Disturbed post-movement beta synchronization in Wilson's disease with neurological manifestation

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Wilson's disease is a rare autosomal recessive inherited disorder of copper metabolism leading to chronic hepatopathy, neurological and psychiatric symptoms, corneal, renal, skeletal abnormalities, cardiomyopathy and hormonal dysfunction [27]. Its worldwide prevalence is 30/million [5].

In 40–50% of the Wilson patients the initial clinical findings are neurological and psychiatric symptoms [27]. The most common neurological signs (tremor, Parkinsonism, dystonia, ataxia, chorea, dysarthria) are related to dysfunction of the basal ganglia and the cerebellum [33]. In these patients the typical MRI findings are low signal intensity on the T1-weighted and high signal intensity on the T2-weighted images in the putamen, nucleus caudatus, globus pallidus, nucleus subthalamicus, substantia nigra, thalamus and the cerebellum [10]. However, pathological MRI findings cannot always be demonstrated in patients with neurological manifestations [37].

Post-movement beta synchronization (PMBS) of the electroencephalogram (EEG) is a transient, short increase of power in the beta frequency band, which can be detected above the sensorimotor cortical areas 1–2 s after the termination of the movement [22]. It may be generated in the supplementary [23] and/or sensorimotor cortex [32]; its power and latency can be correlated with the parameters of the preceding movement [21,31]. PMBS may reflect afferent input processing [26] causing active inhibition of the sensorimotor cortex [16]. Activation of proprioceptive fibers was shown to evoke higher beta synchronization than cutaneous inputs [9]. PMBS appears only after closure of a complex motor program and not between the sequences [1].

Earlier studies revealed decreased PMBS power in Parkinson's disease [34], increased latency of PMBS in essential tremor [35]. The aim of the present study was to investigate whether PMBS distinguishes these disorders from Wilson's disease.

EEG was recorded in ten patients (8 males and 2 females, average of disease duration: 14.7 ± 11.17 years) with neurological manifestation of Wilson's disease and in 10 control subjects after obtaining written informed consent. The study has been performed in accordance with the ethical standards of the 1964 Declaration of Helsinki and it was approved by the local Ethics Committee. None of the patients had significant cognitive impairment (score of Mini-Mental State Examination < 28/30), none of them had hepatic

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encephalopathy (peak power frequency of the background activity: 10.5, 10.5, 9.5, 10.5, 9.5, 11, 11, 10, 10.5, 10 Hz; successively) [4]. Clinical data are summarized in Table 1 [8].

During the EEG acquisition subjects were lying in supine position and had to press an on–off button with the thumb of the dominant hand 40 times, in a self paced manner. All of the control subjects were right handed; 3 out of the 10 Wilson patients were left handed. Because in Wilson's disease the abnormalities in the MRI images [11] and the neurological signs are mostly symmetric, and PMBS is influenced by handedness [31]; we examined the movement of only the dominant hand. The time interval between two button presses was at least 10 s.

Nineteen scalp electrodes were positioned according to the international '10–20' system, with accessory C1 and C2 electrodes defined in the modified combinatorial nomenclature [3]. The reference electrode was placed on the tip of the nose. For off-line EEG analysis we used the common average reference method. To detect muscle activity, a surface EMG electrode was placed above the flexor pollicis brevis muscle referenced to the electrode lying above processus styloideus radii. Electrode impedances were kept below 5 kΩ.

We analyzed the parameters of PMBS at those localizations, where PMBS can typically be observed after short voluntary finger movement, in many studies [19,20,32]. Five electrodes in the region of the sensorimotor cortical areas were selected and renamed according to their localization related to the dominant hand (C: contralateral C3/C4, CM: contralateral medial C1/C2, M: medial Cz, IM: ipsilateral medial C1/C2, I: ipsilateral C3/C4). After rectification of EMG signal we signed the EMG and EEG signal at the beginning of the movements with ON and at the end of the movements with OFF markers. The time delay between ON and OFF markers was defined as movement duration. Six seconds long EEG segments, 3 s before and 3 s after the OFF marker position (as the time 0) were selected.

25–40 segments of each subject were averaged, in which the beginning and the end of the movement could be clearly defined and did not contain visible artifacts in F1, Fz, F2 EEG channels.

The multitaper method [15,17,36] was used for the spectral analysis. The spectrum was estimated by multiplying the data with K different windows (i.e. tapers). This method uses a sliding time window for calculating the power spectrum by discrete Fourier transformation. If y_t is the signal, then the spectral power is calculated as follows [14]:

$$S_{MT}(f) = \frac{1}{K}\sum_{k=1}^{K}|\tilde{Y}_k(f)|^2 \tag{1}$$

$\tilde{Y}_k(f)$ is the Fourier transform of the windowed signal y_t which can be formulated as

$$\tilde{Y}_k(f) = \sum_{t=1}^{N} w_t(k) y_t \ \exp(-2\pi i f t) \tag{2}$$

and the terms $w_t(k)\ \ (k = 1, 2, \ldots, K)$ are the K orthogonal tapers. As orthogonal tapers with good leakage and spectral properties, the discrete prolate spheroidal sequences (DPSS) [30] are applied. The DPSS can be defined as $v_t(k, W, N)$, where the Kth DPSS has a length N and a frequency bandwidth parameter W. The Fourier transform of the sequence $v_t(k, W, N)$ is given as

$$U(f) = \sum_{1}^{N} v_t(k, W, N) \ \exp(-2\pi i f t) \tag{3}$$

The sequences $v_t(k, W, N)$ are determined such that the spectral amplitude $U(f)$ is maximally concentrated in the interval $[-W, W]$,

Table 1
Clinical data of patients with Wilson's disease.

No.	Present neurological symptoms	MRI findings	K–F ring	Child–Pugh scores/class A–C	Serum cerulopl. (0.2–0.6 g/l)	Serum Cu (11–22 μmol/l)	ATP7B gene mutations	Scores for WD (poz: ≥4)
W1	Behavioural disturbance	BG	Yes	5/A	0.16	9	H1069Q/P1273S	9
W2	Dysdiadochokinesia of both hands, gait ataxia	Normal	Yes	5/A	0.17	Not available	H1069Q/H1069Q	9
W3	Ataxia of hands and gait	Normal	Yes	5/A	0.19	Not available	H1069Q/H1069Q	9
W4	No symptom	BG	Yes	6/A	0.16	15.1	H1069Q/H1069Q	11
W5	Dysarthry, rigor, rest tremor of both hands, right sided intention hand tremor, ataxia of gait	BG, T	Yes	5/A	0.05	0.6	H1069Q neg	7
W6	Postural tremor of all extremities, left hand intention tremor	Normal	0	5/A	0.29	24 h urine Cu (<100 μg/24 h): 343 μg/24 h	T977M/V1364V-fs	5
W7	Parkinson-syndrome, bilateral postural hand tremor, dysarthria, gait ataxia, dysthymia	BG, M	Yes	5/A	0.16	2.8	H1069Q/Y715H	11
W8	Parkinson-syndrome, dysarthria, ataxia of gait, bilateral postural and intention hand tremor	BG, M	Yes	5/A	0.1	9.1	H1069Q neg	5
W9	Parkinson-syndrome, choreo-athetosis	BG	Yes	5/A	0.05	4.7	R778G/? H1069Q neg	8
W10	Gait ataxia, bilateral postural and intention hand tremor	BG	Yes	5/A	0.04	3.6	H1069Q/?	7

DD: duration of the disease, M: male, F: female, K–F ring: Kaiser–Fleisher ring, BG: basal ganglia, T: thalamus, M: mesencephalon.

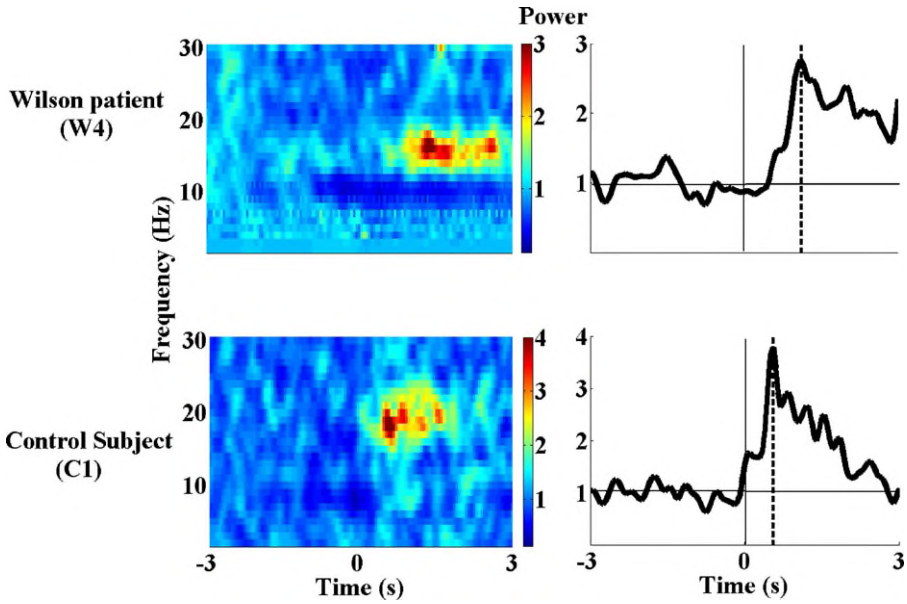

Fig. 1. PMBS of one Wilson patient and one control, contralateral to the movement. Relative time–frequency–power plot and time–power plot in the most reactive beta frequency band are shown. The maximum power and latency values are indicated by dashed line. PMBS power is lower; latency is longer in the patient than in the control.

i.e.,

$$\int_{-W}^{W} |U(f)|^2 \, df \tag{4}$$

is maximised. The maximisation problem is solved by using Lagrange multipliers; this leads to an eigenvalue equation, and the eigenvectors of this equation will be the DPSS. After calculating the absolute power spectra, the event-related power changes, a normalisation with respect to the reference interval were estimated for each frequency. The reference interval was taken from −3 to −2 s. In this study, we used windows of block length 1000 ms with overlapping windows within a frequency band of 2–30 Hz. The signals were sampled at 256 Hz, the time resolution was 50 ms, and the frequency resolution was 1 Hz.

PMBS was measured in the most reactive 4 Hz frequency band, which was determined for all analyzed channels of each patient from the time–frequency–relative power plots (Fig. 1). After calculating the relative power of the most reactive frequency range we chose the maximum beta power value of the 0–2 s period (PMBS power). The latency of PMBS was determined as the time delay between 0 s and the time when this maximum beta synchronization appeared.

We used analysis of variance (ANOVA) for repeated measures to compare the median values of most reactive beta frequency range, the means of the absolute power values of the reference interval, the power and latency of PMBS in the patient and control group. The between subject factor was GROUP: Wilson's disease and control; the within subject factor was the localization (LOC): contralateral, contralateral medial, medial, ipsilateral medial, ipsilateral to the movement. For post hoc comparisons we used Newman–Keuls test. Student's *t*-test for independent samples was performed to show whether the age of subjects and the duration of movement are different in the two groups. The level of significance was $p < 0.05$. Spearman rank correlation was calculated between PMBS power, latency and the duration of the movement.

Overall PMBS power without differentiating between different electrode locations was not significantly different in the patient (W) and control (CL) group. Results of the statistical analysis are summarized in Table 2. The effect of electrode location (LOC) was significant, and PMBS power was significantly higher contralateral to the movement compared to every other electrode location in the combined analysis of both groups. However, there was a significant interaction between Group and electrode location (Group × Loc) with the post hoc comparisons demonstrating that PMBS power is significantly higher in the contralateral (C) electrode in the control group compared to any other positions in both the control and the Wilson's groups. On the one hand this shows that the normal topography of PMBS with contralateral preponderance disappeared in the patient group. On the other hand it indicates that the power of PMBS in the most reactive electrode location in the contralateral central area was significantly lower in the patients with Wilson's disease (Figs. 1 and 2).

The latency of the PMBS was significantly longer in the Wilson group than in the control group. PMBS appeared significantly earlier on the contralateral, the contralateral medial and in medial electrode position than on the ipsilateral electrode localization (Table 2, Fig. 2).

Duration of the examined self-paced button press was longer in the patient group than in the control group, but the difference was not significant. PMBS power and latency did not correlate with movement duration in the 20 examined subjects (power-movement duration: $r = -0.227$, $p_{\text{two tailed}} = 0.335$; latency-movement duration: $r = 0.141$, $p_{\text{two tailed}} = 0.553$).

The median values of the most reactive 4 Hz wide beta frequency band were not different in the Wilson and the control groups in the five electrode localizations above the sensorimotor cortical areas.

The altered parameters of PMBS were not able to predict clinical neurological deficits or structural changes in the MRI. Every patient, who had severe cerebellar and basal ganglia affection had low PMBS power, 3 out of 4 patients had high PMBS latency.

In the present study we examined the magnitude, latency of PMBS in patients with neurologically manifesting Wilson's disease and in controls. Our aim was to analyze, how the parameters of PMBS are altered in Wilson's disease and if these changes are different from the changes observed in Parkinson's disease and in essential tremor.

In studies of other movement disorders, parameters of PMBS above the sensorimotor cortex were affected. It has been reported that reduction of PMBS power in Parkinson's disease is more pro-

Table 2
Results of the statistical analysis.

	Average data Wilson's disease	Average data controls	*t*-test/ANOVA factor effects
Age of subjects (years)	35.2 ± 14.97	25 ± 2.21	*t*-test for independent samples: $p = 0.06$
Duration of movement (s)	0.75 ± 0.17	0.63 ± 0.18	*t*-test for independent samples: $p = 0.902$
Median of the MRBF (Hz)	C: 18.4 ± 2.36, CM: 19.6 ± 2.27, M: 18.8 ± 1.23, IM: 18.9 ± 1.79, I: 18.6 ± 2.06	C: 19 ± 1.05, CM: 19.5 ± 1.27, M: 19.1 ± 2.08, IM: 19.4 ± 1.77, I: 18.8 ± 1.32	GROUP: $p = 0.595$; $F_{1,18} = 0.293$ LOC: $p = 0.275$; $F_{4,72} = 1.308$ GROUP × LOC: $p = 0.95$; $F_{4,72} = 0.178$
Relative power of PMBS	C: 1.9 ± 0.70, CM: 1.5 ± 0.31, M: 1.6 ± 0.32, IM: 1.5 ± 0.43, I: 1.7 ± 0.32	C: 2.5 ± 0.76, CM: 1.72 ± 0.43, M: 1.77 ± 0.43, IM: 1.4 ± 0.24, I: 1.7 ± 0.5	GROUP: $p = 0.37$; $F_{1,18} = 0.85$ LOC: $p < 0.05$; $F_{4,72} = 13.15$ Significant: C-CM; C-M; C-IM; C-I LOC × GROUP: $p = 0.038$; $F_{4,72} = 2.68$ Significant: C_{CL}-C_W; C_{CL}-CM_W; C_{CL}-M_W; C_{CL}-IM_W; C_{CL}-I_W; C_{CL}-CM_{CL}; C_{CL}-M_{CL}; C_{CL}-IM_{CL}; C_{CL}-I_{CL}
Latency of PMBS (s)	C: 1.2 ± 0.55, CM: 1.3 ± 0.48, M: 1.3 ± 0.39, IM: 1.6 ± 0.34, I: 1.21 ± 0.45	C: 0.86 ± 0.42, CM: 0.83 ± 0.31, M: 0.72 ± 0.31, IM: 1.12 ± 0.51, I: 1.12 ± 0.51	GROUP: $p = 0.005$; $F_{1,18} = 10.29$ LOC: $p = 0.017$; $F_{4,72} = 3.23$ significant: C-IM; CM-IM; M-IM LOC × GROUP: $p = 0.19$; $F_{4,72} = 1.56$

MRBF: most reactive beta frequency.

nounced contralateral to the more severe clinical symptoms, on the side of more advanced nigrostriatal neurodegeneration [34]. PMBS latency was measured in the normal range [35]. In hereditary Parkinson's disease PMBS power contralateral to the movement was the smallest in the patient group, larger in the group of asymptomatic first relatives and the largest in the control group [6]. In an earlier study normal PMBS power but increased latency was seen in essential tremor [35], which has been shown to involve cerebello-thalamo-cortical circuits [7,25].

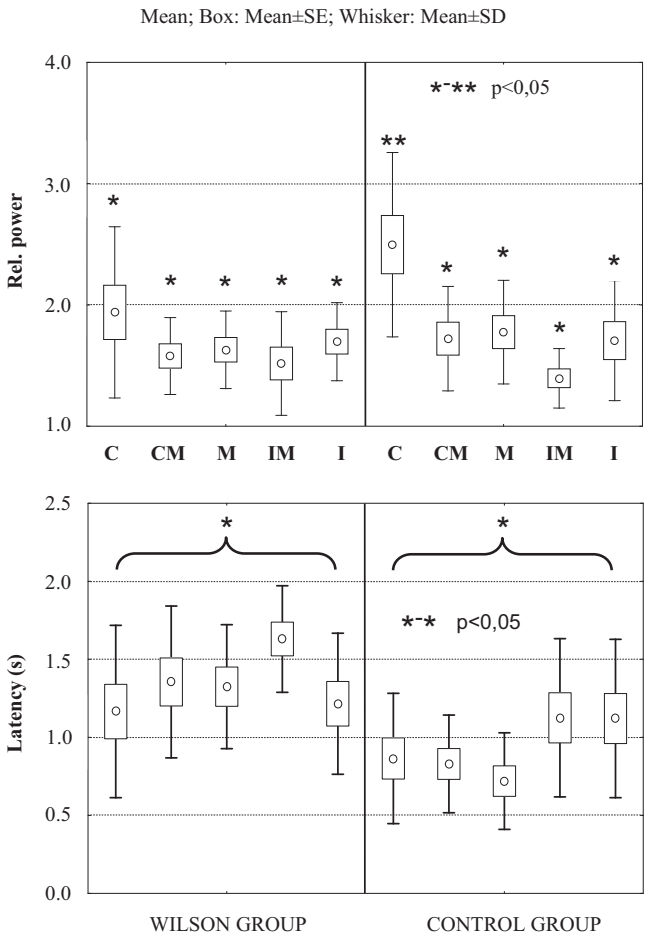

Fig. 2. The relative power and latency of PMBS in the Wilson and control group, in the five electrode localizations. The power of PMBS was significantly largest in the control group, contralateral to the movement. The latency of PMBS was significantly higher in the Wilson group than in controls.

Our present results show that the power of PMBS in the contralateral central electrode was lower in patients with Wilson's disease than in the normal controls. In the control group PMBS power was significantly higher contralateral to the movement (C location) than in the other four localizations corresponding with previous studies [22,31] whereas this contralateral preponderance of PMBS power disappeared in Wilson's disease. The results show that not only the intensity but also the cortical topography of the sensorimotor processing reflected by the PMBS is affected in Wilson's disease. Previous studies indicated that the power of PMBS depends on the level of cortical excitability [1,24,29]. Transcranial magnetic stimulation studies detected alterations of motor cortex excitability in Parkinson's disease [13] and in Wilson's disease [18], which may influence PMBS power. In essential tremor excitability of the motor cortex is normal [28] in line with the observation that PMBS power is also in the normal range in this disease [35]. Our present results support the conclusion that altered cortical excitability can modulate PMBS power in movement disorders due to altered dynamic activation in the cortex-basal ganglia circuitry. This network has common motor information transmission in the beta band, indicated by the fact that simultaneous EEG and local field potential recording in the nucleus subthalamicus showed similar beta movement-related responses [2,12].

PMBS latency was significantly longer in Wilson's disease compared to controls (Fig. 2). In the control group we detected PMBS first at the midline in line with the previous observation [23], but in the Wilson group PMBS could be measured first contralateral to the movement, again, indicating an abnormal temporospatial post-movement processing.

The correlation between MRI-abnormalities, clinical symptoms and functional measures are generally weak [37]. In the present study we grouped the patients according to the dysfunction/lesion of the basal ganglia and cerebellum. We could not find a clear correlation between clinical symptoms or MRI abnormality and the changes of PMBS. However, we have seen a trend towards more marked abnormalities of the PMBS in patients with pathology in both systems.

Our data show that oscillatory activity involved in cortical processing is altered in Wilson's disease. We can conclude that PMBS changes depend on the subcortical motor systems (basal gan-

glia/cerebellar) involved and the overall extent of the affected brain regions, but further studies with larger numbers of patients are needed.

As reduced power of PMBS was found in Parkinson's disease and increased latency in essential tremor, the alteration of both PMBS power and latency in Wilson's disease implies more severe disturbance of sensorimotor integration and may help the differential diagnosis of ambiguous cases.

Conflict of interest

The authors declare that they have no conflict of interest.

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