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Cognitive performance and cardiovascular markers of hyperarousal in primary insomnia

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ABSTRACT

The purpose of the present study was to assess differences in cardiovascular activity and cognitive performance between insomniacs and good sleepers. Sixteen undergraduates participated in the study, eight insomniacs (age 22.9 ± 2.4) enrolled in accord with DSM-IV criteria for primary insomnia, and eight good sleepers (24.8 ± 2.7) were controls. The task employed, Stop Signal Task, assesses motor inhibition processes and was administered in two sessions, before and after a night of polysomnographic recording. During task performance, cardiovascular measures such as heart rate (HR), stroke volume (SV), cardiac output (CO), pre-ejection period (PEP) and left ventricular ejection time (LVET) were continuously recorded by means of impedance cardiography. Performance results showed prolonged Stop Signal Delay (SSD) in the morning in both groups and slower Stop Signal Reaction Time (SSRT) in insomniacs compared with good sleepers, while no effects were observed for performance accuracy. Analyses performed on cardiovascular parameters revealed higher HR and lower LVET values in the insomnia group as compared to healthy controls in the evening. PEP, an index inversely related to sympathetic beta-adrenergic activity, was continuously reduced in insomniacs, indicating constantly enhanced sympathetic activation. These findings suggest a deficit of motor inhibition control in insomnia, matched with high levels of cardiovascular arousal. Overall, our results support the notion that insomniacs suffer from both cognitive deficits and a hyperarousal disorder affecting somatic activity, that contribute to diurnal complaints often referred in addition to sleep disruption.

1. Introduction

According to the DSM-IV criteria (American Psychiatric Association, 1994), primary insomnia refers to difficulties in initiating or maintaining sleep for at least one month, associated with non-refreshing sleep and daytime consequences such as mood disturbance, fatigue, tiredness, and dysphoria. The disorder generates significant distress and functional impairment and is not caused by other medical or psychiatric illness or substance abuse.

Current epidemiological data suggest that insomnia as a symptom is largely widespread while primary insomnia disorder affects 3–5% of the general adult population (Ohayon, 2002), with substantial socioeconomic costs in terms of reduced productivity and life quality, and increased accident risk and health care utilization (Ancoli-Israel and Roth, 1999; Léger and Bayon, 2010; Simon and VonKorff, 1997). Moreover, primary insomnia is a predisposing factor to the development of depressive and anxiety disorders (Chang et al., 1997; Taylor et al., 2005) and it also increases risk for medical conditions such as arthritis, diabetes and cardiovascular diseases (Sateia and Nowell, 2004; Suka et al., 2003; Taylor et al., 2007). Although the pathogenesis of primary insomnia is still elusive, disordered sleep pattern as well as diurnal complaints have been hypothesized to be attributable to a chronic state of hyperarousal (for a review, see Bonnet and Arand, 2010). The hyperarousal perspective postulates that a condition of high activation affects somatic, cortical and cognitive functioning throughout the day as well as at night, leading to nocturnal and diurnal insomnia symptoms (Perlis et al., 1997, 2001b).

Markers of somatic hyperarousal have been identified, particularly in measures of autonomous activity. Several studies documented, indeed, that insomniacs show elevated heart rate compared to good sleepers across the sleep-wake cycle (Bonnet and Arand, 1998; de Zambotti et al., 2010; Freedman and Sattler, 1982; Haynes et al., 1981, 1985; Monroe, 1967; Stepanski et al., 1994). Also, heart rate variability (HRV) analysis revealed higher low frequency power and decreased high frequency power in insomniacs compared to controls across all sleep stages, thus further suggesting elevated sympathetic and reduced parasympathetic nervous system activity (Bonnet and Arand, 1998). In addition, a wide range of other measures support somatic hyperarousal, such as enhanced cortisol secretion (Backhaus et al., 2004; Rodenbeck et al., 2002; Vgontzas et al., 2001), body temperature (Adam et al., 1986; Vgontzas et al., 1998) whole-body metabolic rate (Bonnet and Arand, 1995) and blood pressure (Lanfranchi et al., 2009).

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Cortical hyperactivity has been shown in increased alpha and beta EEG frequencies both in wake and sleep (Buysse, 2008; Krystal et al., 2002; Merica et al., 1998; Perlis et al., 2001a,b). Furthermore, neuroimaging studies observed elevated brain metabolism (Nofzinger et al., 2004) and abnormal intracortical excitability (Van Der Werf et al., 2010).

Finally, cognitive hyperarousal is indicated in worries and intrusive thoughts during the pre-sleep period interfering with the sleep onset (Harvey, 2000; Morin et al., 2003; Wicklow and Espie, 2000).

Patients suffering from insomnia disorders often complain of cognitive impairment. Despite an extensive investigation into many cognitive domains such as vigilance (Altena et al., 2008b; Schneider-Helmert, 1987; Varkevisser and Kerkhof, 2005), selective attention (Broman et al., 1992; Edinger et al., 2000, 2008; Hauri, 1997; Mendelson et al., 1984; Schneider et al., 2004; Vignola et al., 2000), working memory (Bonnet and Arand, 1995; Broman et al., 1992; Varkevisser and Kerkhof, 2005; Vignola et al., 2000), and memory consolidation (Backhaus et al., 2006; Nissen et al., 2006), executive functions have been poorly investigated in insomniacs. Fang et al. (2008) and Vignola et al. (2000) employed the Wisconsin Card Sorting Test, but failed in finding impaired performance among insomnia sufferers. On the other hand, Edinger et al. (2008) found significant group differences between insomniacs and good sleepers in a switch task, which assesses attention, concentration and response inhibition. Consistently, by employing the Porteus maze test, Randazzo et al. (2000) also observed a poorer performance in insomniacs than in healthy controls.

The domain of executive functioning includes higher level cognitive processes such as planning, inhibition, reasoning and problem solving. Examining brain activity through fMRI during a fluency task, Altena et al. (2008a) reported hypoactivation of the prefrontal cortex – a key area for executive functions – in insomnia sufferers relative to controls, despite a lack of performance differences. These findings suggest a differential recruitment of cerebral resources for successful task completion.

Among executive functions, motor inhibition plays an important role in everyday life. Many situations require the interruption of an ongoing action before another, more appropriate to the context demands, can begin.

The efficiency and latency of the motor inhibition process can be assessed by the Stop Signal Task (SST), a two-choice reaction time task in which, occasionally and unexpectedly, a stop signal occurs requiring response inhibition (Logan and Cowan, 1984; Logan et al., 1984). The rationale underlying the Stop Signal paradigm is provided by the *horse race model* (Logan and Cowan, 1984). This model postulates a competition between two sets of mutually independent processes, one producing responses to the primary task (Go process) and the other responding to the stop signal (Stop process). If the Go process ends before the Stop process, response is given and inhibition fails. In contrast, if the Stop process finishes before the Go process, the response is suppressed. Therefore, correct response inhibition depends on the relative end-time of the two processes. Importantly, the Stop Signal Task allows the latency of the unobservable inhibition process to be inferred (Logan, 1994; Logan and Cowan, 1984; Logan et al., 1997).

The stop signal paradigm has been used to assess inhibitory control in a broad range of disorders, such as schizophrenia (Badcock et al., 2002; Enticott et al., 2008), attention deficit and hyperactivity disorder (Bekker et al., 2005; Overtoom et al., 2002; Rubia et al., 1998), obsessive–compulsive disorder (Krikorian et al., 2004; Menzies et al., 2007), Parkinson's disease (Gauggel et al., 2004) and eating disorders (Claes et al., 2006). To our knowledge, only one study (Sagaspe et al., 2007) has applied Stop Signal paradigm to the study of insomnia. Specifically, Sagaspe et al. (2007) investigated cognitive performance by using this task in patients suffering from obstructive sleep apnea syndrome (OSAS) and insomnia, and compared the patient groups to good sleepers. They found poorer performance in the OSAS group compared to controls, but failed in finding significant differences between insomniacs and healthy controls.

Since few studies have been focused on cardiovascular reactivity to the task in primary insomnia and most of them have analyzed blood pressure, heart rate and measures derived from HRV analysis, the direct sympathetic nervous system influence on the heart is still unknown. The most validated and reliable non-invasive measure of the sympathetic beta-adrenergic influence on heart is the pre-ejection period (PEP), the interval between electric and mechanical systole (Sherwood, 1993). PEP is commonly assessed by impedance cardiography, a non-invasive technique which allows the recording of a wide range of cardiac parameters.

The purpose of the current study was to investigate response inhibition in insomniacs employing the Stop Signal paradigm coupled with non-invasive cardiovascular recordings using impedance cardiography, thus assessing the involvement of hyperarousal in cognitive impairment in primary insomnia. Moreover, in order to investigate hyperactivity markers and mutual links, associations between physiological, cognitive and subjective measures were also examined.

2. Materials and methods

2.1. Participants

Eight normal sleepers (4 males; mean age = 24.8 years, S.D. = 2.7) and eight insomniacs (4 males; mean age = 22.9 years, S.D. = 2.4) participated in this study. All subjects were recruited through advertisements placed in the Faculty of Psychology of Padova University.

Screening procedures included the administration of questionnaires to assess sleep quality (Pittsburgh Sleep Quality Index; Buysse et al., 1989), insomnia symptoms (Insomnia Severity Index; Morin, 1993, and Athens Insomnia Scale; Soldatos et al., 2000), depression (Beck Depression Inventory-II; Beck et al., 1996), anxiety (State Trait Anxiety Inventory-Y2; Spielberger et al., 1983), sleepiness (Epworth Sleepiness Scale; Johns, 1991) and hyperactivation (Hyperarousal Scale; Regestein et al., 1996). Subjects also completed a semi-structured interview to collect anamnesis and investigate sleep history, medical and psychological state. The interview also covered items included in DSM-IV (American Psychiatric Association, 1994) criteria for primary insomnia.

Insomniacs satisfying DSM-IV (American Psychiatric Association, 1994) diagnostic criteria for primary insomnia for at least one year before the experiment and achieving scores ≥ 6 on the Pittsburgh Sleep Quality Index, ≥ 6 on the Athens Insomnia Scale and ≥ 11 on the Insomnia Severity Index, were enrolled in the study.

Subjects showing scores below cut-off on sleep questionnaires and reporting no sleep complaints were selected as normal sleepers. Exclusion criteria for both groups were: severe depressive or anxiety symptoms, as evidenced by scores higher than 95 percentile on the Beck Depression Inventory-II and the State Trait Anxiety Inventory-Y2; Body Mass Index (BMI) ≥ 30 kg/m^2; excessive alcohol or caffeine consumption; drugs or medication consumption; medical and psychiatric diseases or other sleep disorders; shift work and travel across time zones during the past three months prior to participation.

Subjects were instructed to wear a wrist actigraph (Octagonal Basic Motionlogger, Ambulatory Monitoring, Inc., Ardsley, NY) and keep a sleep diary during the seven days prior to the experiment to exclude circadian disorders. Moreover, all participants reported no hearing problems and had normal or corrected-to-normal vision.

The experimental protocol was approved by the Ethics Committee of Padova University. Each subject signed an informed consent and received 100 Euros for participation.