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Research report

Alpha absolute power measurement in panic disorder with agoraphobia patients

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ABSTRACT

Background: Panic attacks are thought to be a result from a dysfunctional coordination of cortical and brainstem sensory information leading to heightened amygdala activity with subsequent neuroendocrine, autonomic and behavioral activation. Prefrontal areas may be responsible for inhibitory top-down control processes and alpha synchronization seems to reflect this modulation. The objective of this study was to measure frontal absolute alpha-power with qEEG in 24 subjects with panic disorder and agoraphobia (PDA) compared to 21 healthy controls.
Methods: qEEG data were acquired while participants watched a computer simulation, consisting of moments classified as "high anxiety"(HAM) and "low anxiety" (LAM). qEEG data were also acquired during two rest conditions, before and after the computer simulation display.
Results: We observed a higher absolute alpha-power in controls when compared to the PDA patients while watching the computer simulation. The main finding was an interaction between the moment and group factors on frontal cortex. Our findings suggest that the decreased alpha-power in the frontal cortex for the PDA group may reflect a state of high excitability.
Conclusions: Our results suggest a possible deficiency in top-down control processes of anxiety reflected by a low absolute alpha-power in the PDA group while watching the computer simulation and they highlight that prefrontal regions and frontal region nearby the temporal area are recruited during the exposure to anxiogenic stimuli.

1. Introduction

Panic attacks (PA) are defined as sudden periods of intense fear or discomfort, where various somatic and cognitive symptoms are experienced, such as accelerated heart rate, sweating, trembling, smothering, chest pain, nausea, dizziness, fear of losing control, and fear of dying. Panic disorder (PD) patients experience recurrent PA and fear their future repetition and consequences (APA, 2000). The PA also produces behavioral changes and decrease the quality of life of those with PD (APA, 2000). PD subjects have elevated prevalence of comorbid mental disorders (Goodwin and Gotlib, 2004). Agoraphobia (AG) is associated with substantial clinical severity and impairment relative to those with PD uncomplicated by agoraphobia (Pollack and Smoller, 1995; White and Barlow, 2002; Kessler et al., 2006). PD affects 3–4% of the general population and the lifetime prevalence estimates are 22.7% for PA, 3.7% for PD without AG and 1.1% for Panic Disorder with Agoraphobia (PDA) (Kessler et al., 2006).

Gorman et al. (2000) developed one of the most influential hypotheses of the PD neurocircuitry. They suggest that PA result from a dysfunctional coordination of cortical and brainstem sensory information leading to a heightened amygdala activity

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with subsequent neuroendocrine, autonomic and behavioral activation. Gorman et al. (2000) states that the medial prefrontal cortex (PFC), along with other cortical sites that process higher order sensory information is important in modulating anxiety responses and inhibiting amygdala firing. The PFC's activity is associated with attempts to regulate the outcome of attentional, interpretive and associative processes triggered by the occurrence of potentially threat related cues. Amygdala activity (directly activated by the thalamus or activated by the lack of PFC inhibition), in turn, may trigger activity of some subcortical sites, typical of PA symptoms (Dresler et al., 2013; Martin et al., 2009; De Carvalho et al., 2010; Stein, 2005). Lateral PFC and orbitofrontal cortex have been associated with cognitive strategies to regulate emotion, such as reappraisal; dorsolateral prefrontal activity has been related to the use of proactive metacognitive strategies aimed at self-regulating the fear and anxiety evoked by the anxiogenic stimuli (Aupperle et al., 2009). More generally, the PFC is believed to govern executive functioning, which refers to a heterogeneous and wide-ranging set of cognitive operations, including attention allocation, inhibitory control, hypothesis generation, and self-monitoring, as well as other skills (Mohlman, 2005).

The alpha band (8–13 Hz) reflects top-down, inhibitory control processes (Klimesch et al., 2007). Moreover, a decrease in absolute alpha-power is related to neural excitation, such as cognitive processing (Klimesch et al., 2007). The major findings about alpha band showed a low alpha rhythm in anxiety (Siciliani et al., 1975; Enoch et al., 1995; Kalashnikova and Sorokina, 1995; Wiedemann et al., 1998; Gordeev, 2008; Wise et al., 2011). Thus, an absolute alpha-power decrease in the frontal cortex observed in PD may reflect a dysfunction in thalamic–cortical circuits that is associated with incapacity to inhibit irrelevant information, role played especially by the PFC (Klimesch et al., 2007).

In this context, the aim of this study is to observe absolute alpha-power in the scalp frontal region as a whole (F3, F7, Fz, F4, F8, Fp1, Fp2 electrodes) in PDA patients compared to healthy controls while watching an anxiogenic computer simulation (Freire et al., 2010) comprised of high anxiety moments (HAM) and low anxiety moments (LAM). We were expecting a low absolute alpha-power in PDA patients on all electrodes when compared to healthy controls. Moreover, we formulated the hypothesis that, in high anxiogenic moments, absolute alpha-power may be different than in low anxiety moments.

2. Methods

2.1. Participants

We selected a sample by convenience of 24 PDA patients (8 male and 16 female; ages varying between 25 and 61 years old, mean: 38.75, SD: ± 10.09), who were in psychopharmacological treatment at the Laboratory of Panic and Respiration at the Institute of Psychiatry and were evaluated in the Department of Applied Psychology at the Institute of Psychology before treatment; these are both institutes of the Federal University of Rio de Janeiro (UFRJ). The recruitment of subjects was done through posters with information about the research in the outpatient institute of psychiatry and psychology at UFRJ. All patients that met the study inclusion criteria were invited to participate. The patients were interviewed with the M.I.N.I. 5.0 (Sheehan et al., 1998; Amorim, 2000) and fulfilled DSM-IV [1] criteria for PDA. Another inclusion criterion was the occurrence of at least two panic attacks in a 30-day period before the visit. Patients with comorbid dysthymia ($n=1$), generalized anxiety disorder ($n=2$), social phobia ($n=1$) or depression ($n=3$) were included only when PDA was judged to be the primary diagnosis. Some of them began the treatment unmedicated ($n=7$), while others were already taking antidepressants ($n=3$), benzodiazepines ($n=5$) or both antidepressants and benzodiazepines ($n=9$). The patients performed three self-evaluation questionnaires to measure the severity of anxiety, depression and PDA symptoms: Beck Anxiety Inventory (BAI) (Beck et al., 1988) (mean score: 22.68 and SD: ± 14,17; which means moderate anxiety); Beck Depression Inventory (BDI) (Beck et al., 1961) (mean score: 16.37 and SD: ± 10,99; which means mild depression). Seven of the 24 subjects had BDI scores above the relevant clinical threshold for depression and Panic and Agoraphobia Scale (PAS) (Bandelow, 1995) (mean score: 23.82 and SD: ± 9.96; which means moderate PDA symptoms).

There was also a control group with 21 healthy participants (4 male and 17 female; ages from 23 to 61 years old, mean: 40.52, SD: ± 12.47) who were screened with the M.I.N.I. 5.0 (Sheehan et al., 1998; Amorim, 2000) and did not fulfill criteria for any psychiatric disorder. Subjects with other psychiatric disorders, neurological, cardiologic or respiratory diseases were not included in this study, neither in the patient nor in the control group. Patient and control group did not differ from each other in age ($p=0.848$).Our local Ethics Committee (Comitê de Ética em Pesquisa do Instituto de Psiquiatria da Universidade Federal do Rio de Janeiro—CEP-IPUB/UFRJ) approved the protocol, which complied with the principles of the Declaration of Helsinki. After the experiment was fully explained, the subjects signed a voluntary written consent.

2.2. Computer simulation

The simulation consisted of a 4-min three-dimensional computer animation developed by Triptyque LAB (www.triptyquelab.com). Two 30-s periods in which a gray screen was displayed, one before and the other after the animation per se, were included in this animation. This was in a first person perspective (a graphical perspective rendered from the viewpoint of observer of the computer simulation) and there was a camera movement as if the subject was walking inside/outside a bus and looking at different directions during a bus ride. The animation starts at a bus stop: the bus arrives, the subject gets on the bus and sits down, the bus moves through city streets, it stops again and is filled by many people, it moves through the streets, goes in a tunnel, stops inside the tunnel because of traffic, it starts moving again, gets out the tunnel, stops at a bus stop, and the subject gets off the bus and watches the bus leave. The simulation included sound, which consisted of ordinary street noises associated with the images (Freire et al., 2010). In a previous study, this computer simulation demonstrated to be a useful method to induce anxiety and somatic symptoms in PDA patients. Compared to health controls, they had higher scores in anxiety self-evaluation scales and had higher skin conductance level, electrodermal response magnitude, respiratory rate, tidal volume, and respiratory rate irregularities. Two of 10 patients had PA. The heart rate means were higher for PDA patients who had PA (Freire et al., 2010).

The computer simulation consisted of situations classified as “high anxiety” and “low anxiety”. They were classified as being “high” or “low anxiety” by patients that participated in the cited previous study (Freire et al., 2010). The high anxiety situations were when the bus gets filled with people, when the bus gets in a tunnel and when it stops inside the tunnel because of traffic. And the low anxiety situations were those when the camera just moves around and the subject sees the bus, when the bus moves through the streets but is not filled with people, when the bus leaves the tunnel and there is no traffic and when the subject gets off the bus and watches the bus go away. These low anxiety situations refer to the situations where the difficulty of exposure to anxiogenic events tends to be smaller (but it still exists), that is, moments when the patient is about to leave the situations of greater discomfort and for this reason may experiment