Biological Psychology 95 (2014) 126–134

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

Enhancing sleep quality and memory in insomnia using instrumental sensorimotor rhythm conditioning

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ARTICLE INFO

Article history:
Received 12 October 2012
Accepted 20 February 2013
Available online 30 March 2013

Keywords:
Insomnia
EEG
Hyperarousal
Neurofeedback
Memory
Consolidation

ABSTRACT

EEG recordings over the sensorimotor cortex show a prominent oscillatory pattern in a frequency range between 12 and 15 Hz (sensorimotor rhythm, SMR) under quiet but alert wakefulness. This frequency range is also abundant during sleep, and overlaps with the sleep spindle frequency band. In the present pilot study we tested whether instrumental conditioning of SMR during wakefulness can enhance sleep and cognitive performance in insomnia.

Twenty-four subjects with clinical symptoms of primary insomnia were tested in a counterbalanced within-subjects-design. Each patient participated in a SMR- as well as a sham-conditioning training block. Polysomnographic sleep recordings were scheduled before and after the training blocks.

Results indicate a significant increase of 12–15 Hz activity over the course of ten SMR training sessions. Concomitantly, the number of awakenings decreased and slow-wave sleep as well as subjective sleep quality increased. Interestingly, SMR-training enhancement was also found to be associated with overnight memory consolidation and sleep spindle changes indicating a beneficial cognitive effect of the SMR training protocol for SMR "responders" (16 out of 24 participants). Although results are promising it has to be concluded that current results are of a preliminary nature and await further proof before SMR-training can be promoted as a non-pharmacological approach for improving sleep quality and memory performance.

1. Introduction

Insomnia is characterized by a complaint of difficulty initiating sleep, maintaining sleep, and/or non-restorative sleep that causes clinically significant distress or impairment in social, occupational or other important areas of functioning (Littner et al., 2003; Riemann et al., 2010). From a psychological perspective insomnia patients typically complain of being unable to stop their reverberating thoughts and "rest their mind" which prevents them from sleeping. Insomnia is considered a significant complaint and is associated with decreased quality of life, absenteeism, increased work and car accidents, as well as increased general health care utilization. Epidemiological research shows the high prevalence of insomnia with about 30% of the general population complaining about some insomnia symptoms and 10% of the population fulfilling criteria for an insomnia syndrome with classical symptoms such as negative daytime consequences (Morin, LeBlanc, Daley, Gregoire, & Merette, 2006). According to DSM-IV criteria the proportion of primary insomnia is estimated to be around 3% (Gallup-Organization, 1995) to 6% (Ohayon, 2002). Empirical data demonstrate that insomnia is most often a chronic condition, defined as an inability to consistently sleep well for a period of at least 1 month. The consequences of chronic insomnia are severe and include adverse effects such as deficits in cognitive efficiency (Nissen et al., 2011), social discomfort and non-specific physical complaints (Gallup-Organization, 1995; Morin, Bootzin, et al., 2006; Stepanski et al., 1989). In addition to the high

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http://dx.doi.org/10.1016/j.biopsycho.2013.02.020

rates of past or present psychopathology, insomnia patients also have an increased risk for the development of *further* psychiatric illnesses (Buysse, 2004; Morawetz, 2003; Weissman, Greenwald, NinoMurcia, & Dement, 1997).

In summary, insomnia is a prevalent and clinically important problem. In fact it is the most commonly reported sleep problem in industrialized nations worldwide (Sateia, Doghramji, Hauri, & Morin, 2000).

Reports from patients with insomnia suggest that the disorder often starts as a stress-related phenomenon (Hauri & Fisher, 1986) with the individual's emotional and behavioral response to the condition playing an important role in the final outcome of the situation. These maladaptive cognitive, behavioral and emotional responses – precipitating and perpetuating insomnia – may be well dealt with non-pharmacological treatments. Indeed, there is promising evidence that non-pharmacological interventions besides hypnotics can be (i) efficient in treating insomnia symptoms (i.e., improving objective sleep measures such as sleep onset latency [SOL], wake after sleep onset [WASO], or total sleep time [TST]) and can also (ii) lead to *subjective* alleviation of patient complaints, with higher measurable quality of life after treatment (Ebben & Spielman, 2009; Morin, Bootzin, et al., 2006; Perlis, Smith, Cacialli, Nowakowski, & Orff, 2003; Van Houdenhove, Buyse, Gabriels, & Van den Bergh, 2011).

According to Freedman (1986) and more recently Perlis, Giles, Mendelson, Bootzin, and Wyatt (1997); Perlis, Kehr, et al. (2001); Perlis, Merica, Smith, and Giles (2001); Perlis, Smith, Andrews, Orff, and Giles (2001) the cognitive hyperarousal associated with insomnia (for review also see Riemann et al., 2010) is reflected in fast brain oscillations (including beta and gamma activity) which are elevated at sleep onset and during shallow NREM sleep stages (e.g., Buysse et al., 2008). The "Neurocognitive Model of Insomnia" (Perlis et al., 1997) proposes that the increase in central nervous system tone results in increased and persistent sensory and cognitive processing also during sleep where under normal circumstances such processes would be vastly attenuated or inhibited. According to the model increased sensory processing and perception thus account for difficulties in sleep initiation and sleep maintenance. This view is also in accordance with positron emission tomography (PET) data from Nofzinger and colleagues (Nofzinger et al., 2006) which show greater brain metabolism in arousal systems during the night in these patients. It is assumed that this cognitive hyperarousal and concomitantly elevated beta and gamma frequencies can be influenced and diminished using instrumental EEG conditioning of slower frequencies.

In the present study we therefore specifically focus on the instrumental conditioning of 12–15 Hz oscillations for improving sleep quality and memory performance in a population of young primary insomnia patients. These 12–15 Hz oscillations are prominent over the sensorimotor cortex – therefore termed sensorimotor rhythm (SMR) – and show a very distinctive pattern. They are (i) dominant during quiet but alert wakefulness and (ii) synchronize when motor behavior is inhibited (Sterman, Howe, & Macdonald, 1970). Interestingly, oscillations in the same frequency range are also abundant during light non-rapid eye movement (NREM) sleep, and overlap with the sleep spindle frequency band. Sterman et al. (1970) were the first to demonstrate that instrumental SMR conditioning (ISC) during wakefulness can improve subsequent sleep in cats. Hauri then used instrumental conditioning of various EEG parameters to treat disordered human sleep (Hauri, 1981; Hauri, Percy, Hellekson, Hartmann, & Russ, 1982) and demonstrated that patients suffering from primary insomnia specifically benefited from the SMR training protocol if they did not also suffer from physiological hyperarousal (i.e., enhanced muscle tension) at study intake. Given these findings (Hauri, 1981; Hauri et al., 1982; Sterman et al., 1970) as well as a meta-analysis on the efficacy of SMR biofeedback for epilepsy (Tan et al., 2009) we followed the rationale that ISC is in good place to directly counteract cognitive hyperarousal by attenuating associated high-frequency EEG oscillations.

In addition we recently found that instrumental SMR conditioning (as compared to a "placebo" randomized-frequency-conditioning protocol) can exert positive effects on sleep quality and even on declarative memory performance in *healthy* individuals (Hoedlmoser et al., 2008). Interestingly, and in accordance with previous literature, sleep spindles were found to be elevated after waking SMR conditioning (Sterman et al., 1970). This is in so far important, as a vast amount of literature points to the direct significance of sleep spindles for "offline" memory consolidation (e.g., Griessenberger et al., 2012; Gais, Mölle, Helms, & Born, 2002; Schabus et al., 2004; Tamaki, Matsuoka, Nittono, & Hori, 2008). With respect to insomnia a recent, exploratory study using "tele-neurofeedback" (i.e., done at home but connected with the therapist via an internet connection) (Cortoos, De Valck, Arns, Breteler, & Cluydts, 2010) indicates that TST improves after SMR tele-neurofeedback (but not after electromyography tele-biofeedback) whereas SOL decreases. Unfortunately, most of these – often termed "neurofeedback" (NFT) – studies lack important controls such as (i) real (sham) conditions or (ii) a convincing demonstration of proposed EEG changes after training.

Therefore, we aim to clarify in a counterbalanced cross-over design the efficacy of instrumental SMR conditioning for insomnia (termed ISC in the following) and start by testing whether patients actually succeed in (waking) SMR enhancement over ISC training sessions and whether such enhancement transfers to spindles during sleep. Last but not least we then assess whether ISC may influence (i) sleep quality and/or (ii) cognitive performance (as evidenced by declarative overnight memory consolidation).

Please note that the present study is best seen as a comprehensive pilot test for upcoming studies addressing the efficacy of ISC training on sleep and memory in an even more controlled manner.

2. Methods and materials

2.1. Participants

Twenty-four subjects (17 female, 7 male) aged between 19 and 50 years ($M = 34.83$, $SD = 10.60$) were included in the study. Subjects were recruited from the public by radio, newspaper and online advertisements as well as announcements on notice boards at the University of Salzburg and the Christian-Doppler hospital (Salzburg, Austria). Subjects gave written informed consent on study entrance and were refunded €200 – on study completion. Furthermore, participants had to refrain from any drug/medication and limit caffeine use (no caffeine intake for at least 5–6 h prior to sleep laboratory visits) as well as smoking (to a maximum of 4 cigarettes a day) during the complete study period. Subjects were preselected based on questionnaires concerning sleep quality (Pittsburgh Sleep Quality Index, PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), anxiety (BAI; Beck, Epstein, Brown, & Steer, 1988) and depression (BDI-II; Beck, Steer, & Brown, 1996). On entrance subjects were required to (i) have a global score > 5 on the PSQI to ensure a minimum level of insomnia severity, (ii) have a daytime impairment related to the nighttime sleep difficulty, and show (iii) no signs of a current or past mental or psychiatric disorder (concurrent with insomnia). The latter two criteria are in accordance with the research diagnostic criteria for "primary insomnia" of the American Academy of Sleep Medicine (AASM; Edinger et al., 2004). Furthermore, subjects needed to have a problem in initiating or maintaining sleep, in waking up too early or reporting chronically non-restorative sleep (despite adequate opportunity and circumstances for sleep). Subjects complying with these pre-screening criteria were further evaluated by sleep diaries and a structured clinical interview for sleep disorders (SIS-D; Schramm et al., 1993). Finally, only patients with a primary insomnia complaint (at least 3 times a week over the course of a month) according to SIS-D criteria were included in the study. The mean duration of the current insomnia episode was reported to be about 4 years ($M = 4.08$, $SD = 2.33$). Lastly, we set a minimum criterion for subjectively impaired sleep quality (SOL or WASO greater than 20 min as derived from sleep diaries). At the subsequent entrance examination patients were asked to fill out the Freiburger Personality Inventory (FPI; Fahrenberg, Hampel, & Selg, 1984), the Wechsler Memory Scale – Revised (WMS-R; Wechsler, 1987), the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983) as well as the WHO