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Research report

Phase-locked alpha and theta oscillations generate the P1–N1 complex and are related to memory performance

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Abstract

An oscillatory phase resetting model is presented and data are reported which indicate that early components of the event-related potential are due to the superposition of evoked oscillations. The following hypotheses were tested and could be confirmed: (i) theta and alpha show a significant increase in phase locking during the time window of the P1 and N1 as compared to a prestimulus reference, (ii) the dynamics of event-related changes in evoked theta and alpha power obey the same principles as are known from event-related de-/synchronization research, and (iii) latency measures of the P1–N1 complex are negatively correlated with individual alpha frequency. In addition, we have found that theta phase locking is larger during encoding than recognition and that good memory performers show a larger increase in theta and alpha phase locking during recognition in the time window of the N1. Our general conclusion is that the P1–N1 complex is generated primarily by evoked alpha and theta oscillations reflecting the synchronous activation of a working- and semantic memory system.

Theme: Neural basis of behavior
Topic: Learning and memory: physiology

Keywords: ERP; Oscillations; P1–N1; Theta; Alpha; Memory

1. Introduction

The aim of the present study is to test the hypothesis whether the P1–N1 complex of event-related potentials (ERPs) is generated by a superposition of evoked oscillations [3,4] in the theta and alpha frequency range. We develop this hypothesis within the framework of an oscillatory phase resetting model for ERP generation and then demonstrate that a superposition of evoked theta and alpha waves synchronized in absolute phase during the N1 latency window can nicely describe the typical P1–N1 waveform. In considering findings from event-related de/synchronization (ERD/ERS) research [22,24,31], the proposed hypothesis allows us to predict that evoked theta and alpha amplitudes show the same principles of event-related changes that are known from traditional bandpower analyses [22].

The typical latencies of the P1–N1 wave already suggest a superposition of evoked theta and alpha. The latency range of the P1 (at posterior sites) for visual stimuli is about 90–120 ms and about 150–190 ms for the N1 [25]. In young, healthy adults alpha has a frequency of about 10 Hz (with a range of about 7–13 Hz). Accordingly, mean latency between two alpha peaks of the same polarity is about 100 ms. Thus, mean latency of the P1 (of about 105 ms) lies in the average alpha frequency range, whereas the interpeak latency (between the P1 at about 105 ms and the N1 at about 170 ms) of about 65 ms (representing a half cycle because the P1 and N1 have different polarities) lies in the slow alpha frequency range, close to the 'transition' of theta (a cycle of $2 \times 65 = 130$ ms represents a frequency of 7.7 Hz). Particularly at posterior sites, the early ERP components consist of sharp positive and negative peaks that form the P1–N1 complex. They appear to reflect modality-specific sensory processes that are sensitive to the direction of attention (for a comprehensive review, see Ref. [25]) and are well investigated in the visual

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[15,19,21,26,28,29] and auditory information processing domain [18,20,43–46]. The fact that the P1 and N1 are sharp waves with alternating polarities clearly demonstrates that neural synchrony occurs in narrow time windows and probably alternates between inhibitory and excitatory processes. There is some evidence that the P1 amplitude is associated with the suppression of irrelevant, and the N1 with the processing of the attended, relevant information. Thus, it was suggested that the P1 reflects inhibitory, and the N1 excitatory processes (cf. Ref. [21], p. 23). It is important to emphasize that EEG waves with alternating polarities are due to changes in the (relative) level of depolarization in the (dendritic and somatic) membrane potential of masses of neurons and, thus, reflect phases of low vs. high excitability. This is exactly the way oscillatory processes are operating as was shown by a variety of different investigators analyzing local field potentials and multi-unit activity in animal studies and the EEG in humans (for summaries see, e.g. Ref. [13] and Refs. [3,4], respectively). At the neuronal level, the basic mechanism thereby is that variations in the strength of the local field potential are related to periods of enhanced or suppressed multi-unit activity which reflect bursts of action potentials [8,12].

Although the similarities between the P1–N1 wave and evoked theta and alpha are obvious, it may be objected that this similarity is superficial and that the P1 and N1 are generated as evoked components that are independent from 'background activity' such as ongoing oscillations. Thus, the critical issue is what type of evidence allows us to distinguish between an oscillatory phase resetting and evoked model of ERP generation. The oscillatory phase resetting model (depicted in Fig. 1A) assumes that an

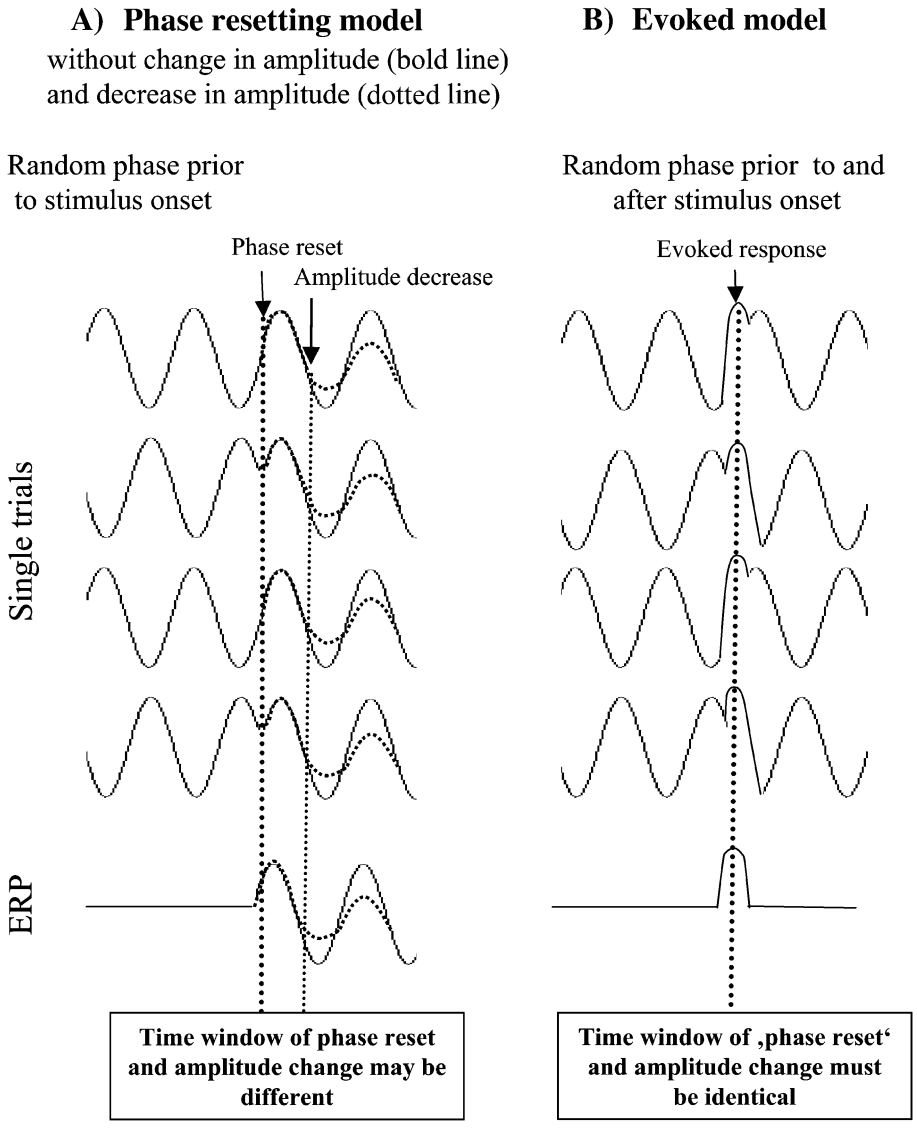


Fig. 1. The oscillatory phase resetting model (A) and the evoked model (B) make different predictions about amplitude changes during or after phase resetting. The generation of an ERP without an increase in amplitudes (or even during an event-related decrease in amplitudes) can be predicted from the oscillatory model only. This latter case is typical for alpha as our results (cf. Figs. 4 and 6) indicate.

ongoing oscillation (of a given frequency domain) undergoes an (i) event-related modulation of phase (i.e. exhibits phase resetting) and, in addition, may also undergo an (ii) event-related modulation in amplitude. The evoked model (shown in Fig. 1B) assumes that superimposed on 'background activity' (depicted as an ongoing oscillation in the frequency domain of interest with random phase between trials and no resetting) a fixed latency and polarity evoked component is generated in response to a stimulus and/or task. It is important to note that phase resetting per se (which is well documented [9,27,37,41,42]) does not allow us to distinguish between the oscillatory and evoked model. A fixed polarity fixed latency component superimposed on a (random) oscillation would also lead to a transient reduction in the intertrial phase variability and, thus, mimic phase resetting. Nonetheless, the two models can be distinguished when considering—in addition to phase resetting—the type of event-related amplitude modulation which is different for theta and alpha. Alpha is characterized by an event-related decrease in amplitude (i.e. by ERD) whereas theta by an increase in amplitude (ERS). Because in the evoked model, 'phase resetting' can occur only together with an increase in (evoked) amplitude and only during that time window where the evoked component appears, in the oscillatory model, phase resetting can occur independent of the type of amplitude modulation (i.e. increase, decrease or no change in amplitude). Furthermore, in the oscillatory model, the time windows for phase resetting and amplitude changes may vary independent of each other. It should be noted (as depicted in Fig. 1A) that even in a case when amplitudes do not change (and hence there is no single trial evoked component), an ERP will be obtained due to phase resetting.

When evaluating the two models, three different measures (explained in Materials and methods below), one for phase resetting (termed phase locking index or PLI in the following), one for amplitude changes in single trials (termed whole power in the following) and another for the amplitudes of the evoked potential (termed evoked power in the following) will be used. PLI reflects the degree of phase variation between trials (for very similar measures see, e.g. the 'intertrial coherence' suggested by Ref. [27] and the 'phase locking factor' used by Ref. [41]). PLI is a normalized measure with a PLI = 0 reflecting maximal and a PLI = 1 reflecting no phase variability [37]. Whole power (a measure similar but not identical to traditional band power) and evoked power will be determined for each frequency band (in the theta and alpha range) on the basis of Gabor wavelet analyses in order to achieve adequate time-frequency resolution and direct amplitude estimates. Evoked power will be calculated on the basis of individual ERPs. It reflects amplitude estimates of evoked theta and alpha oscillations, respectively. For whole power, single trial power estimates will be obtained which then will be averaged over trials.

Under both models we expect an increase in PLI (due to phase resetting) and an increase in evoked power. But only the oscillatory model is capable of predicting a case where (in addition to an increase in PLI) whole power does not change or decrease, whereas evoked power increases. Or, in other words, in cases of either no change or an ERD, a significant increase in PLI constitutes evidence for an oscillatory phase resetting model. We expect this latter case for the alpha band because it is so well-established that alpha power decreases (whereas theta increases) in response to a stimulus and/or task demands.

The similarity of the P1–N1 waveform with an evoked alpha/theta wave can easily be demonstrated by adding (superimposing) an alpha and a theta wave. The critical point here is that in order to generate a typical P1–N1 waveform and latency pattern, the alpha and theta wave must not be randomly superimposed with respect to their phase. We assume that theta and alpha oscillations reflect the activity in different neural network systems (cf. Ref. [22] for a review) and that the two systems become synchronized with respect to the polarity of their evoked components within a narrow time window that is represented by the N1 component. An example is shown in Fig. 2. Two different evoked oscillatory frequencies, one in the theta range with 6 Hz and a second in the mean alpha frequency range with 10 Hz are considered. The latency of the N1 was assumed with 150 ms poststimulus. From this it follows that in the alpha frequency range, the P1 occurs at exactly 100 ms (cf. Fig. 2A), whereas in the theta range, the P1 occurs earlier at 66.6 ms (cf. Fig. 2B). When the two waves are shown superimposed, it becomes clear that in the resulting ERP the N1 latency will not change, but the P1 will occur earlier (somewhere between 66.6 and 100 ms, depending on the amplitude of the evoked alpha and theta oscillation; cf. Fig. 2C). Furthermore, it should be noted that the first zero crossing of the two waves occurs at the same time, i.e. 25 ms poststimulus. It should be noted that, if there would be no phase resetting, the peaks of the alpha and theta waves would be randomly distributed and would not add up to generate the P1–N1 complex. The result of a simple simulation is shown in Fig. 2D, which depicts the predicted waveform of the P1–N1 complex if two sinus waves with different frequencies (6 and 10 Hz) but the same amplitude are added (bold line in Fig. 2D). Although differences in amplitude modulations for theta and alpha are not considered in the simulation, the resulting waveform already depicts the most important features of the P1–N1 complex.

The proposed model, as outlined in Fig. 2, is focusing on the superposition of evoked oscillations but does not make specific assumptions about the possible neural sources of the P1–N1 complex. The general view—based on findings from animal research in particular—is that multiple structures contribute to any surface ERP (cf. the work by Schroeder et al. [39,40]).

Our main hypotheses refers to the modulation of phase and amplitude of theta and alpha oscillations by the presen-

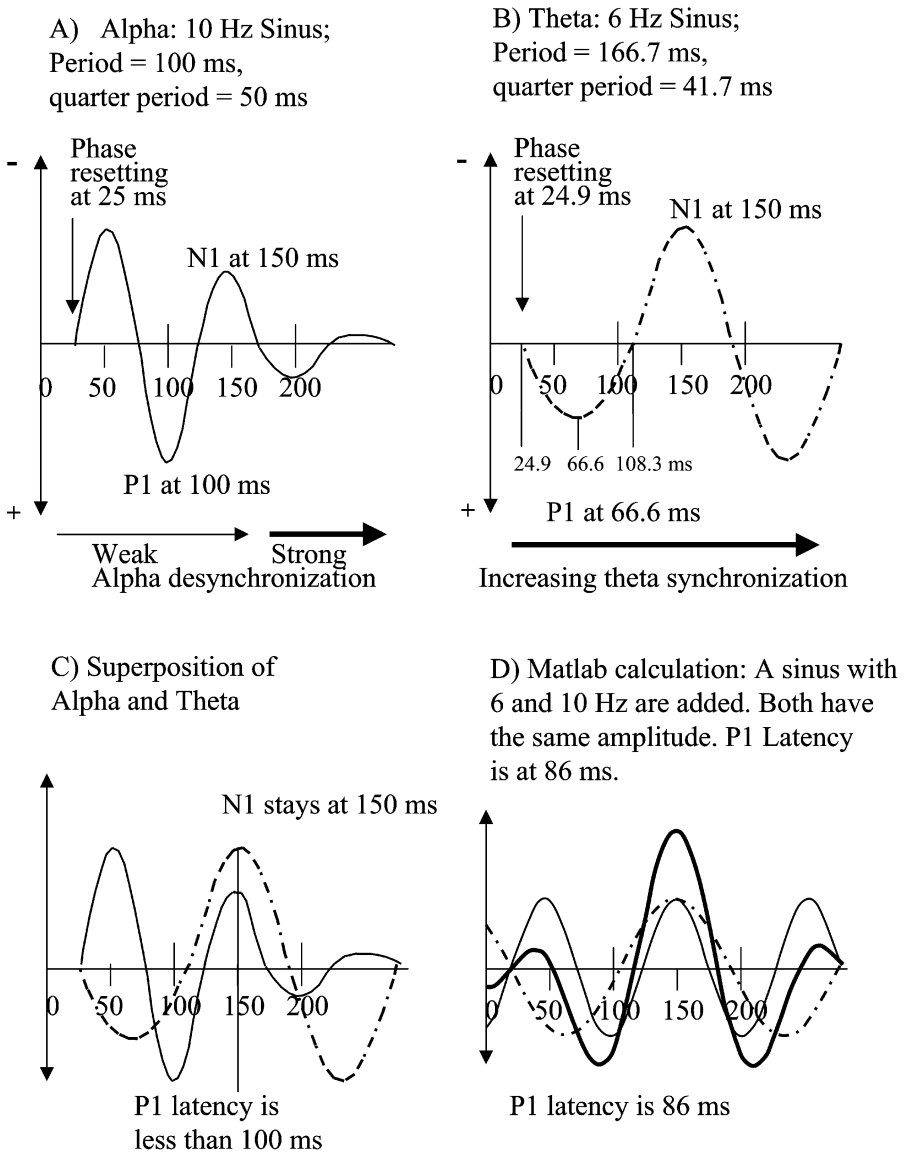


Fig. 2. The P1–N1 complex can be described by a superposition of a theta and alpha wave with a frequency of 6 and 10 Hz, respectively. (A, B) The basic assumption is that in the time window of the N1 latency the alpha and theta wave become phase-locked (i.e. have peaks with the same polarity). (C) When superimposing the two waves, we see that P1 latency will be affected by the amplitudes of both, theta and alpha waves. (D) Adding two sinus waves with the same amplitude but frequencies in the theta and alpha domain (with 6 and 10 Hz, respectively) shows a waveform (bold line) that resembles the basic features of the P1–N1 complex.

tation of a stimulus in a memory task during the time window of the P1–N1 complex. We can make the following predictions:

(1) Theta and alpha will show a significant increase in PLI during the time window of the P1 and N1 as compared to a prestimulus reference. During this time window, the event-related modulation of theta and alpha amplitudes will differ. Theta amplitudes are expected to increase (i.e. exhibit ERS) but alpha amplitudes are expected to decrease (i.e. show ERD).
(2) Due to the influence of ERS, we expect that the increase in evoked theta—from the P1 to the N1 time window—will be larger than the increase in evoked alpha. Within this time window we do not assume differential effects of the PLI for alpha and theta.

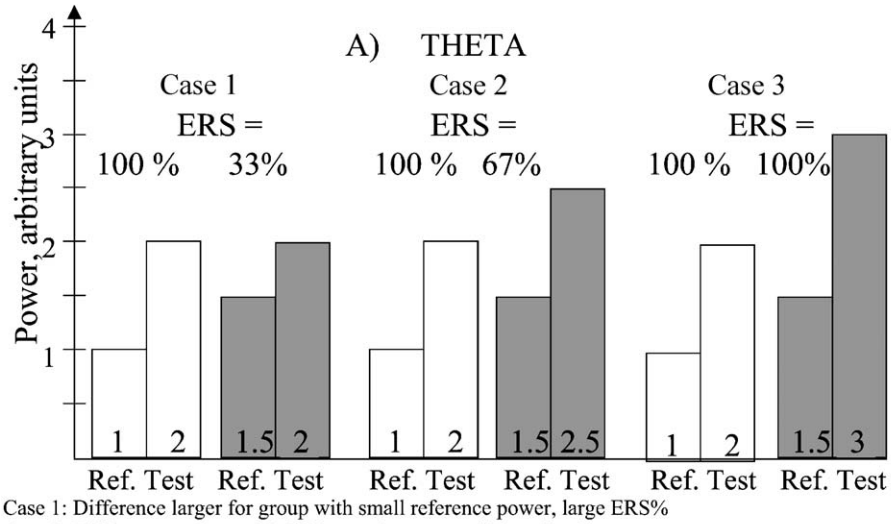
Because we have found repeatedly that theta and alpha play an important role for memory [22], we will in addition focus on the question whether interindividual differences in memory performances are reflected by differences in PLI, evoked or whole power.

If the P1–N1 complex is generated at least in part by alpha and theta oscillations, the amplitudes are expected to behave in a similar way as is known for ERD/ERS. Thus, when estimating P1 and N1 amplitudes by evoked theta and alpha power, we assume that the dynamics of changes in ERD/ERS with respect to reference power are similar for

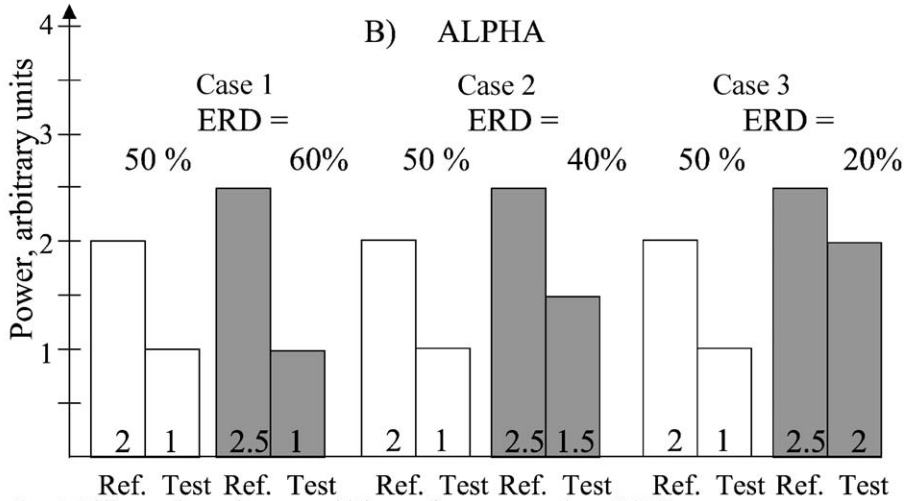
evoked power. The relevant observation here is that the extent of ERS and ERD depends on prestimulus (reference or resting) power but in different ways for theta and alpha [14,22]. Whereas small theta resting power enhances ERS, large alpha resting power enhances ERD. Thus, we predict a similar relationship between pre- and poststimulus power also for evoked theta and alpha oscillations. In particular we predict the following hypotheses:

(3) Small theta resting power will enhance evoked theta (when calculated in a similar way as for ERS, i.e. as a percentage of a power change; cf. Fig. 3A).
(4) Large resting alpha will attenuate evoked alpha (due to power suppression, cf. Fig. 3B).

The experimental evaluation of the latter two hypotheses requires some methodological clarifications with respect to the type of power measurements and the relationship between reference (ref.) power and event-related changes in power. ERD/ERS is the percentage of a change in whole (band) power during a 'test' interval (i.e. some selected interval during the actual processing of a stimulus and/or task) with respect to a reference interval (i.e. a time period preceding the processing of a stimulus and/or task) according to the formula: ERD/ERS %=((ref. power − test power)/(ref. power)) × 100. When relating ERD/ERS to absolute ref. power, we have to consider three different cases for the alpha and theta band as depicted in Fig. 3. For theta the typical observation is that for a group of subjects with small



Case 1: Difference larger for group with small reference power, large ERS%
Case 2: Difference constant; ERS% larger for group with small reference power
Case 3: Difference smaller for group with small reference power, constant or small ERS%



Case 1: Difference larger for group with large reference power, large ERD%
Case 2: Difference constant; ERD% smaller for group with large reference power
Case 3: Difference smaller for group with large reference power, small ERD%

Fig. 3. The magnitude of ERS and ERD depends on absolute resting or reference power but in different ways for theta and alpha. (A) For theta, large reference power attenuates ERS (cf. case 1 and 1). (B) For alpha, large reference power increases ERD (cf. case 1). Case 3 in panel A and cases 2 and 3 in panel B represent mathematically possible relationships between pre- and poststimulus power, not expected under our hypotheses.

ref. power (cf. the white bars in Fig. 3A) the percentage of an event-related increase in power (ERS) is larger than for subjects with large reference power (cf. cases 1 and 2 in Fig. 3A). For evoked theta, we expect the same relationship. We will test this relationship by analyses of variance (ANOVA's) with one grouping factor (termed factor POWER: subjects with small vs. large theta resting power) and one within subject factor (termed TIME: ref. power vs. test power). According to hypothesis 3 (and referring to Fig. 3A) we predict either a significant interaction between factor POWER and TIME (in the direction as predicted by case 1) or no interaction (as indicated by case 2). We predict this relationship for theta whole power as well as for evoked theta. For alpha the typical observation is that for a group of subjects with large ref. power (cf. the gray bars in Fig. 3B) the percentage of an event-related decrease in power (ERD) is larger than for subjects with small reference power (cf. case 1 Fig. 3B). Thus, for alpha power we predict—according to hypothesis 4—a significant interaction as indicated by case 1 in Fig. 3B. The rationale underlying this hypothesis is that a suppression of alpha reduces evoked power, despite the expected significant increase in PLI. Thus, for alpha we have to consider opposite effects between PLI and power suppression on evoked power. Due to phase locking, evoked alpha is expected to increase from reference to test but the extent of evoked alpha will be reduced by a suppression of power. Because desynchronization depends on resting power (case 1 in Fig. 3B), the prediction is that large reference power should lead to only a small increase in evoked power from reference to test.

Finally, according to our basic assumption that alpha is involved in the generation of the P1–N1 complex and that this involvement is particularly clear for the P1 (because during an earlier time window theta is comparatively less important than for the N1), we predict a fifth hypothesis.

(5) Individual alpha frequency (IAF) and individual P1 latency will exhibit a significant covariation.

2. Materials and methods

2.1. Subjects

After informed consent, a sample of 36 right-handed students (10 males, mean age = 25.3; SD = 2.8; 26 females, mean age = 22.1; SD = 3.6) participated voluntarily in the present experiment.

2.2. Materials and design

A set of 180 pictures was taken randomly from a large sample of line drawings [36]. Stimuli were presented at the center of a computer monitor, placed 1.2 m in front of the subjects. The line drawings cover a visual angle of $9.5 \times 5°$ and were presented in white on a black background.

The experimental design consisted of four sessions, two resting periods, study and recognition session. Subjects were asked to relax and the resting EEG was recorded first for 3 min with open eyes and then for 3 min with closed eyes. At the beginning of the study session, subjects were instructed that a series of pictures will be presented which they will have to identify in a later following recognition task. In the study session 70 target pictures were presented. Because the first and last five pictures were not used for data analysis (in order to eliminate primacy and recency effects), only 60 pictures were considered targets. After an interval of about 6 min in which subjects received further instructions, the recognition task was performed. A set of 180 pictures (consisting of the 60 targets and 120 distractors, presented in a random sequence) was shown. Subjects were asked to make a recognition judgment for each of 180 pictures by verbally responding 'yes' or 'no'.

During task performance, a single trial consisted of a blank interval of 4 s (0–4000 ms), the presentation of a picture for 1 s (4000–5000 ms) and another blank interval of 2 s in the study and 3 s in the recognition task. Targets and distractors were presented on a computer monitor in randomized order.

2.3. Apparatus

EEG-signals were amplified by a 32-channel biosignal amplifier system (frequency response: 0.16–30 Hz), subjected to an anti-aliasing filterbank (cut-off frequency: 30 Hz, 110 dB/octave) and were then converted to a digital format. Sampling rate was 128 Hz.

2.3.1. Electrophysiological recordings

A set of 17 silver electrodes were placed according to the International Electrode (10–20) Placement system, at F3, F4, Fz, C3, C4, Cz, T3, T4, P3, P4, Pz, PO1, PO2, PO3, PO4, O1 and O2. In addition, two earlobe electrodes were attached to the left and right ear. Data were recorded against a common reference placed on the nose and off-line algebraically re-referenced to averaged earlobes. For data analysis, only the following six electrodes were selected: Fz, Pz, P3, P4, O1 and O2. The electrooculogram (EOG) was recorded from two pairs of leads in order to register horizontal and vertical eye movements.

All epochs were carefully checked individually for artifacts and were categorized with respect to correctly identified targets (hits) and correctly rejected distractors. For the study session, epochs were also categorized with respect to those pictures which were later correctly identified. After rejecting artifacts and erroneous trials, an average of 37 epochs remained for correctly identified targets in the study and 43 epochs in the recognition task. Only these items were used for statistical analyses.

2.3.2. Electrophysiological variables and data analysis

Five different electrophysiological variables were calculated: (i) individual alpha frequency (IAF), spectral estimates for (ii) whole and (iii) evoked power, (iv) phase locking index (PLI) and (v) latencies of the P1 and N1 of event-related potentials (ERPs).

Estimates for whole and evoked theta and alpha power were calculated for three different time intervals, a prestimulus interval (-3500 to -2500 ms) and two poststimulus intervals for the P1 and N1 time window (25–125 ms and 125–225 ms, respectively). For PLI, the prestimulus interval was -750 to -250 ms, the poststimulus intervals were the same as for whole and evoked power. In addition, for the calculation of resting power in each frequency band the resting period (of 3 min, eyes open), was segmented in epochs with a length of 4 s. For these epochs, whole theta and alpha power was determined and then averaged for each recording site and subject.

IAF was determined for each subject and each of the six recording sites as the highest spectral estimate within a frequency range of 7–13 Hz. Spectral estimates were calculated in steps of 0.25 Hz for the resting period (of 3 min) with eyes closed.

2.3.3. Estimates for whole and evoked power; IAF adjusted frequency bands

Because we have shown repeatedly that the use of fixed frequency bands leads to distorted results [19] we define frequency bands individually by using IAF for each subject (i) (averaged for the six recording sites) as cut off point, for the following four frequency bands with a width of 2 Hz each. We distinguish between an upper alpha band, termed alpha2 (falling above IAF(i)), a lower alpha band, termed alpha1, and two theta bands (falling below IAF(i) in consecutive steps of 2 Hz; termed theta2 and theta1, respectively). Power was determined on the basis of Gabor wavelet analyses which were calculated in small frequency bins of 0.5 Hz and then averaged to obtain frequency bands with a width of 2 Hz. Whole power was determined on a single trial basis and then averaged across trials. Evoked power was calculated on the basis of individual ERPs for each recording site which then was averaged for all subjects.

2.3.4. Phase locking index (PLI)

PLI is a measure evaluating phase variability in relation to stimulus onset. The application of Gabor expansion to a signal yields a transformation into a complex time-frequency signal $x(t)$ yields a transformation into a complex time-frequency signal $y(f_n,t)$ for all frequencies f_n of interest (for more details, see Ref. [38]). Thus, the amplitude $A(f_n,t)$ and phase $\Phi(f_n,t)$ of the signal may be obtained as functions of frequency and time:

$$A(f_n,t) = |y(f_n,t)|$$

$$\Phi(f_n,t) = \arg\{Re[y(f_n,t)], Im[y(f_n,t)]\}.$$

$$\Phi^k(f_n,t),\ k = 1,\ldots,K$$

Evoked power was calculated from individual ERPs according to

$$S(f_n,t) = A^2(f_n,t) = |y(f_n,t)|^2.$$

In order to rate the phase variation with respect to stimulus onset, the phases were calculated for each single trial k. The phase locking index (PLI) is defined by

$$\mathrm{PLI}(f_n,t) = |\langle e^{j\cdot\Phi^k(f_n,t)}\rangle|, j = \sqrt{-1}.$$

The PLI ranges between zero and one with an increased PLI indicating stronger phase locking. The PLI was determined for each time point and each frequency ($0 < f_n < 45$ Hz) for all recording sites, separately per subject.

2.3.5. Latencies of the P1–N1 complex

The time window for determining the P1 was 60–140 ms and 95–205 ms for the N1. Latencies were determined computer-aided and visually inspected for artificial results. In cases where the P1 or N1 component could not be identified, the respective group mean was used as estimate for the missing value. For all subjects and recording sites the percentage of missing values lies between 0% and 17%.

2.4. Statistical data analyses

Four-way ANOVA's were used to test hypotheses 1 and 2. Dependent measures were whole power, evoked power and PLI. The factors and their levels are related to frequency bands (FREQ: theta1, theta2, alpha1, alpha2), time intervals (TIME: prestimulus, P1 window, N1 window), recording location (LOC: Fz, Pz, P3, P4, O1, O2) and experimental session (RECO: recognition vs. encoding). To assess the influence of memory performance, four-way ANOVA's were calculated for the data of the recognition session only. The four factors were FREQ, TIME, LOC (as for the analyses described above but with the exception that TIME comprised only two levels: P1 and N1 window) and MEMORY (subjects with below and above average performance). The latter is a grouping factor obtained by splitting subjects into two groups on the basis of the sample mean d'. Hypotheses 3 and 4 were evaluated by two-way ANOVA's with one grouping factor (POWER: subjects with small vs. large theta resting power) and one within subject factor (TIME: ref. power vs. power in the N1 window). The procedure for grouping subjects was the following. First, for each frequency band, the recording site with the largest resting power (for eyes open) was determined. This was Fz for the two theta and the mean of P3, Pz and P4 for the two alpha bands. Then, the median for resting power was determined for the sample of subjects at these sites and used to split subjects into groups with large vs. small resting power. This procedure was done separate-

ly for each frequency band. The Greenhouse–Geisser procedure was used to compensate for violations of sphericity or circularity. For repeated measurement factors with more than two levels, the adjusted tail probabilities are reported below. For hypothesis 5, correlations were calculated between IAF, number of hits, d' and latencies of the P1–N1 complex.

3. Results

3.1. Behavioral data

On the average, subjects correctly identified 45.7 targets (76% correct). The number of correctly identified distractors was 107.4 (89% correct). The number of incorrect responses to targets (misses) was 12.1 and the number of incorrect responses to distractors (false alarms) was 12.3. Average d' was 2.14 (SD = 0.60).

3.2. Whole power

The results of a four-way ANOVA shows significant main effects for FREQ ($F(3, 105) = 5.96$; $p < 0.01$), LOC ($F(5, 175) = 10.04$; $p < 0.01$) and TIME ($F(2, 70) = 5.50$; $p < 0.05$) but not for RECO. Because, in addition, no significant interactions were obtained with RECO, the findings indicate that whole power does not differentiate between recognition and encoding. The significant main effects for FREQ, LOC and TIME and the significant interactions found for FREQ × TIME ($F(6, 210) = 54.85$; $p < 0.001$), FREQ × LOC ($F(15, 525) = 18.82$; $p < 0.001$) are embedded and, thus, must be interpreted on the basis of the higher order interaction FREQ × TIME × LOC ($F(30, 1050) = 4.84$; $p < 0.01$). Inspection of the respective means (cf. second and fifth column in Fig. 4 for an example) indicates that (i) alpha ref. power (particularly for upper alpha) is larger at posterior than frontal sites whereas the opposite holds true for theta, that (ii) the event-related

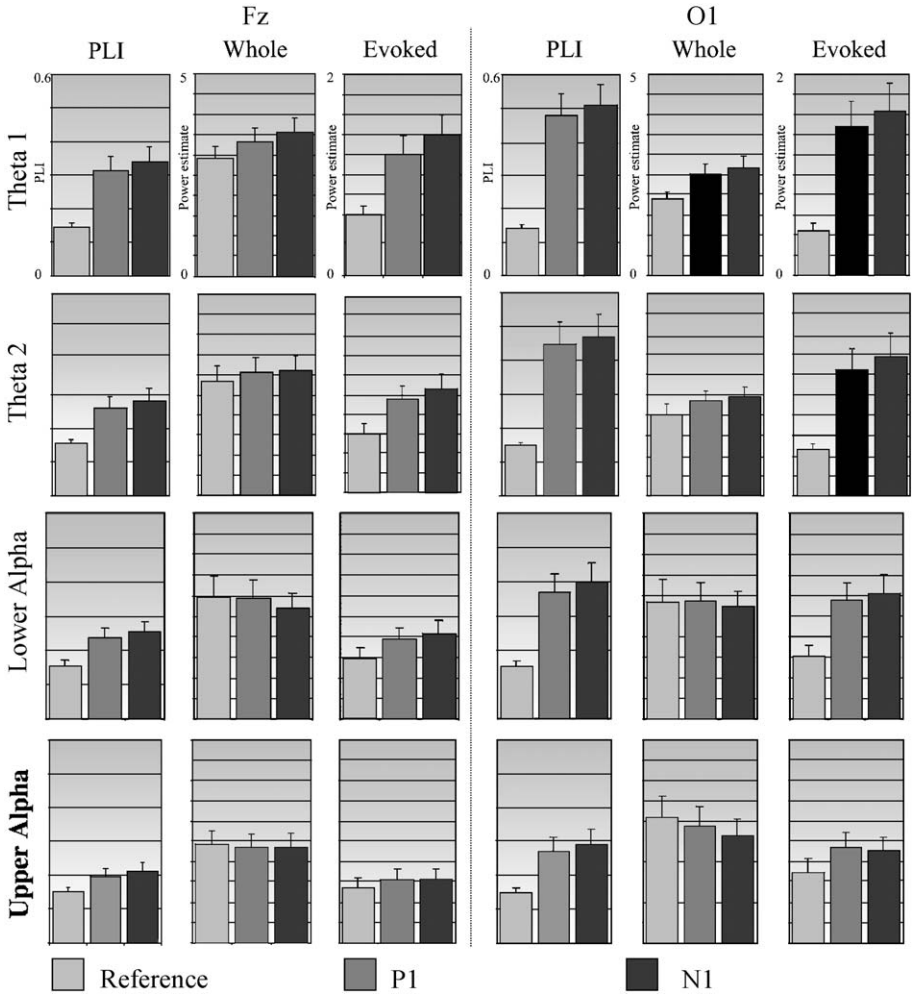


Fig. 4. The increase in evoked power and PLI is particularly large for theta1 and at posterior sites such as O1. The results for whole power reflect the well-known event-related behavior of theta and alpha, whereas theta increases (synchronizes) with cognitive demands from reference to task performance, alpha decreases (desynchronizes).

change for theta and alpha goes in different directions (alpha power decreases from ref. to the P1, N1 window, whereas the opposite holds true for theta) and that (iii) the direction of a change differs at different recording sites (alpha2 power suppression is larger at posterior than anterior sites, whereas the increase in theta1 power is larger at anterior than posterior sites).

3.3. Evoked power

The four-way ANOVA shows significant main effects for FREQ ($F(3, 105) = 15.62$; $p < 0.001$), LOC ($F(5, 175) = 6.92$; $p < 0.001$) and TIME ($F(2, 70) = 140.88$; $p < 0.001$) but not for RECO. No significant interactions were obtained with RECO. Thus, in a similar way as for whole power, evoked power does not differentiate between recognition and encoding. Significant interactions were found for FREQ × TIME ($F(6, 210) = 59.46$; $p < 0.001$), FREQ × LOC ($F(15, 525) = 3.16$; $p < 0.01$) and TIME × LOC ($F(10, 350) = 11.16$; $p < 0.001$). They indicate that (i) the increase in evoked power is larger for theta than alpha (ii) evoked power is larger at posterior than anterior sites and that this difference is more pronounced for theta than alpha, and that (iii) the increase in evoked power is generally larger at posterior than anterior sites.

Differences in evoked power in the P1 and N1 time window were evaluated by a three-way ANOVA with data collapsed over factor RECO and with factor TIME comprising only two levels (P1, N1 window). Here, we focus only on findings related to TIME and interactions with FREQ. The results show a highly significant main effect for TIME ($F(1, 35 = 59.53$; $p < 0.001$) that is embedded in the interaction TIME × FREQ ($F(3, 105) = 27.47$; $p < 0.001$) indicating that the increase in evoked power from the P1 to the N1 is much larger for theta than alpha (cf. the third and sixth column in Fig. 4). No other interactions with factor TIME reached significance. This finding clearly supports hypothesis 2.

3.4. Phase locking

The four-way ANOVA reveals highly significant effects for FREQ ($F(3, 105) = 27.79$; $p < 0.001$), LOC ($F(5, 175) = 20.18$; $p < 0.001$) and TIME ($F(2, 70) = 134.69$; $p < 0.001$) but—again—not for RECO. However, two significant higher order interactions, RECO × FREQ × LOC ($F(15, 525) = 2.22$; $p < 0.05$) and RECO × FREQ × LOC × TIME ($F(30, 1050) = 2.43$; $p < 0.05$) were found. Inspection of the respective means reveals that the increase in phase locking is larger during encoding for the theta band at O2 and P4.

The significant main effects for FREQ, TIME, and LOC must be considered together with the significant interactions obtained for FREQ × TIME ($F(6, 210) = 28.85$; $p < 0.001$), FREQ × LOC ($F(15, 525) = 4.31$; $p < 0.001$) and TIME × LOC ($F(10, 350) = 21.00$; $p < 0.001$). Analogous to evoked power, these interactions indicate that (i) the increase in phase locking is larger for theta than alpha (ii) phase locking is larger at posterior than anterior sites and that this difference is more pronounced for theta than alpha, and that (iii) the increase in phase locking (from the reference to the P1–N1 window) is generally larger at posterior than anterior sites (cf. first and fifth column in Fig. 4).

In order to assess frequency related differences in the P1 and N1 time window, a four-way ANOVA with the same factors but with TIME comprising only two levels was carried out. In this analysis, we only focus on TIME (P1 vs. N1 window) and interactions with TIME. The findings show a significant increase in PLI from the P1 to the N1 time window (TIME, $F(1, 70) = 97.92$; $p < 0.01$). Because TIME is not involved in a significant interaction with FREQ (or any other factor), the increase in PLI does not differ between frequency bands.

3.5. Memory performance, event-related power, evoked power and phase locking

The influence of memory performance was tested for analyzing the data of the recognition task with four-way ANOVA's. Instead of factor RECO, the grouping factor MEMORY was introduced and for factor TIME only two levels (P1, N1) were used. The results for whole and evoked power show no significant findings with respect to the main effect of MEMORY or its involvement in any of the interactions. However, for phase locking as the dependent measure, we obtained a significant interaction MEMORY × TIME ($F(1, 35) = 5.08$; $p < 0.05$) indicating that during recognition the increase in phase locking from the P1 to the N1 window is larger for good memory performers (largely independent of location and frequency; cf. Fig. 5 for an illustration).

3.6. Event-related modulation of whole and evoked power

The type of event-related modulation of evoked as compared to whole power was assessed by two-way ANOVA's with the grouping factor POWER (subjects with large or small resting power) and the within subject factor TIME (reference vs. time window of N1), carried out separately for the two tasks (encoding and recognition), four frequencies and six locations. Thus, for each task, 24 ANOVA's were carried for evoked and whole power. The variance source of interest is the interaction between the two factors, POWER and TIME. In addition, the direction of the interactions are determined and classified with respect to the three cases depicted in Fig. 3.

For the two theta bands none but one interaction yielded significance. POWER × TIME ($F(1, 35) = 6.4$; $p < 0.05$) yielded significance for theta 2 evoked power at Fz. Thus with this single exception (showing an example of case 1) all other findings are equivalent to case 2.

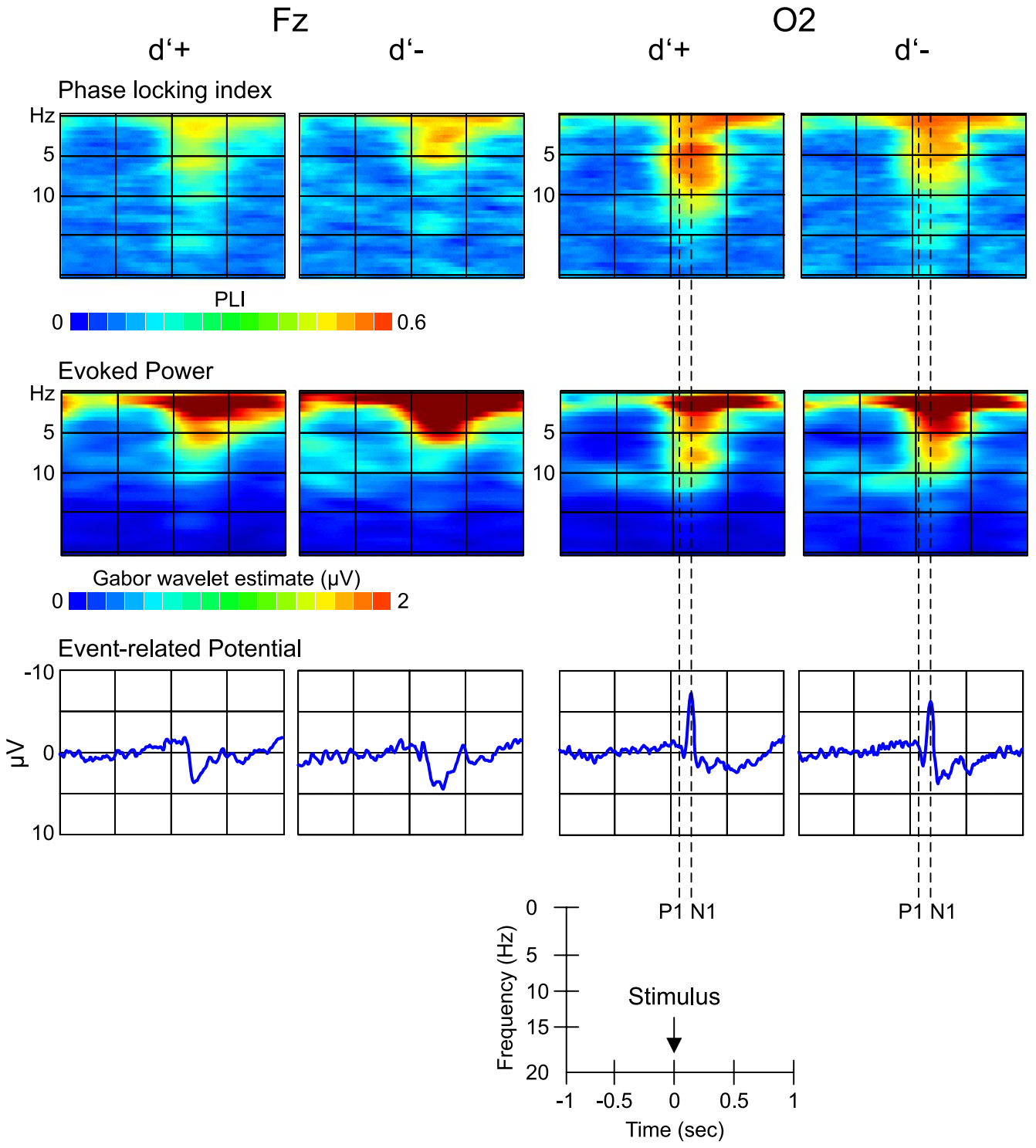


Fig. 5. Examples of time-frequency distributions of phase locking (PLI) and evoked power (EP) in relation to standard ERP's and memory performance (good and bad performers; denoted $d'+$ and $d'-$, respectively). All data are for hits during recognition at Fz and O2. Note that good performers show a larger increase in PLI from the P1 to the N1 window. Vertical dotted lines mark the time window of the P1 and N1, respectively.

For the two alpha bands, all of the findings for whole power represent examples of case 1 (i.e. large ref. power leads to an increase in ERD; cf. Fig. 3). From these findings, 10 (out of 12) are significant at or beyond the 5% level for the lower and 8 (out of 12) for the upper alpha band. For evoked power, a smaller increase from reference to the N1 window was found for the POWER+ as compared to the POWER − group in all cases. The interaction reached significance at or beyond the 5% level in 6 (out of 12) cases for the lower alpha and in 2 (out of 12) cases for the upper

Table 1
Summary of significant findings for hypothesis 4

		Alpha1						Alpha2					
		Fz	P3	Pz	P4	O1	O2	Fz	P3	Pz	P4	O1	O2
Whole	Enc	–	12,4*	–	7,3^{+}	10,2*	11,7*	–	–	–	6,3+	5,6+	7,8*
	Re	7,8*	27,7*	16,7*	33,7*	22,5*	24,4*	4,9+	7,8*	–	12,9*	5,7+	8,1*
Evoked	Enc	–	6,6+	–	–	4,5+	5,2+	–	14,4*	–	–	7,8*	–
	Re	–	–	–	–	7,7*	4,5+	–	–	–	–	–	–

* Significant beyond the 1% level.
$^{+}$ Significant beyond the 5% level.

alpha band. Statistical findings are summarized in Table 1 and examples of the interactions are depicted in Fig. 6.

3.7. IAF, memory performance and latencies of the P1–N1 complex

Intercorrelations between IAF at different recording sites and memory performance (hits and d') are shown in Table 2. Alpha frequency is significantly associated with the number of hits but not d'. The relationship between the 'speed' of alpha, appearance of the P1 and N1 peak amplitudes and memory performance is depicted in Table 3. Whereas during encoding only one significant (negative) correlation was obtained between IAF and the latencies of P1 or N1, four cases were found during recognition. The negative correlations indicate that fast alpha (high frequency) is related to

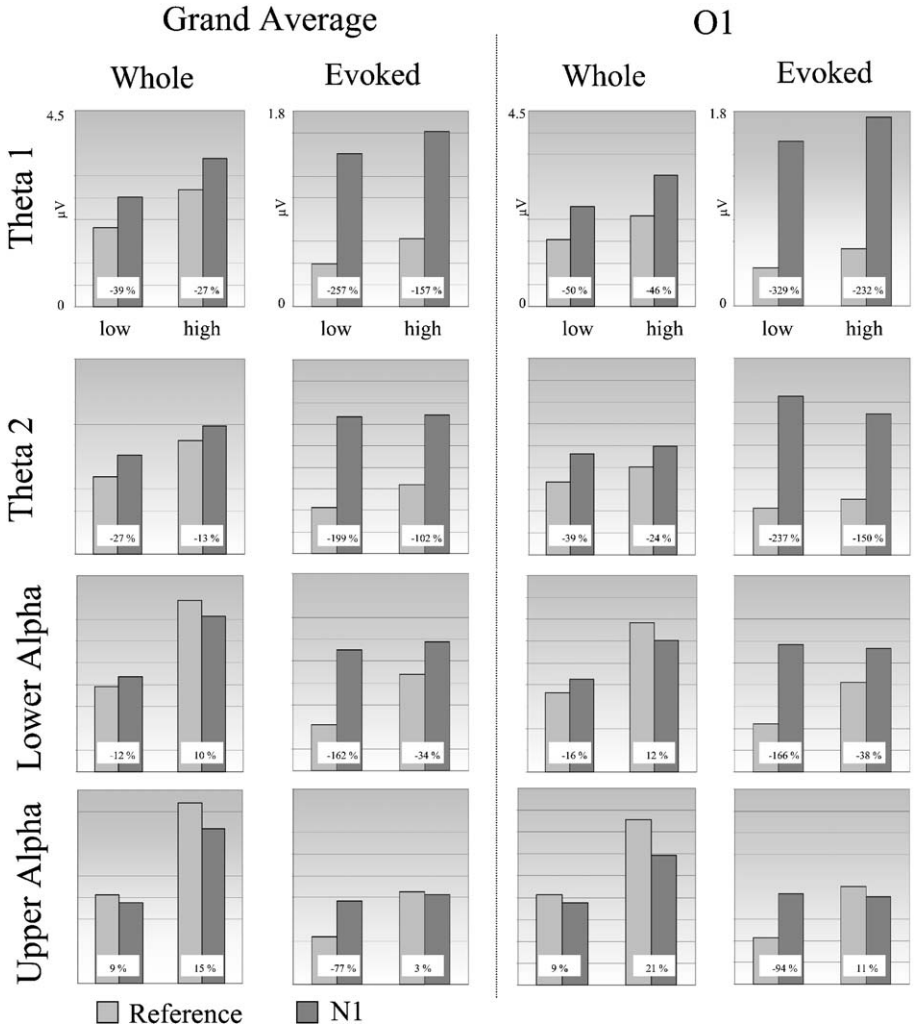


Fig. 6. Examples of results for hypotheses 3 and 4 reflecting cases 1 and 2 for theta and case 1 for alpha (cf. Fig. 3). For theta, large resting power attenuates ERS (note that ERS is scaled negatively, due to the formula: ERS/ERD%=((ref. power − test power)/ref. power). For alpha the findings indicate that large resting power increases ERD (cf. case 1 in Fig. 2A). It is interesting to note that for lower alpha subjects with small resting power show even ERS but not ERD. Alpha evoked power clearly is reduced for subjects with large resting power (due to a pronounced desynchronization).

Table 2
Intercorrelations between IAF and memory performance (M)

	IAF Fz	IAF Pz	IAF P3	IAF P4	IAF O1	IAF O2	Hits	d'
IAF Fz							0.288*	0.165
IAF Pz	0.507**						0.261	0.164
IAF P3	0.358*	0.681**					0.295*	0.117
IAF P4	0.283*	0.656**	0.895**				0.271	0.246
IAF O1	0.415**	0.730**	0.917**	0.799**			0.419**	0.159
IAF O2	0.384*	0.749**	0.910**	0.892**	0.926**		0.381*	0.191
Mean	9.61	10.31	10.37	10.33	10.47	10.44	45.69	2.14
SD	1.24	1.13	1.03	0.97	1.0	0.89	6.94	0.6

One-tailed; $N = 36$.
* 5% level of significance.
** 1% level of significance.

short latencies. It is interesting to note that for recognition, IAF is significantly related to the latency of the (early) time window of the P1 at Pz but to the (late) time window of the N1 at Fz. In addition, during recognition a significant negative correlation between the N1 latency and d' at Pz was obtained.

4. Discussion

The findings of the present study support the hypotheses derived from our model. Most importantly, the finding that for alpha a significant PLI is accompanied by a decrease in amplitudes (as measured by whole power) clearly suggests the validity of the oscillatory phase resetting model (cf. Fig. 1A). More specifically, as predicted, (i) theta and alpha show a significant increase in PLI during the time window of the P1 and N1 as compared to a prestimulus reference (cf. hypothesis 1), (ii) the largest increase in evoked power occurs during the time window of the N1 and is due to theta activity (cf. hypothesis 2), (iii) the dynamics of event-related changes in evoked theta and alpha power obey the same principles as are known from ERD/ERS research (cf. hypotheses 3–4) and (vi) latency measures of the P1–N1 complex are negatively correlated with IAF (cf. hypothesis 5).

Although a significant increase in PLI was found for all four frequency bands, the extent of phase locking is much larger for theta than alpha. In good agreement with the typical topography of the P1–N1 complex our findings indicate that for each frequency band phase locking is generally larger at posterior as compared to more anterior sites (cf. Fig. 4).

Phase locking and evoked power do show differential effects as predicted by hypothesis 2. The argument here is that (in contrast to alpha) theta amplitudes will increase from the P1 to the N1 time window. Phase locking on the other hand is not expected to increase differentially between alpha and theta because for both frequencies, the time window for phase resetting should be similar. In agreement with hypothesis 2, the results show a constant increase in PLI from the P1 to the N1 window for all frequency bands but an increase in evoked power particularly for theta. This finding clearly indicates that evoked power is the result of two factors, phase locking and the way amplitudes of evoked oscillations are modulated during stimulus presentation and/or task performance.

With respect to memory performance, the findings show that theta phase locking is larger during encoding (as compared to recognition) and that phase locking for theta and alpha oscillations during recognition shows a larger increase from the P1 to the N1 window for good memory performers (cf. Fig. 5). Thus, PLI is a sensitive measure to reflect differences in task demands and memory performance. The validity of this interpretation is underlined by the fact that neither evoked nor whole power reflects differences in task or cognitive demands. The importance of phase locking within the alpha and theta frequency range for memory was also documented in studies by Halgren et al. [17] and Rizzutto et al. [35].

The importance of theta for encoding is well in line with research from our and other laboratories demonstrating that successful episodic encoding is reflected by an increase in theta activity [22]. Recent work shows in addition that gamma oscillations and phase coupling between different brain regions also play an important role for the encoding of new information [7,16,30]. It is also interesting to note that during recognition, good memory performance is related to a general increase in PLI, regardless of frequency which is more pronounced in the N1 as compared to the P1 window. This latter finding agrees with the idea that during the N1 window theta and alpha oscillations become synchronized in phase (cf. Fig. 2). Thus, memory performance appears to be related to the activation of at least two different neuronal networks, one oscillating in theta, the other in alpha frequency, a conclusion well in line with previous findings from our laboratory [22]. Finally, the finding that latencies of the P1–N1 are related to IAF and memory performance (cf. Tables 2 and 3) provide additional support for the view that alpha oscillations play an important role for memory [23].

Another important argument that the P1–N1 complex is due to evoked alpha and theta oscillations comes from the finding that evoked power in the P1 and N1 time window reflects the same dependency on absolute power measurements as ERD/ERS does (cf. hypotheses 3 and 4). As is

Table 3
Correlations between IAF and peak latency (L) and between peak latency and memory performance (hits, d') during encoding (E) and recognition (R)

	IAF Fz			IAF Pz			IAF P3			IAF P4			IAF O1			IAF O2		
	L	hits	d'	L	hits	d'	L	hits	d'	L	hits	d'	L	hits	d'	L	hits	d'
P1 E	−0.23	−0.01	0.15	−0.13	−0.10	−0.01	−0.01	0.02	0.01	0.07	−0.25	−0.24	−0.13	−0.14	0.10	−0.34*	−0.36*	−0.27
N1 E	−0.20	−0.20	−0.00	−0.17	−0.03	−0.02	−0.14	−0.21	−0.21	−0.15	−0.32*	−0.37*	−0.10	−0.07	−0.01	−0.13	−0.11	−0.04
P1 R	0.02	0.05	−0.13	−0.51**	−0.29*	−0.17	−0.33*	−0.19	0.13	−0.18	−0.32*	−0.14	−0.22	−0.05	0.06	−0.36*	−0.42**	−0.12
N1 R	−0.34*	0.21	−0.07	−0.06	−0.14	−0.40**	−0.02	−0.12	−0.19	−0.16	−0.29*	−0.28*	−0.07	−0.22	−0.07	−0.15	−0.25	−0.03

One-tailed; N=36.
* 5% level of significance.
** 1% level of significance.

known for theta ERS, subjects with small resting theta power show a larger percent increase in evoked power than subjects with large resting theta power (cf. Fig. 6). On the other hand, for alpha ERD, subjects with small power show a small ERD (cf. last row in Fig. 6) but a large increase in evoked power. Conversely, subjects with large alpha power show a large ERD but either only a small or even no increase in evoked power at all (cf. last row in Fig. 6). This relationship between power suppression and evoked power appears complex but is easy to interpret when keeping in mind that evoked power is the result of phase locking and amplitude size. When phase locking remains constant between the two groups of subjects, differences in evoked power are solely due to differences in the amplitude of alpha. As an example, if power suppression is negligible (as in the case for subjects with small upper alpha power; cf. the first and third diagram in the last row of Fig. 6), the increase in evoked alpha is due to the influence of phase locking. If, however, alpha power becomes suppressed, the influence of alpha amplitudes on evoked power decreases and counteracts the influence of phase locking. Thus, evoked power may even decrease poststimulus (compared to the prestimulus reference) as is the case for subjects with large alpha power (cf. the second and fourth diagram in the last row of Fig. 6). In other words, alpha suppression reduces evoked power and this effect depends on the size of resting power. It should be noted that this finding provides additional evidence against the evoked model (cf. Fig. 1B).

A variety of studies have related the background EEG with the amplitude of different ERP components, particularly the P3 (cf. Ref. [32] for a review and comprehensive study). Probably the most-studied relationship is that between prestimulus alpha power and the amplitudes of the N1. Basar et al. [2,5,6,33,34] have carried out extensive research on this issue and have found that alpha and ERP amplitudes are negatively correlated. Other researchers (particularly Brandt et al. [9–11], but also Ref. [1]); however, have reported a positive relationship (with increasing prestimulus alpha power ERP amplitudes increase). On the basis of our findings these different results are not surprising because P1 and N1 amplitude size is differentially affected by the event-related modulation of theta and alpha, which in turn depends on absolute resting power. Furthermore, our measure of evoked theta and alpha represents only part of the P1 or N1 amplitudes which might in addition be influenced by amplitudes of slower frequencies. Thus, the obtained findings will depend very much on the type of measure (for the background EEG or power in different frequencies and estimate for ERP amplitudes). As an example, Basar et al. defined the enhancement factor (EF) as the ratio of the largest poststimulus ERP peak-to-peak amplitude to the root mean square prestimulus alpha power. Because this ratio decreases when prestimulus alpha power is large, the negative correlation between EF and ERP amplitudes found by this group is in line with our finding that large alpha power tends to reduce evoked power.

In summarizing, our findings clearly support the hypothesis that the P1–N1 complex is generated by alpha and theta oscillations. This is further evidence for the more general assumption, proposed by Basar et al. that ERP's can be explained by a superposition of different frequencies ranging from slow delta to fast gamma oscillations.

Acknowledgements

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