



Electroencephalographic and autonomic alterations in subjects with frequent nightmares during pre- and post-REM periods



Péter Simor^{a,b,*}, János Körmendi^c, Klára Horváth^d, Ferenc Gombos^e, Péter P. Ujma^c, Róbert Bódizs^{c,e}

^a Department of Cognitive Sciences, Budapest University of Technology and Economics, H-1111, Egry József u. 1, T. épület/V, Budapest, Hungary

^b Nyíró Gyula Hospital, National Institute of Psychiatry and Addictions, Budapest, Hungary

^c Institute of Behavioural Sciences, Semmelweis University, H-1089, Nagyvárad tér 4, Budapest, Hungary

^d Department of Experimental Psychology, University of Oxford, South Parks Road, OX1 3UD Oxford, UK

^e Department of General Psychology, Pázmány Péter Catholic University, 2087, Egyetem u. 1, Piliscsaba, Hungary

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ABSTRACT

Abnormal arousal processes, sympathetic influences, as well as wake-like alpha activity during sleep were reported as pathophysiological features of Nightmare Disorder. We hypothesized that in Nightmare Disorder, wake-like cortical activity and peripheral measures linked to arousals would be triggered by physiological processes related to the initiation of REM periods. Therefore, we examined electroencephalographic (EEG), motor and autonomous (cardiac) activity in a group of nightmare (NM) and healthy control (CTL) subjects during sleep-state-transitions while controlling for the confounding effects of trait anxiety. Based on the second-nights' polysomnographic recordings of 19 Nightmare Disordered (NM) and 21 control (CTL) subjects, we examined the absolute power spectra focusing on the alpha range, measures of heart rate variability (HRV) and motor (muscle tone) activity during pre-REM and post-REM periods, separately. According to our results, the NM group exhibited increased alpha power during pre-REM, but not in post-REM, or stable, non-transitory periods. While CTL subjects showed increased HRV during pre-REM periods in contrast to post-REM ones, NM subjects did not exhibit such sleep state-specific differences in HRV, but showed more stable values across the examined sleep stages and less overall variability reflecting generally attenuated parasympathetic activity during sleep-state-transitions and during stable, non-transitory NREM states. These differences were not mediated by waking levels of trait anxiety. Moreover, in both groups, significant differences emerged regarding cortical and motor (muscle tone) activity between pre-REM and post-REM conditions, reflecting the heterogeneity of NREM sleep. Our findings indicate that NM subjects' sleep is compromised during NREM-REM transitions, but relatively stabilized after REM periods. The coexistence of sleep-like and wake-like cortical activity in NM subjects seems to be triggered by REM/WAKE promoting neural activity. We propose that increased arousal-related phenomena in NREM-REM transitions might reflect altered emotional processing in NM subjects.

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Abbreviations: AI, atonia index; ANCOVA, analysis of covariance; CAP, cyclic alternating pattern; CTL, control; DQQ, Dream Quality Questionnaire; ECG, electrocardiogram; EEG, electroencephalography; HF, high frequency measure of heart rate variability; HRV, heart rate variability; LF, low frequency measure of heart rate variability; NM, nightmare; NN, normal sinus to normal sinus; PTSD, post traumatic stress disorder; REM, non-rapid eye movement sleep; REM, rapid eye movement sleep; RMSSD, square root of the mean of the squares of differences between adjacent normal sinus to normal sinus intervals; VDAS-H, the Hungarian version of the Van Dream Anxiety Scale.

* Corresponding author at: Department of Cognitive Sciences, Budapest University of Technology and Economics, H-1111, Egry József u. 1, T. épület/V, Budapest, Hungary. Fax: +36 1 463 1072.

E-mail addresses: psimor@cogsci.bme.hu, petersimor@gmail.com (P. Simor).

1. Introduction

Although at the behavioral level sleep is apparently a global, whole-brain phenomenon that is governed by central mechanisms (Saper, Chou, & Scammell, 2001), neural oscillations during sleep often indicate the coexistence of markedly different EEG patterns, especially during the transitions between different states of vigilance (Bódizs, Simor, Csóka, Bérdi, & Kopp, 2008; Bódizs, Sverteczki, & Mészáros, 2008; De Gennaro, Ferrara, Curcio, & Cristiani, 2001; Nobili et al., 2011). The coexistence of different sleep-like and wake-like electroencephalographic (EEG) oscillations was shown to be related to lucid dream experiences (Tyson, Ogilvie, & Hunt, 1984; Voss, Holzmann, Tuin, & Hobson, 2009),

arousal parasomnias (Terzaghi et al., 2009), occasional sleep paralysis (Mahowald, Cramer Bornemann, & Schenck, 2011), and sleep paralysis within the context of narcolepsy (Terzaghi, Ratti, Manni, & Manni, 2012), or REM Behavior Disorder (Manni, Terzaghi, & Glorioso, 2009).

More recently, a relatively less emphasized sleep disorder, Nightmare Disorder was also characterized by the combination of wake-like and sleep-like EEG features during REM sleep (Simor, Horváth, Ujma, Gombos, & Bódizs, 2013). Subjects with Nightmare Disorder report at least one emotionally negative dream on a weekly basis; which experiences may result in abrupt awakenings from (usually) late-night sleep (ICSD-II; American Sleep Disorders Association, 2005). Higher arousability, and lower awakening thresholds in Nightmare Disorder seem to be related to the qualitative aspects of dream experiences. For instance, nightmares that end with abrupt awakenings in contrast to dysphoric dreams without awakenings were rated by dreamers as emotionally more intense, containing a higher rate of physical aggression and more negative scenario (e.g. failures and unfortunate endings) (Robert & Zadra, 2014; Zadra, Pilon, & Donderi, 2006). Frequent nightmare complaints are highly prevalent, affecting approximately four percent of the adult population (Spoormaker, Schredl, & van den Bout, 2006). The clinical relevance of nightmares should not be underestimated given their high incidence in psychiatric populations (Swart, van Schagen, Lancee, & van den Bout, 2013), co-morbidity with insomniac symptoms (Schredl, 2009; Schredl, Schafer, Weber, & Heuser, 1998) and their association with waking affective (Levin, Fireman, Spendlove, & Pope, 2011; Sjöström, Hetta, & Waern, 2009; Van Liempt, van Zuiden, Westenberg, Super, & Vermetten, 2013) as well as cognitive (Simor, Pajkossy, Horváth, & Bódizs, 2012) dysfunctions.

Polysomnographic studies suggest that subjects with frequent nightmares in comparison with controls are characterized by disturbed sleep patterns, comprising frequent nocturnal awakenings (Simor, Horváth, Gombos, Takács, & Bódizs, 2012) and enhanced microarousals (Simor, Bódizs, Horváth, & Ferri, 2013) during NREM sleep, increased sympathetic (cardiac) activation (Nielsen et al., 2010) and heightened wake-like alpha power during REM sleep (Simor, Horváth, et al., 2013), as well as elevated indices of periodic leg movements (Germain & Nielsen, 2003) during both sleep stages. However, the underlying mechanism of disrupted sleep in relation to frequent nightmares is far from being fully elucidated.

Sleep stage transitions are specifically “sensitive” periods for the presence of microarousals, muscle activity and EEG desynchronization (Pilcher & Schulz, 1987). These transitory periods clearly show the heterogeneity of sleep as well as the constraints of defining discrete sleep stages. The ambiguity of sleep stages is particularly prevalent in children’s (Grigg-Damberger et al., 2007) and in clinical patient’s sleep recordings (Mahowald et al., 2011). The coexistence of different sleep and awake states is relatively frequent during the so-called ascending phases of sleep, comprising the transition from NREM to REM sleep (Halász & Bódizs, 2013; Parrino, Ferri, Bruni, & Terzano, 2012). During these periods, the cortex is prone to exhibit phasic arousals involving wake-like EEG (McKinney, Dang-Vu, Buxton, Solet, & Ellenbogen, 2011), increased motor activity (Nobili et al., 2011) and autonomic fluctuations (Halász & Bódizs, 2013). During the so-called ascending phases (pre-REM periods) spontaneous or elicited micro-arousals are more frequent and involve the desynchronization of EEG as well as associated autonomic changes (Parrino et al., 2012). In contrast, during the so-called descending slopes (post-REM periods) phasic activations are rare, and environmental stimulation is more likely to induce a K-complex, a sleep-like (antiarousal) response (Halász & Bódizs, 2013). Moreover, EEG spectral power time-courses over the ultradian sleep cycle (Merica & Fortune, 1997, 2004) indicate that the ascending, in comparison to the descending

phases, are characterized by reduced sleep promotion, increased environmental awareness, and presumably the intensification of mental (dream) experiences (Conduit, Bruck, & Coleman, 1997; De Gennaro et al., 2010).

We assume that nightmare sufferers’ sleep is particularly vulnerable during the ascending, transitory pre-REM periods, and will show signs of covert wake-like activity, in contrast to the descending, post-REM periods, when due to the reduction of REM sleep pressure, and the reinstatement of sleep-promoting influences, the brain gradually advances into deeper and less fragile sleep stages. In order to examine the cortical correlates of wake-like neural activity we focused on the EEG alpha band power that was shown to reveal instantaneous responsiveness to environmental stimulation as well as spontaneous fluctuations in relation to sleep fragility (McKinney et al., 2011). Since alpha band power seems to reflect covert wake-like activity and increased environmental awareness during sleep (McKinney et al., 2011; Terzaghi et al., 2009, 2012), we hypothesized that nightmare (NM) in comparison to control subjects (CTL) would exhibit increased EEG alpha power, especially during the pre-REM periods. In addition, we examined two “peripheral” measures related to arousals and sleep disruption: heart rate variability (HRV) reflecting parasympathetic control on cardiac regulation (Ferini-Strambi et al., 2000) and muscle atonia (Ferri et al., 2010) indexing motor (in)activity during sleep.

The analysis of HRV is a widely used method for evaluating autonomic cardiovascular regulation (Heart Rate Variability, 1996) characterizing different states of vigilance (Elsenbruch, Harnish, & Orr, 1999) and showing alterations in different sleep disorders, such as insomnia (Spiegelhalder et al., 2011; Vgontzas, Fernandez-Mendoza, Liao, & Bixler, 2013), PTSD (Chang et al., 2013) and Nightmare Disorder (Nielsen et al., 2010). Frequency and time domain measures of HRV based on the variability of interbeat intervals are reliable indices of parasympathetic activity related to the vagal tone (Heart Rate Variability, 1996; Reyes del Paso, Langewitz, Mulder, van Roon, & Duschek, 2013). Since wake-like oscillations and arousals during sleep are related to reduced parasympathetic (vagal) tone (Ferini-Strambi et al., 2000), we expected that during pre-REM, in contrast to post-REM periods, NM subjects would exhibit reduced parasympathetic control in comparison with the control group.

The automatic quantification of muscle atonia, based on the submental (chin) muscle EMG recording proved to be an efficient tool to describe abnormal motor activity during REM sleep (Ferri, Bruni, Fulda, Zucconi, & Plazzi, 2012). We hypothesized that NM in comparison to CTL subjects would show signs of abnormal motor activity (reduced muscle atonia) in the pre-REM, but not in the post-REM periods.

Given that waking levels of anxiety were reported to be moderately associated with nightmare frequency (Schredl, 2003), but may also have an effect on EEG spectral power (Mizuki, Kajimura, Nishikori, Imaizumi, & Yamada, 1984; Suetsugi, Mizuki, Ushijima, Yamada, & Imaizumi, 1998), HRV (Dishman et al., 2000; Gorman & Sloan, 2000; Kemp, Quintana, Felmingham, Matthews, & Jelinek, 2012) and muscle activity (Willmann, Langlet, Hainaut, & Bolmont, 2012), we aimed to control for the confounding effects of trait anxiety. Although nightmare frequency and increased anxiety often appear as co-morbid symptoms, research indicates that these variables are indeed independent from each other, and should be treated as separate factors, especially in case of Nightmare Disorder (Coolidge, Segal, Coolidge, Spinath, & Gottschling, 2010; Lancee, Spoormaker, & Van den Bout, 2010; Simor, Horváth, et al., 2012; Spoormaker et al., 2006; Wood & Bootzin, 1990). Therefore, by controlling for the influence of trait anxiety, we could examine the primary relationship between disturbed dreaming and sleep physiology (EEG, HRV and muscle tone), regardless of the effects of waking anxiety.

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