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The best of both worlds: Phase-reset of human EEG alpha activity *and* additive power contribute to ERP generation

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Abstract

Some authors have proposed that event-related potentials (ERPs) are generated by a neuronal response which is additive to and independent of ongoing activity, others demonstrated that they are generated by partial phase-resetting of ongoing activity. We investigated the relationship between event-related oscillatory activity in the alpha band and prestimulus levels of ongoing alpha activity on ERPs. EEG was recorded from 23 participants performing a visual discrimination task. Individuals were assigned to one of three groups according to the amount of prestimulus total alpha activity, and distinct differences of the event-related EEG dynamics between groups were observed. While all groups exhibited an event-related increase in phase-locked (evoked) alpha activity, only individuals with sustained prestimulus alpha activity showed alpha-blocking, that is, a considerable decrease of poststimulus non-phase-locked alpha activity. In contrast, individuals without observable prestimulus total alpha activity showed a concurrent increase of phase-locked and non-phase-locked alpha activity after stimulation. Data from this group seems to be in favor of an additive event-related neuronal response without alpha-blocking. However, the dissociable EEG dynamics of total and evoked alpha activities together with a complementary simulation analysis indicated a partial event-related reorganization of ongoing brain activity. We conclude that both partial phase-resetting and partial additive power contribute dynamically to the generation of ERPs. The prestimulus brain state exerts a prominent influence on event-related brain responses.

Keywords: Additive power; Alpha activity; EEG; ERP; Phase-resetting

1. Introduction

The event-related potential (ERP) is one of the most frequently employed measures of the brain's event-related electrical activity. It is derived by averaging the poststimulus electroencephalogram (EEG) over a sufficient number of trials. However, the generation of ERPs is still an issue of ongoing debate because of the gap between electrophysiological observations at the scalp and the underlying neurophysiological processes (e.g. Makeig et al., 2004). Two alternative (but not necessarily exclusive) mechanisms have been proposed. On the one hand it is assumed that stimulation induces a partial 'phase-resetting' of ongoing EEG rhythms in each trial, and averaging these phase-coherent rhythms produces the ERP (Basar, 1980; Brandt et al., 1991; Jansen et al., 2003; Makeig et al., 2002; Sayers et al., 1974). The alternative additive ERP view suggests that stimulation elicits a neural population response with fixed polarity and latency in each trial which is additive to and independent from ongoing activity (so-called 'additive power

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model'[1]) and that averaging these evoked responses produces the ERP (Jervis et al., 1983; Lopes da Silva, 1999; Schroeder et al., 1995).

Although the mechanisms that are at work in the generation of the ERP have been debated and investigated for decades, researchers are still at odds on the methods which would allow one to distinguish between the 'phase-reset' and the 'additive power' models as outlined above. The consistency of the phase of oscillatory activity across trials can be quantified by means of the so-called 'phase-locking factor (PLF)' (Delorme and Makeig, 2004; Herrmann et al., 2005; Tallon-Baudry and Bertrand, 1999). Although measures of phase consistency across trials have been used to provide evidence for a stimulus induced reorganization of ongoing activity (Brandt, 1997; Jansen et al., 2003), it is important to point out that an increase in phase-locking *per se* is not informative about the generating mechanism because either phase reorganization or additive responses with fixed latency and polarity in each trial can produce an increase of inter-trial phase consistency (Jervis et al., 1983; Klimesch et al., 2004b; Makeig et al., 2004, 2002; Shah et al., 2004; Yeung et al., 2004).

Therefore, for a comprehensive analysis of the event-related EEG dynamics, it appears necessary to consider both phase and amplitude dynamics. Common measures of oscillatory EEG activity comprise evoked and induced activities as well as the phase-locking factor. Evoked activity is computed as the time-frequency representation of the ERP and contains signals that are strongly phase-locked to stimulus onset. Induced activity is a measure of oscillatory power in single trials and captures signals that are not phase-locked to stimulus onset.

In former studies (Klimesch et al., 2004a; Makeig et al., 2002; Shah et al., 2004) it has been stated that a pure phase-reset of ongoing activity would be indicated by changes in evoked activity and phase-locking factor without a change in signal power in single trials. This would be true if background oscillations would not exhibit modulations of amplitude to a stimulus at the same time. However, it is well known that brain oscillations show a variety of amplitude modulations according to a stimulus or task. Whereas some oscillations exhibit a decrease in power, e.g. alpha (Klimesch, 1999), others show an increase in power, e.g. theta (Basar-Eroglu and Demiralp, 2001; Debener et al., 2005) and gamma (Herrmann et al., 2004a). Thus, it appears plausible that an evoked response, superimposed on background EEG, occurs at the same time as a power decrease. This additive component might then elicit an increase in PLF with no observable power increase being visible in the EEG due to the strong simultaneous decrease. This line of argumentation was used by Klimesch et al. (2006) in order to explain why Mäkinen et al. (2005) failed to observe an influence of ongoing brain activity in ERP generation. That is, amplitude variance alone is not sufficient to distinguish between an additional evoked response and phase-resetting of ongoing activity. For these reasons, it is impossible to dissociate between the 'phase-reset' and the 'additive power' models by simply considering poststimulus power changes or phase-locking dynamics if alpha-blocking occurs.

However, under certain conditions, it may be possible to differentiate between the two models. If the alpha power remained constant after stimulation but phase-locking increased compared to baseline this would indicate phase-resetting (Klimesch et al., 2004a; Shah et al., 2004). Therefore, we categorized our subjects into three groups based on the amount of prestimulus alpha activity. Since the amount of ERD depends upon the prestimulus alpha power (Doppelmayr et al., 1998b), we expected to find subjects who showed no poststimulus decrease of alpha but an increase of phase-locking. While our categorization yielded interesting results, however, it did not yield a group of subjects without modulation of alpha power after stimulation as would be required for the differentiation of the two models. Thus, in addition, we applied a simulation analysis to the data of all three groups, as recently suggested by Hanslmayr et al. (2007).

In the present study, we analyzed data recorded in a visual discrimination experiment in order to find evidence for one of the two models of ERP generation. As has been pointed out by Shah et al. (2004), phase-resetting of ongoing activity requires that there be a significant level of activity in the prestimulus baseline, a requirement which is usually fulfilled for the alpha frequency range. We therefore restricted our analysis to activity in the alpha band in the present study. We reasoned that if event-related EEG signals were in fact dependent on ongoing EEG activity it would be plausible that the prestimulus brain state influences the subsequent response. Therefore we investigated differences in the event-related EEG dynamics of three subgroups of subjects, which were categorized according to the amount of prestimulus total alpha activity. Such different amounts of prestimulus alpha activity yielded different levels of ERD (Doppelmayr et al., 1998b), which might also induce a dissociation of the poststimulus patterns of alpha dynamics. Additionally, a simulation was carried out for each of these subgroups to investigate whether all groups show evidence for phase-resetting.

2. Materials and methods

2.1. Subjects and experimental procedure

Twenty-three subjects participated in this study (16 females, mean age 25; range 20–39 years). Subjects gave informed consent prior to the start of the experiment. This research was carried out in accordance with local ethics guidelines and the Declaration of Helsinki (World Medical Association: Ethical Principles for Medical Research Involving Human Subjects, 1964). All subjects had normal or corrected-to-normal vision and were free of neurological or psychiatric disorders. Recordings were made while subjects sat in a dimly lit, sound-attenuated and electrically shielded booth.

Black circles and squares on a white background were used as stimuli. Both types of stimuli appeared with equal probability. Stimuli were presented on a computer monitor placed at a distance of 105 cm in front of the subject. Monitor refresh rate

[1] This model is also referred to as evoked model in the literature (e.g. Fell et al., 2004; Klimesch et al., 2004a; Hanslmayr et al., 2007).

was 100 Hz. Stimuli were displayed at a size of 8° visual angle and were presented centrally for a duration of 250 ms. Subjects were required to always remain centrally fixated. Stimulus presentation was followed by a variable inter-stimulus-interval ranging from 1000 to 1400 ms. Subjects were instructed to press a button with the thumb of one hand if the stimulus was a circle and to press a button with the other hand if the stimulus was a square. In order to analyze reaction times, subjects were asked to press the button as quickly as possible. Response hands were counterbalanced across subjects. The experiment consisted of 90 trials per type of stimulus (circle or square), resulting in a total number of 180 trials. For the purpose of the present analysis data were collapsed across both stimulus types. Two breaks of one minute duration were given in this experimental session.

2.2. Data acquisition

EEG was recorded using a high impedance 64 channel Net Amps 200 system (Electrical Geodesics, Eugene, Oregon) with Ag/AgCl-electrodes placed in an electrode cap (Easycap, Falk Minow Services, Munich) and a nose-tip reference. Sensor impedances were maintained below 20 kΩ prior to data acquisition (Ferree et al., 2001). EEG was analogue filtered from 0.1 to 100 Hz, digitized at 500 Hz and stored for off-line analysis. The present study is a reanalysis of the data published in Busch et al. (2004), which focuses on gamma activity.

Data were epoched from 500 ms before to 1000 ms after stimulus onset. Since the aim of the present study was to investigate the relation of prestimulus EEG activity and poststimulus EEG dynamics, no baseline correction was applied. Automatic artifact rejection excluded trials from averaging if the standard deviation within a moving 200 ms time interval exceeded 30 μV. Subsequently, all epochs were visually inspected for artifacts, and epochs containing eye-movements or electrode drifts were rejected. One subject had to be excluded from further analyses because of poor data quality.

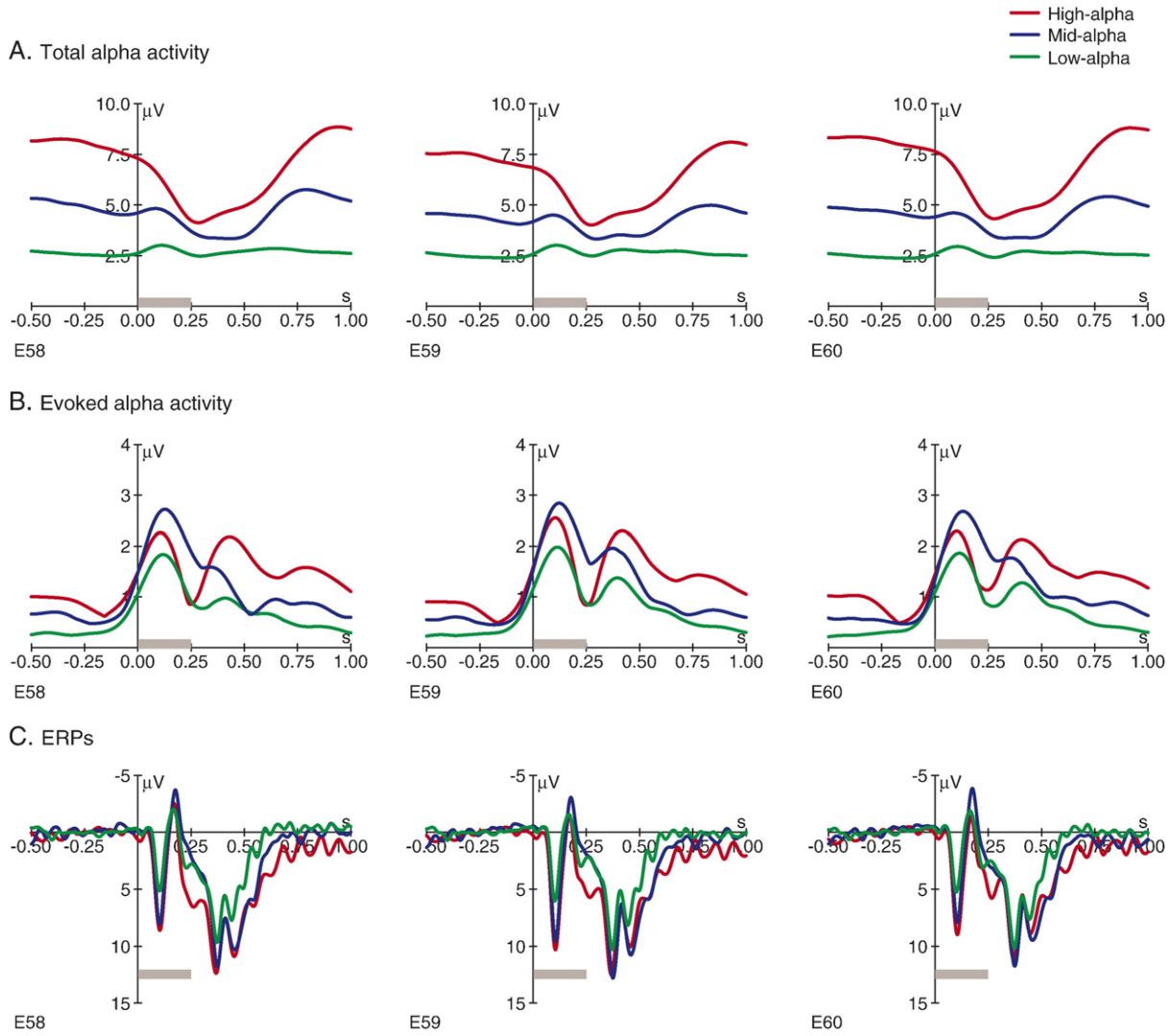

Fig. 1. Grand-averaged time courses of (A) total, (B) evoked alpha activity and (C) ERPs at the occipital electrodes (E58, E59 and E60) for all three groups (red lines: high-alpha group; blue lines: mid-alpha group; green lines: low-alpha group). For displaying ERPs, we performed 20 Hz low-pass filtering and a baseline correction by subtracting mean amplitudes from 300 to 200 ms prestimulus. Gray bars indicate stimulus duration.

2.3. Time-frequency analysis

For investigating the amplitude and time course of oscillatory activity, the EEG signals were convolved with Morlet wavelets (Herrmann et al., 2005; Herrmann and Mecklinger, 2000). The wavelet transform was performed for each individual trial, and the absolute values of the resulting transforms were averaged. This measure of signal amplitude in single trials reflects the total activity for a certain frequency range, irrespective of whether it is phase-locked to the stimulus or not. We will refer to this measure as the total activity, since it comprises evoked as well as induced activity. On the other hand, to compute the evoked activity, which is, by definition, phase-locked to the stimulus, the wavelet transform was applied to the averaged evoked potential. In addition, the degree of phase-locking was calculated by means of the 'phase-locking factor', which reflects the homogeneity of the instantaneous phase across single trials. To this end, the phase of the complex wavelet decomposition in each single trial was represented as a point on the unit circle irrespective of amplitude. Averaging these points yields values between 0 for randomly distributed phases and 1 for phases that are strictly phase-locked to stimulus onset across trials.

2.4. Selection of the individual alpha frequency

It is well known that subjects differ considerably in their 'individual alpha frequency' (IAF; Doppelmayr et al., 1998a; Klimesch, 1999). Therefore, the frequencies used in the wavelet analyses of total and evoked alpha activities were determined individually for every subject. Time-frequency transforms were first computed for electrodes E58, E59 and E60 (equivalent to O1, OZ and O2 in the 10–10 system, respectively) and were subsequently averaged to increase the signal-to-noise ratio. From these averaged time-frequency scalograms individual alpha frequencies were obtained as the maximum of prestimulus total activity in the frequency range between 8 and 13 Hz in a time window from 300 to 200 ms prior to stimulus onset. If there was no detectable alpha peak, 10 Hz was selected as the individual alpha frequency for those subjects. This had to be done for eight subjects which were later assigned to the low-alpha group (see the following section).

2.5. Grouping subjects and statistical analysis

In order to inspect differences in the subsequent responses according to the amount of prestimulus total alpha activity, we categorized our subjects into three subgroups (high-, mid-, and low-alpha) according to their mean amplitude of prestimulus total alpha activity while trying to keep the size of the three groups as equal as possible. The seven subjects with highest amplitudes of prestimulus total alpha activity comprised the high-alpha group (mean amplitudes ranging from 6.2 μV to 9.9 μV). The next seven subjects with lower amplitudes comprised the mid-alpha group (mean amplitudes ranging from 3.9 μV to 5.6 μV) and the remaining eight subjects were assigned to the low-alpha group (mean amplitudes ranging from 2.0 μV to 3.2 μV).

For statistical analyses, total and evoked alpha activities were averaged across electrodes E58, E59 and E60 and analyzed within a time window from 0 to 400 ms after stimulus onset for the poststimulus onset responses. Since we did not apply a baseline correction in our analysis, we instead measured mean values of prestimulus total and evoked alpha activities and compared them statistically with the poststimulus individual amplitude. Baseline activity was measured as the mean of activity in the time window from 300 ms to 200 ms before stimulus onset. Baseline magnitudes were then compared with the maximum amplitude of evoked alpha activity in the time window from 0 to 400 ms poststimulus. For total alpha activity, we chose the same time window and compared baseline activity with minimum amplitudes of the high- and mid-alpha groups and maximum amplitudes for the low-alpha group, as the grand average of the high- and mid-alpha groups demonstrated a decrease, and that of the low-alpha group showed an increase after stimulation, as shown in Fig. 1A. The peak latencies of total and evoked alpha activities were also evaluated. All time windows were determined on the basis of the grand

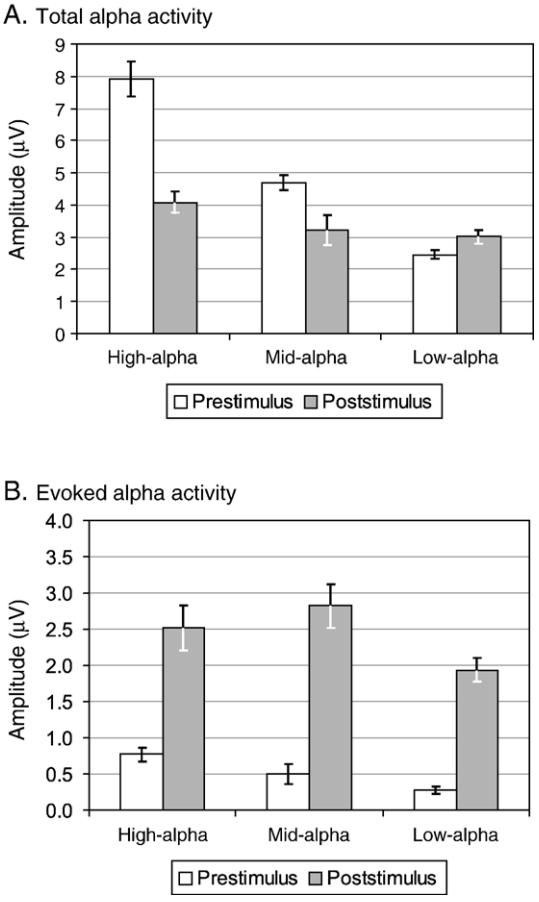

Fig. 2. Categorized bar graphs with error bars for (A) total and (B) evoked alpha activities of both prestimulus and poststimulus activities averaged across electrodes E58, E59 and E60 for all groups. White columns indicate prestimulus activity and gray columns poststimulus activity. Error bars represent ±1 standard error of the mean. All subjects demonstrate an increase of evoked alpha activity in response to stimulation. However, only the high- and mid-alpha groups show a decrease of total alpha activity which is considered alpha-blocking. In the low-alpha group a significant increase of total alpha activity is visible instead of alpha-blocking.

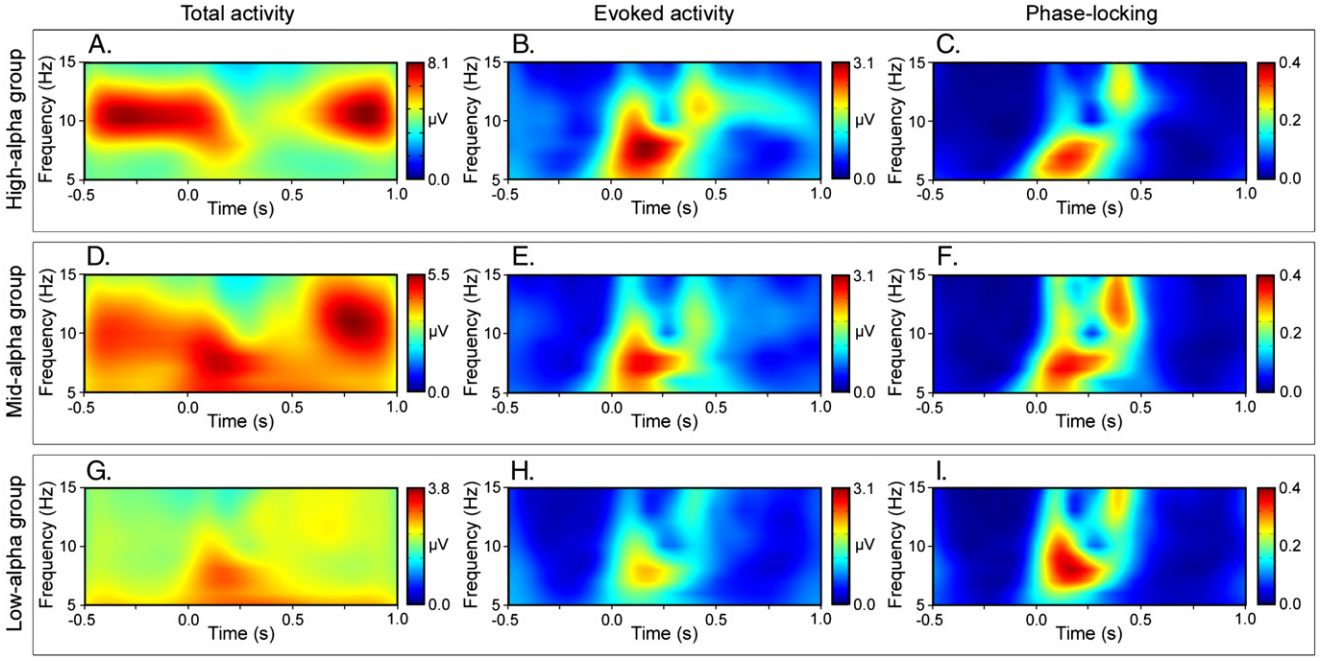

Fig. 3. Time-frequency representations of total activity (A, D and G); evoked activity (B, E and H); phase-locking factor (C, F and I) averaged across electrodes E58, E59 and E60 for all groups. All plots are grand-averaged. High- and mid-alpha groups show prominent prestimulus alpha activity which is almost absent in the low-alpha group (cf. between −500 and 0 ms in A, D and G). Stimuli were presented from 0 to 250 ms.

averages (cf. Fig. 1) and individual variances were taken into account.

Reaction times and accuracy (error rates) were also measured for behavioral analysis. In order to compare reaction times, error rates, mean amplitudes of prestimulus total alpha activity, mean frequencies of IAFs among three groups and peak latencies of total and evoked alpha activities, we performed a one-way ANOVA with a 'group' factor (high-alpha vs. mid-alpha vs. low-alpha group). In addition, the amplitudes of total and evoked activities were analyzed with a repeated measures ANOVA comprising the within-subjects factor 'stimulation' (baseline vs. onset response) and the between-subjects factor 'group' (high-alpha vs. mid-alpha vs. low-alpha group). In order to compare the peak latencies of poststimulus total alpha activity with those of poststimulus evoked alpha activity, we conducted a repeated measures ANOVA comprising the within-subjects factor 'latency' (peak latency of total activity vs. of evoked activity) and the between-subjects factor 'group' (high-alpha vs. mid-alpha vs. low-alpha group). Greenhouse–Geisser correction was used where appropriate. All subsequent post-hoc tests were Bonferroni-corrected for multiple comparisons, and only corrected p-values (the threefold of the original p-values) are reported.

2.6. Simulation of additive and non-additive signal generation processes

For our simulation analysis the same procedure as in the study by Hanslmayr et al. (2007) was used. The main idea of this simulation is that for the 'additive power' model the EEG signal, eeg(ij), for each sample point i and trial j can be described by two additive components. First, the background EEG amplitude $b(ij)$ and second, the single trial evoked potential ep(ij) (Eq. (1)).

$$\mathrm{eeg}(ij) = b(ij) + \mathrm{ep}(ij) \tag{1}$$

For the 'phase-reset' model, the lack of an additive component leads to the prediction that the EEG signals remains equal to background EEG (Eq. (2)).

$$\mathrm{eeg}(ij) = b(ij) \tag{2}$$

Note that in Eq. (2), $b(ij)$ is assumed to undergo a phase-reset which is not the case for Eq. (1). As mentioned above, testing for additivity would be easy and straightforward if there was no concurrent amplitude modulation of the background EEG. However, if the background EEG undergoes an amplitude change during the same time as evoked components are generated, the expected increase in amplitudes by evoked components may be masked by an event-related decrease in amplitudes. Therefore, we need to take into account this amplitude decrease in our simulation. As suggested by Hanslmayr et al. (2007) the amplitude of the background EEG can be estimated by subtracting the average ERP, i.e. ep(i), from each single trial. This measure is called 'non-phase-locked activity' (NP; or induced activity; see Kalcher and Pfurtscheller, 1995) and makes the assumption that the single-trial ep(ij) can be estimated by the average ep(i).

The simulation we used (Hanslmayr et al., 2007) comprised three steps:

1. We computed the time course of the 'non-phase-locked activity' (NP) for each individual's alpha activity. This was

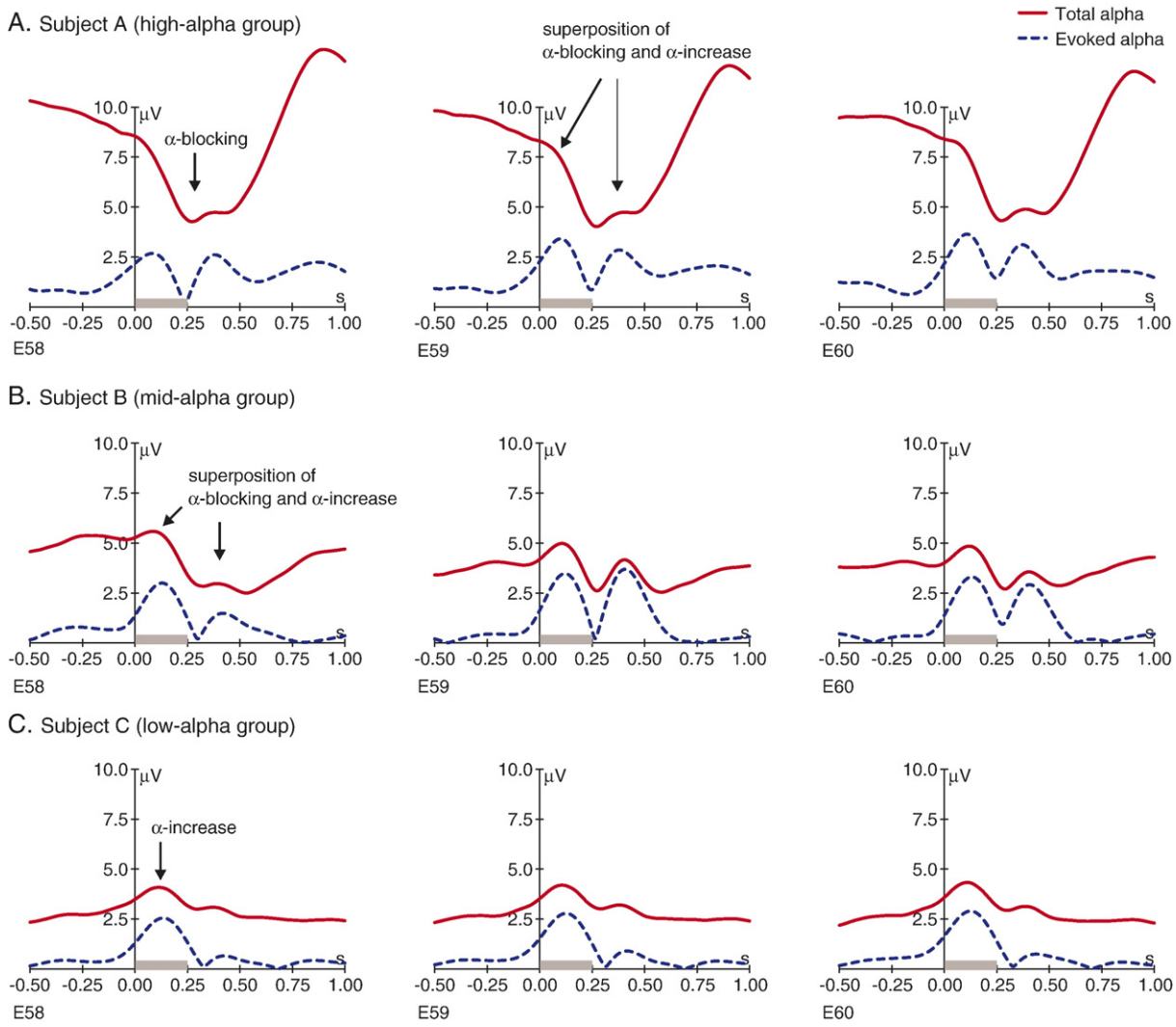

Fig. 4. Total and evoked alpha activities at the occipital electrodes (E58, E59 and E60) of one representative single subject for each group (solid lines: total alpha activity; dashed lines: evoked alpha activity). Gray bars indicate stimulus duration. Subject A of the high-alpha group shows a prominent decrease of total alpha activity while subject C of the low-alpha group reveals an increase of total alpha activity after stimulation. In subject A and B, a superposition of such an increase and a decrease in the poststimulus total alpha activity can be seen.

done by subtracting the average ERP from each single trial and computing the absolute values of all single trials which were then averaged.

2. Next, we generated artificial single trials (sine waves, randomly varying in phase) and multiplied these single trials with the individual NP computed in step 1.
3. Thereafter, we added the average ERP of an individual subject onto the single trials computed in step 2 and calculated the resulting envelope, which will be termed 'estimated activity'. The envelope was computed using the Hilbert transform implemented in 'Matlab software (The MathWorks Inc., USA; version 7.0)'. Finally, the 'estimated activity' was compared with the 'real activity' of the data (see below).

If the 'additive power' model was true, then amplitudes estimated by the simulation should equal the empirically observed amplitudes. If, however, these two values differ there must be non-linear processes involved. Hanslmayr et al. (2007) argued that in the latter case the 'phase-reset' model is more likely, since phase-resetting is a non-linear process.

The simulation was carried out separately for each subject. To estimate the alpha activity for the simulation, the unaveraged EEG data (collapsed across both stimulus types) were first bandpass filtered at the subjects' IAF. We employed a Butterworth filter without phase shift which is implemented in the 'Brain Vision Analyzer software (BrainProducts GmBH, Germany; version 1.05)' with a slope of 48 dB/octave and a bandwidth of 2 Hz. For the analysis of individual alpha frequencies we applied individual filter pass-bands around the subject's individual alpha frequency. Thus, for example, we took cutoff frequencies (−3 dB) at 9 Hz and 11 Hz for subjects with 10 Hz IAF.

For statistical analyses, the following procedures were carried out. At first, real activity and estimated activity were calculated for each subject. Next, *t*-tests were calculated for

each sample point in the poststimulus interval for each group and electrode separately to determine whether the real activity is significantly different from the estimated activity in the simulation. To control for multiple testing, the p-level was set to 0.005 (two-tailed). Additionally, a two-way ANOVA was calculated with the factors 'group' (high-alpha vs. mid-alpha vs. low-alpha group) and 'activity' (real vs. estimated total activity averaged over all three channels from 0 to 500 ms poststimulus).

3. Results

The three groups differed significantly in the amplitude of prestimulus total alpha activity ($F(2,19)=69.315$, $p<.0005$; high-alpha group: 7.9 μV; mid-alpha group: 4.7 μV; low-alpha group: 2.5 μV), while individual alpha frequencies did not differ significantly between groups ($F(2,19)=.638$, *ns*; high-alpha group: 10.4 Hz; mid-alpha group: 9.9 Hz; low-alpha group: 10.0 Hz). No significant differences between the three groups were observed for reaction times ($F(2,19)=.166$, *ns*) or error rates ($F(2,19)=1.109$, *ns*).

Total alpha activity changed in response to stimulation ($F(1,19)=37.638$, $p<.0005$; cf. Figs. 1–4). A strong group effect indicated that the amplitudes of total alpha activity were significantly different between groups ($F(2,19)=44.790$, $p<.0005$). Furthermore, the event-related change of total alpha activity relative to baseline differed between groups ('group' × 'stimulation': $F(2,19)=24.818$, $p<.0005$). Post-hoc tests revealed that

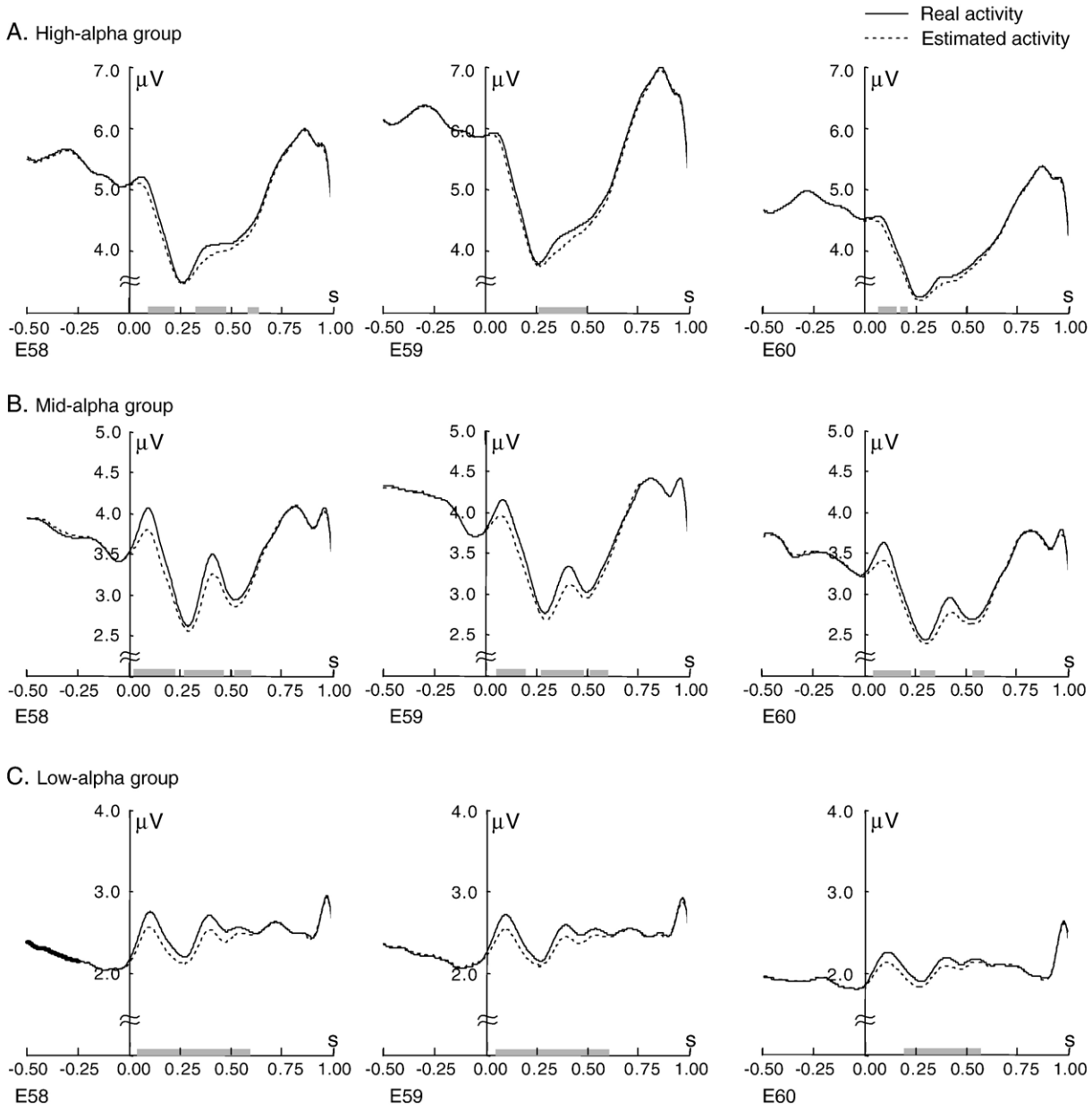

Fig. 5. Results of the simulation of total signal activity for the (A) high-alpha, (B) mid-alpha and (C) low-alpha group are plotted. Solid lines correspond to the real activity and dotted lines correspond to the signal activity estimated by the simulation. Gray bars indicate the time interval at which the real and the estimated activity differ significantly from each other ($p<.005$; two-tailed). Note that the real activity is different from the estimated activity in the simulation in all three groups.

there was a significant decrease of total alpha activity in the high-alpha group ($F(1,6)=35.155$, $p<.005$; baseline: 7.9 μV; onset: 4.1 μV) and in the mid-alpha group ($F(1,6)=10.638$, $p=.051$; baseline: 4.7 μV; onset: 3.2 μV) but a significant increase of total alpha activity in the low-alpha group ($F(1,7)= 11.326$, $p<.05$; baseline: 2.5 μV; onset: 3.0 μV; cf. Figs. 1A and 2A). As for the peak latencies of poststimulus total alpha activity, the three groups differed significantly ($F(2,19)= 61.868$, $p<.0005$). Post-hoc tests indicated that the latencies in the low-alpha group (mean: 106.9 ms) were significantly earlier than those in the high-alpha group ($F(1,13)=103.780$, $p<.005$; mean: 291.0 ms) and in the mid-alpha group ($F(1,13)=111.056$, $p<.005$; mean: 328.2 ms).

On the other hand, evoked alpha activity increased in response to stimulation ($F(1,19)=157.262$, $p<.0005$; cf. Figs. 1–4). Moreover, the three groups differed in the amplitude of evoked alpha activity ($F(2,19)=4.809$, $p<.05$), but there was no significant interaction ('group'×'stimulation': $F(2,19)= 1.836$, *ns*). The peak latencies of evoked alpha activity were not significantly different between groups ($F(2,19)=1.142$, *ns*; high-alpha group: 107.7 ms; mid-alpha group: 129.7 ms; low-alpha group: 118.0 ms).

Furthermore, the peak latencies of evoked alpha activity were significantly different from those of total alpha activity ($F(1,19)=183.991$, $p<.0005$), and there was a significant interaction ('group'×'latency': $F(2,19)=57.364$, $p<.0005$). Post-hoc tests revealed a significant main effect for the factor 'latency' in the high-alpha group ($F(1,6)=222.244$, $p<.0005$) and the mid-alpha group ($F(1,6)=66.762$, $p<.0005$), whereas the low-alpha group showed no significant differences ($F(1,7)= 2.037$, *ns*).

The results of the simulation revealed that the real activity was significantly different from the estimated activity in the simulation for each group and channel (see Fig. 5). The ANOVA showed a significant main effect for the factor 'activity' ($F(1,19)=20.266$, $p<.001$) indicating that the activity estimated by the simulation was smaller than the real activity. A significant main effect for the factor 'group' ($F(2,19)=28.468$, $p<.001$) and a significant 'group'×'activity' interaction ($F(2,19)=10.274$, $p<.001$) showed a stronger difference between real and estimated activities for the low-alpha group compared with the two others.

4. Discussion

In this study, we investigated the relationship between event-related oscillatory activity in the alpha band and prestimulus levels of ongoing alpha activity. Our results demonstrate that the amount of prestimulus alpha activity modulates the subsequent event-related neural response. Similar findings about ERD/ERS in the alpha band (Pfurtscheller, 1992, 2001; Pfurtscheller et al., 1996) and a power increase in intracortical recordings (Fell et al., 2004; Shah et al., 2004) have been reported before. Here we observed different event-related alpha dynamics in terms of total and evoked activities in a single framework with respect to ERP generation to evaluate a plausible model for ERP generation.

As Doppelmayr et al. (1998b) found that the prestimulus level of absolute band power has a strong influence on ERD, in the high- and mid-alpha groups we observed a typical poststimulus alpha-blocking which seemed to be absent in the low-alpha group (cf. Figs. 1A, 2A, 3A, D, G and 4). One possible explanation for this difference is that those subjects in the high- and mid-alpha groups are more susceptible to desynchronization of their synchronized prestimulus alpha activity than those with low levels of prestimulus alpha activity (low-alpha group).

In the high- and mid-alpha groups, we observed significantly later peaks of total than of evoked alpha activity. These findings suggest that there are different underlying mechanisms for phase-locked and non-phase-locked alpha activities.

4.1. Evidence for phase-resetting

The evoked alpha activity of all groups exhibited an obvious increase after stimulation (cf. Fig. 1B). Thus, we found a dissociation between a decrease of total alpha activity and an increase of evoked alpha activity in the high- and mid-alpha groups, as shown in Fig. 1A and B. Since phase-resetting can occur independently of the modulation of total activity, these observations from subjects with higher prestimulus alpha activity can be better explained by poststimulus phase-resetting of the ongoing alpha activity. However, we are reluctant to interpret this poststimulus dissociation between an increase of evoked alpha and a decrease of total alpha activity as evidence for pure phase-resetting, because single subject data in the high- and mid-alpha groups (cf. Fig. 4A and B) demonstrated a tiny increment of the poststimulus total alpha activity superimposed on its decrease when evoked alpha increased. In these cases, a majority of poststimulus total alpha enhancement seemed to be masked by prominent alpha-blocking, as suggested in the Introduction section. Thus, we complementarily referred to our simulation results, which substantiated that there was evidence for a non-linear contribution for ERP generation in all three groups. Accordingly, we assume that phase-resetting contributed here because phase-resetting is a non-linear process whereas additive power is a linear process.

Such an evidence of phase-resetting is in line with the observations and conclusions reached by other previous studies (Brandt, 1997; Gruber et al., 2005; Hanslmayr et al., 2007). For example, Brandt (1997) reported that the first two poststimulus negative peaks of the ERP undergo phase and prestimulus amplitude sensitive latency reorganization during presentation of both visual and auditory paradigms. These findings consistently suggest that stimulation induces a rearrangement of ongoing EEG activity reflected in the attributes of subsequent responses.

A recent report of a failure to find phase-resetting in posterior alpha activity seems to be contradictory to our findings (Mazaheri and Jensen, 2006). Using a measure termed the phase-preservation index the authors reported that in single trials the alpha oscillations after visual stimuli preserved their phase relationship with respect to the phase before the stimuli. However, this study did not show considerable poststimulus phase-locking activity within the alpha band. This may be due to the attributes of their stimuli: wedge-shaped checkerboards displayed at a width of 12° visual angle. Only a small portion of

the stimuli was projected to the macula, and the largest part of the wedge-shaped checkerboards was presented peripherally. These factors of stimulus size and eccentricity may have led to their failure to observe significant phase-locking. If, however, not even significant phase-locking could be observed — which is typically present both in phase-resetting and in additive power, the authors were not likely to find evidence for phase-resetting.

4.2. Evidence for additive power

In addition, we observed that the total alpha activity of the low-alpha group showed an increase while evoked alpha simultaneously increased in response to stimulation (cf. Figs. 1A, B, 2A, B, 3G, and H). Even single subject data (cf. Fig. 4C) revealed a simultaneous increase of poststimulus total *and* evoked alpha activities. Although these findings do not directly imply the validity of an additive component, they seem to be more in accordance with the additive power model since in the additive power model phase synchronization can occur only together with an increase in total power when the evoked component appears.

Beside these observations, results from our simulation suggest that phase-resetting alone could not account for ERP generation because the estimated activity was not always significantly different from the real activity in the simulation. Our time-frequency scalograms of phase-locking activity also display that all three groups exhibit an increase in phase-locking factors irrespective of the amount of prestimulus total alpha activity (cf. Fig. 3). This may imply a poststimulus additive power independent of prestimulus conditions. Moreover, recent reports based on depth recordings consistently demonstrated an obvious increase in EEG power accompanied by phase concentration at the dominant frequency of the ERP (Fell et al., 2004; Shah et al., 2004).

4.3. Coexistence of both models

Taken together, all of these phenomena imply that the 'phase-reset' and the 'additive power' models are evenly plausible within a single framework and able to coexist. We recently demonstrated that phase-locking and amplitude modulations of alpha activity reflect different cognitive mechanisms (Herrmann et al., 2004b). Thus, it seems plausible to assume both modulations to occur, however at different degrees depending on cognitive performance as well as prestimulus activity. An auditory study by Fuentemilla et al. (2006) consistently provided that both models are plausible, depending on the stimulation condition. They found that the 'additive power' model was most likely to explain responses to the first presented stimulus but phase-resetting was a more plausible mechanism when stimuli were presented as second or third in a row. A common interpretation of their and our observations is that the dynamics of the alpha-band response depends upon the prestimulus brain condition.

More generally, these studies fully support a dynamic view of brain function. The recently proposed event-related brain dynamics model (ERBD; Makeig et al., 2004) provides a valuable framework for future research in this field. In this model, phase consistency, additive power and frequency span a 3-dimensional signal space. Within this 3-D space, the ERBD illustrates the relationship between additive ERPs, partial phase-resetting and ERS/ERD. The ERBD model also provides a valuable context for the multimodal integration of EEG and fMRI (Debener et al., 2006; Debener et al., 2005). It is likely that only additive brain responses, that is, some areas of the ERBD signal space, systematically correlate with the fMRI BOLD signal, whereas ERPs related to partial phase-resetting may not correlate with the BOLD signal (Debener et al., 2006; Fell et al., 2004). A detailed analysis of individual differences in EEG alpha activity in this context, as demonstrated in the present study, may provide further insights.

4.4. Functions of prestimulus alpha activity

In line with our findings, effects of prestimulus alpha activity on subsequent brain responses have been documented in previous reports (Brandt and Jansen, 1991; Brandt et al., 1991; Doppelmayr et al., 1998b; Fingelkurts et al., 2002; Price, 1997; Rahn and Basar, 1993). Walter and Walter (1949) reported that different patterns of alpha activity after eye-closing were observed according to the features of spontaneous (or resting) alpha activity. Similarly, we observed that there was even poststimulus enhancement in total alpha activity in the low-alpha group, which seems to be in conflict with the phenomenon of alpha-blocking after stimulation. This corroborates the notion that the level of alpha-blocking depends on the substantial existence of prestimulus alpha activity as Doppelmayr et al. (1998b) reported.

For many years, the background EEG alpha activity had been regarded as representing mere 'idling' of the brain (Adrian and Matthews, 1934; Pfurtscheller et al., 1996). However, it has recently been considered as serving a certain active control with respect to mental processes (Basar et al., 1997; Cooper et al., 2003; Petsche et al., 1986). In addition, it has been proposed that integrative cognitive functions are carried out by large-scale neural networks (Bressler, 1995) and that this global binding of local networks may be accomplished by alpha activity (Nunez et al., 2001; Sauseng et al., 2005). Indeed, a growing body of evidence suggests that prestimulus EEG alpha activity may be involved in higher cognitive functions such as memory performance (Klimesch, 1999), anticipation (Klimesch et al., 1998; Maltseva et al., 2000) and sensory awareness (Palva et al., 2005; Varela et al., 1981). For example, Palva et al. (2005) demonstrated that the phase of ongoing cortical activity biases subsequent perception and that the widespread alpha-band component appears dominant for consciously perceived stimuli. Consistently, non-stimulus-locked alpha-band neural synchrony in the cat cortex was prominent in responses to expected objects but not in those to novel objects, which clearly indicated a role for alpha-band synchrony in top-down modulation (von Stein et al., 2000). Therefore, ongoing alpha activity before stimulation may play a functional role reflecting an aspect of the brain's readiness state relevant to an upcoming stimulus.

Consequently, different levels of prestimulus alpha activity induce different cognitive performance. Indeed, it has recently been reported that low prestimulus alpha leads to a good performance in a perception task (Ergenoglu et al., 2004; Hanslmayr et al., 2005) and a poor performance in a memory task (Hanslmayr et al., 2005). According to our results, this different cognitive performance should also be reflected in different poststimulus alpha dynamics. However, their functional relationship remains an open question and requires further investigation in future research.

5. Conclusion

In sum, at least in the alpha frequency domain, it is plausible that both 'phase-resetting' and 'additive power' could occur together after stimulation. Our observations of poststimulus dissociation between an increase in evoked alpha and a decrease in total alpha seem to be in favor of phase-resetting of ongoing EEG alpha activity, but a minor increase of total activity was detected when evoked activity enhanced even during such a dissociable poststimulus dynamics. Thus, we referred to the simulation results and confirmed that a non-linear contribution occurred after stimulation, which provides us with evidence for phase-resetting. On the other hand, a concurrent enhancement of poststimulus evoked and total alpha activities seems in line with the 'additive power' model. In agreement with the ERBD model (Makeig et al., 2004), both partial phase-resetting and partial additive power contribute dynamically to generate ERPs.

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References

Adrian, E.D., Matthews, B.H.C., 1934. The Berger rhythm: potential changes from the occipital lobes in man. Brain 57, 355–385.

Basar, E., 1980. EEG-Brain Dynamics: Relation Between EEG and Brain Evoked Potentials. Elsevier, New York, pp. 375–391.

Basar, E., Schürmann, M., Basar-Eroglu, C., Karakas, S., 1997. Alpha oscillations in brain functioning: an integrative theory. Int. J. Psychophysiol. 26, 5–29.

Basar-Eroglu, C., Demiralp, T., 2001. Event-related theta oscillations: an integrative and comparative approach in the human and animal brain. Int. J. Psychophysiol. 39, 167–195.

Brandt, M.E., 1997. Visual and auditory evoked phase resetting of the alpha EEG. Int. J. Psychophysiol. 26, 285–298.

Brandt, M.E., Jansen, B.H., 1991. The relationship between prestimulus-alpha amplitude and visual evoked potential amplitude. Int. J. Neurosci. 61, 261–268.

Brandt, M.E., Jansen, B.H., Carbonari, J.P., 1991. Pre-stimulus spectral EEG patterns and the visual evoked response. Electroencephalogr. Clin. Neurophysiol. 80, 16–20.

Bressler, S.L., 1995. Large-scale cortical networks and cognition. Brain Res. Brain Res. Rev. 20, 288–304.

Busch, N.A., Debener, S., Kranczioch, C., Engel, A.K., Herrmann, C.S., 2004. Size matters: effects of stimulus size, duration and eccentricity on the visual gamma-band response. Clin. Neurophysiol. 115, 1810–1820.

Cooper, N.R., Dominey, S.J.J., Burgess, A.P., Gruzelier, J.H., Croft, R.J., 2003. Paradox lost? Exploring the role of alpha oscillations during externally vs. internally directed attention and the implications for idling and inhibition hypotheses. Int. J. Psychophysiol. 47, 65–74.

Debener, S., Ullsperger, M., Siegel, M., Fiehler, K., von Cramon, D.Y., Engel, A.K., 2005. Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. J. Neurosci. 25, 11730–11737.

Debener, S., Ullsperger, M., Siegel, M., Engel, A.K., 2006. Single-trial EEG–fMRI reveals the dynamics of cognitive function. Trends Cogn. Sci. 10, 558–563.

Delorme, A., Makeig, S., 2004. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J. Neurosci. Methods 134, 9–21.

Doppelmayr, M., Klimesch, W., Pachinger, T., Ripper, B., 1998a. Individual differences in brain dynamics: important implications for the calculation of event-related band power. Biol. Cybern. 79, 49–57.

Doppelmayr, M.M., Klimesch, W., Pachinger, T., Ripper, B., 1998b. The functional significance of absolute power with respect to event-related desynchronization. Brain Topogr. 11, 133–140.

Ergenoglu, T., Demiralp, T., Bayraktaroglu, Z., Ergen, M., Beydagi, H., Uresin, Y., 2004. Alpha rhythm of the EEG modulates visual detection performance in humans. Brain Res. Cogn. Brain Res. 20, 376–383.

Fell, J., Dietl, T., Grunwald, T., Kurthen, M., Klaver, P., Trautner, P., Schaller, C., Elger, C.E., Fernandez, G., 2004. Neural bases of cognitive ERPs: more than phase reset. J. Cogn. Neurosci. 16, 1595–1604.

Ferree, T.C., Luu, P., Russell, G.S., Tucker, D.M., 2001. Scalp electrode impedance, infection risk, and EEG data quality. Clin. Neurophysiol. 112, 536–544.

Fingelkurts, A.A., Fingelkurts, A.A., Krause, C.M., Sams, M., 2002. Probability interrelations between pre-/post-stimulus intervals and ERD/ERS during a memory task. Clin. Neurophysiol. 113, 826–843.

Fuentemilla, L., Marco-Pallares, J., Grau, C., 2006. Modulation of spectral power and of phase resetting of EEG contributes differentially to the generation of auditory event-related potentials. Neuroimage 30, 909–916.

Gruber, W.R., Klimesch, W., Sauseng, P., Doppelmayr, M., 2005. Alpha phase synchronization predicts P1 and N1 latency and amplitude size. Cereb. Cortex 15, 371–377.

Hanslmayr, S., Klimesch, W., Sauseng, P., Gruber, W., Doppelmayr, M., Freunberger, R., Pecherstorfer, T., 2005. Visual discrimination performance is related to decreased alpha amplitude but increased phase locking. Neurosci. Lett. 375, 64–68.

Hanslmayr, S., Klimesch, W., Sauseng, P., Gruber, W., Doppelmayr, M., Freunberger, R., Pecherstorfer, T., Birbaumer, N., 2007. Alpha phase reset contributes to the generation of ERPs. Cereb. Cortex 17, 1–8.

Herrmann, C.S., Mecklinger, A., 2000. Magnetoencephalographic responses to illusory figures: early evoked gamma is affected by processing of stimulus features. Int. J. Psychophysiol. 38, 265–281.

Herrmann, C.S., Munk, M.H., Engel, A.K., 2004a. Cognitive functions of gamma-band activity: memory match and utilization. Trends Cogn. Sci. 8, 347–355.

Herrmann, C.S., Senkowski, D., Rottger, S., 2004b. Phase-locking and amplitude modulations of EEG alpha: two measures reflect different cognitive processes in a working memory task. Exp. Psychol. 51, 311–318.

Herrmann, C.S., Grigutsch, M., Busch, N.A., 2005. EEG oscillations and wavelet analysis. In: Handy, T.C. (Ed.), Event-Related Potentials: a Methods Handbook. The MIT Press, Cambridge, pp. 229–259.

Jansen, B.H., Agarwal, G., Hegde, A., Boutros, N.N., 2003. Phase synchronization of the ongoing EEG and auditory EP generation. Clin. Neurophysiol. 114, 79–85.

Jervis, B.W., Nichols, M.J., Johnson, T.E., Allen, E., Hudson, N.R., 1983. A fundamental investigation of the composition of auditory evoked potentials. IEEE Trans. Biomed. Eng. 30, 43–50.

Kalcher, J., Pfurtscheller, G., 1995. Discrimination between phase-locked and non-phase-locked event-related EEG activity. Electroencephalogr. Clin. Neurophysiol. 94, 381–384.

Klimesch, W., 1999. EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis. Brain Res. Brain Res. Rev. 29, 169–195.

Klimesch, W., Doppelmayr, M., Russegger, H., Pachinger, T., Schwaiger, J., 1998. Induced alpha band power changes in the human EEG and attention. Neurosci. Lett. 244, 73–76.

Klimesch, W., Schabus, M., Doppelmayr, M., Gruber, W., Sauseng, P., 2004a. Evoked oscillations and early components of event-related potentials: an analysis. Int. J. Bifurc. Chaos Appl. Sci. Eng. 14, 705–718.

Klimesch, W., Schack, B., Schabus, M., Doppelmayr, M., Gruber, W., Sauseng, P., 2004b. Phase-locked alpha and theta oscillations generate the P1–N1 complex and are related to memory performance. Brain Res. Cogn. Brain Res. 19, 302–316.

Klimesch, W., Hanslmayr, S., Sauseng, P., Gruber, W.R., 2006. Distinguishing the evoked response from phase reset: a comment to Mäkinen et al. NeuroImage 29, 808–811.

Lopes da Silva, F.H., 1999. Event-related potentials: methodology and quantification. In: Niedermeyer, E., Lopes da Silva, F.H. (Eds.), Electroencephalography: Basic Principles, Clinical Applications, and Related Fields. Williams & Wilkins, Baltimore, pp. 947–957.

Makeig, S., Westerfield, M., Jung, T.P., Enghoff, S., Townsend, J., Courchesne, E., Sejnowski, T.J., 2002. Dynamic brain sources of visual evoked responses. Science 295, 690–694.

Makeig, S., Debener, S., Onton, J., Delorme, A., 2004. Mining event-related brain dynamics. Trends Cogn. Sci. 8, 204–210.

Mäkinen, V., Tiitinen, H., May, P., 2005. Auditory event-related responses are generated independently of ongoing brain activity. Neuroimage 24, 961–968.

Maltseva, I., Geissler, H.G., Basar, E., 2000. Alpha oscillations as an indicator of dynamic memory operations — anticipation of omitted stimuli. Int. J. Psychophysiol. 36, 185–197.

Mazaheri, A., Jensen, O., 2006. Posterior alpha activity is not phase-reset by visual stimuli. Proc. Natl. Acad. Sci. U. S. A. 103, 2948–2952.

Nunez, P.L., Wingeier, B.M., Silberstein, R.B., 2001. Spatial–temporal structures of human alpha rhythms: theory, microcurrent sources, multiscale measurements, and global binding of local networks. Hum. Brain Mapp. 13, 125–164.

Palva, S., Linkenkaer-Hansen, K., Näätänen, R., Palva, J.M., 2005. Early neural correlates of conscious somatosensory perception. J. Neurosci. 25, 5248–5258.

Petsche, H., Pockberger, H., Rappelsberger, P., 1986. EEG topography and mental performance. In: Duffy, F.H. (Ed.), Topographic Mapping of the Brain. Butterworths, Boston, pp. 63–98.

Pfurtscheller, G., 1992. Event-related synchronization (ERS): an electrophysiological correlate of cortical areas at rest. Electroencephalogr. Clin. Neurophysiol. 83, 62–69.

Pfurtscheller, G., 2001. Functional brain imaging based on ERD/ERS. Vision Res. 41, 1257–1260.

Pfurtscheller, G., Stancák Jr., A., Neuper, C., 1996. Event-related synchronization (ERS) in the alpha band — an electrophysiological correlate of cortical idling: a review. Int. J. Psychophysiol. 24, 39–46.

Price, G.W., 1997. The effect of pre-stimulus alpha activity on the auditory P300 paradigm: a prospective study. Brain Topogr. 9, 169–176.

Rahn, E., Basar, E., 1993. Prestimulus EEG-activity strongly influences the auditory evoked vertex response: a new method for selective averaging. Int. J. Neurosci. 69, 207–220.

Sauseng, P., Klimesch, W., Doppelmayr, M., Pecherstorfer, T., Freunberger, R., Hanslmayr, S., 2005. EEG alpha synchronization and functional coupling during top–down processing in a working memory task. Hum. Brain Mapp. 26, 148–155.

Sayers, B.M., Beagley, H.A., Henshall, W.R., 1974. The mechanism of auditory evoked EEG responses. Nature 247, 481–483.

Schroeder, C.E., Steinschneider, M., Javitt, D.C., Tenke, C.E., Givre, S.J., Mehta, A.D., Simpson, G.V., Arezzo, J.C., Vaughan Jr., H.G., 1995. Localization of ERP generators and identification of underlying neural processes. Electroencephalogr. Clin. Neurophysiol., Suppl. 44, 55–75.

Shah, A.S., Bressler, S.L., Knuth, K.H., Ding, M., Mehta, A.D., Ulbert, I., Schroeder, C.E., 2004. Neural dynamics and the fundamental mechanisms of event-related brain potentials. Cereb. Cortex 14, 476–483.

Tallon-Baudry, C., Bertrand, O., 1999. Oscillatory gamma activity in humans and its role in object representation. Trends Cogn. Sci. 3, 151–162.

Varela, F.J., Toro, A., John, E.R., Schwartz, E.L., 1981. Perceptual framing and cortical alpha rhythm. Neuropsychologia 19, 675–686.

von Stein, A., Chiang, C., König, P., 2000. Top-down processing mediated by interareal synchronization. Proc. Natl. Acad. Sci. U. S. A. 97, 14748–14753.

Walter, V.J., Walter, W.G., 1949. The central effects of rhythmic sensory stimulation. Electroencephalogr. Clin. Neurophysiol. 1, 57–86.

Yeung, N., Bogacz, R., Holroyd, C.B., Cohen, J.D., 2004. Detection of synchronized oscillations in the electroencephalogram: an evaluation of methods. Psychophysiology 41, 822–832.