FEATURE ARTICLE
Alpha Phase Reset Contributes to the Generation of ERPs

An unresolved question in electroencephalogram (EEG) research is whether event-related potentials (ERPs) are generated by phase-reset or evoked response. We analyzed data of a visual feature detection task and will show 1) phase concentration in the alpha frequency range, 2) ongoing alpha activity prior to stimulus onset, 3) evoked alpha oscillation in the ERP, 4) lack of power increase during phase concentration, 5) decrease in amplitude variance during early evoked components preceding a decrease in power, and 6) the same cortical sources for induced prestimulus power and evoked poststimulus power. Because none of these data provide unequivocal evidence for phase reset, we additionally tested the basic assumption of the evoked model, which is the additivity of the evoked response on the basis of a simulation approach. Our findings suggest that nonadditive processes—typical for a phase reset—are involved in the generation of the ERP. Thus, together with the other findings this study provides unequivocal evidence for phase resetting in the human EEG.

Keywords: alpha, EEG, ERP, oscillations, phase, phase resetting

Introduction
A current debate in electroencephalogram (EEG) research refers to the question what the mechanisms are that underlie the generation of event-related potentials (ERPs). According to the classical view (termed evoked model), ERPs reflect transient, fixed latency, and fixed polarity (evoked) responses to a stimulus and/or event that are superimposed on the background EEG as illustrated in Figure 1a.

A competing view (termed phase-reset model) states that at least part of the ERP is generated by a reorganization of ongoing oscillations in the EEG (Sayers and others 1974; Basar 1999; Penny and others 2002; Jansen and others 2003; Barry and others 2004; Fell and others 2004; David and others 2005). The core assumption, illustrated in Figure 1b, is that oscillations undergo a phase reset that generates evoked components in response to a stimulus. For illustrating reasons, Figure 1b depicts a perfect phase reset without any jitter.

For the phase-reset model, a variety of different predictions can be made. Because the concept of phase reset implies the existence of an ongoing oscillation and because in healthy human subjects alpha is the most prominent EEG oscillation during relaxed wakefulness, the following predictions apply particularly to this frequency range. (p1) Phase concentration is expected during an early time window poststimulus where usually the occurrence of the P1–N1 complex can be observed. (p2) Phase concentration must take place at least in that frequency range that is evident as a dominant ongoing oscillation already during a prestimulus interval. But it should be considered that alpha phase reset may be functionally related to phase reset in other frequencies. (p3) The frequency characteristics of evoked activity (i.e., the ERP) have to resemble those of the ongoing oscillation. As a simple example, if dominant frequency is at 10 Hz, predicted interpeak latency between early evoked components of the same polarity is about 100 ms. (p4) During the time interval of the event-related response, power does not increase. (p5) As was suggested by Mäkinen and others (2005), a significant decrease in amplitude variance is to be expected during a phase reset. In assuming that event-related changes in band power become effective after a phase reset, the drop in amplitude variance must occur earlier than a change in power. (p6) Because EEG activity picked up by any scalp electrode sums up the activity of many different sources in the brain, we propose that the source of the ongoing activity must be the same as the source of the evoked activity if phase reset of the ongoing oscillation contribute to the signal generation of ERPs.

The basic problem when trying to evaluate the phase-reset model is that none of its predictions provides unambiguous evidence for the phase-reset model. As an example, Makeig and others (2002) showed that during the time window of the N1 component, phase concentration or phase locking can be observed in the alpha range. However, an evoked component that is superimposed on random, ongoing oscillations mimics phase reset. Filtering the EEG, which is necessary to calculate phase, "treats" the evoked response as a transient change in the phase of an ongoing oscillation. Thus, phase concentration does not provide clear evidence against the evoked model (Yeung and others 2004; Mäkinen and others 2005). As another example, it was suggested to consider changes in power between a pre- and poststimulus period in addition to phase (Shah and others 2004). Because evoked responses are superimposed on the background EEG, poststimulus power must be larger than prestimulus power. Phase concentration during the absence of a power increase appears to provide good evidence for the phase-reset model (Klimesch and others 2004; Makeig and others 2004). Nonetheless, it can still be argued that phase concentration is due to an evoked response masked by a decrease in power or with an amplitude too small to cause a significant increase in power. As a final example, a decrease in amplitude variance may be due to an event-related decrease in power and not phase reset (Klimesch and others 2006). Similar arguments apply to the delayed decrease in power (cf., p5), which may be due to the influence of evoked components.

The aim of the present study is 2-fold. On the one hand, we want to show whether for one and the same experimental data, positive evidence for all the 6 predictions of the phase-reset model can be obtained. Previous research focused only on one or a few of the above described predictions. On the other hand,
we suggest a simulation approach to test the most critical assumption of the evoked model which is the additivity of the background EEG and the evoked component (p7, cf., the summary of the predictions in Table 1). We will apply this test of additivity to data from a previous study (Hanslmayr and others 2005) where phase concentration and event-related decrease (ERD) in the alpha band were observed in a visual target detection task. Our argument is that lack of additivity will provide unequivocal evidence for a phase reset.

Materials and Methods

The EEG was recorded from 25 healthy subjects (12 males, 13 females) aged between 20 and 35 years with a mean age of 22.5 years. A total of 19 Ag-AgCl electrodes (positioned according to the international 10-20 system) against a linked earlobe reference were used. The vertical and horizontal electrooculogram were recorded from 2 additional channels to control for eye movements and blinks. Impedances were kept below 8 kOhm. A Neuroscan Synamps 32-channel amplifier was used for data acquisition. Sampling rate was 500 Hz. Frequencies between 0.15 and 70 Hz with a Notch filter at 50 Hz were recorded.

In the visual target detection task, 2 different target letters had to be distinguished. Target letter was either a "p" or a "q" which was shown for 67 ms. Thereafter a mask consisting of both letters "qp" was presented for 500 ms. A fixation cross was shown during the interstimulus interval (ISI). To control for expectancy effects, the latencies for the ISI varied randomly between 1000 and 1760 ms. The task consisted of 150 trials, the proportion between p and q was 1:1, and the sequence was randomized. Subjects were instructed to press the right cursor button on a conventional computer keyboard with their right middle finger when a "p" was presented and to press the left cursor button when a "q" was shown. Stimuli were presented on a computer screen with 75 Hz refresh rate in 1.3 m distance and 3.3° x 1.4° visual angle.

Source analyses were calculated by using low resolution electromagnetic tomography (Pasqual-Marqui and others 1994). All other analyses were carried out using the software package Matlab version 7.0.1 by Mathworks. To test whether phase distribution prior to stimulus presentation and after stimulus presentation differs significantly from a uniform distribution consistently in all subjects, the Kuiper test was used. This algorithm tests the hypothesis whether circular data (e.g., phase angles) differ significantly from uniformity or not. If the Kuiper V is greater than 2, it differs significantly from uniformity. This test has already been used by others to determine phase concentration (Jansen and others 2004; Shah and others 2004).

In order to investigate whether there is significant prestimulus activity in the alpha frequency range (450--50 ms prior to stimulus onset), power for each of the selected electrodes was calculated. Then, a peak detection for each subject and electrode was run to determine peak frequency and power of the dominant oscillation. These data were collapsed over all electrodes, and the power of the peak frequency was compared with the mean power of the surrounding frequency bands (4--7 and 15--20 Hz) by a paired t-test.

To evaluate whether the evoked power resembles the activity of the ongoing prestimulus activity, a correlation was calculated between the peak frequency of the prestimulus power and the peak frequency of the poststimulus evoked power. Peak frequency of poststimulus evoked power was determined for a time interval ranging from 100
to 400 ms after stimulus onset with the same procedure as for
prestimulus peak frequency.

Stimulus-induced changes in signal power were calculated by
applying T-tests between prestimulus (450–50 ms prior stimulus onset)
and poststimulus (0–200 ms) power in the alpha frequency range
(8–12 Hz), calculated for each electrode. To extract power and phase,
the Gabor transformation was used for all above described analyses.
The application of Gabor transformation to a given signal returns a complex
time–frequency matrix of which power and phase can be extracted.
This filter was used because it has a very good time–frequency
resolution for the frequency range used here (explained in detail
elsewhere, Gruber and others 2005).

To determine the time point of a significant change in power or
variance, the following procedure was used. 1) For each subject, the raw
data were band-pass filtered using a butterworth filter (with a slope of
48 db/decade) in 2 Hz bands from 2 to 18 Hz. 2) The filtered data were
segmented into 1 s time windows (starting at 500 ms prestimulus
and ending at 500 ms poststimulus). Segments containing artifacts were
excluded from the analysis. 3) Mean power estimates (averaged absolute
µV values) for all single trials were calculated for each subject. 4) Amplitude
variance was calculated for each subject. 5) Time course of power estimates and amplitude variance was smoothed using a moving
average with a window length of 50 ms using the “filtfilt” function
(induces no phase shift) in Matlab. 6) Power estimates and amplitude
variance during the prestimulus interval (~500 ms to stimulus onset)
were used to calculate boundaries for 95% confidence intervals. Con-
fidence intervals were calculated assuming a chi-square distribution. 7)
The 1st sample point at which power or amplitude variance was outside
of the confidence boundaries (for 6–18 Hz, it had to be lower, for 2–6 Hz
higher) was determined for each subject and electrode. 8) This time point
was then used to estimate whether there is a significant difference
between onset of decrease (or increase) of amplitude variance and
power. Paired t-tests (two tailed) were used for statistics. To compen-
sate for multiple testing, the P level was set to 0.001. Only posterior
electrode positions (T5, P3, P4, T6, O1, and O2) were included in the
analysis because early evoked components were largest at these sites.

To determine whether the sources of the prestimulus alpha and
and poststimulus evoked alpha activity are the same, a source analysis
was run. This was done for the spectral power ranging from 8 to 10 Hz for
a time interval of 500 ms prior and 500 ms after stimulus presentation.

For statistical examination of the simulation data following procedure
was applied. At first total-power (TP) (termed total power in former
publications), induced power (IP), and ERP were calculated for each
subject. IP was determined by subtracting the ERP from the single trials
run. This was done for the spectral power ranging from 8 to 12 Hz for
a time interval of 500 ms prior stimulus onset which is due to the
time smear of filtering) including the P1 time window, 120 ms
after stimulus presentation (P < 0.01, Fig. 2a). A significant phase
concentration could also be observed for the delta (2–4 Hz, cf.,
Fig. 2b) frequency band, which is associated with the slow-
positive component on which evoked alpha appears superimposed (P < 0.01, Fig. 2d). The power spectra shows a clear
peak around 10 Hz. The power of the alpha peak frequency
(10.22 Hz) was significantly higher than the power of the surrounding frequency bands (F2d = 6.37, P < 0.001) reflecting
ongoing alpha oscillation prior to stimulus presentation (Fig. 2e).

These 2 results confirm the 1st and 2nd prediction (Table 1).

The ERP (Fig. 2d) shows a prominent P1 around 120 ms
and N1 around 160 ms component followed by P2, N2, and P3
components. Because all positive components have interpeak
latencies close to 100 ms, they resemble evoked alpha activity
(cf., Fig. 2e). Consequently, evoked power exhibits large values
in a frequency range around 10 Hz, which correlates with the
peak frequency of prestimulus activity (r = 0.46, P < 0.01, one
tailed). These results confirm prediction 3 (Table 1).

The T-tests for stimulus-induced changes in TP revealed
a significant effect only for electrode O2 (F2d = 3.06, P < 0.01)
indicating a power decrease after stimulus presentation in the alpha
(8–12 Hz) range (Fig. 2f). All other electrodes did not
show significant power changes. Power increase was not found
at any site. This result confirms prediction 4.

Figure 3a depicts the smoothed time course of amplitude
variance and power in the 8– to 10-Hz band. The critical results
for the alpha band are that amplitude variance decreases sig-
ificantly around 90 ms and that amplitude variance decreases
prior to power, which reaches significance 250 ms after stimu-
lus presentation (P < 0.001 in both cases). For the delta band
(2–4 Hz), an increase rather than a decrease can be observed
for power and variance, which do not show a distinguishable
time course (Fig. 3b).

To test whether the decrease in amplitude variance occurs
consistently earlier than that of power (across subjects and for
each electrode), the time point of a significant change (increase or
decrease) in power and variance was determined for each
subject, frequency band (2 Hz bins from 2 to 18 Hz), and
electrode. The results of T-tests, calculated for the obtained
latencies (for variance and power) are summarized in Table 2.
Latencies are shown only for those cases where amplitude
variance decreases significantly earlier than power (in none of
the cases power decreased earlier than variance). In the theta
and delta frequency range, only an increase in variance and
power could be observed which did not differ in time. Note that
in the 8– to 12-Hz range, the decrease in variance occurs earlier
at occipital sites as compared with temporal and parietal
electrodes. These results confirm prediction 5 (Table 1).

The results of the source analysis for the induced prestimulus
and evoked poststimulus alpha activity (8–10 Hz) are depicted
in Figure 3c,d. The sources of these 2 measures are nearly
indistinguishable which confirms prediction 6.

For the evoked model, the EEG signal, eeg(j), for each sample
point t and trial j can be described by 2 additive components,
signal strength s(j) of the background EEG and the single
trial-evoked potential crp(j): eeg(j) = s(j) + crp(j). For the
phase-reset model, the lack of an additive component leads to

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the prediction that the EEG signals remain equal to signal strength: \( eeg(i) = s(i) \).

Testing for additivity would be easy and straightforward if there would be no concurrent change in the background EEG. But there is clear evidence that the event-related EEG response is characterized by an increase or decrease in power (depending on the frequency range and task type). The most prominent examples are that alpha power decreases but theta power increases. For simplicity, we restrict our considerations to the alpha frequency range.
Figure 3. The smoothed time course of amplitude variance (red line) and power (blue line) is depicted for the alpha (a) and delta frequency range (b). Dashed lines indicate the lower (for alpha) and upper (for delta) confidence limit for variance (red) and power (blue). Arrows indicate onset of a significant decrease or increase in amplitude variance and power. In (c) the source of the prestimulus alpha power is plotted, and in (d) the poststimulus evoked alpha source is depicted.
If the background EEG undergoes an event-related power change during the same time evoked components are generated, the expected increase in amplitudes by evoked components may be masked by an ERD (desynchronization) in amplitudes. In order to explain this, let us consider the simulation example for the evoked model in Figure 4a. Superimposed on the (random) background EEG an ERP is added, and with the onset of the ERP, the background EEG starts to decrease according to a function, which we will term ERD (cf., the 2nd and 3rd row in Fig. 4a).

For the evoked model, the power of the background EEG can be estimated by IP (IP(i) = \sum_j|\langle s(j) - \text{erp}(i) \rangle|/m; \text{erp}(i) = \sum_j|\langle s(j) + \text{erp}(j) \rangle|/m; \text{erp}(i) = 0; j = 1, \ldots, m). In other words, as illustrated by Figure 4a (3rd-5th row), the background EEG can be estimated by subtracting the ERP from the EEG signal on a single-trial basis. The power of the EEG signal, termed TP (TP(i) = \sum_j|\langle s(j) - \text{erp}(j) \rangle|/m; \text{erp}(j) = 0; j = 1, \ldots, m), exhibits a slight increase during the 1st ERP component but a considerable drop (relative to prestimulus) during the 2nd ERP peak. This illustrates that the expected increase in power (post-stimulus compared with prestimulus) may be masked partially (cf., the 1st component of the ERP) or completely (cf., the 2nd component of the ERP) by ERD. Thus, for real data, the sizes of the ERP components relative to the strength of the ERD are important factors that will determine whether a significant increase in TP can empirically be detected.

Now, the crucial idea is that IP and ERP are not additive if event-related EEG data are generated by a phase reset. As is shown in Figure 4b (5th row), IP underestimated the power of the EEG to a large extent. In contrast, for the evoked model, subtracting the ERP gives an undistorted estimate of the modulation of the background EEG. In other words, in the latter case, IP is identical to ERD.

In order to examine whether for a real data set the event-related EEG is generated by an evoked response or phase reset, we suggest calculating first the ERP, IP, and TP, and then using these parameters to simulate the data according to the evoked or phase-reset model. This procedure is illustrated in Figure 4c in assuming the data used for Figure 4a as "real data." The general idea is that in case these parameters were taken from a data-generating process obeying the principles of the evoked model, the predicted TP for the simulated and real data will be identical. If, however, the parameters are taken from a data set generated by a phase reset, the predicted TP will be smaller than that of the real data (Fig. 4d, 5th row).

This procedure applied to the data of the present experiment clearly shows that the extent of predicted TP is much smaller than that actually obtained (Fig. 4e). This simulation was carried out separately for each subject and revealed that TP is consistently underestimated by the simulation ($t_{24} = 8.49, P < 0.001$) which confirms prediction 7.

Discussion

The findings clearly demonstrate the validity of the phase-reset model. The data show 1) phase concentration at the dominant frequency, 2) ongoing alpha oscillation prior to stimulus presentation, 3) interpeak latencies of early ERP components equals period of dominant frequency, 4) lack of power increase during phase concentration, 5) significant decrease in amplitude variance preceding a decrease in power, and 6) the same sources of induced prestimulus and evoked poststimulus alpha activity. As we already have emphasized, none of these findings provides unequivocal evidence for a phase reset. This even holds true—at least in part—for the dissociation between the onset of a decrease in amplitude variance and power. It still can be argued that the delayed decrease in power is caused by an evoked response, superimposed on the background EEG. Nonetheless, we want to emphasize that only a "constant" evoked response would not affect amplitude variance at the same time. If the evoked response is "variable," which must be assumed in a physiological process, it will not only increase power but also increase amplitude variance as well. Therefore, a variable evoked response would affect both measures power and variance. Thus, it is very unlikely that the delayed decrease in power is caused by the influence of an evoked response. Nonetheless, a quantification of the parameters involved would be extremely difficult.

For an unequivocal evaluation of the phase-reset model, we suggested a different approach that is based on the fact that only for the evoked model the signal strength can be simulated by 2 additive components but is underestimated if phase reset is involved in the signal generation. The simulation revealed that the evoked model can be excluded as the only signal generation mechanism (p7). Thus, we conclude that the present study provides clear evidence for phase resetting in the human EEG contributing to early ERP components. This evidence, however, was obtained for the alpha frequency range only. In the delta range, we observed significant phase concentration which resembled the positive component reaching a maximum around 300 ms. But because no distinguishable time course between variance and amplitude was obtained for this frequency range, we cannot rule out that the later component in the ERP rather reflects processes more consistent with the evoked model. This conclusion is also consistent with other
studies, which reported that the P300 in an oddball paradigm is generated by additive mechanisms (Fell and others 2004). The reasons why we found phase reset for the alpha frequency range only may be due to the visual nature of the analyzed task. Several studies have shown that the lower frequency bands are more related to working memory processes (Klimesch 1999). Thus, it remains an open question whether in these tasks (which usually show prominent evoked

Figure 4. Results for the simulation of linear and nonlinear signal generation processes. (a) Signal-generating processes for the evoked model are simulated. Representative for the 50 single trials the 1st row shows 2 sine waves (10 Hz), which are modulated by an ERD function (2nd row) and then an ERP is added onto the single trials (3rd row). The resulting ERP is shown in the 4th row, and the time course of TP and IP are depicted in the 5th row. In (b) the signal-generating processes for the phase-reset model are simulated. The 1st row shows again 2 sine waves exhibiting phase reset; these single trials are modulated by the same ERD function as for the evoked model (2nd row). Due to the lack of an additive component the 3rd row is empty, and the resulting ERP is shown in the 4th row. TP and IP are shown in the last row; note that IP in (b) is much less than the IP for the evoked model (a). In (c) the data from (a) are used for the test of additivity. Again 50 sine waves are generated (1st row), and then these sine waves are modulated by IP obtained from the last row in (a). Again the ERP is added onto these single trials (3rd row) and the resulting ERP is shown in the 4th row. TP from (a) and the predicted (new) TP are plotted in the 5th row, note that these 2 lines are identical. In (d) the same steps as in (c) were applied with the exception that the IP from the last row in (b) was taken for amplitude modulation. As it can easily be seen in the last row the predicted TP is now underestimated. In (e) the same simulation approach was used for the data in this experiment (electrode O2, 8–10 Hz.). The critical result is that TP is in fact underestimated.
theta and delta power in the ERP) phase reset mechanisms can be identified. Finally, we wish to emphasize that it is not the intention of this paper to argue that phase resetting is the only mechanism generating the ERP. The present results allow us to argue only that the generation of ERPs cannot solely be explained by the evoked model. To what extent phase reset and evoked responses contribute to the generation of the ERP remains an open question that should be addressed in future research.

Notes
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Address correspondence to Univ. Prof. Dr Wolfgang Klimesch, Department of Physiological Psychology, Institute of Psychology, University of Salzburg, Hellbrunnerstrasse 34, A-5020 Salzburg, Austria. Email: wolfgang.klimesch@sbg.ac.at.

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