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Coupling of infraslow fluctuations in autonomic and central vigilance markers: Skin temperature, EEG beta power and ERP P300 latency

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ABSTRACT

Even under thermoneutral conditions, skin temperature fluctuates spontaneously, most prominently at distal parts of the body. These fluctuations were shown to be associated with fluctuations in vigilance: mild manipulation of skin temperature during nocturnal sleep affects sleep depth and the power spectral density of the electroencephalogram (EEG), and fluctuations in skin temperature during daytime wakefulness are related to sleep propensity and task performance. The association of daytime skin temperature fluctuations with EEG markers of vigilance has not previously been investigated. Therefore, the present study aimed to evaluate the association between daytime fluctuations in skin temperature with those in two quantitative EEG measures: the power spectral density of background EEG, and the event related potential (ERP) elicited by visual stimuli.

High-density EEG and skin temperature were obtained in eight healthy adults five times a day while they performed a visual sustained-attention task. Assessments were made after a night of normal sleep and after the challenge of a night of total sleep deprivation.

Fluctuations in the distal-to-proximal skin temperature gradient measured from the earlobe and mastoid were associated with fluctuations in parieto-occipital high beta band (20–40 Hz) power of the pre-stimulus background EEG, but only after sleep deprivation. The temperature fluctuations were moreover associated with fluctuations in the latency of the P300 elicited by the stimulus.

The findings demonstrate close association between fluctuations in an autonomic correlate of the vigilance state (i.e. the distal-to-proximal skin temperature gradient), and fluctuations in central nervous system correlates of the vigilance state (i.e. background EEG and ERP). The findings are of theoretical and practical relevance for the assessment and manipulation of vigilance.

1. Introduction

The temperature of the skin of the human body shows spontaneous fluctuations, even under thermoneutral conditions (Romanovsky, 2007). Especially the skin of the distal extremities fluctuates considerably, for example over a range of about 2 °C for the finger, measured under strictly controlled conditions (Huizenga et al., 2004; van Marken Lichtenbelt et al., 2006). Both in controlled laboratory settings (Kräuchi and Wirz-Justice, 1994) and in everyday life (Van Someren, 2006), the most pronounced fluctuation in skin temperature is a rhythm of 24-hour. Body temperature rhythms are among the first rhythms to appear during early development and are driven by the hypothalamic suprachiasmatic nucleus, the biological clock of the brain (Swaab et al., 1996). Skin temperature also shows ultradian fluctuations that are associated with variability in subjective, physiological and behavioral indices of sleep propensity and vigilance (Fronczek et al., 2006; Romeijn and Van Someren, 2011; Romeijn et al., 2012). Vigilance may be most strongly associated with the distal-to-proximal gradient, i.e. the temperature gradient between distal skin areas of the extremities relative to proximal skin areas (DPG, Kräuchi et al., 1999, 2000). This association is most reliably found if the gradient is measured between the ear lobe (distal) and the nearby mastoid (proximal) (Romeijn et al., 2012). The gradients reflect sympathetic regulation of distal blood flow (Rubinstein and Sessler,

Abbreviations: BSRT, Brief-Stimulus Reaction Time Task; DPG, Distal-to-Proximal Gradient; EEG, Electroencephalogram; ERP, Event-Related Potential; NS, Normal Sleep; P300, P300 component; SD, Sleep Deprived.

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1990) and thus provide a window on the activity of the autonomic nervous system. It has been proposed that the association is mediated by signaling of skin thermoreceptors to thermosensitive neurons in the preoptic area–anterior hypothalamus (POAH) and other areas that are involved in the regulation of sleep and vigilance (Van Someren, 2006).

A number of studies provide behavioral and physiological support for the proposed causal contribution of fluctuations in skin temperature to variability in sleep and vigilance.

When applied during sleep, mild skin warming affects the macrostructure of sleep, in the sense that it increases the percentage of time spent in deep sleep (Fronczek et al., 2008a; Raymann et al., 2008). Additionally, mild skin warming during sleep changes quantitative sleep measures: the electroencephalogram (EEG) power spectrum shows enhancement of the lower delta and theta oscillations that are typical of sleep, as well as suppression of the higher beta oscillations that are associated with arousal (Raymann et al., 2008).

When applied during wakefulness, mild skin warming accelerates sleep onset latency (Fronczek et al., 2008b; Raymann et al., 2005). It also deteriorates the performance on sustained attention tasks, as indicated by reaction time slowing and an increase in the number of lapses (Raymann and Van Someren, 2007). However, it is not known whether daytime fluctuations of skin temperature are associated with quantitative changes in the background EEG, or in event-related potentials (ERPs). The hypothesis of skin temperature affecting vigilance regulation in the central nervous system would be strongly supported by a covariation with electrophysiological markers of the vigilance state. The present study therefore aimed to evaluate the presence of an association between daytime fluctuations in skin temperature and those in two quantitative EEG measures: the power spectral density of background EEG and the P300 component of the ERP elicited by visual stimuli.

Spectral changes in background EEG in association to changes in vigilance have been demonstrated in numerous studies, often using sleep deprivation as a tool to manipulate vigilance level. Fluctuations in vigilance have been associated with resting-state EEG power fluctuations in the alpha band (8–12 Hz) (Corsi-Cabrera et al., 1996, 1992, 2003; Ferreira et al., 2006; Galliaud et al., 2008; Gast et al., 2011; Strijkstra et al., 2003), theta band (4–8 Hz) (Caldwell et al., 2003; Ferreira et al., 2006; Galliaud et al., 2008; Hoedlmoser et al., 2010) and delta band (Ferreira et al., 2006; Hoedlmoser et al., 2010). Similar findings were reported in studies that assessed sleep deprivation-induced changes in EEG during task performance instead of during relaxed wakefulness (Caldwell et al., 2003; Corsi-Cabrera et al., 1996; Hoedlmoser et al., 2010). Sleep deprivation has also been reported to increase beta band (13–30 Hz) power (Smulders et al., 1997). Because beta band power is mostly regarded as indicating excitatory cortical activity and a state of high arousal, the increase after sleep deprivation has been interpreted as a compensatory effort to maintain vigilance (Smulders et al., 1997; Tsuno et al., 2002).

With respect to changes in ERPs associated with fluctuations in vigilance during normal wakefulness or extended wakefulness (i.e. following sleep deprivation), several studies reported associations with P1, N1 and P300 amplitude or latency (Corsi-Cabrera et al., 1999; Gosselin et al., 2005; Hoedlmoser et al., 2010; Trujillo et al., 2009). These potentials are thought to reflect sensory and higher order processing and are sensitive to modulations in attention and arousal. Sleep deprivation may most consistently affect the P300. A sleep deprivation-induced delay in P300 peak latency has been interpreted as indicating either a slower processing speed or a reduced allocation of attention (Lee et al., 2003). A sleep deprivation-induced attenuation of P300 peak amplitude has been interpreted as a reduction in the resources allocated either to attention or to stimulus evaluation processes, depending on the nature of task used (Corsi-Cabrera et al., 1999; Cote et al., 2008; Gosselin et al., 2005). Other interpretations include lapses, general fatigue, and changes in alertness (Morris et al., 1992). An increase in effort to successfully perform the task ameliorates the effects of sleep deprivation on the peak latency and amplitude of the P300 (Colrain and Campbell, 2007).

The response of background EEG and ERPs to changes in temperature has been studied less extensively. Morris et al. (1992) reported that sleep deprivation-induced changes in the P300 correlate with core body temperature. We are not aware of previous studies that have investigated associations of EEG or ERPs with skin temperature. It would be of interest to evaluate whether central nervous system markers of vigilance (EEG and ERPs) covary with an autonomic nervous system marker of vigilance (distal-to-proximal skin temperature gradient). The present study therefore assessed whether these quantitative EEG markers of vigilance vary in synchrony with daytime fluctuations in skin temperature. It was hypothesized that these central nervous system readout variables indicate lower vigilance during periods of a more elevated distal-to-proximal skin temperature gradient. The association was evaluated both in a well-rested state after a normal night of sleep, and in a state of challenged vigilance induced by a night of sleep deprivation.

2. Methods

2.1. Participants

Eight healthy adults (3 females) gave their written informed consent to participate. Their age ranged from 20 to 26 years (mean = 22.0, sd = 1.77). All were right-handed and had normal or corrected-to-normal vision. Participants received money for their participation. All procedures complied with the declaration of Helsinki and medical ethical approval was obtained from the medical ethical committee of the Academic Medical Center of the University of Amsterdam and the Medical Ethics Committee of the VU University.

2.2. Inclusion criteria

All subjects were non-smokers and free of any medication known to affect sleep or the circadian system, cardiovascular medication, and psychotropic medication. None of the subjects had a history of sleep-related disorders. All subjects were reported to be in good health and free of any physical or mental disorder. None of the female subjects used hormonal contraceptives and they all participated between day 4 and day 13 of the menstrual cycle (follicular phase).

2.3. Study design and procedure

The design and procedure have been described in detail previously (Romeijn et al., 2012). Subjects were instructed to keep a regular sleep–wake pattern by minimizing variability in bedtime and wake-up time in the week before the experiment, which was screened using a sleep diary and actigraphy (Actiwatch, Cambridge Neuro-Technology, Cambridge, UK). Subjects were also instructed to refrain from caffeine, alcohol, heavy medicine, and intensive physical exercise for 12 h before arriving at the sleep laboratory, and to refrain from eating for at least 4 h before arrival.

The experiment consisted of a series of tasks and assessments on two days: after a night of normal sleep (NS) and, in counterbalanced order, after a night of total sleep deprivation (SD). The interval between the two assessment days allowed for at least two nights of normal sleep in between. On assessment days, subjects arrived at the sleep laboratory at 08.30 for setup and EEG preparation. From 10:00 until 17:30 h, participants completed 5 identical experimental blocks while seated under dim-light conditions (<15 lx). Assessments during these blocks included an EEG-ERPs participants, comfortably seated, performed a simple reaction time sustained attention task, the Brief-Stimulus Reaction Time Task (BSRT, Romeijn and Van Someren, 2011), which commenced at 10:35, 12:05, 13:35, 15:05, and 16:35 h.