tremor.⁴ As this feature is commonly seen in cerebellar disease, we proposed the hypothesis that patients with intention tremor show more pronounced electrophysiological and kinematic abnormalities than those with postural tremor only. To test this hypothesis, we compared the triphasic pattern in two groups of patients with essential tremor: those without (ET_{PT}) and those with an intention tremor component (ET_{IT}) accompanying the classical postural tremor. The ET_{IT} group was different from ET_{PT} in that they had a tendency to longer disease duration, were significantly older, and had higher amplitudes of postural tremor on quantitative tremor analysis, indicating an advanced stage of disease. These two groups were compared with a group of healthy controls. If a slowly progressing cerebellar dysfunction underlies essential tremor and is responsible for the development of intention tremor, the abnormalities of ballistic arm movements can be expected to be more pronounced in the ET_{IT} group.

Abbreviations: AG₁, first agonist burst within the triphasic electromyographic (EMG) pattern; AG₂, second agonist burst within the triphasic EMG pattern; A_{max}, maximum acceleration of movement; ANT, first antagonistic burst within the triphasic EMG pattern; ET_{IT}, essential tremor with an additional intention tremor component; ET_{PT}, essential tremor with an isolated postural tremor; T₁, time required to accelerate the movement from zero to maximum velocity; T₂, time required to decelerate the movement from maximum velocity to zero; TP, tremor period (duration of one tremor cycle); TP_{ball}, period of tremor induced by a ballistic movement; TP_{post}, period of postural tremor derived from spectral analysis; V_{max}, maximum velocity of movement

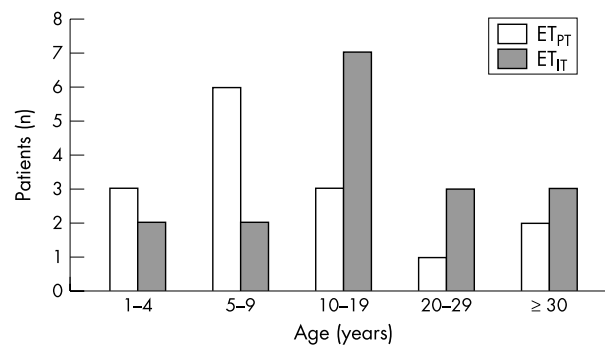


Figure 1 Distribution of disease duration. All patients are assigned to categories of increasing disease duration. A tendency towards a shorter duration in the ET_{PT} group (white columns) and a longer duration in ET_{IT} group (grey columns) can be seen, though the differences failed to reach statistical significance.

Some of these data have been published as preliminary results.²¹

METHODS

Subjects

Patients with the diagnosis of “definite essential tremor” according to the criteria published by Findley and Koller²² were recruited from the outpatient department for movement disorders of the Freiburg University neurological clinic. However, we diverged from those criteria in that we reduced the minimum disease duration from five to two years in order to include patients in the early stages of essential tremor. Seventeen patients with isolated postural tremor and 15 with additional intention tremor were assigned to the ET_{PT} and ET_{IT} group, respectively. In addition to tremor, we found mildly to moderately disturbed tandem walking in two patients in the ET_{PT} group and eight patients of the ET_{IT} group. One ET_{PT} patient and 12 ET_{IT} patients had mild to moderate dysdiadochokinesis. However, it may be difficult to distinguish dysdiadochokinesis from an interference of tremor with hand function. Thus this sign was only judged as positive when, in the light of clinical experience, the dysdiadochokinesis was clearly more severe than could be explained by a mere superimposition of tremor. Patients showing bradykinesia or disturbed postural reflexes were excluded. Treatment with β blocking agents or primidone did not preclude inclusion.

Two patients with intention tremor and one without intention tremor were excluded from the study before the analyses because they were unable to perform the monophasic ballistic movements required for our task adequately. In addition, 14 approximately age matched, healthy controls were examined.

Mean (SD) ages were 62.5 (12.4) years (range 40 to 90) for the controls; 64.3 (11.3) years (50 to 84) for the ET_{PT} group; and 71.0 (9.2) years (49 to 82) for ET_{IT} group. The controls and the ET_{PT} group did not differ significantly in age, but the ET_{IT} group was older ($p < 0.05$). Disease duration tended to be shorter in the ET_{PT} group (12.9 (10.8) years) than in the ET_{IT} group (17.6 (11.8) years). However, this difference did not reach statistical significance. Figure 1 shows the predominance of shorter disease durations in the ET_{PT} group and the longer disease course in the ET_{IT} group. All subjects were informed about the experimental procedures and gave consent to their participation in the study.

Quantitative analysis of postural tremor

Postural upper limb tremor is commonly measured in the distal rather than the proximal segments.²³ We therefore analysed postural tremor at the wrists. Tremor was recorded with both forearms supported and with the unloaded hands raised horizontally against gravity. Accelerometers were taped to the

backs of both hands. Tremor was measured at time intervals of 30 seconds. The methods of data acquisition and subsequent processing by spectral analysis have been described in detail by Timmer *et al.*²⁴

Ballistic arm movements

As cerebellar dysmetria is usually more pronounced in proximal than distal segments of an individual limb, we decided to analyse ballistic arm movements at the elbow joint rather than at the wrist. The patients sat comfortably in a chair. One arm was fixed to a manipulandum of low inertia. The manipulandum was pivoted next to the elbow joint, allowing free comfortable flexion-extension movements of the forearm. The mechanical load of the manipulandum was small and was functionally minimised by performing the movements in the horizontal plane. The angular displacements of the manipulandum were constantly registered by a goniometer and fed back visually by a moving point on a computer screen. The subjects had to move this point between two targets. These targets alternated between right and left on the screen at a distance of 15 cm (6 in), corresponding to an elbow movement of 40° in amplitude. The screen target alternated at intervals randomised between two and three seconds in order to avoid automated movements. Movement onsets were self timed within these periods by the subjects.

The subjects were instructed to perform the movements as rapidly as possible (primary goal) and to perform the movements with high accuracy (secondary goal). Before the experiments were started, the subjects performed several training sweeps. Fifteen flexion movements of each subject's dominant arm were analysed. Maximum velocity (V_{max}) and acceleration (A_{max}) were obtained off-line by digital differentiation of the position signal. According to the approach described in previous studies,^{19,20} movement time was divided into two subunits: T_1 as the time required from movement onset until peak velocity is reached (acceleration), and T_2 as the time from peak velocity to a velocity of zero (deceleration). The ratio T_1 to T_2 was determined. Errors of movement amplitude were also analysed.

Electromyographic pattern of activation

During the ballistic movements, EMG activity was recorded by surface electrodes attached over the agonist (biceps) and antagonist (triceps) muscle of movement. EMG signals were amplified and full wave rectified. Data were analysed off-line by inspecting the single trials on a computer screen. The burst onsets and offsets were marked by an experienced researcher (BG), following the criteria applied by Britton *et al.*²⁰ The onset of a burst was defined as the point when EMG activity abruptly exceeded the baseline noise level. The burst onsets and offsets were sufficiently clear to be determined visually in the great majority of trials. However, at variance to the procedure of Britton *et al.*²⁰ if occasionally a burst onset or offset could not be clearly determined, the respective trial was excluded from analysis. It was then replaced by a subsequent sweep which was recorded as a reserve in order to get the full number of 15 sweeps for each case. The individual burst latencies related to movement onset and burst durations were then determined.

Statistical analysis

The data were tested for group differences by the non-parametric Kruskal-Wallis test; a posteriori tests were performed by the corrected distribution for pairwise comparisons. The level of statistical significance was set at 5%. Correlations were determined using linear regression analysis based on Pearson's product moment coefficient.

Tremor periods

In 10 of the ET_{PT} patients and 14 of the ET_{IT} patients three or more EMG oscillations were induced by the ballistic elbow

Table 1 Data from spectral analysis

	ET _{PT}	ET _{IT}
Total power (mg ²)	6.85 (11.6)	200.3 (599.1)
Frequency (Hz)	6.42 (0.65)	5.10 (0.69)

Values are mean (SD).

The mean amplitudes (total power) and peak frequencies of postural tremor are given for the two patient groups. Tremor was recorded in the more affected hand of each patient. Significant group differences were found for both variables ($p < 0.05$).

movements. These could be identified and clearly delimited from baseline noise after averaging 15 sweeps of elbow flexion. Analyses of the durations of individual single electromyographic tremor cycles (tremor periods, TP_{ball}) derived from the individual ballistic movements were done off-line for each patient. The duration of each tremor cycle was determined between two subsequent ascending zero crossings of the curve. In addition, another TP value (TP_{post}) was determined, based on recordings of postural tremor at the wrist. The EMG time series of postural tremor recorded by tremor analysis were transformed by spectral analysis. We determined TP_{post} from the resulting peak spectral frequencies (f) by calculating $1/f$. In order to evaluate the dependency between tremor frequency and the timing of individual EMG bursts during ballistic movements, we analysed the correlations between burst latencies and these two types of tremor period, using linear regression analysis.

RESULTS

Spectral analysis of the tremor

Spectral analysis of the accelerometric data during postural activation showed significantly lower tremor frequencies and

higher amplitudes in the ET_{IT} group than in the ET_{PT} group ($p < 0.05$; table 1).

Kinematic parameters of the ballistic movements

After careful instruction and several training sweeps, all subjects performed monophasic rapid arm movements. The three groups produced averaged movement overshoots of 5.6° (controls), 6.3° (ET_{PT}), and 7.2° (ET_{IT}), respectively. There were no significant group differences ($p > 0.05$); this can be attributed to the high variance of these data. V_{max} was not significantly different between the three groups, while A_{max} was lower in the ET_{IT} group than in the ET_{PT} group or the controls (table 2). The ratio T_1 to T_2 , reflecting the relation between the times required for movement acceleration and deceleration, was significantly higher for ET_{PT} than for the controls, and for ET_{IT} than for ET_{PT} (table 3), indicating a more abrupt deceleration phase in the latter groups.

Triphasic EMG patterns

All movements were accompanied by the characteristic triphasic EMG pattern consisting of the bursts AG₁, ANT, and AG₂. Figure 2 shows the typical kinematics and triphasic patterns of ballistic movements in a healthy subject (A) and in a patient with ET_{IT}. An overview of burst durations and latencies is given in table 2. In summary, the AG₁ bursts were longer in the ET_{IT} group than in the ET_{PT} group, and in the ET_{PT} group than in the controls, while the durations of ANT and AG₂ were not different. The latencies of ANT were significantly prolonged in the ET_{IT} group and only slightly in the ET_{PT} group, without reaching significance. The ET_{PT} group showed a significant delay in AG₂ compared with the controls.

Correlations between tremor periods and burst latencies

As mentioned above, tremor periods (TP) were analysed in two different ways, from a different database and from

Table 2 Group analysis of ballistic elbow movements. Left panel: individual group values; right panel: group differences

	Mean values (SD)			Group differences		
	Control	ET _{PT}	ET _{IT}	Control v ET _{PT}	Control v ET _{IT}	ET _{PT} v ET _{IT}
Duration AG ₁ (ms)	114.0 (13.5)	142.5 (25.9)	178.1 (41.7)	*	*	**
Duration ANT (ms)	140.9 (22.0)	120.2 (31.1)	122.9 (25.6)	NS	NS	NS
Duration AG ₂ (ms)	129.6 (35.4)	128.6 (32.9)	164.4 (32.9)	NS	NS	NS
Latency AG ₁ (ms)	-77.4 (13.5)	-82.7 (17.7)	-83.7 (14.4)	NS	NS	NS
Latency ANT (ms)	55.3 (18.6)	67.6 (27.6)	95.3 (41.7)	NS	*	NS
Latency AG ₂ (ms)	125.5 (14.2)	159.8 (32.2)	199 (42.8)	**	**	*
Diff AG ₂ -ANT (ms)	70.3 (19.0)	92.2 (31.3)	103.7 (27.8)	NS	**	NS

Analyses are of average burst durations and latencies.

* $p < 0.05$; ** $p < 0.001$.

AG₁, first agonist burst within the triphasic electromyographic (EMG) pattern; AG₂, second agonist burst within the triphasic EMG pattern; ANT, first antagonistic burst within the triphasic EMG pattern; diff, difference between the latency of two respective bursts; ET_{IT}, essential tremor with additional intention tremor component; ET_{PT}, essential tremor with isolated postural tremor.

Table 3 Analysis of kinematic parameters

	Mean values (SD)			Group differences		
	Control	ET _{PT}	ET _{IT}	Control v ET _{PT}	Control v ET _{IT}	ET _{PT} v ET _{IT}
V_{max} (°/s)	333.6 (70.0)	336.3 (56.0)	310.1 (61.1)	NS	NS	NS
A_{max} (°/s ²)	4320.6 (1574.7)	4067.5 (1345.9)	3089.7 (669.3)	NS	**	*
T_1 (ms)	93.86 (16.4)	104.7 (18.0)	131.1 (30.8)	*	**	*
T_2 (ms)	146.2 (31.8)	142.4 (29.5)	141.2 (36.6)	NS	NS	NS
$T_1:T_2$ ratio	0.66 (0.21)	0.78 (0.24)	1.02 (0.42)	*	*	*

* $p < 0.05$, ** $p < 0.001$.

A_{max} , maximum acceleration of movement; ET_{IT}, essential tremor with additional intention tremor component; ET_{PT}, essential tremor with isolated postural tremor; T_1 , time required to accelerate the movement from zero to maximum velocity; T_2 , time required to decelerate the movement from maximum velocity to zero; V_{max} , maximum velocity of movement.

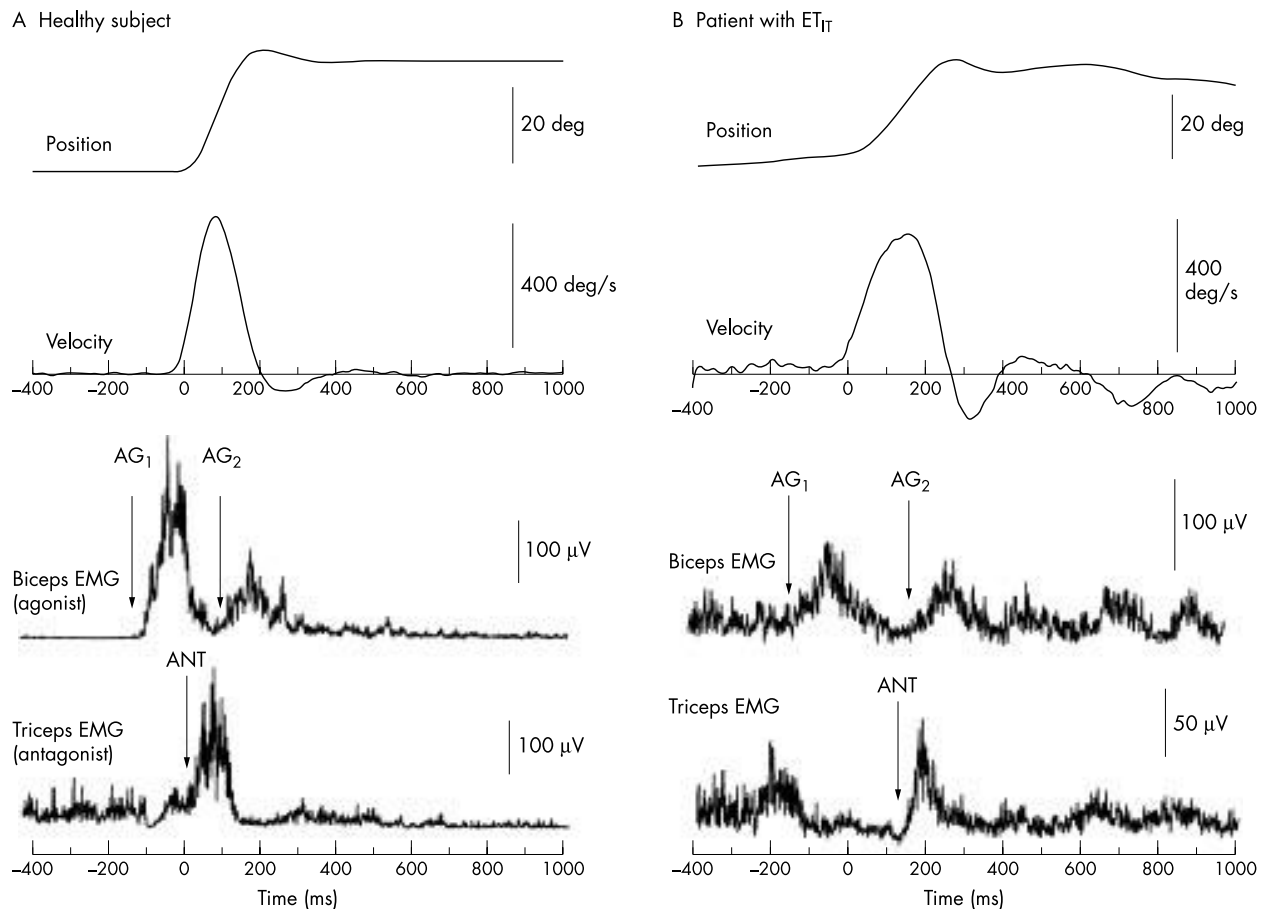


Figure 2 Ballistic elbow movements: representative examples. The agonist and antagonist EMG (lower half) is displayed in relation to the position and velocity signal of the ballistic elbow movements in a healthy control subject (A) and a patient with ET_{pr} (B) (average of 15 movements). The ET_{pr} patient shows the typical abnormalities of the velocity profile and of the triphasic EMG pattern. For a detailed explanation see the text.

different limb segments: (1) directly, by measuring the EMG bursts of tremor induced by the ballistic elbow movements (TP_{ball}); (2) indirectly, by calculating TP from the peak spectral frequency of the EMG time series recorded under postural conditions (TP_{post}). The latter data were recorded from the wrist extensor muscles.

The correlations between these tremor periods and the latencies of individual EMG bursts within the triphasic EMG pattern were determined by linear regression analysis (fig 3). In summary, significant correlations were reached for TP_{ball} between the latencies of ANT and AG_2 for the ET_{pr} group alone as well as for the ET_{pr} and ET_{tr} groups analysed together. For the ET_{tr} group alone, this correlation failed to reach significance, which can be attributed to a greater variability in the data in that group. In relation to TP_{post} group analysis of $ET_{pr} + ET_{tr}$ showed a significant level of correlation with the latency of AG_2 .

DISCUSSION

The clinical signs often present in advanced essential tremor—such as disturbed tandem walking^{9,10} and intention tremor⁴⁻⁷—suggest disordered cerebellar function in this condition. This assumption is supported by studies using positron emission tomography²⁵⁻²⁹ or functional magnetic resonance imaging³⁰ in humans with essential tremor. The results point to an altered state of activation in the olivo-cerebellar system. The triphasic EMG pattern accompanying rapid joint movements is preprogrammed in the central nervous system and reflects cerebellar timing function.^{11,13-16,31} We used this

paradigm in order to investigate cerebellar function in patients with essential tremor with and without an intention tremor component.

Kinematic and electromyographic abnormalities in essential postural tremor

Crucial results suggesting a direct functional relation between cerebellar dysfunction and tremor stem from a study by Britton *et al.*²⁰ who investigated the kinematic parameters and triphasic EMG patterns of ballistic movements in 17 patients with essential tremor. Their chief findings were higher movement overshoots in patients with essential tremor compared with healthy controls, as well as asymmetrical velocity profiles caused by an excessive movement deceleration. In addition, they found a delay of AG_2 , which was correlated with the frequency of the tremor induced by the movements. These investigators concluded that the abnormalities were caused by disturbed cerebellar timing function. Investigating both humans and animals, other investigators have reported similar kinematic findings in cerebellar disorders.¹⁷⁻¹⁹ However, the underlying abnormalities of the triphasic pattern were different in some respects, as studies in established cerebellar lesions have consistently shown prolonged AG_1 burst durations and delayed ANT bursts. Both variables were reported to be normal in the patients with essential tremor examined by Britton *et al.*²⁰

In contrast to their results, our patients did not show significantly increased movement overshoots compared with the controls, which may be explained by the large variance in the data. In some respects, however, our ET_{pr} group was

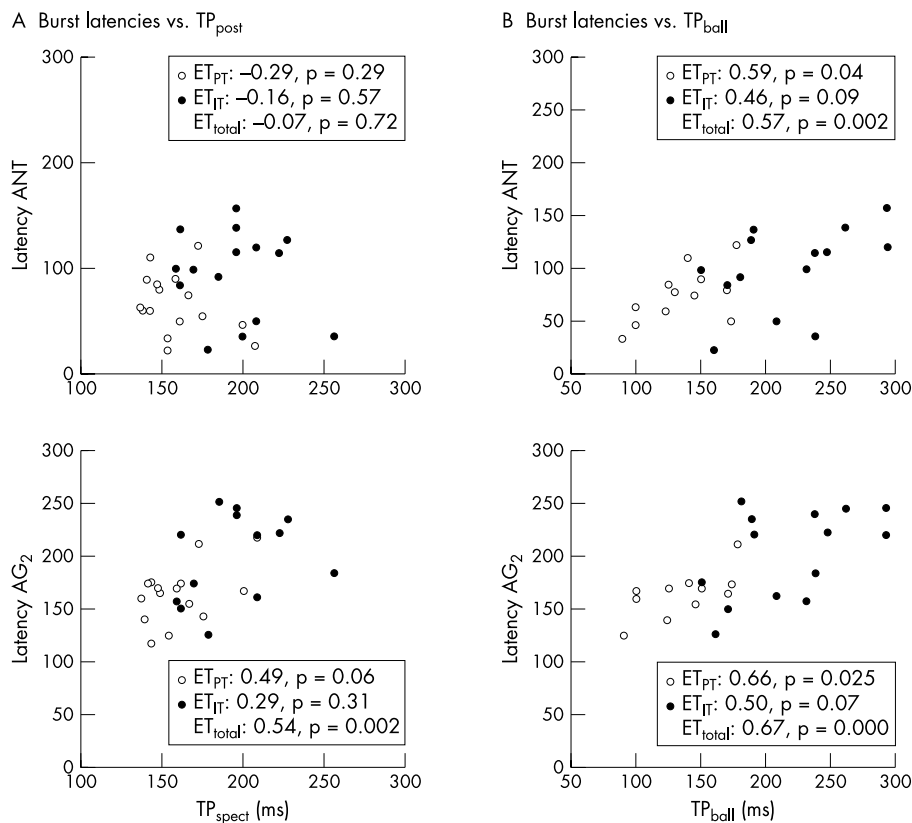


Figure 3 Correlation between tremor periods and burst latencies. The results of linear regression analysis are displayed. The tremor periods following ballistic movements (TP_{ball} , panel A) and the tremor periods derived from spectral analysis of postural tremor (TP_{post} , panel B) were correlated with the latencies of ANT (upper panels) and AG_2 (lower panels) of the triphasic electromyographic pattern. Not all patients had a detectable tremor induced by ballistic movements. Thus the number of data points in the right panel does not equal the total number of patients. The correlation coefficients, significances, and symbols are displayed in the individual sections of the figure.

remarkably similar to the patients with essential tremor investigated by Britton *et al.*,²⁰ as they also had a delayed second agonist burst during ballistic joint movements, and asymmetric velocity profiles with a relatively shortened deceleration phase. Thus their patients, who obviously had a monosymptomatic postural tremor, appear comparable to our ET_{IT} group. However, in contrast with the findings of Britton *et al.*,²⁰ the antagonist bursts seemed slightly delayed, as in previous reports of cerebellar lesions.^{17–19} The functional relevance of this delay and its relation to tremor pathophysiology is suggested by the significant correlation between the delay and the periods of tremor induced by the ballistic movements.

Abnormalities in patients with additional intention tremor

Intention tremor is commonly attributed to a failure of cerebellar function from various causes. We were particularly interested to examine whether this hypothesis, which is based on clinical observations, also accounts for intention tremor in patients with essential tremor. To investigate this, we compared the performance of ballistic arm movements in our ET_{IT} patients with results of previous studies in animals and humans with known cerebellar lesions. In line with a possible cerebellar deficit, our ET_{IT} group had more clinical cerebellar signs—such as dysdiadochokinesia and disturbed tandem walking—than the ET_{PT} group. In our experimental setting, the ET_{IT} patients were characterised by more pronounced abnormalities of their ballistic movements than the ET_{PT} group, and the disproportion between the deceleration phase (T_2) and the acceleration phase (T_1) was more prominent. Furthermore, we found a prolonged duration of AG_1 and a delayed ANT in the ET_{IT} patients, while these variables did not differ

from the controls in ET_{PT} group. Taken together, these abnormalities of our ET_{IT} patients closely resemble those described in studies on laboratory primates or human beings with cerebellar lesions.^{17–19} Thus the hypothesis of disturbed cerebellar function linked to the presence of intention tremor in patients with essential tremor is supported by our data.

Elble *et al.* have previously reported a negative logarithmic relation between the amplitude and frequency of tremor in 44 patients with essential tremor; their subsequent longitudinal study showed a decline in tremor frequency in the course of essential tremor.^{32–33} Our ET_{IT} patients were older, showed lower tremor frequencies and higher amplitudes, and tended to have a longer duration of disease than the ET_{PT} group. They probably represent an advanced stage of the disorder. A contribution of decreased muscle force to the kinematic alterations in the older ET_{IT} patients cannot be ruled out. However, tremor power—requiring muscle force—was higher in this group. Moreover, the relation between acceleration and deceleration of movement (T_1/T_2), both associated with active muscle contraction,³⁴ were not changed equally, but their pattern was distorted in a way characteristic of cerebellar disease. Thus altered muscle properties are unlikely to have affected our results.

Quantitative or qualitative differences between ET_{PT} and ET_{IT} ?

In the ET_{IT} patients only the AG_2 burst was significantly delayed, while ANT showed only a tendency to be later than in the healthy controls. From our data, we do not believe that the mistiming of AG_2 is specific for cerebellar dysfunction in the ET_{IT} group, with sparing of the ANT burst. Whether the latter is found to be significantly delayed may simply be a matter of

the extent to which cerebellar timing is disturbed. A mild shift of all bursts within the triphasic pattern may lead to delays that do not reach the level of significance for the ANT burst, as it occurs early in the sequence. The delay of AG₂, however, may reach significance at a milder stage of cerebellar disturbance. In line with this interpretation, ANT was significantly delayed in the ET_{IT} group, possibly because cerebellar dysfunction was more marked in these patients. Thus the differences found between ET_{IT} and ET_{IT} may reflect quantitatively different expressions within a spectrum of disturbed timing function related to the severity of essential tremor.

How is tremor functionally related to the abnormalities of the triphasic pattern?

An only minimally delayed ANT in the ET_{IT} group may still allow effective deceleration of movement with only mildly disturbed timing. However, the braking process is more abrupt, as the counteraction of AG₂ is delayed and thus inefficient. As a result, AG₂ fails to dampen the resulting counter-movement. This mechanism may reflect the first impulse of an oscillatory process which is maintained owing to the delay in the respective subsequent bursts. If these occur late enough in the sequence, they may accelerate rather than dampen the resulting oscillatory process because they could occur in phase with the respective ongoing tremor cycle. This pathophysiological process would be more pronounced with increasing abnormalities of burst timing and may underlie the crescendo of tremor during voluntary movement characterising intention tremor. The high correlation between the delays of ANT and AG₂ and the periods of tremor induced by the ballistic movements support such a pathophysiological link. Whether this finding points to a distinct pathophysiology underlying postural tremor cannot be answered definitively by our data.

There was no significant correlation between the tremor periods obtained from spectral analysis of postural tremor and the latency of AG₂ when considering the ET_{IT} and ET_{IT} patients individually. However, when these two groups were pooled together, this correlation was clearly significant. In this regard, postural tremor and the tremor induced by the ballistic movements seem to share the same characteristics. As the latter form of tremor is associated with movement, a relation to intention tremor may be assumed. Thus postural tremor and intention tremor could be linked to the same pathological mechanisms in essential tremor. An alternative interpretation of the correlation between tremor and the triphasic pattern is proposed by Elble *et al.*³⁵: the essential tremor rhythm initiated by AG₂ could be the cause rather than the consequence of the delayed AG₂ and ANT burst. From our data it is not possible to choose between these opposing interpretations.

Conclusions

We conclude from our data that a disturbance of cerebellar timing function is present in essential tremor, though this is only mild in monosymptomatic postural essential tremor. Patients with an additional intentional tremor component tend to be older and to have a longer duration of disease, clinical signs of cerebellar dysfunction, higher tremor amplitudes, lower tremor frequencies, and a more pronounced disturbance of cerebellar timing. Thus these patients may represent a more advanced stage of disease. Essential tremor and cerebellar function are interlinked. However, we cannot say with certainty whether cerebellar dysfunction is the cause of the tremor or a consequence of it.

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