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EEG alpha phase shifts during transition from wakefulness to drowsiness

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ABSTRACT

Phases of alpha oscillations recorded by EEG were typically studied in the context of event or task related experiments, rarely during spontaneous alpha activity and in different brain states. During wake-to-drowsy transition they change unevenly, depending on the brain region. To explore their dynamics, we recorded ten adult healthy individuals in these two states. Alpha waves were treated as stable frequency and variable amplitude signals with one carrier frequency (*CF*). A method for calculating their *CF* phase shifts (*CFPS*) and *CF* phase potentials (*CFPP*) was developed and verified on surrogate signals as more accurate than phase shifts of Fourier components. Probability density estimate (*PDE*) of *CFPS*, *CFPP* and *CF* phase locking showed that frontal and fronto-temporal areas of the cortex underwent more extensive changes than posterior regions. The greatest differences were found between pairs of channels involving F7, F8, F3 and F4 (*PDE* of *CFPS*); F7, F8, T3 and T4 (*CFPP*); F7, F8, F3, F4, C3, C4 and T3 (decrease in *CF* phase locking). A topographic distribution of channels with above the average phase locking in the wake state revealed two separate regions occupying anterior and posterior brain areas (with intra regional and inter hemispheric connections). These regions merged and became mutually phase locked longitudinally in the drowsy state. Changes occurring primarily in the frontal and fronto-temporal regions correlated with an early decrease of alertness. Areas of increased phase locking might be correlated with topography of synchronous neuronal assemblies conceptualized within neural correlates of consciousness.

1. Introduction

The hypnagogic state, as a transitional phase between the wake state and stage N1 of NREM sleep (Iber et al., 2007) had been receiving an increased attention in recent decades (Schacter, 1976; Ogilvie and Wilkinson, 1984; Hori et al., 1990; Ogilvie et al., 1991; Hasan et al., 1993; Harsh et al., 1994; Hori et al., 1994; Inouye et al., 1994; Wright et al., 1995; Tanaka et al., 1996, 1997; De Gennaro et al., 2001; Nielsen et al., 2005). A number of complex physiological transitional phenomena between these two brain states were tracked with ever more sophisticated instrumentation and analysis techniques. Classical spectral analysis (Värri et al., 1992; Jung et al., 1997; Morikawa et al., 1997; Cantero et al., 1999; Cantero and Atienza, 2000; Bódizs et al., 2008) and nonlinear techniques (Accardo et al., 1997; Pereda et al., 1998, 1999; Kobayashi et al., 1999; Acharya et al., 2005; Šušmáková and Krakovská, 2008; Bojić et al., 2010) revealed that various brain regions differ electrophysiologically during the wake-to-sleep transition. Three groups of different but complementary results could be summarized based on the most frequently used methods: a) those using Fourier amplitude or power topography (Ota et al., 1996; Jung et al., 1997; Cantero and Atienza, 2000) where changes in the activity of separate cortical regions were measured; b) analyses which reveal strength of intracortical connections between pairs of EEG channels, such as correlation and coherence, (Tanaka et al., 1996; Cantero et al., 2002; Cover et al., 2004; Koenig et al., 2005); c) phase, synchronicity and time delay measurements which offer an insight into time-related electrophysiological events between different cortical regions (Govindan et al., 2005, 2006; Jann et al., 2009). While the first two groups of methods are more frequent, not many studies cover the third issue, particularly in experimental paradigms related to different spontaneous brain states, such as wake and drowsy.

Alpha oscillations were extensively studied in event related experiments, where changes of alpha activity were time or phase locked to an event, including event related synchronization/desynchronization (ERS/ERD), involving changes in alpha amplitudes (e.g. Pfurtscheller and Aranibar, 1977; Klimesch et al., 2007). Similarly, alpha phase analyses, which recently drew more attention, mainly concentrated on event or task dependent measurements (Makeig et al., 2002; Naruse et al., 2006; Klimesch et al., 2007; Palva and Palva, 2007). A typical example is the notion of the alpha phase reset, as an emerging interpretation for the properties of P1 (Klimesch et al., 2007) and N1 (Palva and Palva, 2007) of ERP waveforms. However, phase locking of

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spontaneous alpha EEG activity was rarely studied, especially its topographic distribution, even more so in brain state (not task) related experimental designs.

In our previous study (Bojić et al., 2010) we investigated, in ten adult healthy individuals, changes in amplitudes of alpha activity during transition between two spontaneous brain states, wake and drowsy. We demonstrated that alpha amplitude decrease was related to an increase in fractal dimension of the corresponding signal, reflecting a simultaneous topographic reorganization of channel clusters.

The aim of the present study, performed on the same set of signals, is to investigate changes in topographic distribution of alpha phase shifts, through changes in their mean values and intensity of phase locking. We hope that differences in the measured quantities, detected in different cortical areas, might be linked to their role in the process of decrease of alertness, present during this transition. We also expect that our approach could help elucidate, at least to some extent, topography of brain structures and patterns of neural activity associated with these two states (Rees et al., 2002; Palva and Palva, 2007). Within the current frameworks of neural correlates of consciousness (NCC, Crick and Koch, 2003), global neuronal workspace (GNW, Dehaene et al., 1998), and the inhibition-timing hypothesis (Klimesch et al., 2007) we hypothesize that topography of spontaneously phase locked areas, containing a great number of neurons simultaneously excited and inhibited, might be correlated with the topography of transient neural assemblies recruited in the two states.

The main methodological novelty of the study is the concept of alpha carrier frequency (*CF*) which increases the accuracy of the phase measurements. Namely, whenever a single physiological oscillation is present in the signal, although the cortex is oscillating at one alpha *CF* with a variable amplitude, the corresponding Fourier amplitude spectrum consists of a finite width amplitude peak (usually superimposed on the background activity, Eke et al., 2000; Bojić et al., 2010). We devise a method for measuring, as accurately as possible, phase shifts between *CF* oscillations recorded at two cortical locations. Therefore, we treat signals mathematically as amplitude modulated *CF* oscillations, where the exact *CF* may be lying between two nearest Fourier components (*FC*). We calculate *CFPS* for each available channel pair, using phase shifts of alpha range *FC*. Since *CF* is expected to be different among individuals, ("Individual alpha frequency", Klimesch, 1999), alpha range limits have to be determined individually. Further, we introduce a new measure — carrier frequency phase potential (*CFPP*), as a statistical measure of *CF* phase for each particular EEG channel in the montage. Contrary to initial phase of an oscillation, *CFPP* is not dependent on the chosen moment of observation, making it suitable for analysis of spontaneous EEG activity. We present how *CFPP* is derivable from the corresponding *CFPS*. The term "potential" is suggested because all EEG channels in the montage could be organized in a series (order), according to their *CFPP* values: channel with the highest value of *CFPP* is the one leading in phase to all other channels, while the last channel follows all others. Finally, phases and phase shifts are treated as angular random variables and directional angular arithmetic is used to calculate their basic statistical properties. Accordingly, in our measurements, intensity of alpha *CF* phase locking is inversely correlated with angular standard deviation.

2. Methods

2.1. Subjects and data preparation

The signals were recorded during experiments presented in Vuckovic et al. (2002). All recordings were performed in accordance with the medical ethical standards after the subjects signed the informed consent form approved by the local ethical committee. Ten adult healthy human individuals (seven males, three females), age 25–35 (mean 28.3 ± 6.5) years, without mental disorders, were recorded after passing a neurological screening. The subjects were lying in a dark room with their eyes closed. A neurologist was monitoring their state of alertness and preventing them to fall asleep beyond N1 of NREM sleep. The tested individuals were not previously subjected to any sleep deprivation or deviation from their circadian cycles and have not been taking any medicine. EEG electrodes were positioned at 14 locations (F7, F8, T3, T4, T5, T6, F3, F4, C3, C4, P3, P4, O1 and O2) according to the International 10–20 System with an average reference. Signals were sampled at a rate of 256 samples/s, band pass filtered between 0.5 and 70 Hz (with a software 50 Hz notch) and artifacts were removed manually based on a visual inspection. Other details about data collection and preparation can be found in Vuckovic et al. (2002). One second signal epochs from all individuals were classified into wake and drowsy periods independently by two neurologists. Only those signal sequences for which both experts agreed as being either clearly awake or drowsy were used in the study (60 s for each state and each subject). The 60 s of the alert and the drowsy state was obtained by concatenating sixty 1 s long intervals of alert and drowsy states respectively.

2.2. Data analysis

2.2.1. Calculation of carrier frequency phase shifts

All available 280 signals, recorded from the ten individuals at fourteen scalp locations in the two states, were subjected to an FFT algorithm using a 1 s non-overlapping moving window. As each state was 60 s long, the same numbers of real and imaginary parts of *FC* were stored in separate files for each of the 280 signals. Further calculations were done according to the procedure contained in our previous reports (Kalauzi et al., 1998, 2009) and explained in detail in Appendices A and B. Thus, for each pair of channels (here conveniently denoted with $c1$ and $c2$), respective EEG recordings were treated as signals with variable amplitudes and equal *CF*. Their *CFPS* were calculated according to Eqs. (B.6) and (B.9) in Appendix B. Particularly, we applied a weighted version of the angular mean (Eq. (A.3)), where weighting coefficients W_i present amplitudes at particular *FC* frequencies

$$\Delta\varphi_c \approx \arctan\left(\frac{\sum_{i=k1}^{k2} W_i \sin(\Delta\varphi_i)}{\sum_{i=k1}^{k2} W_i \cos(\Delta\varphi_i)}\right)$$

$$= \arctan\left(\frac{\sum_{i=k1}^{k2} A_{c1,i}A_{c2,i}\left(\sin\left(\varphi_{c1,i}\right)\cos\left(\varphi_{c2,i}\right) - \cos\left(\varphi_{c1,i}\right)\sin\left(\varphi_{c2,i}\right)\right)}{\sum_{i=k1}^{k2} A_{c1,i}A_{c2,i}\left(\cos\left(\varphi_{c1,i}\right)\cos\left(\varphi_{c2,i}\right) + \sin\left(\varphi_{c1,i}\right)\sin\left(\varphi_{c2,i}\right)\right)}\right) \quad (1)$$

where $A_{c1,i}$, $A_{c2,i}$, $\varphi_{c1,i}$ and $\varphi_{c2,i}$ stand for amplitude and initial phase of the ith *FC* of signals $c1$ and $c2$, respectively. Eq. (1) can be reduced to

$$\Delta\varphi_c \approx \arctan\left(\frac{\sum_{i=k1}^{k2}\left(a_{c1,i}b_{c2,i} - b_{c1,i}a_{c2,i}\right)}{\sum_{i=k1}^{k2}\left(a_{c1,i}a_{c2,i} + b_{c1,i}b_{c2,i}\right)}\right) \quad (2)$$

where $a_{c1,i}$, $a_{c2,i}$, $b_{c1,i}$ and $b_{c2,i}$ denote real and imaginary parts of the ith *FC* of signals $c1$ and $c2$ respectively, while $k1$ and $k2$