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Brain Event - Related Oscillations in Parkinsonian Patients During Discrimination Task Conditions

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1. Introduction

Parkinson's disease (PD) is caused by a disruption of dopaminergic neurotransmission in the basal ganglia, which serve as an integrative centre for the sensory and cognitive processing of information and as a mutual link between this processing and disturbed motor performance. The basal ganglia and the cerebellum transmit information via the thalamus to the cerebral cortex in order to regulate movement. The neurotransmitter changes affect the output of the striatum into the globus pallidus as well as into the thalamus and cerebral cortex beyond. The disease is a common and disabling disorder of movement characterized by poverty, slowness and impaired scaling of voluntary movements (akinesia and bradykinesia), muscle rigidity, and tremor of the limbs at rest. Alterations of the basal ganglia with proven neuronal degenerative disorders of dopaminergic neurons and a reduction in activity in frontostriatal neural circuitry have been suggested to play a role in the executive dysfunction of PD (Taylor et al., 1990; Innis et al., 1993; Lewis et al., 2003; Owen, 2004; Leblois et al., 2006; Anik et al., 2007). The slowed information processing, insufficient encoding strategies and planning, and attentional set-shifting are related to memory deficits and cognitive impairment in PD (Daum et al., 1995; Pillon et al., 1997; Knoke et al., 1998; Robertson & Empson, 1999; Sawamoto et al., 2002; Cools, 2006). Neuropsychological studies of PD patients report cognitive deficits even during the early stages of the disease (van Spaendonck et al., 2006). The primary working memory deficit in PD is associated with impaired free recall performances (Higginson et al., 2003).

Many electroencephalographic (EEG) studies on PD have used the event-related potential (ERP) method, where the early modal dependent and obligatory N1 and P2 components permit analysis of sensory events while the later N2 and P3 potentials reflect the cognitive processes involving the assessment of stimuli, decision making, strategy selection and recognition memory. ERP investigations have shown P3 predominantly with prolonged latencies and/or diminished amplitudes for Parkinsonian patients (PP) when compared to healthy subjects (HS) (Evarts et al., 1981; Tachibana et al., 1992; Philipova et al., 1997; Wascher et al., 1997; Minamoto et al., 2001; Antal et al., 2002; Wang et al., 2002). Such results have been interpreted as electrophysiological signs of cognitive slowing with respect to stimulus classification and attentional processing (Robertson & Empson, 1999).

One valuable means of assessing deviations from the normal state in PD is to study oscillatory brain processes. In the ERP method, however, the functional significance of the

responses in different frequency bands is lost. More clarification could be expected when attentional processes in PD during a representation of discrimination tasks (Vieregge et al., 1994) are examined using event-related desynchronization/synchronization (ERD/ERS) method. In the early stages, PD also affects cognitive functions (Cools, 2006). Cognitive processes require transient integration between different brain areas. Hence, dynamic links are formed, mediated by the ERS or ERD of neuronal assemblies. ERD is defined as a relative decrease in the power of a certain frequency band during stimulus processing, while ERS is a relative increase in the power of the same frequency (Pfurtscheller & Klimesch, 1991). The ERD/ERS method has been used to study auditory and visual working memory encoding and categorization processes in PP; studies indicate less theta-ERS and upper alpha-ERD reflected disturbance of both the basal ganglia activity as well as activity related to their thalamo-cortical neuronal nets at frontal electrode locations (Schmiedt et al., 2005; Ellfolk et al., 2006). The encoding of auditory stimuli elicits alpha- and theta-ERS, while memory retrieval during the presentation of a target stimulus elicits theta-ERS and alpha-ERD (Karrasch et al., 1998, 2004; Krause et al., 1996, 1999). Oscillations in the beta frequency band are associated with cognitive control of behaviour or "executive functions" (Pfurtscheller & Lopes da Silva, 1999; Engel et al., 2001). By means of an auditory stop-signal task, the differential participation of beta subbands in voluntary motor control can be revealed: ERD in the 20–30 Hz band is related to initiation of movement, while ERS in a low frequency beta band (12–16 Hz) is exclusively linked to the stopping of planned action (Pfurtscheller & Lopes da Silva, 1999; Engel et al., 2001). One proposed hypothesis for these observations is that lower-frequency beta subbands represent inhibitory components of cognitive control and are more generalized, while higher frequency beta subbands take part in response choice and activation and are more specialized in terms of both function and cortical distribution.

Some recent investigations (Basar, 2001; Ozgoren et al., 2005; Sutoh et al., 2000; Gurtubay et al., 2001) propose that beta and gamma cortical rhythms may serve cognitive processes such as linking perception to action or movement planning (Donoghue et al., 1998). Research both on animals and humans has suggested that gamma-frequency activity also plays an important role in attention as well as working and long-term memory (Herrmann et al., 2004). Current investigations using intracranial and high-density electro- and magnetoencephalographic recordings explore the involvement of gamma-band synchronization in various cognitive paradigms in humans (Engel et al., 2001; Basar et al., 2001; Herrmann et al., 2004; Pantev, 1995; Tallon-Baudry et al., 1996; Farmer, 1998; Fries et al., 2001). Other works associate the changes in EEG spectral power in the gamma frequency band to interactions between the cortex and basal ganglia (Gatev & Wichmann, 2008). Additionally, akinesia in PP has been related in some studies to abnormally increased beta (15–30 Hz) and decreased gamma (35–80 Hz) synchronous oscillatory activity in the basal ganglia (Weinberger et al., 2006). Other results suggest that resting tremor in PD is associated with an altered balance between beta and gamma oscillations in the motor circuits of the subthalamic nucleus (STN) and is exhibited as increased oscillatory activity in the low gamma frequency range (35–55 Hz) during periods with stronger tremor (Weinberger et al., 2008). Therapeutic doses of dopaminergic medication in PP attenuate the beta band power in the STN, giving rise to the hypothesis that the beta prominence is pathological in PD (Cassidy et al, 2002; Kühn et al., 2006; Levy et al., 2001; 2002; Priori et al., 2002). Treatment of PP with dopaminergic therapy leads to increased gamma band activity in the basal ganglia and thus to improvement in motor performance (Brown et al., 2001),

hence the suggestion that synchronization of the activity of populations of basal ganglia neurons in the gamma band may facilitate motor processing (Brown, 2003). Investigations based on local field potentials recorded from the STN in PP show increased power in the beta range (13–35 Hz) while the patient is at rest (Cassidy et al., 2002; Levy et al., 2002; Brown et al., 2001; Priori et al., 2004). This suggests that there is excessive synchrony in the basal ganglia networks in PD and some of the clinical signs of the disease, it is proposed, stem from this abnormal synchrony between basal ganglia and cortical circuits (Brown, 2003; Marsden et al., 2001).

The aim of the present study was to investigate the functional relationships between oscillatory EEG-dominant components with ERD/ERS method for PP and HS during auditory discrimination tasks within two poststimulus intervals of 0–250 and 250–600 ms. We first focused on time-frequency analysis of delta, theta and alpha rhythms, the appearance of which is well established in PD and is thought to reflect the degree of cortical activation during the information processing. We later shift our focus to the beta and gamma bands, where our aim is to assess the differences between PP and HS in these frequency bands and check an assumption that some PP clinical symptoms stem from excessive synchrony between the basal ganglia and cortical circuits. This investigation of the oscillatory processes and ERD/ERS in HS and PP could contribute to clarification of the disturbances of the neurophysiological mechanisms of this disease.

2. Methods

2.1 Experimental procedure

2.1.1 Subjects

We investigated eleven voluntary patients with a mean age of 61 ± 12.2 years (±s.d.; 7 males, 4 females) with a diagnosis of idiopathic Parkinson's disease for no longer than 2.8 years, assessed by a neurologist at the University Neurological Hospital, with score of I on the Hoehn–Yahr scale of motor function (Hoehn & Yahr, 1967). Patients receiving levodopa (L–dopa) drugs (Sinemet) were included in order to reduce the heterogeneity in the medication. During the experimental session, all patients were in off-phase of the medication. None of the patients had dementia, depression, a presence of atherosclerosis, attendant neurological complications or pronounced tremor. The same number of healthy volunteers was included as aged-matched healthy controls with a mean age of 59.5 ± 9.5 years. Screening confirmed that subjects were free of past or current psychiatric and neurological disorders. All participants were right handed and without deficits in hearing. Handedness was assessed by a questionnaire adapted from the Edinburgh Handedness Inventory (Oldfield, 1971). The study was performed with the approval of the local ethics committee. The subjects were introduced to the nature of the investigation and their informed written consent was obtained according to the declaration of Helsinki.

2.1.2 Stimuli and task

Each subject was comfortably seated in an ergonomically designed chair inside a Faraday cage, monitored by a Canon Video system. The experimental design included a binary sensory-motor reaction task. Each sensory-motor series consisted of 50 computer generated low frequency (LT – 800 Hz) and 50 high frequency (HT – 1000 Hz) acoustic stimuli with an intensity of 60 dB, duration of 50 ms, and an inter-stimulus interval of 2.5–3.5 s presented to the subject in a randomized order. PP and HS were asked to press a key with the index

finger of each hand and make rapid and accurate choice responses with the left hand to the high frequency (HT) or with the right hand to the low frequency (LT) tone. The movement performance from the stimulus presentation to the onset of voluntary force production (onset of reaction time) and from the stimulus presentation to the force peak (force peak latency) were measured by a force transducer. A surface electromyographic activity pattern of the first dorsal interosseus muscles was also registered.

2.1.3 EEG recording

An electroencephalogram (EEG) was recorded from Fz, Cz, Pz, C3' and C4' (10/20, system), using Ag/AgCl Nihon-Kohden electrodes with a reference to both processi mastoidei and a ground electrode, placed on the forehead. An oculogram (EOG) was recorded from m. orbicularis oculi dex. We placed two EOG electrodes next to the eyes to register eye movements. EEG and EOG data were recorded using a Nihon-Kohden EEG-4314F (cut-off frequencies of 0.3–70 Hz) and recorded together with markers of the movement performance as a force profile and a surface electromyographic activity pattern of the first dorsal interosseus muscles (bandpass filtered 0.03–500 Hz). The signals were digitized on-line (10 bit A/D converter, 256 samples/s). The data recordings for the sensorimotor task were synchronized to the marker of the stimulus onset (-0.2 s before and 0.8 s after the stimulus). Only recordings that were artifact-free with respect to event-related potentials were processed. We applied a Chebyshev Type II bandpass filter (1-70 Hz) and second-order notch filter at frequency 50 Hz (AC) component. We defined an independent reference interval in order to quantify the changes in the time-frequency energy density of the signal. We used stimulus-nonrelated subepochs within the resting condition series, distant enough (-1.4 s) from the stimulus onset, not including event-related properties, and exceeding the period of the lowest frequency studied in the signal (1.5 Hz, 0.67 s). We preselected trials by applying a bootstrap estimation within the reference period and a false discovery rate correction for multiple comparisons (0.05) to the available data across the indexes corresponding to time and number of the trial, eliminating the need for a strict assumption of ergodicity (Durka et al., 2004).

2.2 Analysis

The time-frequency analysis (TF) represented the power of a continuous EEG signal as a function of both time and frequency (Matlab®, Mathworks, Inc.). For time amplitude-frequency distributions, the filtered signal was analyzed with a sliding-window fast Fourier transform with length 200 ms and step 10 ms. The amplitude was computed for every time window t and frequency bin f by the real and imaginary Fourier coefficients. The amplitude modulations obtained for each frequency band for each subject in a group were added across trials in order to compare amplitude changes in the post-stimulus intervals with respect to pre-stimulus interval reference amplitudes, i.e. to derive ERD/ERS. This method characterizes the relative amplitude decrement/increment of the given frequency during the post-stimulus period in relation to pre-stimulus amplitude modulation of the same frequency (Pfurtscheller G, Klimesch, 1991). This resulted in ERD/ERS values which could then be presented as percentage changes with respect to the reference interval. Negative values indicate a relative power decrease (ERD), whereas positive values point to a relative power increase (ERS). Relatively, sensory processing takes place during the first post-stimulus interval (T1: 0–250 ms) and cognitive processing during the second post-stimulus

interval (T2: 250–600 ms), defined as beginning when a tone ends. The peak amplitude modulations, defined in 10 ms bins, were specified as dominating components. Afterwards, the high amplitudes in each frequency band, respectively, were added over trials and across subjects to compare their amplitude changes in the post-stimulus intervals with those in the reference interval. Thus, we calculated the ERD/ERS of delta (δ ~ 1.5–4 Hz), theta (θ ~ 4.1–7 Hz) and alpha (α ~ 7.1–13 Hz) waves as percentage power differences in each frequency band compared to the reference interval for both 0–250 ms and 250–600 ms post-stimulus intervals. We also defined the ERD/ERS of beta (β_1 ~ 13.1–20 Hz), (β_2 ~ 20.1–32 Hz) and gamma (γ ~ 32.1–50 Hz) frequency rhythms during the post-stimulus intervals T1 (0–250 ms) and T2 (250–600 ms).

We employed the detection of the temporally dynamic processes similar to the approach applied by Foffani et al. (Foffani et al., 2005) that describes the behaviour of β_1 -, β_2 -, γ - ERS rhythms in zones which vary both in amplitude and frequency. Each zone *i* is characterized by a value *ERSi* (*t*) and a frequency value *Fi* (*t*), both dependent on time, which separately describe event-related synchronization and corresponding frequency modulations for the beta1, beta2 and gamma rhythms in two post-stimulus time windows. The significance of the observed ERS values was tested for each frequency band and EEG channel using a permutation test, including corrections for multiple comparisons between time points for time course analysis (Mason & Newton, 1990). The latencies of ERS and corresponding frequency (relative to stimulus onset) were measured as the last zero-crossing before a significant modulation, after subtracting the baseline mean ERS value. Although the ERS clearly occurred, the relationship between the ERS peaks and the maximum of the average event-related synchronization (AERS) was not always evident. Since the AERSs of different channels were not identical, an exact coincidence between the peaks times was not observed. The probabilities for the amplitudes and latencies were not uniform and the activity distribution was clearly not Gaussian. We estimated the latency shift between the largest peaks for each pair of channels.

2.2.1 Statistics

We performed statistical analyses of the ERD/ERS for the two post-stimulus intervals and assessed the statistical difference between the groups (PP and HS) for each tone type and interval by means of a bootstrap nonparametric procedure (Mason & Newton, 1990). The characteristics were grouped by tone, interval, patients and healthy controls and analyzed by means of a permutation test for multiple comparisons (Mason & Newton, 1990). The computed random distribution for interval was analyzed with a nonparametric test (Kruskal-Wallis [KW] test, $p < 0.05$) for pairs comparison of the scalp leads between patient and control group. This procedure reduces the influence of random variations in experimental conditions between trials. The ERD/ERS analysis served to identify the most robust differences between groups and was generally done for the two time windows. The parameters of the movement performance (onset of reaction time, force peak latency and error of performance) were processed statistically by Mann–Whitney U test.

3. Experimental results

3.1 Response parameters

Parkinson's patients showed a longer reaction time onset, but the difference between the two groups was not significant: in response to a low tone—440.5 ± 135.8 ms in HS and 508.4

± 148.5 ms in PP (mean ± S.D., $p > 0.05$), in response to a high tone—455.7 ± 134.6 ms in HS and 500.4 ± 146.5 ms in PP ($p > 0.05$). Parkinson's patients had significantly longer force peak latency (FPL) in response to the two tone types. The FPLs were 672.8 ± 154.7 ms in HS and 919.96 ± 163.7 ms in PP in response to the low tone (mean ± s.d, $p < 0.02$). In response to the high tone, the FPLs were 690.7 ± 148.7 ms in HS and 934.9 ± 160.2 ms in PP ($p < 0.05$). The mean errors (false and missing responses) were 4.5 and 5.1 in PP, respectively, in response to the low and high tone. The mean errors were 3.5 and 4.4 in HS, respectively, in response to the low and high tone. The differences of errors between the two groups were not significant ($p > 0.05$).

3.2 Frequency components

The grand average ERD/ERS values as a function of time and frequency band at the frontal, central, parietal, left and right motor areas were used for assessment of group means with the standard errors (±SE) for the post-stimulus intervals T1 (0–250 ms) and T2 (250–600 ms). The statistical group comparison for pair channels are shown graphically to illustrate the delta-, theta-, alpha-, beta-, gamma- ERD/ERS following the low frequency tone type (Figs. 1A, 2A) and high frequency tone type (Figs. 1B, 2B) for the early (0–250 ms) and late (250–600 ms) post-stimulus intervals.

3.2.1 Delta

The patterns of δ-ERD/ERS were different between the groups for both intervals in response to both tone types (Dushanova et al., 2009). In the early post-stimulus interval, central δ-ERS amplitude responses were most pronounced in the HS after both tones (Cz, Fig. 1A, B, 1st row, left plots) and in the PP at the frontal side for the high tone (Fig. 1B, left) and parietal area for the low tone (Fig. 1A, left). The least pronounced δ-ERS were those appearing at the frontal side in the HS and in the left motor area in PP following both tones (Fig. 1A, B, left). The comparison by the bootstrap procedure of δ-ERS between the two groups after the low frequency tone determined that the control δ-ERS was significantly higher than that of the PP for all channels (Fz, Pz, $p < 0.05$; Cz, C3′, C4′, $p < 0.001$). Both groups displayed δ-ERS following the high frequency tone (Fig. 1B, left). This was significantly higher for the HS than the PP at centro-parietal, left and right motor areas (Cz, C3′, C4′ $p < 0.001$; Pz, $p < 0.05$). The PD patients' frontal δ-ERS, however, was greater than that of the HS ($p < 0.001$).

The HS maintained δ-ERS at all electrodes during the late post-stimulus interval T2 following the low frequency tone, while in the PP the early post-stimulus δ-ERS was reversed to become δ-ERD in the late post-stimulus interval (Fig. 1A, B, 1st row, right). The highest δ-ERS was located at parietal side for the HS, whereas the PP had a less enhanced parietal δ-ERD. The PP showed the most enhanced δ-ERD at the left motor area (Fig. 1B, right). Following the high frequency tone, δ-ERS was elicited at all electrodes in the HS (Fig. 1B, right). The PP group, in comparison with the HS, showed a less pronounced central δ-ERS (Cz, $p < 0.001$) and specific δ-ERD at parietal and left motor areas ($p < 0.001$).

3.2.2 Theta

In the first post-stimulus interval following both tone types, the θ-ERS responses were most prominent at parietal electrodes for both groups (Fig. 1, A, B, 2nd row, left). Following the low tone, the θ-ERS elicited was significantly higher for HS than for PP at frontal, left and

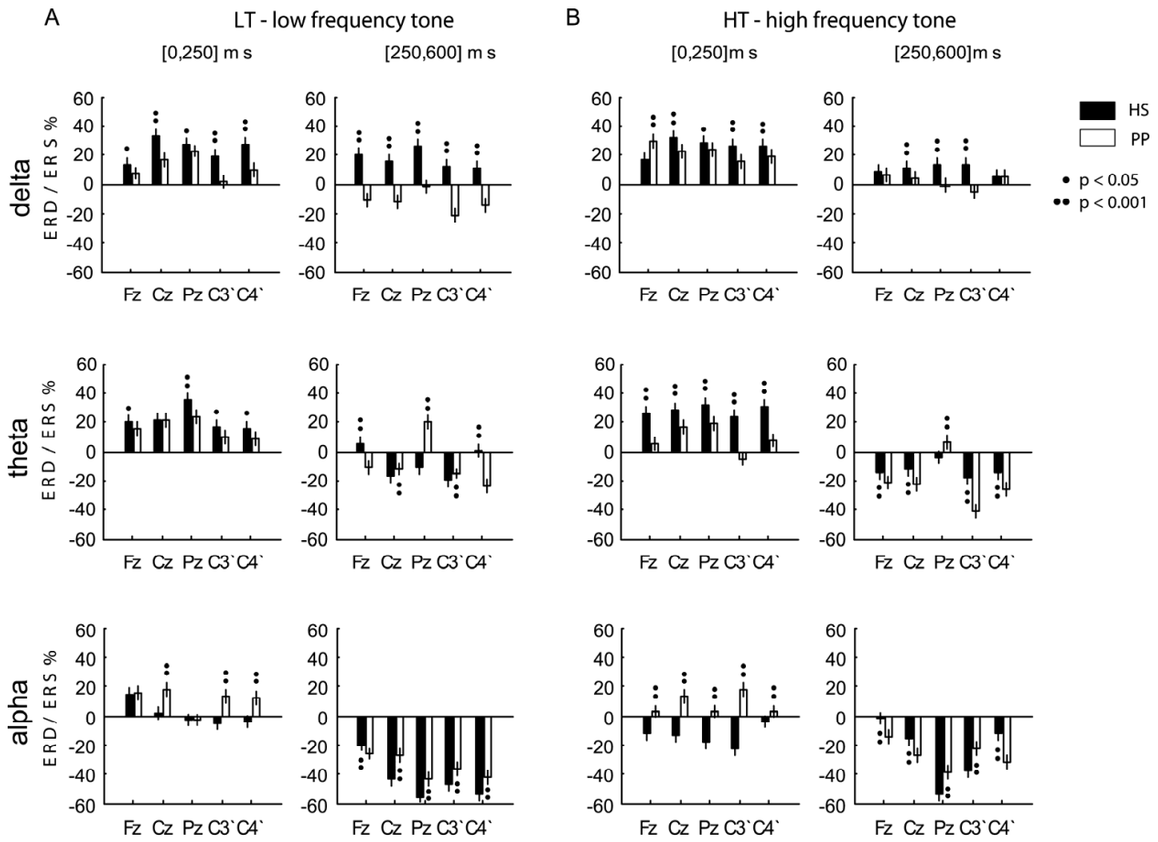

Fig. 1. Group means (±SE) and statistical results of δ-, θ-, α-ERD/ERS over all HS and PP trials after the low tone (A) and high tone type (B) for the early (left) and late time period (right) at all channels.

right motor areas ($p < 0.05$), parietal side ($p < 0.001$), and centrally was non-significantly different (Cz, $p > 0.05$; Fig. 1A, 2nd row, left). Following the high frequency tone, PP produced a significantly lower θ-ERS than the HS at right motor area, fronto-central and parietal sides ($p < 0.001$; Fig. 1B, left). The left motor area showed a pronounced θ-ERS for the HS and a weak θ-ERD in the PP following the high frequency tone.

The comparison of the groups during the late period following the low frequency tone showed θ-ERD in both groups at most electrodes with the following exceptions. PP recorded a large parietal θ-ERS while a less prominent θ-ERS appeared in HS at frontal and right motor areas (Fig. 1A, 2nd row, right). The most pronounced θ-ERDs for HS were at central and left motor areas ($p < 0.001$). The signal at the parietal area was characterized by a very prominent θ-ERS in the patients but by θ-ERD in the control group. In response to the high frequency tone, we found a significant θ-ERD for the PP as compared with the HS at fronto-central, left and right motor areas ($p < 0.001$; Fig. 1B, right). The signal at the parietal area was characterized in a similar manner to that elicited by a low tone, but with a less pronounced θ-ERS in the PP and smaller θ-ERD in the HS.

3.2.3 Alpha

The PP showed fronto-central, left and right motor α-ERS responses following the LT, while the HS had synchronization only at the fronto-central sides but α-ERD at the parietal side,

right and left motor areas (Fig. 1A, 3rd row, left). The alpha frequency band in the first interval after the LT showed an enhanced central α-ERS for the PP in comparison with the HS ($p < 0.001$; Fig. 1A, left). The right and left motor areas manifested different reversal alteration as α-ERS for the PP and weakly elicited α-ERD for the HS. Following the high tone, we detected significantly different processes, α-ERS in the PP contrasted with α-ERD in the HS at all electrodes, most prominently at the left motor side (Fig. 1B, left).
Alpha-ERD differences between the groups were observed after low as well as high frequency tones during the late period (Fig. 1A, B, right). After the low tone, the HS α-ERD means were significantly more pronounced than those of the PP at centro-parietal sides, left and right motor areas ($p < 0.001$; Fig. 1A, right). The PP displayed a higher frontal α-ERD than that in the HS ($p < 0.001$). Alpha-ERD differences were observed between the groups following the high tone (Fig. 1B, right). The α-ERD signals of HS at parietal and left motor areas were more pronounced than in the PP ($p < 0.001$). The PP fronto-central and right motor α-ERD signals had greater respective magnitudes than those in HS ($p < 0.001$).

3.2.4 Beta1

During the early post-stimulus interval T1, ERD/ERS β1 patterns appeared in the lower frequency portions of this band for each electrode and group (Dushanova et al., 2010). The maximum synchronized β1 bursts across the channels were localized over fronto-central sides for both groups after LT, but had significantly higher amplitude and shorter durations in PP than HS ($p < 0.001$, bootstrap, KW test; Table 1, **a**). Later β1 ERS bursts were found only in PP over the frontal side and left motor area (Table 1, LT (T1), **b**). Synchronized β1 bursts were centered on right motor side for PP and fronto-central sides for HS, following HT (Table 1, HT (T1)**, a**, **b**). In PP, the peaks were of a significantly lower amplitude and peaked later at centro-parietal sides than in HS (Table 1, HT (T1)**, a**). During the late post-stimulus interval T2, frontal synchronized β1 peaks at 20 Hz were extracted only in HS after HT and had a prolonged latency of 176 ± 11.5 ms (Table 1, HT (T2)**, f**).

3.2.5 Beta2

During T1, ERS β2 bursts were present only in PP after either tone (Table 2, LT(T1), HT(T1)). Their maximum amplitude across the scalp was localized over frontal side for either tone, right and left motor areas respectively for LT and HT (Table 2, **c**). They peaked earlier at the right than at the left motor area, fronto-central leads (Table 2, **c**), and parietal side (Table 2, **d**) for either tone, but in higher β2 frequency range after LT than after HT. During T2, the maximum value of β2 bursts across all recorded areas was centered on right motor area in PP (Table 2, **g**) but on parietal side in HS following LT (Table 2, LT(T2), **j**). More widely distributed, prolonged β2 ERS bursts were pronounced over all locations for PP following HT, but limited to frontal-central and right motor areas for HS (Table 2, HT(T2)). The maximum scalp β2 burst was localized at the right motor area in PP and frontal area in HS after HT (Table 2, **g - j**). At the right motor area, spectral peaks of low β2 exhibited significantly more exaggerated and prolonged bursts in PP than in HS ($p < 0.001$, Table 2, **g - j**). The frontal synchronized high frequency β2 bursts appeared in PP during two subintervals, the first one with a significantly shorter latency than the low frequency β2 bursts in HS ($p < 0.001$, Table 2, **g**). In PP, synchronized bursts of high β2 were generated on the left motor area (Table 2, **g**) earlier than larger synchronized bursts at low β2 (Table 2, **i**, **j**).

Channels	**Fz** **ERS(%)/ F/ t** (±SE) **D**	**Cz** **ERS(%)/ F/ t** (±SE) **D**	**Pz** **ERS(%)/ F/ t** (±SE) **D**	**C3′** **ERS(%)/ F/ t** (±SE) **D**	**C4′** **ERS(%)/F /t** (±SE) **D**
Subjects **Tone(period)**			**HS**		
LT (T1)	**64.1 (±1.2)/** 13 Hz 40 (±7.3) ms [8–72] ms (**a**)	50.1 (±5.8) /13 Hz 36 (±6.9) ms [8–64] ms (**a**)	18.8 (±0.5)/13 Hz 20 (±5.2) ms [8–32] ms (**a**)	41 (±3.3)* /13 Hz 36 (±28) ms [8–80] ms (**a**)	44.9(±5.2)*/13 Hz 32.0 (±6.5)** ms [8–72] ms (**a**)
HT (T1)	**68.6(±10.2)**/13.9(±0.3)Hz [13–15] Hz 36 (±6.9) ms [8–64] ms (**a**)	62.7(±8.5)*/13.7(±0.3)Hz [13–15] Hz 32 (±6.5)** ms [8–56] ms (**a**)	30.49(±3.3)*/13Hz 28(±6.1)** ms [8–48] ms (**a**)	54.7(±8.3)*/13.5(±0.3)Hz [13–15] Hz 32(±6.5) ms [8–56] ms (**a**)	50.4(±6.6)*/13 Hz 32(±6.5) ms [8–56] ms (**a**)
LT (T2)	–	–	–	–	–
HT (T2)	**51 (±3.2)**/20 Hz 516 (±11.5) ms [424–600] ms (**f**)	–	–	–	–
Subjects **Tone(period)**			**PP**		
LT (T1)	**69.4(±0.9)*/** 13 Hz 12 (±4)** ms [8–80] ms (**a**)	57.2(±3)*/13 Hz 16 (±4.6)** ms [8–64] ms (**a**)	–	36.8(±2.8)/13 Hz 12 (±4)** ms [8–16] ms (**a**)	33.9(±5)/13Hz 48 (±6.5) ms [24–72] ms (**a**)
	15.8(±0.2)/14(±0.6) Hz 124 (±5.2) ms [112–136] ms (**b**)	–	–	19.3(±1.3)/18 Hz 148 (±5.2) ms [136–160] ms (**b**)	–
HT (T1)	–	34.4(±3.8)/13.8(±0.3) Hz 53.6 (±11.2) ms [8–104] ms (**a**)	21.3(±0.8)/ 13 Hz 46 (±8.7) ms [8–88] ms (**a**)	35(±1.9)/ 13 Hz 20(±5.2)** ms [8–32] ms (**a**)	**40.2(±2.8)**/13 Hz 24 (±5.7) ** ms [8–40] ms (**a**)
	–	–	–	–	25(±1.1)/16 Hz 92 (±5.2) ms [80–112] ms (**b**)
LT (T2)	–	–	–	–	–
HT (T2)	–	–	–	–	–

Note: Bold-marked mean ERS β1 bursts (%) (±SE) are maximum across the channels for each condition and subject separately for LT(T1), HT(T1), LT(T2), HT(T2); **F** (Hz) – mean frequency peaks (±SE) of the maximum ERS β1 bursts across trials; **t** (ms) – mean times (±SE) of maximum ERS β1 bursts across trials during T1 (or T2) with respect to stimulus onset; **D** (ms) – time duration of these short–term zones with ERS β1 bursts; [1] * =a significant difference in ERS between the groups HS, PP for each channel and tone separately, marked the higher value ($p<0.001$, KW test); [2] ** =a significant difference in **t** between the groups for each channel and tone separately, marked the shorter value ($p<0.001$, KW test). Lower case letters in Table 1 marked consecutive sub-intervals with ERS β1 bursts: **a**, **b** during T1; **f** during T2.

Table 1. Mean ERS β1 bursts (%) across the trials with mean frequency peaks **F (**Hz) for HS and PP after LT and HT presented in short–term zones **D** (ms) during early T1 and late T2 period.

Channels	**Fz** **ERS(%)/ F/ t** (±SE) **D**	**Cz** **ERS(%)/F/ t** (±SE) **D**	**Pz** **ERS(%)/ F/ t** (±SE) **D**	**C3′** **ERS(%)/ F/ t** (±SE) **D**	**C4′** **ERS(%)/F /t** (±SE) **D**
Subjects tone(period)			**HS**		
LT (T1)	–	–	–	–	–
HT (T1)	–	–	–	–	–
LT (T2)	–	–	**25.8(±1.6)**/32Hz 584 (±6.5) ms [560–600] ms (**j**)	19.6(±1.3)/30.5(±0.2)Hz 588 (±6.1) ms [576–600] ms (**j**)	–
HT (T2)	**43.9(±3.1)**/23.1(±0.1)Hz 516 (±11.5) ms [424–600] ms (**g–j**)	18.1(±0.6)/21Hz 488(±5.7) ms [472–504] ms (**h**)	–	–	28.2(±2.3)/ 32 Hz 384 (±6.5)** ms [360–408] ms (**g**)
	-	–	–	–	40.6(±3.1)/23.4(±0.4)Hz 532 (±10.6) ms [456–600] ms (**h–j**)
Subjects tone(period)			**PP**		
LT (T1)	**61.1 (±1.1)**/ 32 Hz 40 (±5.7) ms [24–56] ms (**c**)	30.6(±4.7)/32Hz 48 (±5.7) ms [32–64] ms (**c**)	23.2(±1.7)/25.2(±1.6)Hz 64 (±9.2) ms [8–120] ms (**c**, **d**)	22.4(±3.7)/29.7(±0.3)Hz 32 (±4.6) ms [24–40] ms (**c**)	**60.3(±1.1)**/32Hz 12 (±4) ms [8–16] ms (**c**)
HT (T1)	41.2(±2)/24.3(±0.5)Hz 60 (±8.8) ms [32–88] ms (**c**)	29.8(±1.1)/23Hz 64 (±4.6) ms [56–72] ms (**c**)	28(±2.7)/24.2(±1.1)Hz 121.6 (±9.1) ms [64–160] ms (**d**)	**46(±2.8)**/23.9(±0.4) Hz 64 (±6.5) ms [40–88] ms (**c**)	38.1(±1.2)/22Hz 56 (±4.6) ms [48–72] ms (**c**)
	–	–	–	19.3(±0.8)/32 Hz 168 (±4.6) ms [160–176] ms (**e**)	–
LT (T2)	–	15.5(±0.1)/32Hz 392 (±4.6) ms [384–400] ms (**g**)	17.9(±1)/ 32 Hz 376 (±4.6) ms [368–384] ms (**g**)	–	**30.7(±2.1)**/32 Hz 368 (±8.6) ms [320–416] ms (**g**)
	–	–	24.7(±1.7)/30.9(±0.1)Hz 528 (±10.8) ms [448–600] ms (**h**)	26(±1.5)/29.3(±0.5)Hz 492 (±9.5) ms [432–552] ms (**h**)	–
HT (T2)	36.7(±2.9)/ 32Hz 388(±8.9)** ms [336–440] ms (**g**)	23.8(±1.3)*/32Hz 376 (±7.3)** ms [344–408] ms (**g**)	25.3(±0.7)/30(±0.03)Hz 452 (±14.8) ms [296–600] ms (**g-j**)	34.8(±2)/32Hz 352 (±11.8) ms [256–448] ms (**g**)	**60.6(±2)***/22.2(±0.1)Hz 444 (±15.1) ms [280–600] ms (**g–j**)
	26.1(±1.0)/ 32Hz 548 (±9.5) ms [488–600] ms (**i**)	–	–	44.8(±2.5)/25Hz 532 (±10.6) ms [456–600] ms (**i**, **j**)	–

Note: Lower case letters in Table 2 marked consecutive sub-intervals with ERS β2 bursts: **c**, **d**, **e** during T1 and **g**, **h**, **i**, **j** during T2.

Table 2. Mean ERS β2 bursts (%) across the trials with mean frequency peaks **F** (Hz) for HS and PP after LT and HT presented in short–term zones **D** (ms) during early T1 and late T2 period (same format as **Table 1).**

3.2.6 Gamma

The scalp γ burst topography was localized on frontal area for PP following either tone (Table 3, LT(T1), HT(T1), **a**) and for HS – on right motor area after LT and left motor area after HT during T1 (Table 3, **c**). The ERS γ bursts in PP peaked later than in HS at the fronto-central for either tone, and at right and left motor areas after LT (Table 3, **a, b, c**). They were of significantly greater amplitude and more prolonged duration in PP than in HS at the fronto-central and parietal sides after either tone ($p < 0.001$, Table 3). Later γ burst ERS was also found during T1 over right motor and parietal areas in PP after LT (Table 3, **b; c**), and over left motor area in PP, but parietal and right motor sides in HS, after HT (Table 3, **d**). During T2, synchronized frontal γ bursts were extracted from both groups after either tone (Table 3, **f**) and peaked later in PP. The scalp γ bursts were with significantly higher amplitudes in PP than the equivalent responses from HS ($p < 0.001$, Table 3).

In sum, despite the early short-term β1 synchrony during the two periods, both groups exhibited mean β1 ERD following either tone type and interval (Fig. 2A, B, 1st row), which was significantly greater for HS in comparison with PP in all channels ($p < 0.001$, bootstrap, KW test), except frontal mean β1 ERS for the control group following HT during T2 (Fig. 2B, 1st row; Pz, C3', C4', $p < 0.001$; Cz, $p < 0.05$). The prolonged β2 synchronized bursts for PP during T1 had an effect on the β2 band behavior during the entire early time period (T1, Fig. 2, 2nd row, left plot). The mean β2 ERS were prominent only in PP at frontal-parietal and right motor areas after LT and at parietal and left motor areas following HT during T1 (Fig. 2A, B). The comparison of the groups also showed mean β2 ERD in HS and β2 ERS in PP during T2 following either tone in all channels except for the frontal area, which showed mean β2 ERD in both groups after LT, significantly more prominently in HS (LT, $p < 0.05$; Fig. 2A, B). The results for γ closely resembled the β2-frequency band behaviour. The mean γ ERS were more prominent than those for β2 in PP. During the sensory processing (T1), PP showed mean γ ERS responses at fronto-parietal and right motor areas after LT, but not after HT (Fig. 2A, B, 3rd row, left). The γ ERD in HS and γ ERS in PP were observed in all channels after either tone during the cognitive processing (T2), except frontal γ ERS after HT, which was more pronounced in HS than in PP ($p < 0.001$, Fig. 2B, right plot).

Channels	**Fz**	**Cz**	**Pz**	**C3′**	**C4′**
	ERS(%)/ F/ t (±SE) **D**	**ERS(%)/ F/ t** (±SE) **D**	**ERS(%)/ F/ t** (±SE) **D**	**ERS(%)/ F/ t** (±SE) **D**	**ERS(%)/F /t** (±SE) **D**
Subjects tone(period)			**HS**		
LT (T1)	22.7(±2)/ 47 Hz (**c**) 20 (±5.2)** ms [8–32] ms	19.8(±2.8)/ 33 Hz (**a**) 12 (±4)** ms [8–16] ms	20.4(±1.1)/39.4(±1.3)Hz [35–48] Hz (**a, b, c**) 86.4 (±9.9) ms [24–144] ms	18.9(±1.4)/41(±5.2) Hz [32,50] Hz (**a, c**) 28 (±9.5)** ms [8–48] ms	**30.7(±1.9)**/47.6(±0.4)Hz [46–49] Hz (**c**) 40 (±7.3)** ms [8–72] ms
HT (T1)	18.4(±1)/42.2(±2.5)Hz [36,37,47,48] Hz (**a**, **b**) 28 (±6.1)** ms [8–48] ms – 	19.4(±1.4)/36 Hz (**a**) 20 (±5.2)** ms [8–32] ms –	19.1(±0.5)/40.8(±0.2)Hz 72 (±8.6)** ms (**b**) [24–120] ms 32.2(±4)/ 42.6(±0.4) Hz 208 (±8.6) ms [160–250] ms (**d**)	**33.8(±2)/**43.8(±0.4) Hz 120 (±12.6) ms (**c**) [8–250] ms –	30.1(±2.3)*/39.9(±0.5)Hz 44 (±7.7) ms (**b**) [8–80] ms 32.8(±3.3)/41.5(±0.2) Hz 216(±8) ms [176–250] ms (**d**)

LT (T2)	16.9(±0.5)/ 38 Hz 260 (±4)** ms [250–264] ms (**f**) 24.6(±2)/ 35 Hz 568 (±6.5) ms [544–592] ms (**j**)	21.8(±1.2)/40 (±0.5) Hz [38, 42] Hz 548 (±9.5) ms [488–600] ms (**i-j**) –	**36.8(±1.6)**/38.8(±0.4)Hz [35–41] Hz 464 (±14.2) ms [320–600] ms (**g-j**) –	24.3(±1.6)/34.1(±0.3)Hz [33–35] Hz 552.6 (±9.9) ms [496–600] ms (**i-j**) –	16.6(±1.1)/39Hz 524(±5.2) ms [512-536] ms (**h**) –
HT (T2)	36.3(±1.4)/43(±0.6)Hz [37,38,40,46,47] Hz 432 (±15.7) ms [250–600] ms (**f**, **g**, **j**) – –	25.6(±2.2)/45.9(±0.6) Hz [45, 49] Hz 312 (±7.3)** ms [280–344] ms (**f**) 39(±3)/ 45 Hz 444 (±7.7) ms [408–488] ms (**g**) 48(±7)/ 38.6 (±0.2) Hz 572 (±7.7) ms [536–600] ms (**j**)	53.7(±2)/ 43.2(±0.2) Hz 432(±15.7) ms [250–600] ms (**f**, **j**) – –	**55(±3)**/ 44.1(±0.3) Hz [39–45] Hz 432 (±15.7) ms [250–600] ms (**f**, **g**, **i**, **j**) – –	32.4(±1.9)/40.7(±0.4) Hz [39–44] Hz 376 (±13.1)** ms [250–496] ms (**f**) 46(±5.7)/39.3(±0.6) Hz 568 (±8) ms [528–600] ms (**j**) –
Subjects tone(period)			**PP**		
LT (T1)	**49(±5.5)***/ 32 Hz (**a**) 44 (±7.7) ms [8–80] ms –	36.4(±4)*/32.3(±0.7)Hz(**a**) 36 (±6.9) ms [8–64] ms –	32.5(±2.1)*/45.5(±1.1)Hz [42–50] Hz (**c**) 56 (±8.6)** ms [8–104] ms 21.9(±0.8)/47.8(±1.4)Hz [33,49,50] Hz (**c**) 196 (±9.5) ms [136–250] ms	28.2(±3.4)*/37.5(±1.6)Hz [34,35,43] Hz (**a**) 36 (±6.9) ms [8–64] ms –	32.4(±4.3)/43.2(±2.2) Hz [32,46,48,49,50]Hz (**a**,**d**) 56 (±8.6) ms [8–104] ms 30.1(±2.7)/37.9(±0.1) Hz 192 (±8) ms (**b**) [152–232] ms
HT (T1)	**49.7(±3.5)***/34 Hz (**a**) 40 (±7.3) ms [8–72] ms –	27.6(±2.4)*/34.9(±0.1)Hz 44 (±7) ms (**a**) [16–72] ms –	34.3(±3)*/ 34.1 (±0.1) Hz 112 (±11.8) ms (**a**) [16–208] ms –	29.5(±2.6)/34.4(±0.2) Hz 36 (±7)** ms (**a**) [8–64] ms 29.9(±1.5)/33.4(±0.1) Hz 188 (±10.1) ms (**d**) [120–250] ms	21.1(±2)/41(±3.5)Hz [34,36,47] Hz (**a**) 20 (±5.2)** ms [8–32] ms –
LT (T2)	39(±2.6)*/42.7(±0.9)Hz [34–42] Hz 468 (±14.0) ms [328–600] ms (**g-j**)	48.8(±4.2)*/44.3(±1.2)Hz [35–50] Hz 468 (±14) ms [328–600] ms (**g-j**)	68.9(±5.4) */ 45(±0.7) Hz [35–50] Hz 432 (±15.7) ms [250–600] ms (**g-j**)	**82.1(±5.2)** */47(±0.7) Hz [36–50] Hz 468 (±14.0) ms [328–600] ms (**g-j**)	54.3(±2.9)*/41.6(±0.7)Hz [38–50] Hz 452 (±14.8) ms [296–600] ms (**g-j**)
HT (T2)	**58(±3.5)***/40.3(±1.1)Hz [34,48,49] Hz 444 (±15.1)ms [280–600] ms (**f**, **g**, **j**)	43(±1.5)/44.9(±0.7)Hz, [47,50, 35,38,39] Hz 432 (±15.7) ms [250–600] ms (**f**, **g**, **i**, **j**)	52(±2.1)/ 37.8(±0.9) Hz [34,35, 41,45,49,50] Hz 436 (±15.5) ms [264–600] ms (**g**, **i**, **j**)	**55.5(±3.2)**/36.5(±0.9) Hz [33, 50] Hz 432 (±15.7) ms [250–600] ms (**f**, **g**, **i**, **j**)	41.6(±2.3)/42.4(±1.2)Hz [34–36, 49,50] Hz 448 (±15) ms [288–600] ms (**g**, **h**, **i**, **j**)

Note: Lower case letters in Table 3 marked consecutive sub-intervals with ERS γ bursts: **a**, **b**, **c** during T1 and **f**, **g**, **h**, **i**, **j** during T2.

Table 3. Mean ERS γ bursts (%) across the trials with mean spectral peaks **F** (Hz) for HS and PP after LT and HT presented in short–term zones **D** (ms) during early T1 and late T2 period (same format as **Table 1).**

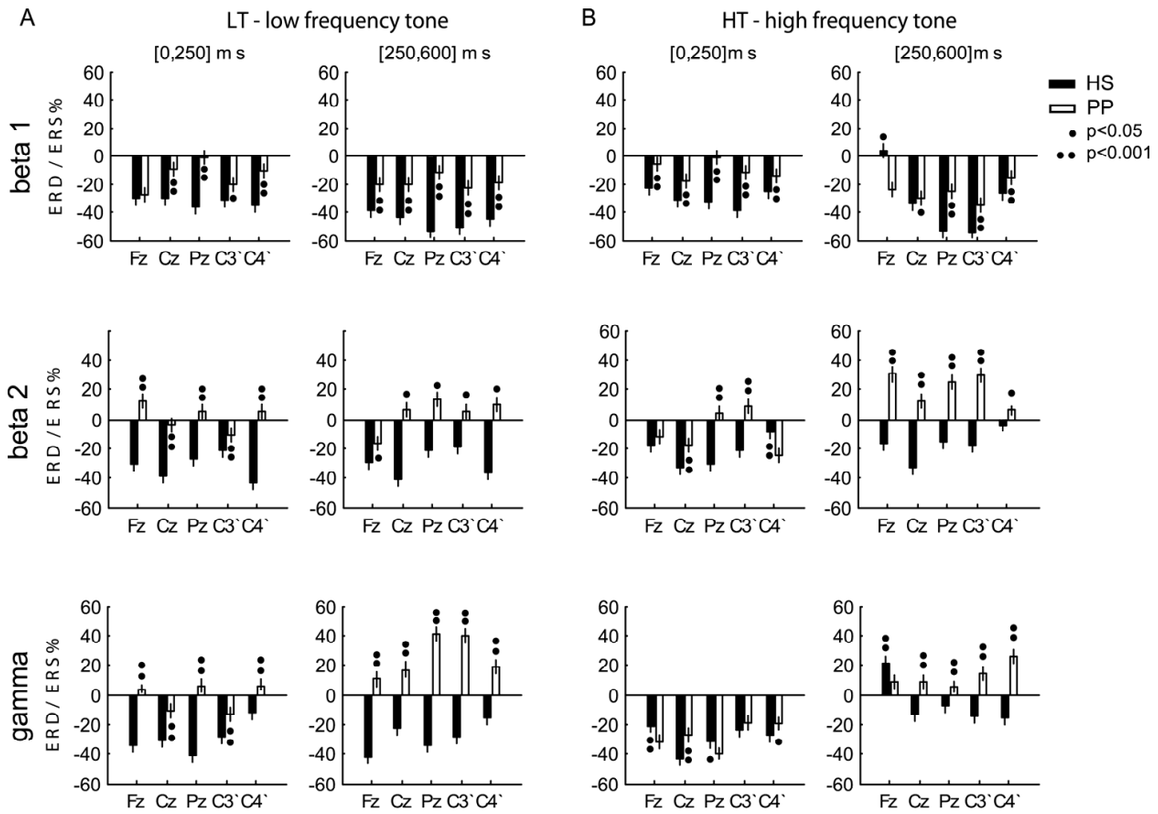

Fig. 2. Beta 1, Beta 2 and gamma band ERD/ERS over T1 and T2. Group means (±SE) are shown graphically to illustrate statistical results of β1-, β2-, γ-ERD/ERS following LT (A) and HT (B) for the early T1 (left) and late T2 (right) time periods. The significant difference in the ERD/ERS of HS and PP are presented for each pair of channels and marked by * (p <0.05) or ** (p <0.001, KW test).

4. Discussion

The data obtained confirmed that event-related oscillatory responses in different frequency bands vary with sensory and cognitive processes. We found functional differences between event-related oscillatory activity for cognitive and sensory-motor information processing, and a clear distinction between PP and HS in both the stimuli encoding (0–250 ms) and cognitive processing (250–600 ms) intervals. Attended stimuli produced theta response synchronizations in both groups, more markedly in HS, in the first period up to 250 ms after stimulation. Enhanced theta waves in the early period (up to 250 ms) of visual and auditory stimuli have also been described by Basar (1980), Schurmann and Basar-Eroglu (1994). Theta frequency rhythms are dominant oscillations within the hippocampal formation, which is of crucial importance for the encoding of new information (Klimesch, 1997; Klimesch et al., 2005). In the late post-stimulus period functionally related to cognitive processing, θ-ERD response was predominant, excluding the parietal θ-ERS in PP in response to both frequency tones and the frontal θ-ERS in HS in response to the low frequency tone.

Prominent differences in the α-ERD/ERS responses between the groups were observed during the first time period of 0–250 ms. A widely distributed fronto-central α-ERS

manifested in PP in response to both tone types during the first 250 ms after stimulus was absent in HS. In the second period after stimulus absence, α-ERD was found in both groups in response to both tone types. This was generally more prominent in HS, with the exception of frontal side in response to either a high or low frequency tone. Alpha-ERD was more prominent at central and right motor areas in response to the high frequency tone in PP.

Theta and alpha frequency ERD/ERS were significantly different between subject groups. It is known that the oscillatory alterations to θ-ERS are related to memory encoding (Klimesch et al., 2001; Jensen & Tesche, 2002).

Alpha-ERS most probably demonstrates active working memory or attentional processes (Klimesch, 1997; Jensen et al., 2002), whereas α-ERD is functionally related to mental activity (Basar, 1980) and reflects memory search processes (Klimesch, 1997; Klimesch et al., 2005; Pesonen et al., 2006). The recognition of auditory stimuli elicits widespread α-ERD responses (Krause et al., 1994). It is accepted that alpha oscillations are mainly generated by cortico-cortical and thalamo-cortical neuronal networks (Lopes da Silva et al., 1980; Schmiedt et al., 2005; Ellfolk et al., 2006). This fact, together with the changes in the metabolic patterns of thalamic, premotor and prefrontal cortex, parieto-occipital regions, etc., that occur in PP (Fukuda et al., 2001) could explain the abnormality of early time period α-ERS in the PP compared to the HS. Observed slight activity of the basal ganglia–thalamic and cerebellar–thalamic pathways might be implicated in the development of parkinsonian symptoms (Rolland et al., 2007).

Schmiedt et al. (2005) also found differences between PP and HS in the θ- and α-frequency ERD/ERS responses during working memory encoding but in a visual working memory paradigm. We cannot draw direct parallels between their results and ours in the present study because of the different stimulus modality. The early and late δ post-stimulus activities were enhanced in HS. The late period, related to cognitive information processing, exhibited δ-ERS in HS and δ-ERD in PP at most electrodes in response to a low frequency tone, and at parietal and left motor areas in response to the high frequency tone. Many authors agree that the main power of P300 is in the delta range (Demiralp et al., 1999; Karakas et al., 2000; Klimesch et al., 2000; Klimesch et al., 2006). The lower δ-ERS in PP in the late post-stimulus period, which becomes δ-ERD in some recordings, could explain the lower P3 amplitude observed in PP (Philipova et al., 1997). Our patients were medicated by L-dopa drugs and this medication may have had some effect on the present findings. One of the models (Leblois et al., 2006) supposed that high dopamine depletion could modify the network dynamic state from an imbalance between the feedbacks and lead to synchronous oscillations driven by a hyperdirect loop appearing in basal ganglia after inactivation of the striatum.

A reduction in this α-ERD/ERS abnormality and a consequent improvement in PP performance during working memory tasks have been found as the result of L-dopa (Lewis et al., 2003; Marini et al., 2003; Shohamy et al., 2005; Devos et al., 2004). Nevertheless, we found some differences between the two groups. The memory related and stimulus categorized ERD/ERS responses at all these frequencies reflected different underlying neuropathological and cognitive changes in this neurodegenerative disease. Theta activity is suggested to be mostly engaged in memory operations (Klimesch et al., 1996; Karakas et al., 2000; Jensen et al., 2002b) and this θ pathological synchronized enhancement in PD could explain the cognitive dysfunction commonly occurring even in the early stages of Parkinson's disease (Lewis et al., 2003). We found specific significant differences at left

motor area as θ-ERS in the HS and θ-ERD in the PP during sensory-motor processing (early period) following the high frequency tone. We also detected different processes of θ-ERS for the PP and ERD for the HS in the parietal lead during the cognitive information processing (late period) following both tones, which reflects different task-related activation of the associative posterior cortex.

These findings are probably due to the auditory cortex being located in the dorsal and lateral part of the superior temporal gyrus as well as in the inferior parietal lobule (Konig et al., 2005). The absence of α-ERD at the frontal electrode locations in the patients with PD indicated that the PP, compared with HS, used different cognitive strategies for stimulus response processing which are normally implemented by fronto-striatal circuits (Krause, 2006). The late higher fronto-central α-ERD in PP accompanied by a lower P3 component amplitude, especially in the fronto-central sides, reflects a disturbance in the frontal regulation of attentional processes as well a disturbance of the basal ganglia activity and their related thalamo-cortical neuronal nets (Stam et al., 1993; Piccirilli et al., 1989; Schmiedt et al., 2005).

In PP, we found hemispheric lateralization for sensory and cognitive processing concerning θ-ERD/ERS at left and right motor areas as well as a significantly higher α-ERS at left compared to right motor area. This finding corresponds with the results of Magnani et al., 1998, Defebvre et al., 1996. These authors suggested that other cortical areas may be activated both to compensate for a dysfunction of motor preparation and to increase the level of cortical activity necessary for the realization of the movement. Another possible explanation is that this hemispheric lateralization is connected with auditory attention and hemispheric differences in the processing of high and low frequencies (Ivry & Robertson, 1998).

Post-stimulus β1 ERD was elicited from both groups during sensory (T1) and cognitive information (T2) processing, though this was significantly more pronounced in HS in response to both tone types at all electrodes. The greater β1 ERD in HS can be explained by the increased excitability level of the neurons (Pfurtscheller & Lopes da Silva, 1999; Brown & Marsden, 1998). Late post-stimulus frontal β1 ERS (T2) was evident only in HS following HT. This HS ERS, comprising components in the band between 13 and 20 Hz, may represent an inhibited frontal cortical network, at least under certain circumstances (Pfurtscheller & Lopes da Silva, 199; Engel et al., 2001).

A frontal β2 ERD was maintained in both groups during the cognitive information processing (T2) following LT, though this was weaker in PP. β2 ERS was only observed in PP. These were weakly elicited during the sensory stimuli processing (T1) and appeared at fronto-parietal and left motor areas (LT: Fz, C3', Pz; HT: C3', Pz). β2 ERS in PP was more prominent during cognitive processing (T2) after either tone type, but particularly so following HT. The β2 change reversals compared to β1 which we observed for the PD patients support the hypothesis of Marceglia et al. (2009), that two distinct information channels in the cortico-basal ganglia–thalamo-cortical loop, involved in motor and non-motor information processing, are formed in the parkinsonian brain. The frontal β synchronization at 20–30 Hz arises both from communication with, and also from within, the STN (Williams et al., 2003). The β synchrony has been ascribed predominantly to a lack of dopaminergic activity in the striatum which, together with the STN, is the recipient of cortical input to the basal ganglia (Fogelson et al., 2006; Williams et al., 2002). Studies with unmedicated PD patients have revealed prominent oscillations in 'basal ganglia β frequency band' (Weinberger et al., 2006; Kühn et al., 2006; Priori et al., 2004; Fogelson et al., 2006). The

engagement of the basal ganglia in β band synchronization is found when there is acute or chronic dopaminergic hypoactivity, and while primarily associated with bradykinesia and rigidity, it has also been associated with impairments to complex movements and motor related cognitive behaviour because of the widespread basal ganglia connectivity with the cerebral cortex (Terman et al., 2002). Further, the pathological β synchrony in the cerebellum might lead to a purer breakdown of simple motor tasks because of more focal cerebellothalamic projections into the cerebral cortex that are concentrated on the primary motor cortex (Leblois et al., 2007). A relative functional division between activities in the β band might be supported by the evidence for different patterns of pharmacological sensitivity (Priori et al., 2004) and cortico-subthalamic coupling (Fogelson et al., 2006). The dopaminergic drug treatment suppressed mainly β1 synchrony, graded by the amount of drug-induced suppression in the STN (Kühn et al., 2006; Wang et al., 2005) and cerebral cortex, correlating with the level of improvement in bradykinesia and rigidity but not in parkinsonian rest tremor (Weinberger et al., 2006; Silberstein et al., 2005), the latter of which probably has an independent pathophysiological substrate (Rivlin-Etzion et al., 2006).

Our group of patients showed a significantly reduced γ–ERD compared with HS over central and left motor areas, and only PP showed γ–ERS over fronto-parietal and right motor areas following LT during the sensory stimuli processing (T1). A widespread γ–ERS appeared during later cognitive processing (T2), and then only in PP, following either tone type, with the exception of a more prominent frontal γ–ERS in HS following HT. In our study, we observed switches between cortical activity in the β2 and γ band oscillations. Hence we concluded that a reduction in β2-band synchronized activity allows higher frequency oscillatory activity in the γ range leading to its synchronization. The observed energy changes in the β2 and γ bands indicate that an increase in one is accompanied by a decrease in the other. These T2 changes in PP were more pronounced in the motor cortex than in the parietal and even frontal cortex data. In the parkinsonian state, there was a tendency towards increased synchronized higher frequency fluctuations, specifically in the motor cortex, where instances of peaks were found after both tone types. Except for the β2 band series of data during cognitive processing (T2) after HT, the difference between magnitude of the peaks in the frontal, parietal and contralateral motor areas did not reach significance. Recent data demonstrate that the disruptions of the beta and gamma range cortical rhythms are based on the disturbed temporal relationship between cortical oscillatory activity and basal ganglia activity in Parkinsonism (Gatev & Wichmann, 2008). This finding is also in agreement with studies of PP following dopaminergic medication, which promoted synchronized oscillatory activity at higher frequencies (γ) predominantly at the level of the frontal cortex and striatum (Levy et al., 2001; 2002; Brown et al., 2001; Williams et al., 2002; Leblois et al., 2007).

In recent MEG investigations of various cognitive and sensory tasks (Kaiser et al., 2003; Lutzenberger et al., 2002) the reported γ band activity over the higher sensory areas has not shown a sustained activation, but rather, a peaking activity. In our sensori-motor study, these transient responses were functionally dissociable between the two groups. We observed stimulus-specific γ band activity components over the fronto-parietal cortex, but this was differently manifest in each group and varied over the time course. The topography was compatible with the notion of an auditory dorsal space processing stream involving the posterior temporal, parietal and superior frontal cortex (Rauschecker, 1998; Arnott et al., 2004). If the cognitive processing (T2) γ band activity components represent similar

anticipatory activations both for LT and HT, one might assume that the same cortical networks should underlie the same stimulus representations. However, while all components were mainly localized over fronto-central areas, there was some variation between the conditions, showing significant effects on the parietal γ components for LT, but not for parietal γ activity for HT. This suggests that networks encoding the stimulus features are not fixed, and may vary with task demands.

The assessing EEG stimulus-specific oscillatory activity yielded insights into the temporal dynamics of sound processing in short-term memory. Contrasting oscillatory γ activity between the two stimuli, such as between LT ERS and HT ERD during the sensory processing (T1) in PP, as well as between LT ERD and HT ERS during the cognitive processing (T2) in HS, revealed stimulus-specific γ activity behavior in the 30–50 Hz range over the HS's frontal and PP's fronto-parietal cortex. This suggests that γ band activity reflects the general involvement of cortical networks in particular tasks but may index the specific content of short-term memory in each group.

The pronounced and well-synchronized γ burst in HS was present in the very short-term phases around 25–60 ms after the stimulus onset, with spectral peaks ranging from 30 to 45 Hz (Gurtubay et al., 2001). Despite this short-term high synchrony in HS, the common γ behavior during sensory stimuli processing (T1) was desynchronization. However, the PP processes were with higher short-term energy, which is a prerequisite for all maintenance-related processes, and thus defined a persistent synchronization during sensory stimuli processing, mainly at fronto-parietal and right motor areas following LT. It is clear that there is a difference between groups in the early and well-synchronized response that is basically a sensory phenomenon important to preparing the brain for the subsequent processing. This evidence suggests that γ oscillations may be modulated by attentional processes. Several cognitive paradigms for the auditory system have shown early spectral peak responses in the γ band between 30 and 40 Hz at around 25 ms after stimulus onset that last for about 100 ms (Pantev, 1995; Arnott et al., 2004).

Later peaking activity has been recorded in the 200–400 ms interval following an experimental task. The latency and scalp topography vary according to the type of stimulus, indicating task-dependent local network activation. The significantly varying magnitudes of differentiation demonstrated that the topography of stimulus-specific γ-band activity is also task-dependent within the groups (Tiitinen et al., 1993). We found restricted energy changes over all recorded areas in HS, but not in the frontal area after HT. The significant differences between groups in T2 recorded after both stimuli could be due to memory retrieval processes that are activated during the performance of the paradigms. The lower energy in HS during cognitive processing (T2) could be related to fewer attentional processes required to eventually perform a task. The relative strength of differentiation in the γ–band may suggest that performance depends on the different group's ability to retain a representation in memory of the relevant stimulus feature and thus to be able to neglect the irrelevant stimuli. The acquisition and retention of sound frequency information was accompanied by frontal gamma band activity components (Karakaş & Başar, 1998). The high-frequency stimuli were accompanied by more exaggerated, well–synchronized frontal γ band components in performing the tasks. Memory for low versus high frequency tones selectively enhanced oscillatory activity for the posterior versus the frontal components, thus directly demonstrating differentiation of the group's modulation of cortical activity by task demands.

A mechanism that underlies many of the immediately aforementioned cognitive functions is the match of sensory information with memory contents (Kaiser et al., 2009a; 2009b; Visscher et al., 2007). The 'early' γ-band activity occurring 150 ms after stimulus presentation reflects such a match with memory. The 'late' γ activity, which typically emerges with a latency of more than 150 ms, is a temporal signature of utilization processes such as response selection or context updating. We also found a later (250–400 ms) ERS γ response, following only HT in HS, over the frontal location, where this activity peaked in the 33–45 Hz range. In PP, the specific β2 and γ bursts (30–38 Hz) exhibited maximal scalp projection covering areas to the left and right of the motor areas**,** and with frontal, central or parietal participation that depended on the stimuli. This oscillatory burst reflects a later stimulus context process, although it has also been associated with the motor responses later in the task (Brown, 2003; Kaiser et al., 2009b). The β2/γ oscillation in the groups points to a direct relation to aspects of post-discrimination processes related to the P300 wave **(**Haig et al., 2000). This oscillatory burst (letters **g-j** for intervals from 320 to 550 ms) also showed a variable relationship to attention, as it was significantly different during the HT and LT task. The results also showed that EEG activity in the frontal, parietal and motor cortex is significantly different between groups, not only in temporal variations (always with a delay in PP) but also in frequency shifts (β2/γ ERD in HS compared to the ERS in PP). Although these shifts do not follow a simple pattern, they are significantly different from HS, raising the possibility that the interactions between basal ganglia activity and cortical rhythms are functionally relevant. Therefore, the normal higher frequency relationships between cortical and basal ganglia activity are strongly altered in the parkinsonian state (Gatev & Wichmann, 2008). The shifts of β/γ patterns occurring in the groups are probably associated with specific types of basal ganglia events related to transitions between cortical idling and more active states (Williams et al., 2003).

5. Conclusion

Our investigation further demonstrates the close relationship between physiological abnormalities in PD and disturbances in the EEG frequency characteristics. The results of this investigation in PD patients of both sensory and cognitive processing of auditory stimuli suggests that PD should be characterized by multiple impairments in oscillatory networks, which in turn indicates the presence of task-specific disturbances in the temporal and regional integration of all frequency components.

6. References

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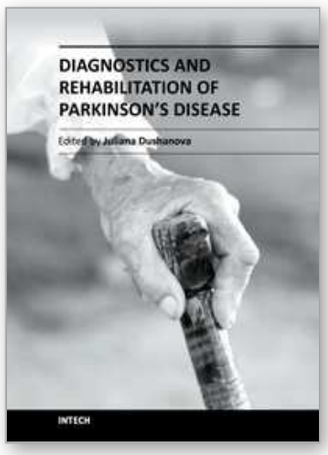

Diagnostics and Rehabilitation of Parkinson's Disease
Edited by Dr. Juliana Dushanova

ISBN 978-953-307-791-8
Hard cover, 528 pages
Publisher InTech
Published online 07, December, 2011
Published in print edition December, 2011

Diagnostics and Rehabilitation of Parkinson's Disease presents the most current information pertaining to news-making topics relating to this disease, including etiology, early biomarkers for the diagnostics, novel methods to evaluate symptoms, research, multidisciplinary rehabilitation, new applications of brain imaging and invasive methods to the study of Parkinson's disease. Researchers have only recently begun to focus on the non-motor symptoms of Parkinson's disease, which are poorly recognized and inadequately treated by clinicians. The non-motor symptoms of Parkinson's disease have a significant impact on patient quality of life and mortality and include cognitive impairments, autonomic, gastrointestinal, and sensory symptoms. In-depth discussion of the use of imaging tools to study disease mechanisms is also provided, with emphasis on the abnormal network organization in parkinsonism. Deep brain stimulation management is a paradigm-shifting therapy for Parkinson's disease, essential tremor, and dystonia. In the recent years, new approaches of early diagnostics, training programmes and treatments have vastly improved the lives of people with Parkinson's disease, substantially reducing symptoms and significantly delaying disability. Written by leading scientists on movement and neurological disorders, this comprehensive book should appeal to a multidisciplinary audience and help people cope with medical, emotional, and practical challenges.

How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Juliana Dushanova (2011). Brain Event - Related Oscillations in Parkinsonian Patients During Discrimination Task Conditions, Diagnostics and Rehabilitation of Parkinson's Disease, Dr. Juliana Dushanova (Ed.), ISBN: 978-953-307-791-8, InTech, Available from: http://www.intechopen.com/books/diagnostics-and-rehabilitation-of-parkinson-s-disease/brain-event-related-oscillations-in-parkinsonian-patients-during-discrimination-task-conditions

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