Parkinsonism and Related Disorders 65 (2019) 153–158

Contents lists available at ScienceDirect

Parkinsonism and Related Disorders

journal homepage: www.elsevier.com/locate/parkreldis

A peek into premonitory urges in Tourette syndrome: Temporal evolution of neurophysiological oscillatory signatures

Valentina Niccolai[a,*], Silvana Korczok[b], Jennifer Finis[a], Melanie Jonas[c], Götz Thomalla[d], Hartwig Roman Siebner[e,f], Kirsten Müller-Vahl[g], Alexander Münchau[h], Alfons Schnitzler[a], Katja Biermann-Ruben[a]

[a] Institute of Clinical Neuroscience and Medical Psychology, Medical Faculty, Düsseldorf University, Germany
[b] General Internal Medicine, Infectiology, Pneumology, Osteology, Leverkusen Hospital, Germany
[c] Department of Human Resources, Health and Social Affairs, Fachhochschule des Mittelstands, University of Applied Sciences, Cologne, Germany
[d] Department of Neurology, University Medical Center Hamburg-Eppendorf (UKE), Germany
[e] Danish Research Centre for Magnetic Resonance, Centre for Functional and Diagnostic Imaging and Research, Copenhagen University Hospital Hvidovre, Denmark
[f] Department of Neurology, Copenhagen University Hospital Bispebjerg, Copenhagen, Denmark
[g] Clinic of Psychiatry, Socialpsychiatry and Psychotherapy, Hannover Medical School, Germany
[h] Department of Pediatric and Adult Movement Disorders and Neuropsychiatry, Institute of Neurogenetics, University of Luebeck, Germany

ARTICLE INFO

Keywords:
Tourette syndrome
Oscillations
Beta
Urge
Intention

ABSTRACT

Background: Tics are the core symptom of patients with Gilles de la Tourette syndrome, yet the spatial-temporal dynamics of neural activity causing a tic remains to be determined.
Objective: Identification of cortical events preceding tic onset.
Methods: In twelve patients with Tourette syndrome we performed magnetoencephalography to trace the time course of beta oscillations (15–30 Hz) in motor cortical areas before tic onset.
Results: Patients showed a biphasic modulation of cortical beta activity during the second before tic onset. We observed an initial increase of beta power over the left-hemispheric channels overlying the motor cortex. This increase was subsequently replaced by a decrease in beta power. The beta decrease close to tic onset resembled the typical pattern accompanying preparation of voluntary movements. Only the initial increase in beta power positively correlated with the intensity of motor urges preceding tics.
Conclusions: The spatial-temporal dynamics of cortical activity suggests a voluntary component of tics that might be triggered by a failure of compensatory motor inhibitory mechanisms.

1. Introduction

Gilles de la Tourette syndrome (TS) is a neurodevelopmental disorder characterized by multiple motor and vocal tics present for at least one year with onset before the age of 18 (DSM-5 [1]). Tics are sudden, rapid, recurrent, patterned non-rhythmic movements or vocalizations that can be suppressed for brief intervals [2]. There have been some investigations targeting the neurophysiological correlates of tic initiation. For instance, functional magnetic resonance imaging (fMRI) studies showed activation of the motor, premotor, and supplementary motor area during the 2 s preceding tics [3,4]. Inconsistent results emerge from animal models of TS. While no motor cortical activity was observed in inter-tic intervals in one study [5], motor cortical activation was found to determine the occurrence of tics in another study [6]. Taken together, investigations of the period preceding tic onset point to a crucial involvement of premotor and motor cortical areas in tic initiation.

When focusing on the temporal cortical dynamics preceding tics, the potential impact of premonitory urges needs to be considered, as these sensory phenomena often immediately precede tics in most adult TS patients [7,8] and are considered a force driving tics [9]. Urges can differ in sensory and motor-related qualities. An urge or impulse to move has most frequently been reported [10], thus pointing to a relevant motor component of urges. Moreover, the close temporal relationship between urges and tics could imply that the latter are some sort of voluntary responses to relieve unpleasant sensations, although

* Corresponding author. Institute of Clinical Neuroscience and Medical Psychology, Heinrich-Heine University, Building 23.02.03.47, Universitaetstrasse 1, 40225, Duesseldorf, Germany.
E-mail address: Valentina.Niccolai@hhu.de (V. Niccolai).

https://doi.org/10.1016/j.parkreldis.2019.05.039
Received 12 March 2019; Received in revised form 13 May 2019; Accepted 30 May 2019

some behavioral results argue for independence between urges and tics [11,12]. Indeed, electrophysiological patterns of voluntary movement preparation were not consistently observed prior to tics [13,14]. Addressing the neurophysiological motor correlates of tic initiation using an oscillatory-based approach might contribute to the issue of a possible voluntary tic control aimed at relieving inner tension.

In the present study, we focused on the evolution of tics exploiting the high temporal resolution of magnetoencephalography (MEG) and the well-known oscillatory correlates of motor preparation, namely beta oscillations (15–30 Hz) in motor cortical areas. Here, beta power typically decreases during movement preparation and execution [15]. Deep brain stimulation (DBS) postoperative data from two TS patients showed beta power increase in one and decrease in the other subject occurring about 500 ms prior to a tic in the motor area [16]. We investigated beta power and its relationship with subjective measures of urge intensity in two 500 ms long time-windows preceding tic onset in a sample of twelve TS patients. Beta power modulation across time was expected to disclose the motor cortical state preceding tic onset and possibly point to a neurophysiological signature of premonitory urges and tic inhibition.

2. Methods

2.1. Participants

The study was in accordance with the Declaration of Helsinki and was approved by the responsible Ethics Committee (Hamburg Medical Association, study number 2514). Twelve adult TS patients aged between 22 and 54 years (mean 37 ± 9 years, 2 female) were recruited from three specialized TS outpatient clinics (Departments of Neurology of the University Hospitals Hamburg and Düsseldorf and Department of Psychiatry, Socialpsychiatry and Psychotherapy of the Hannover Medical School). All patients met the diagnostic criteria of TS according to the DSM-5. To assess symptom severity, the Yale Global Tic Severity Scale (YGTSS) and the Modified Rush Video-based Scale for tic rating were used (MRVS); the diagnostic confidence index was applied to confirm the diagnosis (DCI). To assess attention deficit hyperactivity disorder the short version of the Wender Utah Rating Scale was used (WURS-k). Self-injurious behavior and obsessive-compulsive behavior were assessed by neurologists with experience in the assessment and care of TS patients. Premonitory urge was evaluated by means of the 9 items version of the Premonitory Urge for Tics Scale (PUTS). Handedness was assessed with the Edinburgh Inventory and the Annett Hand Preference Questionnaire. Descriptive data and clinical scores of the sample studied have been reported previously [17]. Patients had no clinically significant comorbidity with any disorder. Five TS patients had never received any medication to treat tics; the remaining 7 were off medication for at least 6 months (n = 1) or 1–10 years (n = 6). Mean tic onset was 9 (± 3) years and mean duration was 29 (± 9) years. All participants had normal or corrected-to-normal vision and provided written informed consent prior to the MEG.

2.2. Data acquisition

We used a MEG data set that had been recorded to study cortical activity during a Go-NoGo task [17]. We extracted the MEG epochs recorded between a cue and a Go-NoGo target as this was the longest time interval without visually evoked (cue/prompt-related) and motor (response-related) activity. This time span varied from 2 to 6 s in steps of 1 s in the experiment. Descriptions of stimuli and task procedure as well as behavioral results were reported previously [17]. Participants were instructed not to suppress tics. Vertical and horizontal electrooculogram (EOG) was used to monitor eye movements and blinks. To detect tics, patients were videotaped. In addition, bipolar electromyographic (EMG) electrodes were applied to the Musculus frontalis, orbicularis oculi, and orbicularis oris of the right side of the face. These electrodes were referenced to electrodes on the jaw. To monitor shoulder tics, two EMG electrodes were attached to the Musculus trapezius bilaterally and were referenced to the clavicles. Neuromagnetic brain activity was continuously recorded with a 122-channel MEG system (Elekta Neuromag, Helsinki, Finland). Four coils were attached to the subject's head bilaterally on the forehead and behind the ears. The position of these coils and prominent anatomical landmarks (right and left preauricular points and nasion) on the subject's head were digitized (Polhemus Isotrak) to map functional MEG data to individual anatomy. MEG data were digitized at 1000 Hz, band-pass filtered from 0.03 to 330 Hz online, and stored on a computer hard disk.

2.3. Data analysis

Data were analyzed with Matlab (Mathworks, Natick, MA, USA) and FieldTrip (http://fieldtrip.fcdonders.nl), a Matlab software toolbox for MEG and EEG analyses.

2.4. MEG data pre-processing

The exclusion of stimulus- and repose-related time-windows, which could have induced visual and motor processes, respectively, led to the selection of episodes consisting of 1.5 s long time-window preceding tic onset and 100 ms after tic onset. Data were filtered with a high-pass filter of 2 Hz and with band-stop filters at 49–51, 99–101, 149–151 Hz; a Butterworth IIR zero-phase forward and reverse filter was used. Artefacts due to heartrate, eye movements and blinks were excluded from analysis by means of independent component analysis (ICA); on average 3.5 components were rejected per subject. Additional visual semi-automatic muscular artefact rejection resulted in an average of 31 ± 13 SD trials per subject. Considering that the overall number of trials in the Go-NoGo task was 300, tics were observed in about 10% of the selected trials, these having an average length of 1600 ms. Due to large artefacts the dataset of one participant was not included in the analysis.

2.5. Time-frequency representation (TFR)

TFRs were calculated by means of a fast Fourier transform (FFT). An adaptive window including 5 cycles was shifted in steps of 50 ms from 1500 ms pre-to 100 ms post-tic onset. Data were padded up to 5 s and a Hanning taper was applied to the epochs. Power was estimated between 5 and 39 Hz in steps of 2 Hz. Temporal-Spectral-Evolution (TSE) for the beta frequency was calculated by filtering the signal with a bandpass filter from 15 to 30 Hz, and by rectifying and averaging the resulting signal. Trials were normalized with respect to the baseline, which encompassed a time-window between 1.5 and 1.1 s preceding tic onset, to subtract the activity related to response preparation. To further control for the tic time-point within the response preparation time-window, an additional analysis was run using tic-free trials of each patient as baseline. For each tic, a time marker indicating time distance between cue and tic onset was used to define a corresponding baseline epoch from tic-free trials including a time-window between 1500 ms pre- and 100 ms post-time marker. The same amount of tic-free trials as tic trials was selected by means of randomization and tic-free episodes went through the same preprocessing steps and TFR calculation as tic episodes.

2.6. Statistical analysis of MEG data

Considering the multidimensionality of the MEG data, a procedure which corrects for multiple comparisons was used [18]. Beta (15–30 Hz) power values were measured for the average of eight channels presumably situated above the motor and premotor cortex of each hemisphere and for each time point (see Fig. 4). For every time-frequency sample two baseline-corrected time-windows (Tw) preceding

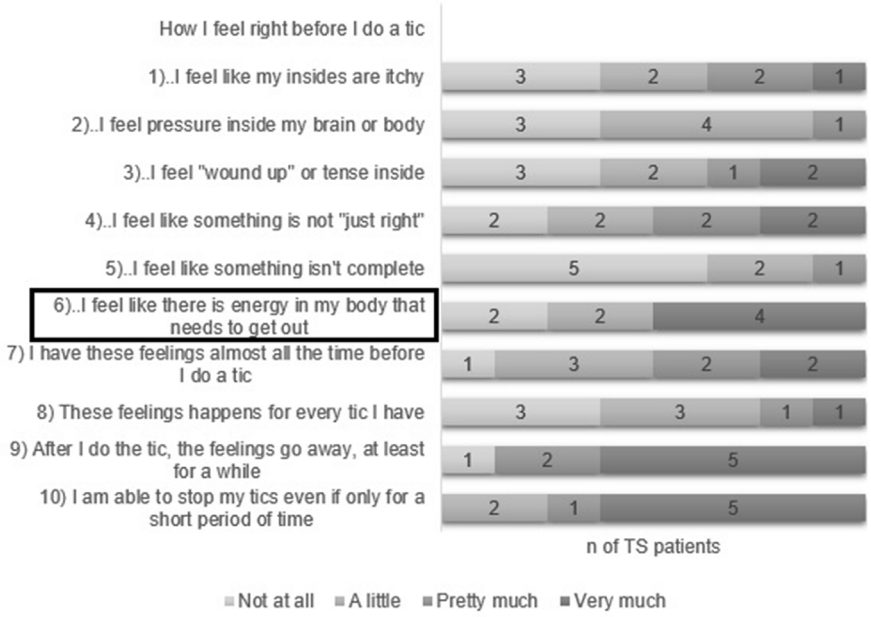

Fig. 1. Scores for each item of the PUTS across eight patients. Item number 6 was selected for motor urge scores.

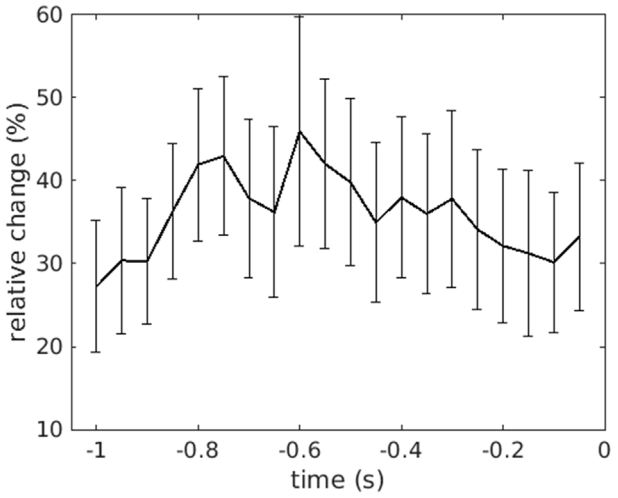

Fig. 2. Schematic layout of epoch definition and temporal spectral evolution (TSE) of relative beta power preceding tic onset (time = 0) with bars indicating standard error of the mean; average of eight left-hemispheric motor channels as shown in the topographical plot on the right.

tic onset were defined as Tw1 (−1 to −0.5 s before tic onset) and Tw2 (−0.5 to 0 s before tic onset) and were compared by means of a *t*-test for dependent samples. All time-frequency samples with *t*-value larger than the threshold ($p < .05$) were selected and clustered with temporally and spectrally adjacent bins. A cluster-level statistic was then calculated by taking the sum of the t-values of the time-frequency samples within every cluster. Nonparametric permutation testing, which consisted in computing 1000 random sets of permutations between Tw1 and Tw2, was used to obtain a distribution of cluster statistics and the significance level of the observed cluster ($p < .05$). Analyses were done separately for each hemisphere. Similarly, TFRs of tic-related trials were compared with tic-free trials in the selected channels of each hemisphere between −1 and 0 s before time marker/tic onset with the cluster-based permutation test as described above.

2.7. Clinical and behavioral correlation

Individual changes in beta power across all head channels were calculated by means of a linear regression based on 100 ms-long time steps for each of the two time-windows preceding tic onset. The resulting regression slope values were then correlated with individual total PUTS scores as well as with scores of the item that prevalently refers to motor aspects of the urge (Fig. 1, item 6) by means of a Spearman rank test. This item was specifically selected to identify a possible relationship with the power modulation of the beta frequency, which is a neurophysiological marker of motor related processes. Further, the selection of the motor item was motivated by the fact that an urge to move is most commonly reported [10]. Correlation between individual average beta power and behavioral accuracy was calculated by means of Pearson correlation test.

3. Results

3.1. PUTS

PUTS scores were available for 8 of the 11 subjects entering the MEG data-analysis. Results showed a mean score of 21 (± 6 SD), 36 being the maximum possible value. The overall urge intensity ranged from high in four cases (score 25 to 30.5) to middle in three cases (score 12.5 to 24.5) and low in one case (score 9 to 12) (classification according to https://depts.washington.edu/dbpeds/Screening%20Tools/ScreeningTools.html). Patients' motor urge spanned from no urge at all to little and high urge intensity (Fig. 1, item 6). All patients who completed the PUTS reported the ability to suppress tics to some degree (Fig. 1, item 10) and a majority (87.5%) reported that urges disappeared following tic execution (Fig. 1, item 9).

3.2. MEG data

Temporal spectral evolution from 11 TS patients showed beta power increase compared to baseline across the first time-window (−1 to −0.5 s) and a subsequent decrease in the second time-window (−0.5 to 0 s) immediately before tic onset in the left-hemispheric selected channels (Fig. 2). Beta power was overall increased in Tw1 and Tw2 compared to baseline but was significantly stronger in Tw1 compared

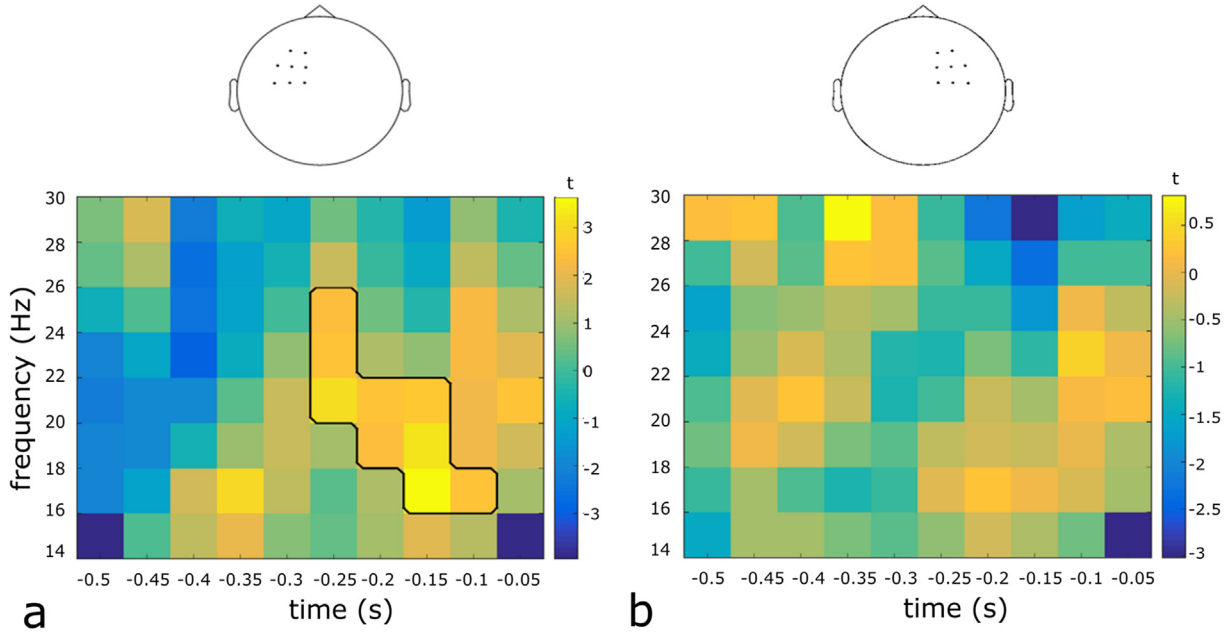

Fig. 3. Time-frequency statistical cluster showing significant difference between Tw1 and Tw2 preceding tic onset (significant cluster outlined) in the selected left-hemisperic channels (a) and no significant cluster in the corresponding right-hemispheric channels (b).

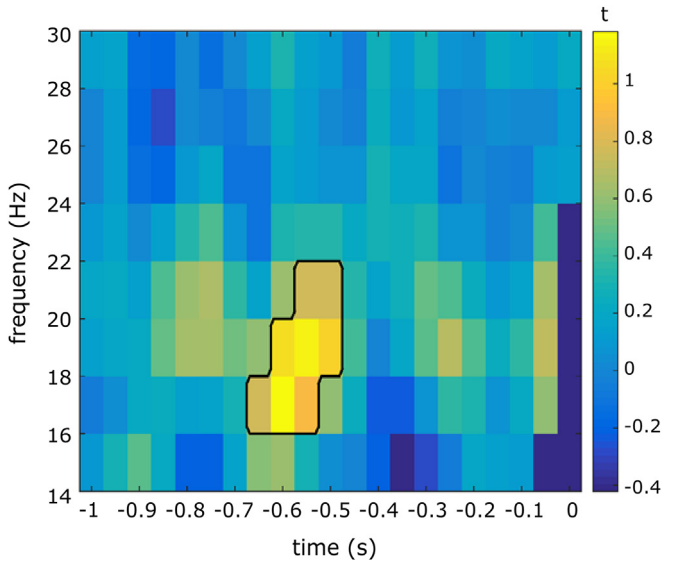

Fig. 4. Time-frequency statistical cluster showing significant difference in the selected left-hemisperic channels between 1 s long pre-tic and corresponding tic-free episodes (0 = time marker and tic onset, respectively; significant cluster outlined).

to Tw2 (p = .024; Fig. 3a). In contrast, there was no significant effect in the corresponding right-hemispheric channels (p = .196; Fig. 3b). The tic-related vs. tic-free trials contrast confirmed a significant power increase in the selected left-hemispheric channels in the same frequency and time range as in the aforementioned analysis (p = .040, Fig. 4).

3.3. Clinical and behavioral correlation

The correlation between the beta power slope and total PUTS score did not survive Bonferroni correction for multiple comparisons in Tw1 (rho = 0.710, p = .055) and was not significant in Tw2 (p = .564). Instead, the correlation between beta power slope across the Tw1 and the selected motor item of the PUTS was significant (rho = 0.928, p = .004) surviving Bonferroni correction for multiple comparisons. This indicates that increased beta power is associated with stronger motor urges (Fig. 5). No such correlation was found in the Tw2 (p = .184).

4. Discussion

Two adjacent time-windows preceding tic onset by 1 s and 0.5 s, respectively, were investigated in TS patients by focusing on oscillatory patterns of the motor cortical areas. Results show that beta power increased across Tw1 in left-hemispheric cortical motor channels and that the intensity of motor urge positively correlated with the overall increase of beta power. In Tw2 beta power decreased significantly compared to Tw1 thus resembling the typical pattern that accompanies preparation of voluntary movements, while there was no correlation with motor urge intensity.

Considering that tic episodes were extracted from a task requiring response preparation and that the latter is typically related to beta power decrease [15], the power increase observed in the baseline-corrected Tw1 may depend on active response inhibition processes. We controlled for a possible effect of time by contrasting tic-related trials with tic-free trials of the same patient within the same time-window on trial level. Results confirmed a beta power increase in the second half of the first 500 ms, corresponding to Tw1 in the previous analysis, and between 16 and 22 Hz. Moreover, previous comparison with matched healthy controls during the same (tic-free) motor preparation phase showed that the beta power increase was larger in these TS patients in motor areas [19]. These findings indicate that other processes than task-related motor preparation are responsible for this effect. Beta power increase was previously shown to be related to movement inhibition in healthy individuals [20]. Pre-tic beta power increase may depend on motor suppression mechanisms related to the occurrence of a tic. The presence of compensatory inhibitory mechanisms in TS has been repeatedly suggested (see Ref. [21] for a review), particularly on the basis of increased inhibitory activity preceding tic onset [22], BOLD signal modulation during tic suppression [23], and age dependent changes of brain morphology [24,25]. The SMA playing a crucial role in action inhibition [26], showed increased connectivity with motor areas in TS patients both during tic suppression compared to resting state and during the suppression of a voluntary movement compared to healthy

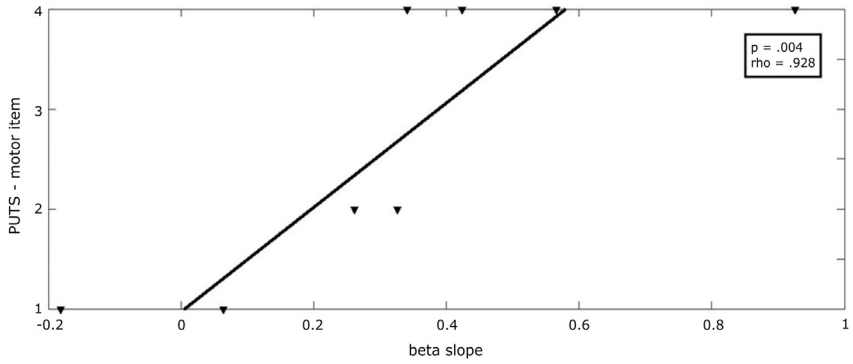

Fig. 5. Correlation between beta power slope in Tw1 and motor urge intensity.

controls [19,22]. Although participants were instructed not to suppress tics in the present study, TS patients often do so automatically to improve performance [27]. It is likely that this was also the case in the current sample because most patients were able to suppress tics (Fig. 1, item 10). Enhanced beta power in the earlier time-window preceding tic onset may thus constitute the neurophysiological cortical correlate of motor suppression mechanisms in TS patients.

In Tw2, immediately preceding tic onset, beta power decreased, which resembles the typical oscillatory pattern that accompanies the preparation of voluntary movements [15]. This may indicate a neural cortical signature of motor initiation and a failure or an interruption of compensatory mechanisms. The fact that TS patients often describe motor tics as a voluntary motor response to an involuntary sensation [10] raises the question of whether the oscillatory similarity between tics and other movements perceived as voluntary may be indicative of some sort of motor control in tics, at least partly. Of note, the typical beta suppression accompanying movement preparation and execution does not occur when a movement is not preceded by an intention such as in unexpected passive movements [28]. Available fMRI evidence points to motor cortical engagement underpinning both tics and voluntary movements [29]. The readiness potential physiologically preceding the onset of a voluntary movement, was observed in a minority of TS patients and with different latency prior to tic onset [13,14]. However, readiness potential and beta power suppression likely do not reflect identical cortical mechanisms [30]. Also, it is possible that the intention to tic in order to reduce or eliminate intense sensory or motor urge might still differ from the intention to start a normal movement.

Premonitory urges appear temporally and causally related to tic occurrence [8]. Beta modulation across the two time-windows preceding tic onset might reflect a conversion from a state of motor inhibition to voluntary motor preparation of a tic as urge increases. In line with this, there was a correlation between motor urge intensity and the amount of beta power increase across time (regression slope). A steeper increase in compensatory inhibition as suggested by the slope may thus be a response to stronger motor urge. The fact that this relationship was limited to the motor item of the PUTS and disappeared when considering the total PUTS score possibly depends on beta oscillations being the main neurophysiological correlate of motor processes. Interestingly, positive correlation between PUTS values and attentional difficulties suggests that urges, while aiding in tic suppression, likely also engage attentional resources in TS patient thus bearing some costs [31]. Also, tic suppression was shown to be associated with poorer quality of life, possibly depending on the constant effort required to manage the urge [31]. Current correlational results can also suggest that stronger tic suppression may lead to enhanced urge intensity. Tic suppression would not benefit premonitory sensations, i.e. despite or because of tic suppression, the urge is enhanced. The fact that the first but not the second time-window preceding tic onset showed this correlation indicates a qualitative difference between the cortical processes occurring in these two time-windows (i.e., tic inhibition and intention to perform a tic) and reflecting the development of premonitory urges in time. This sensorimotor relationship probably depends on the impairment of the cortico-striatal-thalamocortical pathways in TS patients [32]. A recent study based on a Bayesian framework suggests the sensorimotor regions of the putamen as the origin of tics and premonitory sensations, which depend on aberrant striatal synaptic integration of cortical inputs [33]. Although tic inhibition appears to produce a continuing intensification of the urge [8] that can only be eliminated by the execution of a tic, the fact that not all tics are preceded by urges ([10] and Fig. 1, item 8) and that premonitory urge did not change from pre- to post-tic suppression periods [12] calls for further real-time investigations of a possible influence of tic suppression on motor urge.

The main limitation of the study is the rather small sample used for PUTS-related analysis. Therefore, caution is needed when interpreting correlational results. Another limitation of the study is the debatable selection of a specific item of the PUTS targeting motor aspects of urges. This choice was motivated by the significance of beta oscillations with respect to movements and by the fact that an urge to move is most commonly reported [10]. Still, the development of more accurate real-time measures of motor urge is required to adequately address this issue.

In conclusion, the present result of a cortical beta power shift from a farther to a closer time with respect to tic onset hints at the presence and subsequent failure of compensatory motor inhibitory mechanisms.

Acknowledgements

This work was supported by the Deutsche Forschungsgemeinschaft (DFG; MU 1692/2-1, SCHN 592/1-2, FOR 2698, and SFB 991/B03). HRS was supported by Lundbeckfonden. We thank the German Research Foundation, which supported the study. We are grateful to Sofie Burgmer, Dr. Hanneke van Dijk, and Atal Ashna for their technical assistance.

References

[1] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders: DSM-5, fifth ed., American Psychiatric Publ, Washington, DC, 2013.
[2] J.F. Leckman, M.H. Bloch, L. Scahill, R.A. King, Tourette syndrome: the self under siege, J. Child Neurol. 21 (8) (2006) 642–649.
[3] S. Bohlhalter, A. Goldfine, S. Matteson, G. Garraux, T. Hanakawa, K. Kansaku, et al., Neural correlates of tic generation in Tourette syndrome: an event-related functional MRI study, Brain 129 (Pt 8) (2006) 2029–2037 https://doi.org/10.1093/brain/awl050.
[4] I. Neuner, C.J. Werner, J. Arrubla, T. Stocker, C. Ehlen, H.P. Wegener, et al., Imaging the where and when of tic generation and resting state networks in adult Tourette patients, Front. Hum. Neurosci. 8 (2014) 362 https://doi.org/10.3389/fnhum.2014.00362.
[5] K.W. McCairn, A. Iriki, M. Isoda, Global dysrhythmia of cerebro-basal ganglia-cerebellar networks underlies motor tics following striatal disinhibition, J. Neurosci. 33 (2) (2013) 697–708 https://doi.org/10.1523/JNEUROSCI.4018-12.2013.
[6] M. Israelashvili, I. Bar-Gad, Corticostriatal divergent function in determining the temporal and spatial properties of motor tics, J. Neurosci. 35 (50) (2015)

16340–16351 https://doi.org/10.1523/JNEUROSCI.2770-15.2015.
[7] K.R. Muller-Vahl, Tourette-Syndrom und andere Tic-Erkrankungen im Kindes- und Erwachsenenalter, first ed., Medizinisch Wissenschaftliche Verlagsgesellschaft, Berlin, 2010.
[8] V.C. Brandt, C. Beck, V. Sajin, M.K. Baaske, T. Baumer, C. Beste, et al., Temporal relationship between premonitory urges and tics in Gilles de la Tourette syndrome, Cortex 77 (2016) 24–37 https://doi.org/10.1016/j.cortex.2016.01.008.
[9] S.C. Cohen, J.F. Leckman, M.H. Bloch, Clinical assessment of Tourette syndrome and tic disorders, Neurosci. Biobehav. Rev. 37 (6) (2013) 997–1007 https://doi.org/10.1016/j.neubiorev.2012.11.013.
[10] C. Kwak, K. Dat Vuong, J. Jankovic, Premonitory sensory phenomenon in Tourette's syndrome, Mov. Disord. 18 (12) (2003) 1530–1533 https://doi.org/10.1002/mds.10618.
[11] C. Ganos, U. Kahl, O. Schunke, S. Kuhn, P. Haggard, C. Gerloff, et al., Are premonitory urges a prerequisite of tic inhibition in Gilles de la Tourette syndrome? J. Neurol. Neurosurg. Psychiatry 83 (10) (2012) 975–978 https://doi.org/10.1136/jnnp-2012-303033.
[12] K.R. Muller-Vahl, L. Riemann, S. Bokemeyer, Tourette patients' misbelief of a tic rebound is due to overall difficulties in reliable tic rating, J. Psychosom. Res. 76 (6) (2014) 472–476 https://doi.org/10.1016/j.jpsychores.2014.03.003.
[13] J.A. Obeso, J.C. Rothwell, C.D. Marsden, Simple tics in Gilles de la Tourette's syndrome are not prefaced by a normal premovement EEG potential, J. Neurol. Neurosurg. Psychiatry 44 (8) (1981) 735–738.
[14] S.M.A. van der Salm, M.A.J. Tijssen, J.H.T.M. Koelman, A.-F. van Rootselaar, The bereitschaftspotential in jerky movement disorders, J. Neurol. Neurosurg. Psychiatry 83 (12) (2012) 1162–1167 https://doi.org/10.1136/jnnp-2012-303081.
[15] G. Pfurtscheller, F.H. Lopes da Silva, Event-related EEG/MEG synchronization and desynchronization: basic principles, Clin. Neurophysiol. 110 (11) (1999) 1842–1857.
[16] J.B. Shute, M.S. Okun, E. Opri, R. Molina, P.J. Rossi, D. Martinez-Ramirez, et al., Thalamocortical network activity enables chronic tic detection in humans with Tourette syndrome, Neuroimage Clin 12 (2016) 165–172 https://doi.org/10.1016/j.nicl.2016.06.015.
[17] K. Biermann-Ruben, A. Miller, S. Franzkowiak, J. Finis, B. Pollok, C. Wach, et al., Increased sensory feedback in Tourette syndrome, Neuroimage 63 (1) (2012) 119–125 https://doi.org/10.1016/j.neuroimage.2012.06.059.
[18] E. Maris, R. Oostenveld, Nonparametric statistical testing of EEG- and MEG-data, J. Neurosci. Methods 164 (1) (2007) 177–190 https://doi.org/10.1016/j.jneumeth.2007.03.024.
[19] V. Niccolai, H. van Dijk, S. Franzkowiak, J. Finis, M. Sudmeyer, M. Jonas, et al., Increased beta rhythm as an indicator of inhibitory mechanisms in tourette syndrome, Mov. Disord. 31 (3) (2016) 384–392 https://doi.org/10.1002/mds.26454.
[20] N. Swann, N. Tandon, R. Canolty, T.M. Ellmore, L.K. McEvoy, S. Dreyer, et al., Intracranial EEG reveals a time- and frequency-specific role for the right inferior frontal gyrus and primary motor cortex in stopping initiated responses, J. Neurosci. 29 (40) (2009) 12675–12685 https://doi.org/10.1523/JNEUROSCI.3359-09.2009.
[21] G.M. Jackson, A. Draper, K. Dyke, S.E. Pepes, S.R. Jackson, Inhibition, disinhibition, and the control of action in tourette syndrome, Trends Cognit. Sci. 19 (11) (2015) 655–665 https://doi.org/10.1016/j.tics.2015.08.006.
[22] D.J. Serrien, M. Orth, A.H. Evans, A.J. Lees, P. Brown, Motor inhibition in patients with Gilles de la Tourette syndrome: functional activation patterns as revealed by EEG coherence, Brain 128 (Pt 1) (2005) 116–125 https://doi.org/10.1093/brain/awh318.
[23] B.S. Peterson, P. Skudlarski, A.W. Anderson, H. Zhang, J.C. Gatenby, C.M. Lacadie, et al., A functional magnetic resonance imaging study of tic suppression in Tourette syndrome, Arch. Gen. Psychiatr. 55 (4) (1998) 326–333.
[24] K.J. Plessen, T. Wentzel-Larsen, K. Hugdahl, P. Feineigle, J. Klein, L.H. Staib, et al., Altered interhemispheric connectivity in individuals with Tourette's disorder, Am. J. Psychiatry 161 (11) (2004) 2028–2037 https://doi.org/10.1176/appi.ajp.161.11.2028.
[25] B.S. Peterson, L. Staib, L. Scahill, H. Zhang, C. Anderson, J.F. Leckman, et al., Regional brain and ventricular volumes in Tourette syndrome, Arch. Gen. Psychiatr. 58 (5) (2001) 427–440.
[26] C.-Y. Chen, N.G. Muggleton, O.J.L. Tzeng, D.L. Hung, C.-H. Juan, Control of prepotent responses by the superior medial frontal cortex, Neuroimage 44 (2) (2009) 537–545 https://doi.org/10.1016/j.neuroimage.2008.09.005.
[27] V. Roessner, B. Albrecht, P. Dechent, J. Baudewig, A. Rothenberger, Normal response inhibition in boys with Tourette syndrome, Behav. Brain Funct. 4 (2008) 29 https://doi.org/10.1186/1744-9081-4-29.
[28] G.R. Muller, C. Neuper, R. Rupp, C. Keinrath, H.J. Gerner, G. Pfurtscheller, Event-related beta EEG changes during wrist movements induced by functional electrical stimulation of forearm muscles in man, Neurosci. Lett. 340 (2) (2003) 143–147.
[29] Z. Wang, T.V. Maia, R. Marsh, T. Colibazzi, A. Gerber, B.S. Peterson, The neural circuits that generate tics in tourette's syndrome, Aust. J. Pharm. 168 (12) (2011) 1326–1337 https://doi.org/10.1176/appi.ajp.2011.09111692.
[30] H. Shibasaki, M. Hallett, What is the Bereitschaftspotential? Clin. Neurophysiol. 117 (11) (2006) 2341–2356 https://doi.org/10.1016/j.clinph.2006.04.025.
[31] E. Crossley, A.E. Cavanna, Sensory phenomena: clinical correlates and impact on quality of life in adult patients with Tourette syndrome, Psychiatr. Res. 209 (3) (2013) 705–710 https://doi.org/10.1016/j.psychres.2013.04.019.
[32] H.S. Singer, Tourette's syndrome: from behaviour to biology, Lancet Neurol. 4 (3) (2005) 149–159 https://doi.org/10.1016/S1474-4422(05)01012-4.
[33] C.L. Rae, H.D. Critchley, A.K. Seth, A bayesian account of the sensory-motor interactions underlying symptoms of tourette syndrome, Front. Psychiatry 10 (2019) 29 https://doi.org/10.3389/fpsyt.2019.00029.