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**Phase resetting in EEG event-related potentials**

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# 1 Introduction

Brain ERPs (event-related potentials) are an important tool in research and clinical work. They are most often obtained by averaging EEG (electroencephalography) responses to a particular event, for example an auditory stimulus. Usually it is assumed, at least implicitly, that the stimulus evokes a fixed-latency response independent from the rest of EEG. Averaging many stimulus-locked epochs then removes the background EEG activity, considered to be noise. An alternative viewpoint is that the stimulus causes an alteration in the phase of the ongoing electric field potential oscillations, seen as the ERP in the averaging process. In a recent paper, Makeig et al. [4] conclude that the visual ERP can be explained almost entirely by phase resetting of ongoing alpha band (8 to 12 Hz) EEG oscillations. In this project, we examine the validity of their arguments with theoretical considerations and a computer simulation.

## 2 Background

### 2.1 EEG evoked potentials: superposed responses vs. phase resetting

Evoked potentials (EPs) are electrophysiological responses to stimuli. They are most commonly measured using electrodes attached to the skin. The idea of recording evoked potentials (EPs) is many decades old, but only the advent of reliable signal averagers in the 1960s made the expansion of EP research possible. Modern digital computers and signal processing methods have made EP recording and analysis easier still.

EEG evoked potentials arise from stimulus-induced brain activity. EEG amplitudes may reach 60–100  $\mu\text{V}$ , but the amplitude of a scalp EPs is commonly on order of a few  $\mu\text{V}$  or less. [6] The signal-to-noise ratio of EP recordings is therefore small, and EPs are often undetectable in the unprocessed EEG. The “signal” is defined as the evoked response that is summed with the “noise”, which is a sum of background brain activity and other activity due to instrumental noise.

The most common method of improving the signal-to-noise ratio is averaging. Averaging  $N$  signals improves the amplitude signal-to-noise ratio by  $\sqrt{N}$ , at least if the following conditions are fulfilled:

1. the signal does not change over time (phase, amplitude and latency stay the same)
2. there is no correlation between the signal and the noise
3. the variance of the noise is constant

The basic assumption is that the evoked potential represents a response that is independent of the prestimulus EEG.

Since the 1970s an alternative explanation for the generation of EEG evoked potentials has been discussed. The idea is that the stimulus somehow causes a redistribution of phase in the ongoing EEG oscillations, and the nonuniformity of poststimulus phase manifests as the EP in the signal averaging process. In this case no increases in poststimulus signal power should be seen. Usually there is

a clear increase in spectral power following stimulus presentation; nevertheless, with stimulus levels approaching detection threshold (for example barely-audible sounds) increases in spectral power are sometimes not detected. [6] It is not clear whether this reflects phase resetting processes or insensitivity of spectral analysis, possibly combined with stimulus-caused reduction in the power of the spontaneous activity.

For example, in their study of auditory EPs Sayers et al. [1] measure no significant increases in poststimulus signal power with “moderate stimulus levels” and propose a phase resetting mechanism. They note that imposing the phase spectrum of an averaged evoked potential on a length of spontaneous EEG produces a waveform correlated with the original averaged response. More specifically, in a later discussion [2] they claim that “...the evidence for the dominant importance of phase lies not in the extent of any AER (auditory evoked response) power changes but in the finding that imposing the the appropriate phase spectrum (that is, that of a suprathreshold AER) on a length of unstimulated EEG, converts this into a pattern highly correlated with the true suprathreshold response. Repeating the experiment by imposing the amplitude spectrum has no such effect.”

However, a large amount of information in a signal is contained in its phase spectrum and the amplitude spectra of the spontaneous signal and the averaged response are probably quite similar, so this result is hardly surprising. It is known, for example, that imposing the phase spectrum of a digital image on another, unrelated image may result in a quite good reproduction of the original image [3]. It should be noted that the epochs used for spectral analysis by Sayers et al. are quite long (940 ms) compared with the duration of the auditory response, and this affects the sensitivity of the spectral analysis to poststimulus power increases.

## 2.2 Spectrograms and coherence measures

Many digital signal analysis and processing methods are used in EEG analysis. Here we discuss spectrograms and coherence measures employed in [4].

Spectrograms give information about the time-related changes in the power spectrum of the signal. They are usually pseudocolor plots that show relative or absolute signal power as a function of time and frequency. Makeig et al. use the term “event-related spectral perturbation” (ERSP) for spectrograms that show changes in EEG spectral power after stimulus presentation. Spectrograms are traditionally calculated by windowing the signal with a short moving time domain window and calculating FFTs (Fast Fourier Transforms) of the short windowed segments. The amplitude of these FFT segments as a function of time is then the spectrogram. When analyzing an unaveraged EEG signal with many trials of stimulus presentation, an average ERSP of these trials can be calculated. A more modern alternative to the FFT technique is to use sinusoidal wavelets of different frequencies and calculate their correlation with the signal as a function of time. This procedure may offer an improved compromise between frequency and time resolution.

Coherence measures may be used to estimate phase consistency between signals. The idea is essentially to decompose the signals into harmonic components and compare the phase of the components between the signals at each frequency. This is usually done via wavelet methods. Tallon-Baudry et al. [5]

used the term “phase-locking factor” in studying the 40 Hz visual responses in human. Makeig et al. use essentially the same coherence measure, and call it intertrial coherence (ITC). Phase consistency of activity between trials has important implications for averaging. Activities that are not phase-locked will cancel out in the averaging process. As demonstrated in [5], non-phase-locked activities may still be detected from the spectrograms of the nonaveraged signal, provided that the signal-to-noise ratio is high enough.

### 3 Analysis of Makeig et al. [4]

Makeig et al. examined EEG responses to sudden onsets of simple visual stimuli. ERPs evoked by these stimuli obtained by averaging contain a negative peak (N1) with a latency of 150 to 200 ms, followed by slowly decaying 8–12 Hz rhythmic activity. This activity is termed “alpha ringing”, because it occupies the same frequency band as EEG alpha activity. In the ERP data obtained by Makeig et al., the alpha ringing typically seems to continue until at least 600 ms after stimulus.

In brief, Makeig et al. perform various spectral and coherence analyses and phase sorting to the EEG trials, and conclude that the evoked visual responses seen in the averaged EEG are not fixed polarity, fixed latency events summed to the “background EEG”, but arise from phase resetting of the ongoing EEG activity. We now investigate their arguments in detail.

#### 3.1 Power spectra and the ITC

Makeig et al. begin by comparing the poststimulus ERP spectral amplitude with the expected amplitude in a “phase-incoherent” situation. Here “phase-incoherent” means that the phase, relative to stimulus onset, of every frequency component is evenly distributed between trials. The actual ERP spectral amplitude is significantly larger than this expected amplitude in all frequencies up to  $\sim 18$  Hz. However, this finding says nothing about phase resetting of the prestimulus signal. It merely says that there are some phase-locked components in the signal, with frequencies mainly below  $\sim 18$  Hz. This may equally well be interpreted as a superposed evoked response dominated by these frequencies.

Next, Makeig et al. compare the event-related spectral perturbations of the averaged ERP and nonaveraged EEG. The ERSP of the nonaveraged EEG is calculated by first calculating the ERSP of every single trial and then averaging these single-trial ERSPs. They note that no power increases around the 10 Hz frequency are seen in the ERSP of the nonaveraged EEG, while there is a near 15 dB increase of 10 Hz power in the averaged EEG. Their interpretation is that in the single trials, the background EEG oscillations continue with unaltered power after the stimulus, but their phase is altered towards a dominant phase. Then when the single trials are averaged, a strong 10 Hz poststimulus response appears because the single trial poststimulus 10 Hz oscillations have a similar phase.

However, the ERSP plots are in fact consistent with the “superposed evoked responses” model. Suppose there are random phase 10 Hz background oscillations and an evoked response with a strong 10 Hz component phase-locked to the stimulus is summed with them. It is obvious that the poststimulus change

in total 10 Hz power depends on the phase relationship between the background oscillations and the evoked response. If they happen to be in same phase, an increase in 10 Hz power is seen. If they are in the exact opposite phase, the 10 Hz power decreases. And if the phase of the background oscillations relative to the stimulus is uniformly distributed over the trials, the average change in 10 Hz power over all trials turns out to be zero, as observed here.

The event-related intertrial coherence plot shows a significant increase in intertrial phase coherence below  $\sim 18$  Hz. But this is also consistent with a superposed evoked response phase-locked to the stimulus, in a similar way to the deviation from expected ERP spectral amplitude previously discussed. Note that the frequency distribution of coherence increase agrees very well with the ERP power spectrum.

### 3.2 Phase-sorted trials

To test the phase-locking hypothesis, Makeig et al. took 12000 single trials, sorted them according to poststimulus alpha power and selected two 1200-trial subsets, one consisting of trials with the highest alpha power and the other with the lowest. They then phase-sorted the trials and plotted them together with the two ERPs obtained by averaging the low-alpha and the high-alpha subsets. In the resulting plots, the highest alpha subset shows a non-uniform phase distribution, and this is more clearly visible after the stimulus. Also the ERP obtained from the high-alpha subset is large compared to the one obtained from the low-alpha subset. The conclusion of the authors is that “the ERP is produced by stimulus-induced phase resetting of ongoing EEG activity”.

There is an alternative explanation for this result. Let us again suppose that there is an evoked response with a strong 10 Hz component phase-locked to the stimulus, and it is relatively independent of the background EEG activity. If we choose trials that have the largest poststimulus alpha power, we end up selecting the trials where the alpha EEG activity has a similar phase with the evoked response, because then the evoked response and the background alpha activity reinforce each other. This explains the nonuniform phase distribution seen in the high-alpha subset. It also explains why the phase distribution is not uniform before the stimulus. Similarly, the low poststimulus alpha trials are the ones where 1) the alpha activity is in opposite phase with the evoked response, canceling it partially and 2) the evoked response and the alpha activity are relatively small. Note that the spontaneous activity in the low-alpha ERP indeed seems to be at the opposite phase to the high-alpha ERP before stimulus presentation.

### 3.3 Simulation of the phase-sorted data

We simulated the data on a computer to test this alternative hypothesis. The idea was to start from the “superposed evoked responses” assumption, generate simulated data, perform the same phase-sorting and selecting steps as Makeig et al. and see if similar results could be reproduced.

The model EEG trials consist of spontaneous activity, evoked response and noise. The spontaneous activity was modelled as

$$Y(t) = |A| e^{-(t-t_0)^2/T^2} \cos(2\pi ft - \phi) \quad (1)$$

These are “alpha spindles” with a Gaussian envelope. Here  $A$  is the amplitude, normally distributed with parameters  $\mu = 0 \mu\text{V}$ ,  $\sigma = 3.5 \mu\text{V}$ . The frequency  $f$  is also normally distributed with parameters  $\mu = 10.5 \text{ Hz}$ ,  $\sigma = 0.3 \text{ Hz}$ .  $T$  determines the duration of the spindle; the value  $T=0.5 \text{ s}$  was used.  $t_0$  determines the time instant of the maximum amplitude point of the spindle, and it was uniformly distributed within the interval  $[-0.8, 0.8] \text{ (s)}$ . The phase  $\phi$  was uniformly distributed in the interval  $[0, 2\pi]$ . A typical realization of the spontaneous activity model is shown in figure 1.

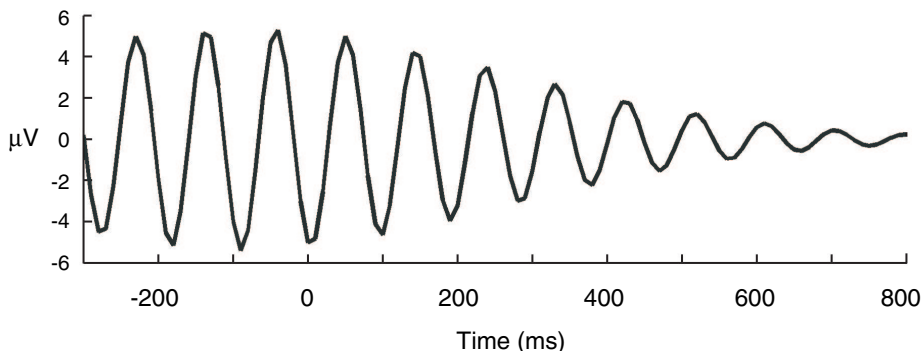


Figure 1: A realization of the spontaneous activity model

The model used for the evoked potential was

$$\begin{aligned} X(t) &= |B| t^3 e^{-\lambda t} \cos(2\pi f t - 3\pi/2), \quad t \geq 0 \\ X(t) &= 0, \quad t < 0 \end{aligned} \tag{2}$$

The enveloped sinusoidal model is simple, but resembles the evoked potentials measured by Makeig et al. closely enough for the purposes of this simulation. The parameter  $\lambda = 14 \text{ s}^{-1}$ ,  $B$  is a random variable scaled so that the maximum amplitude of the potential is normally distributed ( $\mu = 0 \mu\text{V}$ ,  $\sigma = 2.2 \mu\text{V}$ ),  $f$  is constant (10.5 Hz, which is the EP frequency of maximum power in the data of Makeig et al.) One realization of the evoked potential model is shown in figure 2.

For completeness, normally distributed white noise  $N(t)$  was added to the simulated signals, although this would not have been necessary for the demonstration of the present effects. The parameters were  $\mu = 0 \mu\text{V}$ ,  $\sigma = 0.2 \mu\text{V}$ . The standard deviation of the noise was set so that the visual noise level in the plot of the simulated data (figure 3) was roughly similar to that in the original work. The “sampling frequency” of the discrete-time simulation was 100 Hz (time grid spacing = 0.01 s), so the white noise is actually limited to frequencies under 50 Hz. In the work of Makeig et al. the data were low-passed below 40 Hz.

12000 “EEG trials”  $f_i(t)$  were then generated by summing realizations of the spontaneous activity model, the evoked potential model, and the noise model:

$$f_i(t) = y_i(t) + x_i(t) + n_i(t), \quad i = 1, \dots, 12000 \tag{3}$$

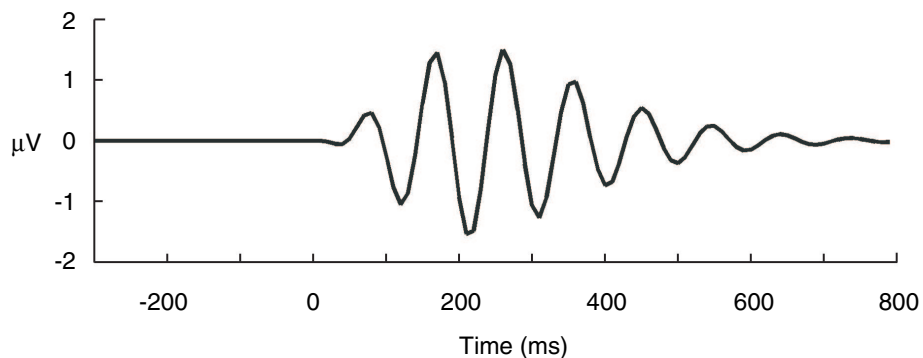


Figure 2: A realization of the evoked potential model

Here  $y_i(t)$ ,  $x_i(t)$  and  $n_i(t)$  are realizations of the random processes  $Y(t)$ ,  $X(t)$  and  $N(t)$ , respectively. These trials were first sorted by variance in the post-stimulus time window (0 to 0.293 s), and two 1200-trial subsets with the largest and smallest variance were chosen. For simplicity, we did not do spectral analysis and choose trials based on the 10 Hz power, because the 10 Hz-components almost completely determine the variance of the simulated data. Then the trials in these subsets were sorted according to their phase relative to 0 s. The ERP averages for both subsets were calculated. The results of the simulation are shown in figure 3.

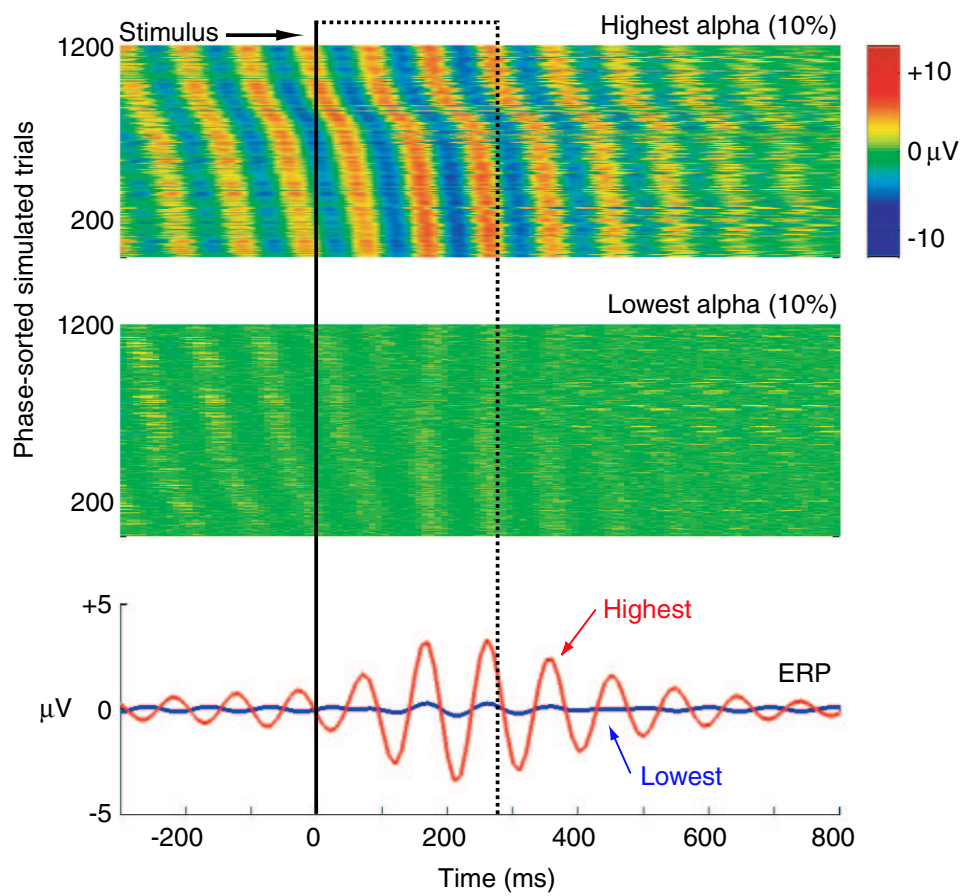


Figure 3: Simulated EEG data. Phase-sorted 1200 trial subsets of highest alpha and lowest alpha. Each horizontal colour-coded trace represents a single trial. The red ERP is an average of the highest-alpha trials, the blue ERP of the lowest-alpha trials. The power of “alpha activity” was estimated using the variance in the poststimulus interval (dotted lines). The solid black line indicates the “stimulus onset time”.

The phase in the high alpha subset is nonuniform in a similar way to the data of Makeig et al. The nonuniformity is also more clearly seen after the stimulus. The reason for this is that the phases of the spontaneous oscillations are most strongly aligned with the phase of the evoked response where the evoked response is strongest, that is, around 200 ms after stimulus. This maximizes the poststimulus alpha power. In both the simulated and the original data, the phase nonuniformity is most clearly visible at 200 ms. Because the frequencies of the spontaneous oscillations vary, they can be exactly in phase only for a short time.

Also in a similar way to the original data, the spontaneous activity in the low-alpha trials in the simulated data seems to be at the exact opposite phase to the spontaneous activity of in the high-alpha trials, as previously explained. The results are visually very close to those presented by Makeig et al., and so it is conceivable that the “phase resetting” they observed in the phase-sorted trials was actually due to the mechanism discussed previously. In particular, the claim made by the authors that “the fixed polarity components of early visual ERP records, if they exist at all, must be small ( $\ll 1 \mu\text{V}$ )” is not supported by their analysis.

## 4 Summary

It seems that Makeig et al. present no conclusive evidence that the visual ERP is caused by phase resetting of ongoing oscillations. Further investigations would be needed to clarify the ERP generation mechanism.

However, this task may not be easy. One way to tell which mechanism actually is responsible for the visual ERP could be to investigate the event-related changes in power spectrum in single trials. If the stimulus consistently causes no change in the 10 Hz spectral power, this could be taken as evidence for phase resetting. As shown, measuring the average change in 10 Hz spectral power is not sufficient, because the changes in the individual trials may average to zero. The effect of the stimulus on the spontaneous activity (for example stimulus caused alpha attenuation) may complicate this kind of investigation, especially if the evoked potentials are small.

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