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Chapter 8

TRAIT ANXIETY AND NEUROPHYSIOLOGY OF EXECUTIVE CONTROL IN THE STOP-SIGNAL PARADIGM

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ABSTRACT

The stop-signal paradigm (SSP) is an experimental method that allows research into brain mechanisms of executive control. In the SSP, subjects either react to a target stimulus or withdraw a reaction when a stop-signal appears after target. In this chapter we investigate neurophysiologic mechanisms underlying regulation of motor responses in healthy subjects given different trait anxiety (TA) levels. SSP experiment with simultaneous EEG recording was performed on 21 subjects. Event-related spectral perturbations (ERSP, Makeig, 1993) were analyzed in order to study brain oscillatory activity in different frequency bands. The data were partitioned into four experimental conditions: successful reaction (Go), successful withdraw (Stop), missed reaction (unsuccessful Go), and failure to stop (unsuccessful Stop).

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In the Go condition, power increase in delta and theta bands (1-7 Hz) was observed between 150-1000 ms after the target onset. Beta (15-25 Hz) desynchronization (power decrease) appeared before button-press (200-700 ms), while posterior alpha and central mu (7-14 Hz) desynchronization were revealed in 200-1200 ms, i.e. both before and after button-press. Successful reaction invited beta "rebound" (power increase, 1000-1600 ms). In the Stop condition additional low-frequency (1-7 Hz) power increase appeared 200-800 ms after the stop-signal onset. The low-frequency synchronization had maximal amplitude in the occipital-parietal region in Go condition, while in the Stop condition, the two maxima were observed – early in the posterior and late in the frontal area, implying co-occurrence of two different processes – activation of sensory areas, and active inhibition of prepared motor activity. Both types of mistakes were associated with prolonged low-frequency power increase (200-1200 ms).

Several components of EEG reactions depended on the TA level. Post-movement desynchronization in alpha and beta bands was significantly stronger in subjects with higher TA, while beta "rebound" had higher amplitude and shorter latency in subjects with lower anxiety. The amplitude of delta (1-4 Hz) synchronization was positively related with TA level in all experimental conditions. We have interpreted these results basing on attentional control theory (Eysenck et al., 2007). We hypothesize that subjects with high TA exert more attention to experimental task, which is reflected by higher amplitudes of alpha and beta desynchronization. On the contrary, subjects with low TA are less predisposed to elevated attentional control, and instead, they are more inclined to relax after a successfully completed task. The latter feature is mirrored in shorter latency and higher amplitude of "beta rebound" after successful performance of the trial in subjects with lower TA.

Keywords: Trait anxiety, Stop-signal paradigm, Brain oscillatory systems, EEG

INTRODUCTION

Executive control or executive functions is a human ability to regulate personal behavior based on the information received from environment and internal motivations. The executive functions are regarded as a "top down" mechanism in contrast to stimulus-driven and more or less automated "bottom up" processes and are related with a choice between several possible behavioral reactions. Some of these reactions are adequate to situations and should be activated, but other ones are inadequate and should be suppressed.

In this reason, one of the important features of executive control is the ability to inhibit responses that are inappropriate in the current context (Ramautar et al., 2006; Verbruggen & Logan, 2008; Verbruggen & Logan, 2009).

The stop-signal paradigm (SSP) (Lappin & Erikson, 1966) is one of widely applicable experimental methods for studying activation and inhibition of responses as the components of executive functions both in clinical patients (Alderson et al., 2007) and healthy subjects (Dimoska et al., 2006; Johnstone et al., 2007; Xue et al., 2008; Congdon et al., 2010). The stop signal task involves two concurrent tasks: a "Go" task and a "Stop" task. The "Go" task is usually a two choice reaction time (CRT) task, when subjects have to react on a presented stimulus (one of two "Go" stimuli with different responses associated with each of them) as fast as possible. But in some randomly chosen trials (typically 20–30%) the "Go" stimulus is followed by presentation of the "Stop-signal", which signals participants to inhibit their response on that trial. The horse-race model (Logan, et al., 1984; Band et al., 2003) provides theoretical interpretation of results obtained using the SSP. According to this model, two alternative neural processes compete after the stop-signal onset – activation and inhibition of response. If activation processes "win the race", subject responds, and otherwise the reaction is successfully inhibited. Besides the studying of response activation and inhibition mechanisms, the SSP could be useful for exploring processes, associated with another important component of executive control – the error monitoring, as design of SSP usually presumes that number of errors made by participants would be relatively large. These erroneous responses include pressing the wrong button, missing button-press (unsuccessful activation or "missed Go" condition) or, in opposite, pressing the button after Stop-signal onset ("missed Stop" or unsuccessful inhibition).

Brain processes providing executive control are a topic of numerous studies, as their violations can be found in many cases of neurological and psychiatric pathology. The majority of studies relating brain mechanisms of activation and inhibition of reactions with personality traits in healthy persons and pathological conditions in patients utilize such constructs as impulsivity and aggressiveness, which are directly associated with this control and specific overt modes of behavior. Particularly, executive function deficits were identified in several neuropsychiatric conditions associated with impulsivity, namely attention deficit hyperactivity disorder (ADHD) (Alderson et al., 2007), Parkinson's disease (Beste et al., 2009), and alcohol dependency (Li et al., 2009). Aggressive, suicidal and violent behaviors have been also associated with impulsive personality and problems in inhibiting responses

(Horn et al., 2003). At the same time, anxiety, which could be defined as an inclination to perceive environment as potentially dangerous and develop corresponding emotional and behavioral reactions, being a more "internal" and "covert" trait, is also closely interrelated with executive functions, though in a more complex way. There are several theories of anxiety; among neurobiologically oriented ones the Gray's BIS/BAS theory, M. Eysenck's theory of attentional control and Davidson's works concerning the "emotional side" of anxiety should be referred to here.

Neuropsychological theory of J. Gray postulates that three principal systems determine human personality: Behavioral Approach and Inhibition Systems (BAS and BIS) and Fight/Flight/Freeze System (FFFS). In the current version of this theory (Gray and McNaughton, 2000; McNaughton and Corr, 2004) anxiety as a state is proposed to be a result of conflict between BAS and FFFS in situations of uncertainty, then person could not predict whether the situation and/or his/her own activity would lead to positive or negative reinforcement. First is associated with expectation of reward or avoided punishment and activates BAS, while second – with expectation of punishment or non-reward and activates FFFS. The "collision" of these two systems activates BIS and its activity causes the state of anxiety. Furthermore, as the uncertainty is seen by most people as something negative, it would itself provoke anxiety, especially when it concerns the possibility of getting a reward or avoiding a punishment. The anxiety as a personality trait would be in this case the outcome of high excitability of BAS, FFFS and especially BIS (while, for example, high excitability of only BAS, with low excitability of BIS would lead to impulsive personality). The prefrontal cortex, amygdala and basal ganglia are proposed to be the neuroanatomical basis of BAS, while the orbital frontal cortex, septohippocampal system and Papez circuitry are described as such basis for BIS (Gray & McNaughton, 2000). Unfortunately, these descriptions are until now mostly speculative as there are yet no verified means to assess BIS or BAS activity/excitability to correlate it with activity of proposed structures. The popular BAS/BIS questionnaire of Carver and White (1994) is based on a previous version of Gray's theory (Gray, 1982) and could give misleading results, especially concerning BIS, as conceptualization of this system was quite changed in the current version. Nevertheless, the BAS/BIS approach is, to our mind, one of the most promising attempts to model neurobiological grounds of state and trait anxiety and to link them with psychological mechanisms and behavioral manifestations of this trait.

Theory of attentional control proposed by M. Eysenck et al. (2007) focuses rather on cognitive aspects of anxiety. According to this theory,

persons with high anxiety are inclined to suppress their behavior in threat-related and novel conditions in comparison with those with lower anxiety. However, behavioral suppression is compensated by increase of attention to external stimuli and to results of one's own reactions. Persons with a high anxiety level try to control situations as much as possible. It can be hypothesized that the differences in brain activity between higher and lower anxiety groups can be found in conditions in which they have to realize or stop an activity.

Finally, R.J. Davidson's approach to anxiety lies in line with other works of his group concerning affective styles and emotionality-related disorders and considers the neuroanatomical background of emotional processes associated with anxiety being based mainly on data on patients with anxiety disorders (Davidson et al., 1999). This approach links anxiety with the amygdala and prefrontal (especially its ventromedial and orbital zones) cortex (Davidson, 2002). Another important region in this circuitry is the anterior cingulate cortex (ACC), which is supposedly involved in control of anxious reactions depending on their relevance in the current situation (Nitschke et al., 2009). Correspondingly, if ACC activity indeed plays such an adaptive role, we could expect that in highly anxious but yet non-patient subjects reactivity in anxiety-provoking situations would be increased both in the amygdala and ACC. In their earlier works Davidson and coauthors (e.g. Wheeler et al., 1993, Sutton and Davidson, 1997, Davidson, 1998) observed pronounced cortical asymmetry in functional specialization of brain hemispheres with positive affect and approach being linked to activity of the left, while negative affect and withdrawal – of the right frontal cortex. However, though these affective dimensions are closely related with anxiety, links between brain asymmetry and this trait are not so clear, especially in non-patient samples. Another important approach of Davidson's group, apparently concerning the subject of this work, is so-called "affective chronometry". Using startle reflex potentiation as a measure of affective reactivity they (Larson et al., 2007) demonstrated that in anxious subjects the state of increased reactivity is much prolonged after offset of aversive (and, moreover, affectively charged with any valence) stimulus in anxious in comparison with normal control subjects. Although the task we used is scarcely provoking strong affects, we still could expect that EEG reactions of subjects with higher anxiety would be more prolonged especially in lower frequencies, which are associated with emotionality (Knyazev et al., 2008a; Knyazev et al., 2009), and particularly reactions related with errors (Knyazev et al., 2008b; Savostyanov et al., 2009).

In general, the three mentioned approaches are not contradicting but rather consider different aspects of anxiety. At the same time all these aspects are more or less directly associated with executive functions. Indeed, the activation/approach and inhibition/withdrawal are the core processes of executive control, which inevitably requires voluntary attention, and although theoretically it could be realized without emotions, in practice they usually accompany it. In the narrower context of this chapter these aspects are also present: the Stop-signal paradigm we used involves activation and inhibition of motor reactions, both requiring attentional control as well as does the error-monitoring, while the errors the participants made and their worry about general successfulness of task completion provide some level of emotionality. Summing up, we could expect that level of anxiety would exert influence on executive control processes, and that this influence would be mediated through neurophysiological systems and processes associated with both executive functions and anxiety.

Thus, the topic of this chapter is a research on the dependency between trait anxiety level and brain processes, which are related with such executive functions as activation and inhibition of motor responses and error monitoring. Studies of Spielberger (see e.g. Spielberger and Reheiser, 2004) demonstrated that trait anxiety is a stable psychological trait with low variability in long-term (several years) period. At the same time, being a stable trait, anxiety is far not always manifested as a state, at least in non-pathological conditions. Therefore, we propose that brain processes/systems associated with anxiety should have a "covert" nature of the same kind, manifesting only in certain situations. The most promising candidates for this role are, in our opinion, oscillatory systems of the brain. The concept of brain oscillatory systems (Stryker, 1989; Basar, 1998; von Stein and Samthein, 2000) is relatively recent in neuroscience. Being based mostly on electroencephalographic and systems electrophysiology findings (Basar et al., 2001; Eckhorn et al., 1988), it is also supported by converging evidences from fields of cellular electrophysiology, neuroanatomy and even functional genomics. Particularly, intrinsic oscillatory properties of neurons (see e.g. Silva et al., 1991) and abundance of small-scale neural circuits in the cerebral cortex (Steriade et al., 1996) provides the basis for "local" high-frequency oscillations, while long-distance cortico-cortical (Nunez, 1989) and cortico-subcortical (Miller, 1991, Steriade et al., 1993) loops are thought to be the substrate of lower-frequency oscillatory activity. And frequency-specific mechanism of some synaptic proteins' genes expression activation by oscillations of postsynaptic potential (Fields et al., 2005) could be the ground for development of such frequency-tuned neural

nets in ontogenesis. As postsynaptic potentials determine excitability of neurons, the oscillations of these potentials, either endogenous or exogenous, provide a core mechanism for temporal binding of synchronously oscillating neurons into one functional system, and at the same time for "filtering out" any non-synchronized (irrelevant) activity from other neural circuits. During the recent three decades much work has been carried out not only for understanding neurophysiological basis of oscillatory systems (Lopes da Silva, 1991), but also to elucidate their roles in organization of behavior, in emotional reactions and cognitive processes (see Wang, 2010 for expansive review in this field). The most widely used tool for the later purposes is the electroencephalography (EEG), therefore, oscillatory systems are commonly named by EEG rhythms of corresponding frequency (e.g. alpha-system, theta-system etc.). According to evolutionary based interpretations of brain oscillatory activity (Basar, 1998; Knyazev & Slobodskaya, 2003; Knyazev, 2007), oscillations in delta band (1-4 Hz) are associated with reward-related motivational structures, theta (4-8 Hz) with emotions and contextual memory, and alpha (8-12 Hz) with semantic memory, perception and attention. We should note that alpha rhythm since pioneering works on EEG (Berger, 1929, Adrian & Matthews, 1934) was for a long time considered just as a sign of general "inhibition" or "idling". However, recent findings suggest that the situation is not that simple. Being indeed the correlate of behaviorally inhibited states, increased alpha oscillations are rather providing the readiness for possible future physical or mental actions (Klimesch, 1999) and/or active selective inhibition of irrelevant perceptions and activities (Klimesch et al., 2007, Knyazev et al., 2006). Delta, theta and alpha rhythms are considered to be "global", providing long-range inter-regional connectivity, while higher frequency bands (beta, 13-30 Hz, and gamma, >30 Hz) are usually characterized as more "local" (Nunez, 1989). Changes in beta band activity have been associated with various functions such as visual attention (Wrobel, 2000), movement and motor imagery related changes (Neuper & Pfurtscheller, 2001), face recognition (Ozgoren et al., 2005), cognitive tasks (Ray and Cole, 1985), etc. Functions of gamma oscillations include perceptual binding (Singer et al., 1997), top-down attentional control (Gonsales Andino et al., 2005), short-term memory (Tallon-Baudry et al., 1996) and other complex cognitive functions (Kaiser & Lutzenberger, 2003). As the oscillatory activity appears to be the one of constitutive principles of brain functioning, the number of corresponding studies grows very rapidly. For the scope of this chapter important data were obtained in works interrelating brain oscillatory activity

with state and trait anxiety and with executive functions such as activation and inhibition of reactions and error monitoring.

EEG correlates of anxiety were found both for certain frequency bands of background EEG and event-related oscillatory reactions. Particularly, several studies demonstrated that anxiety level and BIS reactivity are correlated positively with alpha band spectral power, but negatively related with power of slow-wave (i.e. delta and theta) rhythms in background EEG records (Knyazev et al., 2002, 2003, 2004). Moreover, alpha power further increases (Knyazev et al., 2004, 2005) and theta power further decreases (Aftanas et al., 2003) in anxiogenic situations in highly anxious subjects. At the same time, reduction of anxiety level using anxiolythic drugs (Breimer et al., 1990; Hotz et al., 2000), autogenic training (Jacobs & Lubar, 1989) and other methods (Diego et al., 2004, Field et al., 1996) is accompanied by decrease of alpha power. Event-related oscillatory activity in their dependency from anxiety level corresponds with the notion about reciprocal relationships between background EEG power and phasic EEG reactions (Klimesch, 1999). Particularly, event-related desynchronization (ERD, *reduction* of spectral power) in alpha band was stronger in subjects with higher anxiety (Aftanas et al., 1996; Knyazev et al., 2006), while in slow-wave bands synchronization (*increase* of spectral power) was higher in individuals with high anxiety than in low-anxious persons (Knyazev et al., 2008a). We should note, however, that while findings for alpha-band are quite repeatable across studies, results for theta and delta bands are less consistent and seem to depend on other factors unrelated to anxiety. It has been shown that the emotion-related delta/theta synchronization, in particular, depends on a type of instruction presented to subjects before experiment (Knyazev et al., 2009). Also, this reaction differs among the people from different ethno-cultural groups (Wu et al., 2010).

Dependence between EEG parameters and anxiety was stronger expressed in the conditions of uncertainty, i.e. if individuals were not sure if there unpredictable events could not happen (Knyazev et al., 2005). It is possible to assume that anxiety should influence executive control especially strongly in those situations when the individuals make their decision with time or information lacking for event evaluation. EEG reactions related to activation or inhibition of behavior should show dependence on trait anxiety if individuals are compelled to make fast decisions in conditions of unguaranteed success.

Recently we have studied the brain oscillatory responses using analysis of EEG recorded in conditions of SSP (Levin et al., 2007; Knyazev, et al., 2008b; Savostyanov et al., 2009). We showed that “Go” response is accompanied by

spectral power decrease (desynchronization) in alpha and beta frequency range, whereas during response inhibition ("Stop" trials) the additional increase of spectral power (synchronization) in slow (delta and theta) bands was found. Successful ending of reaction was accompanied by synchronization in beta band or "beta rebound" as Pfurtscheller et al. (1996) named it. Summing up, analysis of EEG oscillations showed that in conditions of SSP there are three kinds of oscillatory changes relating with different frequency ranges– activation of motor response (alpha and beta band desynchronization), inhibition of prepared response (delta and theta band synchronization), and inhibition after successfully ended response (beta band synchronization).

Besides, the dependence between successful activation/inhibition of motor response on anxiety level was obtained. Similarly to the results mentioned above, the amplitude of alpha/beta desynchronization was stronger for higher-anxious subjects in Go and Stop conditions of SSP (Savostyanov et al., 2009). Earlier we didn't find significant correlations between anxiety and low-frequency reaction in SSP, but negative dependence on anxiety was revealed for beta rebound. In that study we didn't explore the dependence between brain oscillatory reactions and anxiety in conditions of unsuccessful responses. Meanwhile, it is plausible to assume that the situation of making an error would, from one side, evoke negative emotional reactions, and from the other side, would force the participant to allocate more voluntary attention to the task completion. Both these factors could be supposed to increase the effects of anxiety. Therefore in this chapter we would like to compare the anxiety influence on successful and unsuccessful regulation of behavior in Go and Stop conditions.

Also, an important topic of this chapter is topological distribution of anxiety-related processes in the different conditions of SSP. The inhibitory control is typically associated with the prefrontal cortex (Ridderinkhof, et al., 2004; Forstmann et al., 2008). However, as soon as inhibitory control is based on analysis of external signals and participates in regulation of motor responses, this process cannot be limited by only the frontal cortex. Specific and nonspecific sensory areas, motor and associative cortices are also involved in response regulation. According to Fuster (2006), behavioral control is provided by long reciprocal cortico-cortical connections between posterior and frontal networks. These connections support the dynamics of the perception-action cycle in sequential behavior. It is possible to assume that dependence of various SSP components on anxiety will have a topological specificity, i.e. this dependence will be related either with anterior or with posterior areas. However, according to other hypothesis the anxiety level is modulated by

activity of deep subcortical structures (Gray and McNaughton, 2000, McNaughton and Corr, 2004). In this reason, the effect of anxiety should not have topological specificity in EEG reactions. Here we would like to check up hypotheses about topological distinctions in reactions related with activation, inhibition or estimation of success of motor responses in the persons with different anxiety levels. Therefore we have made a detailed, multiple-factor analysis of EEG reactions in different time-frequency intervals in the SSP conditions.

Thus, in this chapter we have compared brain reactions in four (successful Go, successful Stop, unsuccessful Go and unsuccessful Stop) conditions of the SSP in the individuals with different levels of trait anxiety. The analysis of topological distribution of reaction on a scalp surface has been made for the time-frequency intervals reflecting different aspects of regulation of the motor responses.

METHODS

Subjects: EEG data were obtained in 21 healthy right-handed subjects (15 males), aged 25-33 years old (with average age 26±3.0 years). All participants gave informed consent before enrollment into the study. The study was approved by the ethics committee in accordance with the Declaration of Helsinki. The Chinese version of the State Trait Anxiety Inventory (Shek, 1993; Spielberger et al., 1970) (cSTAI) was used for measuring the trait anxiety level. A median split was applied to divide subjects into the relatively high- and low-anxiety groups. The cSTAI has been validated and its psychometric characteristics can be found in the study of Shek (1993). The face validity of cSTAI was doubly examined by a senior psychometrician, and we also interviewed several subjects who voluntarily took the cSTAI. The final cSTAI used in the experiment had minor corrections on the Chinese translations of the original STAI based on the interview results.

*Procedure:*_The experimental procedure was described in the paper of Savostyanov et al., (2009). During the experiment, each participant was seated comfortably in a chair with eyes open in a sound-insulated dimly lit chamber. Visual stimuli were presented via a 24.4 x 18.3 cm monitor located 60 cm in front of the subject. The procedure was designed in a form of EEG-interfaced interactive computer game, during which the subject was motivated to get the maximum score. After 15 minutes of background EEG recording, participants performed 30 training trials of a choice reaction task in which they had to

press the left or right button after presentation of one of two pictures (a deer or a tank). The pictures (15 x 10 cm) were presented at the center of the computer monitor for 500 ms. Subjects should choose a weapon to strike a target (a rifle or an antitank rocket launcher) and shoot. Subsequently in 130 trials of the stop-signal task, they had to refrain from pressing any button if a stop-signal (red bar, 3 x 2 cm, 250 ms in duration) was presented after the picture. In the pre-task instruction, the participants were told to respond as fast and accurately as possible. The deer and tank alternated randomly and the inter-stimulus interval randomly varied between 3.5 and 5.5 seconds. The stop-signal was randomly presented in 30 (approximately 23%) trials. The interval between onset of the picture and stop-signal was 250 ms. If response was correct, the subject received additional points, and a deduction of points was applied to mistakes and to insufficient speed of reaction. Thus, four different conditions (successful Go and Stop; unsuccessful Go and Stop) were considered in the experimental procedure. EEGs were registered simultaneously with the game. The target and stop-signal presentations along with the subject's reactions were automatically labeled during EEG recording.

EEGs were recorded in 21 subjects using 132-channels (122 EEG, VEOG, HEOG, EKG, EMG, and 6 face muscle channels) via Ag/AgCl electrodes. The EEG electrodes were placed on 122 head sites according to the extended International 10-10 system (Oostenveld and Praamstra, 2001) and referred to Cz with ground at FzA. The monopolar reference scheme was applied for the montage of electrodes. The Quik-Cap128 NSL was used for electrode fixation. The electrode resistance was maintained below 5 κΩ. The signals were amplified using "Neuroscan (USA)" amplifiers, with 0.1-100 Hz analog bandpass and digitized at 1000 Hz.

Data processing: Event-related spectral perturbations (ERSP) were calculated using the *timef* function in the EEGLAB toolbox (http://sccn.ucsd.edu/eeglab/) (Delorme and Makeig, 2004). The ERSP shows mean log event-locked deviations from baseline-mean power at each frequency. For time-frequency representation of EEG data, the wavelet transformation using the *Morlet* waveform as a mother wavelet was chosen.

Ongoing EEG from 1.0 sec before to 2.5 sec after the target stimulus onset was selected for data analyses. EEG from 1.0 to 0.25 sec before the target stimulus onset was used for baseline-correction. In other words, the "baseline" interval did not contain any task-related activity. EEG was preliminarily band-pass filtered in 1-50 Hz using elliptic filters. Re-reference to average reference and baseline adjustment procedures were performed during data preprocessing using *pop_reref* and *pop_rmbase* EEGlab functions (Delorme and Makeig,

2004). In EEGlab re-reference to average reference is realized as follows: first, the activity in the original reference channels is estimated as minus (sum of all electrodes activity divided by number of electrodes) and, second, this activity is added to each channel. Baseline for each channel and each epoch was removed by subtracting the mean EEG value in the baseline time range from epoch EEG. Some trials (1-7 per person) with non-stereotyped artifacts were removed after visual inspection of EEG records. The presence of eye-blink or eye-movement artifacts was not a criterion for rejection of the episode.

The 80-99 (mean 91.6; SD 8.9) EEG trials were selected in successful Go condition, 17-30 (mean 25.4; SD 3.8) EEG trials in successful Stop condition, 0-33 (mean 4.2; SD 7.5) in unsuccessful Go and 0-10 (mean 2.3; SD 2.5) in unsuccessful Stop conditions. A particular EEG-channel (mean 2.9; SD 1.5) was reconstructed by means of the *EEG-interp* function, if there were irremovable artifacts.

Independent component analysis (ICA) was used for correcting eye blink and eye-movement artifacts (Makeig et al., 1996; Hyvärinen et al., 2010). ICA transforms EEG channels data into "EEG components" representation (with number of components equal to number of channels), which are supposed to reflect activity of sources independent from one another. The components' weights were computed individually for each subject. The components corresponding to eye artifacts were identified by visual inspection of component sets together with electrooculogram records. For each person, 5-15 components were identified as "artifactual". These components had the highest weights in the electrooculographic channels and contained spectral power changes in time intervals corresponding to eye activity in oculogram. After removal of artifactual components EEG for each subject was transformed back to standard representation, but now EEG channels were "cleaned" from electrooculographic artifacts. This approach allows avoiding rejection of trials contaminated by eye-movements and blinks, which is especially important in studies where activity associated with relatively rare events, such as missed stop trials, is to be explored. Then, we computed ERSP-indices separately for each EEG-channel and each subject.

The averaged ERSP patterns were firstly investigated separately for different subject groups without direct statistical comparison between groups and brain areas. Given a subject group, ERSPs were averaged across subjects and all channels. The random permutation method (bootstrap) with $p<0.01$ significance level was applied in the statistical analysis of ERSPs separately for each experimental condition. Here we used the bootstrap statistical method realized in EEGLAB toolbox (*statcond* function) and based on random data re-

sampling. In this method, a surrogate data distribution is constructed by selecting spectral estimates from randomly selected samples and then averaging these. Applying this process 500 times produces a surrogate data distribution where the specified percentiles are then taken as significance thresholds (Delorme and Makeig, 2004). This method was used to visualize significant deviations from baseline random fluctuations that were observed after stimulus presentation, such that non-significant features of the output plots were zeroed out (i.e., plotted in green).

We partitioned 122 EEG scalp channels into 11 regions: the left (10 channels), midline (14 channels), and right frontal (10 channels); left (13 channels), and right temporal (13 channels); and the left (9 channels), midline (14 channels) and right central (9 channels), left (9 channels), midline (12 channels), and right occipital-parietal (9 channels). ERSPs were averaged across channels within each region for each individual subject. The Kolmogorov-Smirnov test did not show significant deviations from normal distribution in our data. Thus, conventional ANOVA was applied for testing between-condition and between-group differences. The Greenhouse–Geisser correction for the sphericity assumption violation was used whenever necessary. Repeated measures ANOVA was applied to testing the main effects of regions (11 levels) and groups (high vs. low-anxious), as well as the interaction effect between regions, groups and conditions.

RESULTS

Behavioral data: The average reaction time in the Go condition was 697.8 ms (SD 68) for the whole group. In high-anxious subjects the average reaction time was 672.7 ms (SD 87.2) and in low-anxious subjects – 716.6 ms (47.7). The inter-group differences in reaction time between high and low-anxious subjects were insignificant. The mean number of missed button-presses (unsuccessful Go reaction) was 4.2 (SD 7.5) for whole group, 5.2 (SD 10.5) in high-anxious subjects and 3.4 (SD 4.6) in low-anxious subjects. The mean number of incorrect button-presses after stop-signal onset (unsuccessful Stop reaction) was 2.3 (SD 2.5) for whole group, 2.2 (SD 3.1) in high-anxious subjects and 2.3 (SD 2.1) in low-anxious subjects. The inter-group differences in the number of behavioral errors were statistically insignificant for all experimental conditions. Thus, the significant differences between high and low-anxious subjects were obtained for no behavioral parameter.

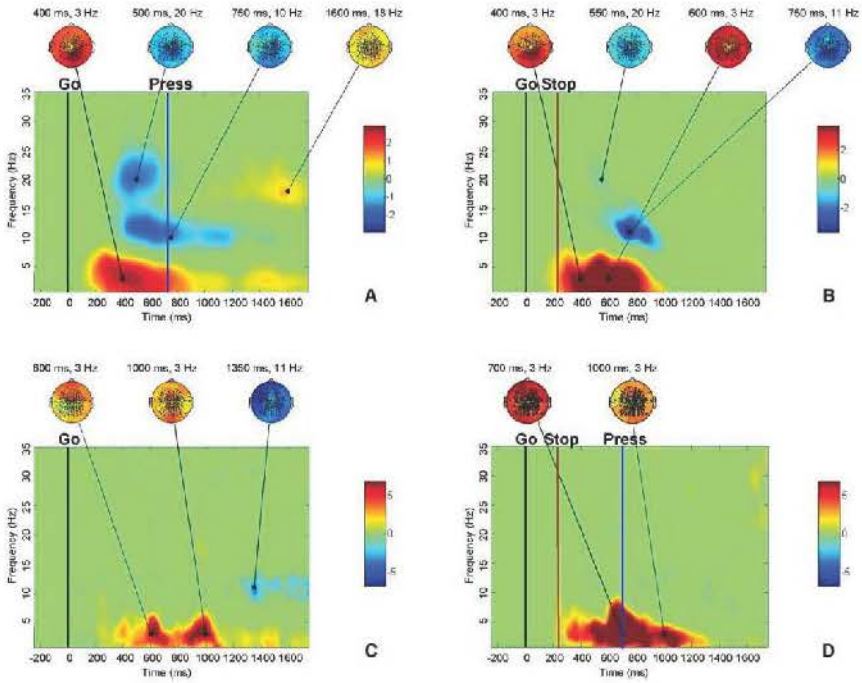

Figure 1. ERSP plots for successful Go (A), successful Stop (B), unsuccessful Go (C) and unseccessful Stop (D) conditions. ERSP values are averaged for whole group. Warm colors mean increase of power; cold colors – decrease. Black vertical line (zero time point) represents time of target stimulus onset. Blue vertical line corresponds to button-press and red vertical line – to Stop-signal onset. $P < 0.01$.

EEG patterns for successful and unsuccessful conditions: EEG patterns in different experimental conditions were firstly investigated without reference to the anxiety level. In the successful Go condition EEG spectral power was increased in 1-7 Hz band during 150-1000 ms after target stimulus onset (See Fig. 1a). A decrease of spectral power was found in EEG between approximately 250-1200 ms after the target stimulus onset. During movement preparation, this reaction covered a wide frequency range in 8-25 Hz corresponding to boundaries of alpha and beta rhythms. After the button press it was sustained at 7-14 Hz frequency range. Increases in spectral power were observed in a frequency range 12-25 Hz during 1000-1600 ms after target stimulus onset (i.e. about 300 – 900 ms after the button press). In the successful Stop condition, significant desynchronization was observed in the frequency range 7-25 Hz during approximately 300-1000 ms after the target stimulus onset (See Fig. 1b). Besides, significant synchronization in the

frequency range of 1-7 Hz was found during 150-1000 ms after the target stimulus onset. The amplitude of alpha/beta power decrease in successful Stop condition was approximately the same as in Go condition, whereas the amplitude of slow-wave synchronization was essentially higher for Stop than that for Go condition. In both unsuccessful conditions only slow-wave synchronization was obtained in 1-7 Hz during 150-1400 ms after target stimulus onset (See Fig. 1c,d). No alpha/beta desynchronization or high-frequency synchronization was found in unsuccessful conditions.

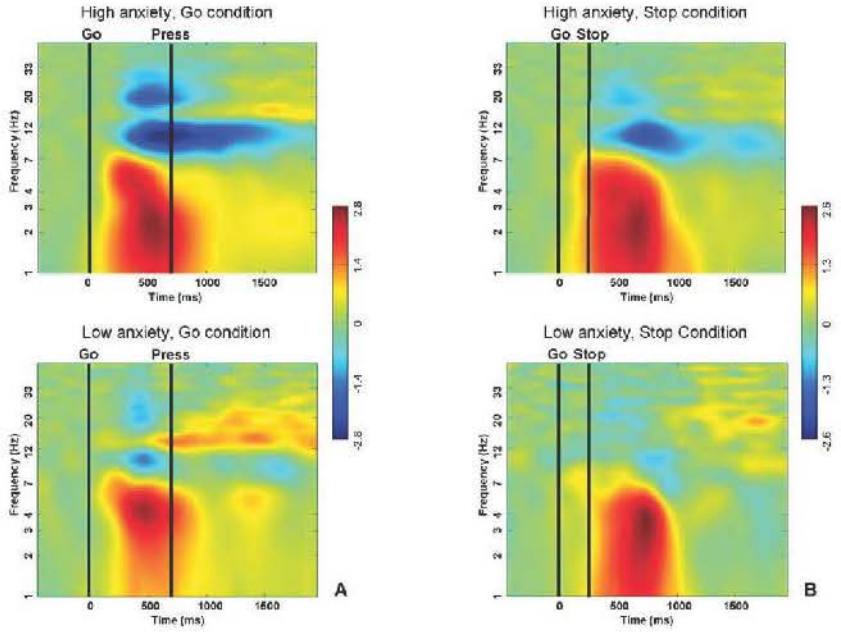

Figure 2. ERSP plots for successful Go (A) and successful Stop (B) conditions in high and low anxiety groups. Upper panels represent high anxiety group, and lower panels – low anxiety group. Warm colors mean increase of power; cold colors – decrease. $P < 0.01$.

For additional control of our results we compared ERSP plots, which had been prepared separately for relatively high and low anxious persons in the successful Go and Stop conditions. In both of these conditions, subjects with higher anxiety showed a higher level of alpha/beta desynchronization (See Fig. 2a,b). The amplitude of slow-wave synchronization was also higher in high-anxious subjects in comparison with low-anxious ones, but those differences were obtained only in delta (1-4 Hz) and not in theta (4-7 Hz) frequency band.

The amplitude of high-frequency synchronization (12-25 Hz) was stronger in low-anxious subjects in comparison with high-anxious ones in both successful conditions. Besides, the latency of this reaction in Go condition was shorter for low-anxious subjects than that in high-anxious subjects (about 600 ms vs about 1400 ms), whereas such differences were not seen in the latencies of Stop condition (See Fig. 2a). No differences between anxiety groups were found by comparison of ERSP plots in unsuccessful conditions.

The ANOVA results: Fourteen time-frequency intervals of interest (1-7 Hz in 200-400 ms; 1-4 Hz in 200-400 ms; 4-7 Hz in 200-400 ms; 1-7 Hz in 400-800 ms; 1-4 Hz in 400-800 Hz; 7-12 Hz in 400-700 ms; 12-16 Hz in 400-700 ms; 16-20 Hz in 400-700 ms; 20-25 Hz in 400-700 ms; 7-12 Hz in 700-1000 ms; 12-16 Hz in 700-1000 ms; 16-20 Hz in 700-1000 ms; 20-25 Hz in 700-1000 ms; 12-25 Hz in 1000-1500 ms) were chosen basing on analysis of ERSP plots described above and further analyzed for all four experimental conditions using repeated measures ANOVA. The significant main effects and interactions obtained in this analysis for different intervals are summarized in Table 1.

In 1-7 Hz and 200-400 ms time-frequency interval main effect of region, $F(10, 670) = 4.38$, $p < 0.00001$, and two-way interaction region × condition, $F(30, 670) = 2.61$, $p < 0.00001$, were highly significant. Planned comparisons showed that the amplitude of synchronization was the strongest in all occipital-parietal areas. These areas significantly ($p = 0.0032$) differed from all other cortical regions, whereas the differences between other regions were insignificant. The main effect of condition was insignificant. In the same time-frequency interval repeated measures ANOVA was made separately for two successful and two unsuccessful conditions. For successful conditions the effect of region, $F(10, 380) = 20.39$, $p < 0.00001$, was significant, but two-way interaction region × condition, $F(10, 380) = 1.14$, $p = 0.33$, was insignificant. Oppositely, for unsuccessful conditions the effect of region, $F(10, 290) = 0.88$, $p = 0.55$, was insignificant, but two-way interaction region × condition, $F(10, 290) = 3.20$, $p = 0.0006$, was significant. Thus, the amplitude of early low-frequency synchronization didn't differ between successful Go and Stop conditions. In these conditions the synchronization had a maximum in occipital-parietal regions. However, the between-region differences were not obtained for all unsuccessful conditions, but posterior synchronization in the unsuccessful Stop condition was stronger in comparison with unsuccessful Go condition. For whole 1-7 Hz frequency band the effect of anxiety was insignificant. However, the main effect of anxiety, $F(1, 67) = 6.86$, $p = 0.011$, was significant for synchronization in delta (1-4 Hz) band, but was

insignificant, $F(1, 67) = 0.144$, $p = 0.71$, for theta (4-7 Hz) band. The amplitude of delta (not theta) synchronization was stronger for high- than that for low-anxious persons in all four conditions. At the same time, the effects of region and conditions for delta and theta bands were similar to the effect for 1-7 Hz band.

In 1-7 Hz and 400-800 ms time-frequency interval main effect of condition, $F(3, 67) = 5.52$, $p = 0.0019$ was significant. Post-hoc comparisons showed that both the Go conditions significantly differed from both the Stop conditions ($p < 0.05$). The differences between successful and unsuccessful Go conditions ($p > 0.7$), and also between successful and unsuccessful Stop conditions ($p > 0.22$) were insignificant. The main effect of region, $F(10, 670) = 4.46$, $p < 0.00001$, was highly significant. To further elucidate the effect of region, ANOVA were run separately in successful and unsuccessful conditions. In both successful conditions the effect of region, $F(10, 380) = 13,85$, $p < 0.00001$ was significant. Planned comparisons show that the amplitude of synchronization was minimal in all central areas. These areas significantly ($p = 0.0012$) differed from all other cortical regions, whereas the differences between other regions were insignificant. The cortical topology of later synchronization did not differ between successful Go and successful Stop conditions. In both unsuccessful conditions the effect of region, $F(10, 290) = 1.09$, $p = 0.37$, was insignificant. Similar to 200-400 ms interval, for later synchronization the main effect of anxiety was insignificant for 1-7 Hz and 4-7 Hz bands, but it was significant for delta (1-4 Hz) band, $F(1, 67) = 5.00$, $p = 0.029$. In this interval (1-4 Hz, 400-800 ms) the amplitude of delta synchronization was stronger for high- than for low-anxious persons in all four conditions.

Thus, for synchronization in 1-7 Hz the differences between conditions were significant only in later (400-800 ms), but not in earlier (200-400 ms) time intervals. The amplitude of later synchronization was stronger for both Stop conditions in comparison with both Go conditions. The successful and unsuccessful conditions didn't differ in amplitude of reaction, but differed in its cortical distribution. In earlier time intervals successful reactions are maximum in occipital-parietal areas, whereas in later intervals two regions with strong amplitude (anterior and posterior, but not central) were found. The between-region differences were insignificant in all unsuccessful conditions in both time intervals. Also, the differences between anxiety groups were significant for all conditions and all time intervals, but only in delta frequency band. The amplitude of delta synchronization always was stronger for high- than that for low-anxious subjects.

For pre-movement (250-700 ms) time interval in alpha (7-12 Hz), beta1 (12-16 Hz), and beta2 (16-20 Hz) frequency bands only main effect of region was significant (See Table 1). The amplitude of desynchronization was the strongest in central cortical areas for all bands. The effects of condition and anxiety in these time-frequency intervals were insignificant. The influence of anxiety was obtained by post-hoc comparisons only for beta1 desynchroniza-tion in successful (both Go and Stop) conditions and only in central cortical areas. In these areas the amplitude of reaction was significantly ($p < 0.01$) stronger for high- than that for low-anxious persons. For beta3 (20-25 Hz) band the effects of condition $F(3, 67) = 12.10$, $p < 0.00001$, and anxiety, $F(1, 67) = 4.10$, $p = 0.047$, were significant. The beta3 desynchroniza-tion was stronger for unsuccessful, than that for successful conditions. Also, the amplitude of beta3 desynchronization was stronger in high-anxious persons for all regions and conditions with between-group differences being stronger for successful, than for unsuccessful conditions.

In post-movement (700-1000 ms) time interval, similar to pre-movement desynchronization, only main effect of region was significant for alpha, beta1 and beta2 bands and the cortical distribution of reaction amplitude also was similar to that in pre-movement time interval. Then, ANOVA were run separately in successful and unsuccessful conditions. The significant effects of region, anxiety and condition were obtained only in successful conditions, whereas all effects and interactions in unsuccessful conditions were insignificant or marginal. In alpha band the effect of region, $F(10, 380) = 3.43$, $p < 0.00001$, was highly significant. In both successful conditions the maximal alpha desynchronization was obtained in central areas. Also, the effect of anxiety, $F(1, 38) = 8.28$, $p = 0.007$, was significant. Desynchronization was stronger in high- in comparison with low-anxious subjects. For beta1 and beta2 bands the effects of region, $F(10, 380) = 7.34$, $p < 0.00001$ (beta1), $F(10, 380) = 5.42$, $p < 0.00001$ (beta2), condition, $F(1, 38) = 5.63$, $p = 0.023$ (beta1), $F(1, 38) = 3.8$, $p = 0.058$ (beta2), and anxiety, $F(1, 38) = 7.66$, $p = 0.0087$ (beta1), $F(1, 38) = 3.80$, $p = 0.052$ (beta2) were significant in successful conditions. The amplitude of beta1 and beta2 desynchronization was stronger for Stop, than that for Go condition, and also stronger in high-anxious subjects in comparison with low-anxious ones. In beta3 (20-25 Hz) frequency band the main effects of region, $F(10, 670) = 4.26$, $p = 0.00001$, and anxiety, $F(1, 67) = 5.27$, $p = 0.025$, were significant in all four experimental conditions. The desynchronization in this band also was stronger in high-anxious group. Besides, the effect of condition, $F(3, 67) = 8.70$, $p = 00006$, was significant.

The desynchronization was stronger for unsuccessful, that that for successful conditions.

Table 1. The ANOVA results

Time-frequency interval	Effect	G-G Epsilon	Degrees of Freedom	MS	F	p
1-7 Hz, 200-400 ms	region	0.34	3.4	228.1	4.38	0.00001
	region* condition	0.34	10.2	228.1	2.61	0.00001
1-4 Hz, 200-400 ms	anxiety		1	67	6.86	0.011
1-7 Hz, 400-800 ms	condition		3	67	5.52	0.002
	region	0.43	4.3	289.7	4.46	0.00001
1-4 Hz, 400-800 ms	anxiety		1	67	5.00	0.029
7-12 Hz, 250-700 ms	region	0.49	5.0	331.5	2.63	0.004
12-16 Hz, 250-700 ms	region	0.49	5.0	325.1	2.19	0.017
16-20 Hz, 250-700 ms	region	0.52	5.2	350.4	1.86	0.048
20-25 Hz, 250-700 ms	anxiety		1	67	4.10	0.047
	condition		3	67	12.10	0.00001
7-12 Hz, 700-1000 ms, successful conditions	anxiety		1	38	8.28	0.007
	region	0.46	4.6	175.4	5.88	0.00001
12-16 Hz, 700-1000 ms, successful conditions	anxiety		1	38	7.66	0.009
	condition		1	38	5.63	0.023
	region	0.44	4.4	168.5	7.34	0.00001
16-20 Hz, 700-1000 ms, successful conditions	anxiety		1	38	3.8	0.052
	condition		1	38	3.8	0.058
	region	0.48	4.8	183.0	5.42	0.00001
20-25 Hz, 700-1000 ms	anxiety		1	67	5.27	0.025
	condition		3	67	8.70	0.00006
	region	0.44	4.4	297.8	4.26	0.00001
12-25 Hz, 1000-1500 ms	anxiety		1	67	9.68	0.0027
	condition		3	67	10.98	0.00001
	region	0.42	4.2	278.3	1.99	0.032
	region* condition	0.42	12.5	278.3	1.77	0.007

Thus, both pre- and post-movement desynchronization had maximal amplitude in central cortical areas. The effects of anxiety were significant in alpha, beta1 and beta2 bands only in post-movement interval and only in successful conditions (both Go and Stop). In beta3 band the effects of anxiety were significant in all conditions and intervals. The amplitude of desynchronization was in all these cases stronger for high-, than that for low-anxious subjects. The two-way interactions anxiety*region were insignificant or only marginal.

For 12-25 Hz and 1000-1500 ms time-frequency interval main effects of both anxiety, $F(1, 67) = 9.68$, $p = 0.003$, and condition, $F(3, 67) = 10.98$, $p < 0.00001$, were significant. In this interval the positive values of ERSPs (synchronization) were obtained for successful conditions (both successful Go and Stop), but negative values (desynchronization) were found for both unsuccessful conditions. Post-hoc comparisons (Fisher LSD) show that each of the successful conditions differed from each of the unsuccessful conditions ($p < 0.0001$), but the conditions with same success did not differ from each other ($p > 0.8$). The amplitude of synchronization was stronger for low- than that for high-anxious persons. Post-hoc comparisons show that the anxiety groups significantly differed only for successful conditions ($p < 0.01$), but for unsuccessful conditions the between-group differences were insignificant ($p > 0.1$). Besides, the main effect of region, $F(10, 670) = 1.99$, $p = 0.03$, and two-way interaction region × condition, $F(30, 670) = 1.77$, $p = 0.007$, were significant. Planned comparisons show that between-region differences were insignificant ($p > 0.8$) for all successful conditions. For all unsuccessful conditions desynchronization in this time-frequency interval was significantly stronger in central regions in comparison with frontal ($p = 0.003$) and occipital-parietal ($p = 0.040$) regions.

DISCUSSION

This chapter was aimed at finding dependencies between brain processes underlying executive control and the level of trait anxiety in healthy subjects. According to the neuropsychological model of Gray and McNaughton (Gray & McNaughton, 2000, McNaughton & Corr, 2004) trait anxiety is associated with inclination to activation of Behavioral Inhibition System, which leads to inhibition of ongoing behavioral patterns if information about environment is insufficient and a person is unable to predict results of own behavior. In our experiment subjects had to choose appropriate reactions to presented stimuli of an unpredictable type, but in some cases (also unpredictable) Stop-signal forced them to stop a prepared or already started reaction. Based on Gray & McNaughton's model we proposed that these uncertain conditions would enhance activity of BIS and strengthen manifestations of neurophysiological differences between subjects with different levels of trait anxiety. Analysis of EEG recorded in these conditions could improve understanding of mechanisms underlying these differences.

Behavioral control is associated with execution of several interacting brain functions, including sensory, motor and cognitive components. We supposed that different brain processes coupled with behavioral control are differentially related with trait anxiety. Some of them could be enhanced in persons with high trait anxiety, while others are manifested more intensively in low-anxious persons. Testing of this hypothesis requires specific measures, sensitive to relatively short-term changes of activity of different functional systems of the brain. Such measures are provided by the concept of brain oscillatory systems (Basar, 1998; Knyazev, 2007). According to this concept different functional activities of the brain are mirrored in EEG oscillations of different frequencies and temporal changes of these oscillations' spectral power could be interpreted as signs of execution of corresponding functions. Besides, EEG is characterized by spatial distribution of reactions. Before considering differences in executive control mechanisms related to anxiety level we would interpret their common patterns observed in this chapter in terms of the above-mentioned characteristics and their functional meanings.

Slow-wave synchronization with maximal amplitude in occipital-parietal region in the Go condition could be interpreted as a sign of activation of the visual cortex after target stimulus appearance (Basar, 1998). Widespread desynchronization in alpha/beta band is presumably related to task non-specific attention concentration, typical to all situations when task-related stimuli are presented (Klimesh et al., 2007). Besides, in the Go condition this desynchronization is more pronounced in the central region (desynchronize-tion of mu-rhythm), mirroring activation of motor areas, associated with hand movement regulation (Neuper and Pfurtscheller, 1999). The power increase in upper beta band after successfully ended motor task was earlier interpreted as so-called "beta rebound" (Pfurtscheller et al., 1996), i.e. brain reaction induced by suppression of task-related activity and relaxation. Our results show that the alpha/beta desynchronization and "beta rebound" is also present in the Stop condition in the same time-frequency intervals and cortical areas as in the Go condition. The amplitude of these reactions was lower than in the Go condition, but statistical significance of this difference was weak. This result could partly be correlated with data of Neuper and Pfurtscheller (1999) on motor imagery, considering that reaction, being not completed due to Stop-signal appearance, was still "imagined". However, if higher frequency (alpha and beta) reactions allow such "imaginary" interpretation, the lower-frequency (delta and theta) Stop-related activity could not be explained this way.

In general, amplitude of low-frequency synchronization was significantly larger in the Stop than in the Go condition. Earlier (Savostyanov et al., 2009)

we interpreted this larger synchronization only as a sign of inhibition of prepared movement after Stop-signal onset. However, analysis of its cortical distribution showed that the situation is more complex. Indeed, quite predictably, in 200 – 400 ms after target stimulus onset differences between conditions were insignificant and maximal synchronization was observed in occipital-parietal regions. But in later interval (400 – 800 ms after target stimulus) differences between the Go and Stop became significant with maximal Stop-related synchronization in parietal, frontal and temporal, but not in the central region. Accordingly, low-frequency synchronization in the Stop condition could be explained as a result of two processes, overlapping in time: sensory activation by "bottom-up" mechanisms and "top-down" inhibition. Though they utilize the same frequency band, spatial distribution and timing of these processes differ. Bottom-up sensory activation is observed in 200 – 1000 ms after target stimulus with maximum in occipital-parietal region. It is stronger in successful (both Go and Stop) trials than in trials with erroneous reaction/non-reaction, but do not differentiate between Go and Stop conditions. We suggest that it is initially generated in the visual cortex and then spreads to more anterior areas. Top-down inhibitory reaction is observed after Stop-signal onset (400 – 1000 ms after target stimulus) with maximum in frontal and temporal areas. It is stronger in Stop (both successful and erroneous) than in Go conditions and spreads from frontal to more posterior regions. Noteworthy, in the Stop condition these two reactions are observed simultaneously in the same frequency band, but in different cortical regions.

In unsuccessful trials only changes in slow-wave activity were revealed as significant compared with the baseline by bootstrap statistics. Having the same amplitude and topography as in successful trials, slow-save synchronization was significantly prolonged both in erroneously missed Go and in unsuccessful Stop. This prolonged reaction was described as error-related activity (Luu et al., 2004; Trujillo and Allen, 2007). However when we compared successful vs. unsuccessful trials, the most significant differences were found for "beta rebound". This reaction occurs only when a result (either button-press or its inhibition) is evaluated as "successful", but it is absent in case of any kind of mistakes. Therefore, "beta rebound" is related not only to motor relaxation as it was suggested earlier (Chen, 1998), but also with self-rating of personal behavior as successful or unsuccessful.

In general, ERSP patterns for all experimental conditions could be explained using the horse-race model proposed by Logan and coauthors (1984, 2003). This model suggests that in SSP a competition exists in the brain between processes regulating behavioral activation and inhibition, and motor

response or its cancellation occurs depending on which of the processes "wins the race". Processes related to activation were mirrored in early posterior low-frequency synchronization and pre- and post-movement alpha and beta desynchronization, without significant differences between Go and Stop trials. Inhibition in Stop trials (both successful and unsuccessful) was associated with late low-frequency synchronization in the frontal region. In successful trials these reactions had clear spatial maximums in functionally relevant regions, while unsuccessful trials were characterized by weaker and more widespread early reactions, but prolonged late low-frequency synchronization. In addition to processes described in the horse-race model (Logan et al, 1984, Band et al., 2003), we observed brain activity, specifically associated with self-rating of own behavior as successful or unsuccessful. "Beta rebound" could not be associated either with activation, or inhibition of response, as it occurred post factum and depends on successfulness of reaction, but not on type of trial.

Significant main effect of trait anxiety was observed in delta (1 – 4 Hz), but not in theta (4 – 7 Hz) frequency band. Both early and late synchronizations were significantly stronger in high- than in low-anxious subjects for all four experimental conditions (Go and Stop, successful and unsuccessful). That is, anxiety-related differences in amplitude of low-frequency synchronization were not specifically associated with either activation or inhibition of motor reaction, or successful or erroneous responses. Also, in successful, but not in unsuccessful conditions (both Go and Stop) desynchronization in alpha and beta bands (7-25 Hz) was significantly stronger in subjects with high than with low trait anxiety level. On the contrary, amplitude of "beta rebound" in successful (and again, not in unsuccessful) conditions was significantly stronger in low-anxious subjects. Besides, as could be seen in ERSP plots (Fig. 2), differences between groups with high and low anxiety are related with not only amplitude, but also timing of reactions. Desynchronization in alpha and beta bands lasts 200 – 300 ms more in high- than in low-anxious subjects, while "beta rebound" occurs in later group about 800 ms earlier than in first. Almost all of the above-mentioned anxiety-related differences had not spatial specificity: interactions Anxiety * Region were insignificant for all reactions.

Therefore, anxiety-related differences could be observed in SSP in components of reaction, related to its activation, inhibition and evaluation. These differences could interact with successfulness of reaction, but not depend on type of trial (Go or Stop). Also, they have "global" properties, and are not specific for any local brain area. This result lies in line with the hypothesis of global brain oscillatory systems integrating separate structures

into a united functional system and regulating all cortical regions (Basar, 1998; Knyazev, 2007). Although initial sources of these oscillations could be localized in some brain regions, they nonetheless spread to wide areas and could influence several brain functions.

The amplitude of slow-wave synchronization (related to both early sensory activation and late top-down inhibition) was positively correlated with trait anxiety in all experimental situations. We could interpret this fact based on the idea that slow-wave oscillations are modulated by (or even originate from) non-specific activity of the thalamus (Aron and Poldrack, 2006). This activity is associated with attention switching and choice between possible behavioral reactions (McFarland and Haber, 2002; Herrero et al., 2002; Li et al., 2008). Accordingly, although the thalamic activity was not directly measured, we could speculate that trait anxiety level could be correlated with non-specific activity of thalamus. This hypothesis could be considered in the future using neuroimaging techniques and/or additional EEG studies.

Background spectral power of alpha- and beta-band activity in most of studies was positively correlated with trait anxiety, as well as did amplitudes of event-related desynchronization in various experimental paradigms, such as face recognition, motor and linguistic tasks (Wu et al., 2010). Earlier (Savostyanov, 2009) we interpreted this dependency based on M. Eysenck's theory of attentional control (Eysenck et al., 2007). According to this theory subjects with high trait anxiety are inclined to enhance their control over a situation using additional allocation of attention to external stimuli. Oscillations in alpha and beta band could be interpreted as a sign of readiness/preparation to possible nearest future tasks (Klimesch, et al., 2007). In conditions of behavioral uncertainty activity in these bands is elevated in high-anxious subjects, as they try to be ready for following actions. During successful completion the SSP task both sensory and motor activation was stronger in subjects with higher anxiety, as mirrored in higher amplitudes of desynchronization in alpha and beta bands. At the same time, in unsuccessful trials there are no significant differences between subjects with high and low anxiety. We suggest that the main reason for errors was the (temporarily) dropped level of attention in some trials. In this case, the attentional control-related differences in brain activity should be leveled for these trials as it happened.

Functions of "beta rebound" are not completely clear yet. Initially it was associated with inhibition of cortical motor activity after task completion (Chen, 1998; Jurkiewicz et al., 2006). Our results demonstrated that this reaction also reflects self-rating of personal behavior. Stronger and faster "beta

rebound" in subjects with lower anxiety correlates well with both of these interpretations. Indeed, subjects with a low anxiety level are more inclined to evaluate a situation (including, as in our case, results of personal behavior) positively and this positive appraisal could allow them to be more relaxed. On the contrary, according to the theory of attentional control, high-anxious subjects are inclined to keep control longer and stay arduous for some time after completion of task.

CONCLUSION

This chapter revealed dependencies between trait anxiety level and brain reactions related to execution of different behavioral control functions. Amplitude of slow-wave synchronization is positively related to anxiety suggesting that subcortical structures, particularly the thalamus, are involved in mechanisms of anxiety. Trait anxiety level is correlated positively with amplitude of alpha and beta desynchronization confirming the idea of anxiety influence on control of specific attention. High-frequency "beta rebound", reflecting self-rating of own behavior and relaxation, associated with evaluating it as successful, is, on the contrary, negatively related to anxiety level suggesting that low-anxious people are more inclined to evaluate the situation positively. Finally, analysis of cortical topology of EEG changes demonstrates that anxiety is linked to "global" parameters of brain activity, being personality trait modulating execution of several behavioral functions.

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