

Characterization of evoked and induced activity in EEG and assessment of intertrial variability

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Abstract— Brain response to an internal or external event, is composed by the superposition of evoked and induced oscillatory activity, which reflect different brain mechanisms involved. Identification of such activations could serve for diagnostic purposes and provide useful tools for brain computer interfaces through insight on the activation of different brain regions. In this paper we study several statistical measures that have been proposed for identifying the nature of the involved activations. All these measures are based on some mean of an appropriate signal attribute over trials in the time/ frequency domain and do not characterize the variability across trials. In order to quantify trial-to-trial variability we consider a measure based on entropy, characterizing the distribution of power across trials. The results indicate that brain activations can be characterized and differentiated by their behavior from trial to trial.

I. INTRODUCTION

BRAIN activity induced by an internal or external stimulus involves the organized activation of different neural assemblies at different brain locations. These activations are captured in the scalp electrodes either as phase-locked (evoked) or non-phase-locked (induced) oscillations. Phase-locked activity can be directly extracted by averaging of the EEG single trials, resulting in the well known Event Related Potential (ERP).

The ERP represents an important and extensively studied brain response. It has been proven extremely useful in clinical and physiological research. There is a rich literature about the functional meaning of the different peaks of ERP (such as the P1, N1 and P3), which are thought to reflect different aspects of information processing in the brain [1]. This classical point of view states that ERPs are generated by fixed latency, phase-locked responses [1]. Its underlying assumption implies that the interesting ERP response is evoked by the task and can be detected by averaging the recorded signals over trials, which increases the signal-to-noise ratio (SNR) in the average signal [2].

Induced activities are expressed through the increase or

decrease of energy in a specific band post-stimulus, denoted as event related synchronization (ERS) or desynchronization (ERD), respectively [3]. Non phase-locked oscillations have been associated with a variety of different functions related to perception and different types of cognitive processes [4]. Extensive findings correlate alpha energy and alpha phase, on stimulus onset with the ERP amplitude, indicating that the ERP and EEG oscillations interact and relate to each other [5]. Furthermore, one part of ERP has been found to associate with phase resetting of ongoing EEG activities [6], which reorganizes the phase at a particular frequency to produce the ERP without implying the generation of a new response. Evoked and induced oscillations may be considered as coupled processes progressing in time, with different spatial localization of origin and partially overlapping frequency content [7].

Many studies suggest that event related brain dynamics entail a variety of activations and oscillations, from phase resetting of ongoing EEG activity in the alpha and theta bands [8] to phase-locked evoked and non-phase-locked induced oscillations especially in delta, theta and gamma bands [2], [7]. Their origins relate to multiple task conditions and many stimulus types engaged during the event presentation and execution of its consequent actions [9], which define distinct brain functions some operating independently and some being coupled [10]. Because of their separate neurophysiologic origins, phase-locked (evoked) and non phase-locked (induced) responses are of different nature [7] and have different functional roles [11], even though they may correlate with similar cognitive events [11], [12]. Furthermore the induced activity has also been characterized as event-related spectral perturbations (ERSP)[13]. ERSP measures have been considered in relation to quantified changes (increase or decrease) in power of specific frequency bands relative to mean prestimulus power, which is also termed as event related desynchronization/ synchronization (ERD/ERS) [3].

A variety of methods to characterize the nature of EEG activity in terms of their major time/frequency activity and topographic origin has been employed. Phase-relevant techniques as intertrial coherence (ITC) and phase intertribal coherence (PIC) have been used as measures to characterize the phase consistency of the detailed TF content throughout trials [14]. Alternatively, the techniques based on average power relative to prestimulus reveal non-phase-locked (induced) activity associated with ERD and ERS[15]. In case

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of evoked (ERP) activity, both phase-relevant (ITC and PIC) and power-related (ERSP) measures show significant change, whereas only phase-relevant measures are sensitive to phase resetting. In order to provide a measure of induced activity directly related to signal consistency over trials, the phase-shift intertribal coherence (PsIC) measure has been introduced. It considers the amplitude (or power) consistency of the post-stimulus part over the trials, without reference to the pre-stimulus. As such, PsIC is sensitive to phase and also frequency resetting, which reorganize and synchronize ongoing processes in terms of either phase or frequency attributes. Also, a method to extract the induced activity out of the global energy is to subtract the ERP out of each trial before applying the measures[16].

Another very interesting aspect of ERP is the trial to trial variability of different activations that take place during the experiment. In general measures using the trial mean of a metric do not characterize the variability of the metric across trials. Trial to trial variability may give more insight into the generation of the brain processes that arise during the response. Reference [6] exhibits significant results identifying processes that despite their equivalent mean dynamics, present strong amplitude variability in the single trial data. Identifying such processes effectively alters the interpretation of their physiological function.

In this work we present a framework to compare measures as the above, which quantify such trial to trial differences. We introduce and propose the notion of intertrial entropy as a measure of power variability between trials. Entropy has been used before for ERP data in the form of wavelet entropy[17],[18] in order to characterize the degree of order/disorder in a multi-frequency response and its time evolution. We also describe and demonstrate the use of the different measures and how they can be used in conjunction in order to reveal different aspects of the underlying activity. In addition we demonstrate the additional information provided by using intertrial entropy.

II. METHODS

A. Time-Frequency Transforms

Time-frequency (TF) transforms have significantly advanced in the recent years. Wavelet approaches decompose signals into constituent time–frequency ranges of energy based on the notion of scale applied to a set of basis functions. The application of wavelet transforms in TF analysis is limited by the tradeoff between frequency and time, since wavelets compute small scale (high-frequency) intervals with shorter time windows and large scale regions (low-frequency) with longer time windows. As a result, they resolve energy in higher scales with high resolution in time but not in frequency. Alternatively, in lower scales they resolve energy in detailed frequency ranges but in larger time intervals.

B. Evoked / Induced activity Metrics

In order to quantify phase locked coherence along the

trials, we can utilize the intertrial coherence (ITC) of TF maps for all channels[14]. This measure reflects the phase-locked consistency among trials and is derived from the analysis of TF maps of individual trials at each specific channel. It takes under consideration only the phase of the signal in each trial, so that any phase-locked activity of either large or small amplitude has the same effect. In order to engage the amplitude along with the phase of each trial, the phase intertrial coherence (PIC) is defined as:

$$C_{PIC}[k, n] = \frac{|\sum_i X_i[k, n]|}{\sum_i |X_i[k, n]|} \leq 1 \quad (1)$$

where $X_i[k, n]$ denotes the frequency coefficient at the i -th trial and the k -th frequency tick[14]. Equality holds if and only if all trials involve the same signal with the same phase, but each trial contributes to the measure according to its amplitude. This metric is expanded to the time-frequency representation of a signal $X_i[k, n]$, with k and n indicating the frequency and time ticks, respectively.

For the quantification of event related but not phase-locked activity, we can use a measure that is based on the energy of single-trial decompositions and highlights frequency bands of increased energy in all trials. More specifically, the phase-shift intertrial coherence (PsIC), which is a variation of the energy measure used in ERD/ERS detection, using only the post-event energy and is defined as:

$$C_{PsIC}[k, n] = \frac{\sum_i |X_i[k, n]|^2}{\max_{k, n} \sum_i |X_i[k, n]|^2} \leq 1 \quad (2)$$

where equality implies the same magnitude of $X[k, n]$, even with different shifts at each trial[14].

These maps, along with the TF energy spectrum, will be used for the characterization of relevant content, since each one emphasizes on different aspects of synchronous activity. The intertrial coherence measures can be computed for the signal itself, or its time-frequency decomposition, deriving a trial-synchronization map complementary to that of the time-frequency energy spectrum. Alternatively, they can be utilized as global metrics on a multi-trial signal, measuring its overall intertrial coherence (preferably at specific bands). In this form, they can be effectively used for significance ranking of components in each band.

ERD/ ERS represent a mean increase or decrease in event-related power. The associated ERSP measure is defined as the percentage of increase or decrease in mean trial power from a mean power baseline, time-locked to an event. The assumption behind this measure implies that there exist brain sources which are inactive in the prestimulus period and triggered by the event. Alternatively implies that the ongoing brain processes, prestimulus, are unaffected by the event and retain the same state in all trials.

The PsIC measure reflects persistent activity in all trials. A frequency band that is active in all trials would have a PsIC close to 1. It can be used in conjunction with the ERD/S measure.

C. Induced and Global Energy

Induced activity is time locked to the event but the exact latency and frequency that this activation occurs in not

known a priori. Thus the measures that use the mean power of trials, actually take under consideration the mixed activity of evoked and induced activations. A method to extract the induced activity out of the global energy is to subtract the average ERP out of each trial before applying the measures[16]. This way we can discriminate between power changes attributed directly to induced and those to evoked activations by comparing results from ERP removed and original data.

D. Intertrial Power Entropy

The Shannon entropy [19] gives a useful criterion for analyzing and comparing probability distributions. It provides a measure of the information of any distribution. We define the intertrial power entropy as:

$$S_{IE}[k, n] = - \sum_{j=1}^{trials} p_j[k, n] \log_2 p_j[k, n] \quad (3)$$

where $p_j[k, n]$ is the probability distribution of the power in each trial for each frequency k time n . The intertrial entropy reflects the order/ disorder of the power levels for each frequency among trials. Equal levels of power among trials would be the equivalent of an ordered state while random power fluctuation from trial to trial can be perceived as a state of disorder. Low values of intertrial entropy reflect the same activation across trials, while high values reflect variability in the power or latency of the activation across trials.

It is apparent that differences between trials are natural to arise in terms of power. Since the brain is always working, receiving information from different sources, we can claim that the recordings entail an inherent level of entropy. Thus, the value of entropy for each time and frequency tick is difficult to be evaluated as an absolute value as is. We define the baseline probability distribution, reflecting the natural disorder in the power between trials, as the mean entropy in the prestimulus time interval. Base in this we define the relative entropy or Kullback - Leibler divergence between trials as

$$S_{RIE}[k, n] = \sum_{j=1}^{trials} p_j[k, n] \log_2 \left(\frac{p_j[k, n]}{q_j[k, n]} \right) \quad (4)$$

where $q_j[k, n]$ is the baseline power distribution. Relative intertrial entropy gives a measure of similarity between two distributions, between $p_j[k, n]$ in respect with $q_j[k, n]$. Relative intertrial entropy is positive and zero only when $p_j[k, n] \equiv q_j[k, n]$. This measure can be used to evaluate divergence of the power distribution poststimulus compared to the power distribution prestimulus. Activations with significant difference to the prestimulus distribution can be identified. In order to evaluate whether this difference refers to an increase or decrease of entropy relative to prestimulus we use the change of intertrial entropy as:

$$S_{IEc}[k, n] = \frac{S_{IE}^{(post)} - S_{IE}^{(pre)}}{S_{IE}^{(pre)}} \quad (5)$$

Used in parallel with the measure of relative entropy we can asses whereas the activation under consideration presents an increase or decrease in entropy.

III. RESULTS

A. Application in real EEG data.

We applied the measures described in 27-channel recordings from a healthy subject performing an auditory oddball experiment. The dataset was provided by the Ecological University of Bucharest, Romania and was obtained after an approved ethics protocol. The whole dataset consists of recordings captured from 9 healthy participants (3 females and 6 males), who had no history of neurological or psychiatric disorder.

Signals were digitally sampled at 1024Hz, with a high pass filter of cut-off frequency 0.016Hz. A stimulator provided 40 2 kHz target tones (20%) and 160 1kHz non-target tones (80%). The inter-stimulus interval was 1.29s. We used 41 trials, corresponding to target tones, in our analysis. The auditory oddball experimental set-up is expected to produce both phase-locked oscillations, especially in the theta and delta bands related to P300 activity (including P3a and P3b components[20]) and especially in channels Cz and Pz, as well non phase-locked (induced) oscillatory activity, particularly related to alpha event related desynchronization (ERD).

The time-frequency transform for the processed data as well the original was computed and each of the measures was calculated. Fig. 1 presents the results on channel CZ for the considered dataset. The latency of the event is presented with black line. The first row presents the measures on the original EEG signal, indicating measures of global activity (mixed evoked and induced). Furthermore, the average ERP was calculated and subtracted from each single trial. Power measures on the resulting signals associate with only induced activity (without ERP) and are presented in the second row of Fig. 1.

We can observe that the data present increased phase locking activity in delta and theta bands following the event. Alpha band presents ERD starting 200ms after the event. Notice the weak phase locking in alpha band at the same latency as the ERD onset. PsIC measure shows that delta and lower theta bands present significantly coherent activity in all trials. PsIC measure accounts for the global power and cannot be associated with evoked or induced activity, as they overlap in time and frequency (Fig. 1a and Fig. 2b). The same applies for the ERS that occurs 300ms (P300) after the event at theta (mainly) and upper delta band. We cannot distinctly associate the power increase with an evoked activation.

During the ERD in alpha band, the relative entropy shows significant divergence from the prestimulus distribution which can be identified as an entropy decrease (80% decrease). This finding indicates that the alpha ERD presents small variations in power and latency from trial to trial and is directly modulated by the event onset. On the other hand,

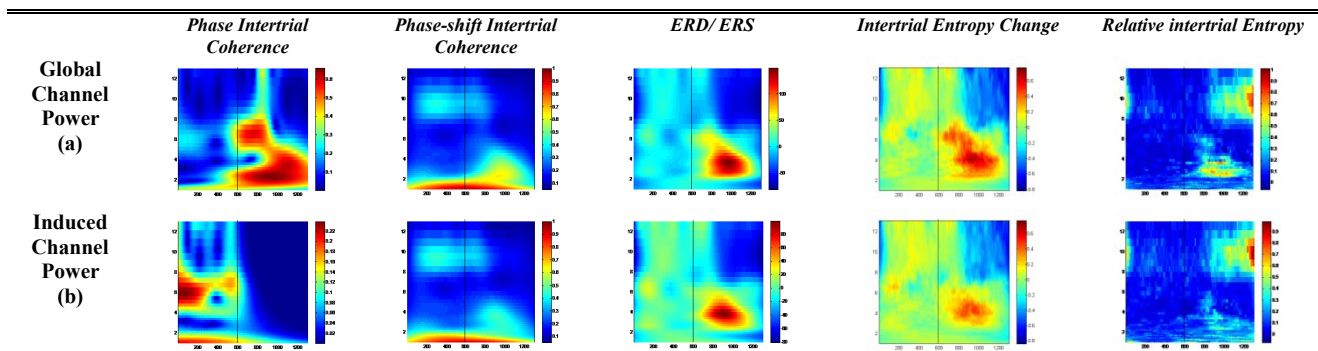


Fig. 1: Results for measures in Channel Cz. First row (a) displays the measures on the original channel, involving both evoked and induced energy. Second row (b) displays the results on the processed (ERP-free) data where the average ERP has been subtracted from the post-stimulus.

the entropy divergence identified in the same region with the ERS, in delta and theta bands, corresponds to an increase (40%) of entropy compared to the baseline. The mean divergence is 0.47. This indicates that this activation is variable from trial to trial and is affected from other factors as fatigue or level of attention.

By linking the above with the results from the ERP-free data we can identify whether the nature of the activity can be identified as evoked or induced. The PIC measure confirms that all evoked activity has been removed. A very interesting finding is that ERS is slightly reduced for the delta and theta bands compared to the original data. This indicates that the increase in power on these bands is mostly related with a non-phase locked (induced) process that takes place at the same time.

Relative entropy shows that the divergence in delta and theta bands is less for the induced activity. More specifically the mean value of relative entropy is 0.16 for the region corresponding to delta and lower theta ERS and corresponds to an increase of 19% compared to baseline. Notice the absence of entropy increase in high theta band for the induced activity. These results indicate that entropy increase is mainly due, to activity of evoked nature.

IV. CONCLUSIONS

We presented a new measure, intertrial entropy, which can be used to investigate the brain dynamics that take place during an ERP experiment, based on their distribution of power in trials. In conjunction with the other, well established measures we can identify and describe the different brain activities recorded by scalp electrodes. The results indicate that the additional information provided by this measure could be used to further understand the nature and mechanisms of brain response to external stimuli.

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