Biofeedback
Volume 45, Issue 1, pp. 19–20
DOI: 10.5298/1081-5937-45.1.08

SPECIAL ISSUE

Neurofeedback as a Potential Nonpharmacological Treatment for Insomnia

Shona L. Halson, PhD

Australian Institute of Sport, Belconnen, ACT, Australia

Keywords: insomnia, sleep, neurofeedback, electroencephalogram, sensory motor rhythm

Insomnia is a significant sleep disorder in today's society and has multiple psychological, physical, general health, and occupational implications. Common treatments for insomnia include pharmacotherapy, cognitive behavior therapy, and over-the-counter products. These treatments have varying degrees of efficacy and/or potential side effects. Based on the neurocognitive model of insomnia whereby persistent sensory and cognitive processing may disturb sleep, neurofeedback is a potential nonpharmacological treatment. Based on the limited but positive research in the area, neurofeedback may be considered as a promising tool for treating insomnia.

Insomnia is a prevalent, complex, and clinically significant problem in modern society. The *International Classification of Sleep Disorders, Version 3* (ICSD; American Academy of Sleep Medicine, 2014) describes chronic insomnia as having one of the following complaints at least three days a week for three months: (1) difficulty initiating sleep, (2) difficulty maintaining sleep, and (3) waking earlier than desired (ISD-10: G 47.00). The prevalence of insomnia is estimated to be 9–22% (Kay-Stacey & Attarian, 2016) and is associated with decreased quality of life, absenteeism, increased motor vehicle accidents, and increased accidents in the workplace (Hoedlmoser et al., 2008).

Treatment of insomnia typically includes pharmacotherapy, cognitive behavior therapy for insomnia (CBT-I), over-the-counter products, and herbal remedies (Hoedlmoser et al., 2008). Pharmacological treatment (hypnotics) are the most widely used treatment for insomnia; however, there are a number of side effects and contraindications. Evidence exists for impaired cognitive and psychomotor skills, increased risk of falls, insomnia rebound, dependence, and potential for abuse (Laudon & Frydman-Marom, 2014) as a result of pharmacological treatment for insomnia. Due to these potential adverse responses, research has focused on both developing new medications as well as investigation into nonpharmacological forms of treatment.

CBT-I is the most common form on nonpharmacological treatment of insomnia and has been consistently shown to have greater efficacy when compared to medication (Kay-Stacey & Attarian, 2016). However, CBT-I is not widely used in many countries due to a lack of practitioners with expertise in this area. Further, CBT-I typically results in an average improvement in sleep that does bring the individual into the "good sleeper" range, but requires patient effort and dedication. Finally, 19–26% of patients do not respond to CBT-I (Cortoos, Verstraeten, & Cluydts, 2006).

The neurocognitive model of insomnia (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997) proposes that increased central nervous system activity and consistent sensory and cognitive processing may result in difficulty initiating and maintaining sleep (Schabus et al., 2014). This theory is supported by findings of increased beta and gamma activity in insomnia patients as well as greater metabolism in arousal systems during sleep measured by positron emission tomography, or PET (Buysse et al., 2008). Based on the above findings, it may be suggested that a nonpharmacological approach that intervenes at the level of cortical arousal and cognitive processing may be efficacious for improving sleep in insomnia patients. As neurofeedback can result in participants' self-regulating parameters of their cortical activity, neurofeedback has been suggested to be a useful nonpharmacological approach to insomnia treatment (Hoedlmoser et al., 2008).

The first work to examine this possibility was by Sterman, Howe, and Macdonald (1970), who found that enhanced sleep (facilitation of sleep spindle bursts and longer periods of undisturbed sleep) was a consequence of facilitating the sensory motor rhythm (SMR) in cats. The authors demonstrated that the cats learned to voluntarily enhance waking EEG of SMR (12–14 Hz activity) via operant conditioning.

Hauri and colleagues (Hauri, 1981; Hauri, Percy, Hellekson, Hartmann, & Russ, 1982) also demonstrated that neurofeedback was effective to treat patients with psychophysiologic insomnia. Patients in one study (Hauri et al., 1982) were allocated to either an EMG + theta feedback or EMG + SMR feedback treatment group, and received 26 sessions over 13 weeks. Home sleep logs revealed that both groups benefited

from the treatment. However, polysomnography (the gold standard of sleep monitoring) demonstrated that patients who were tense and anxious during training only benefited from theta training, while those who were relaxed benefited only from SMR training.

Neurofeedback has also been demonstrated to improve sleep spindle oscillations during sleep and decrease sleep onset latency time in healthy individuals in a randomized placebo controlled trial (Hoedlmoser et al., 2008). Twenty-seven healthy individuals completed 10 NFB sessions (control condition = randomized frequency conditioning), which resulted in an increase in the relative 12–15 Hz amplitude. This increased SMR activity was also expressed during subsequent sleep and purported to be the explanation for improved sleep.

A further randomized controlled trial was performed by Cortoos, Valck, Arns, Breteler, and Cluydts (2010), whereby EMG biofeedback was compared to SMR neurofeedback in patients with insomnia. SMR neurofeedback resulted in a greater increase in total sleep time when compared to EMG biofeedback. Finally, a recent and comprehensive investigation by Schabus et al. (2014) investigated the effects of NFB training compared to sham conditioning in 24 patients with primary insomnia after 10 SMR training sessions. The authors observed an increase in 12–15 Hz activity, which corresponded to a decrease in the number of awakenings (assessed by polysomnography) and an increase in slow wave sleep and subjective sleep quality.

From the limited but positive findings of the available literature, SMR neurofeedback has the potential to positively influence sleep, particularly in patients with poor sleep, via increasing activity in the 12–15 Hz range. Based on available research, a total of between 10–20 sessions is likely required to consolidate effects. While the long-term effects of NFB on insomnia are not clear, based on research in ADHD, it has been postulated that the effects of NFB on insomnia may persist well beyond treatment cessation (Cortoos et al., 2006). Thus, while further research is required, NFB has the potential to be a new tool in the treatment of insomnia.

References

American Academy of Sleep Medicine. (2014). *International classification of sleep disorders, Version 3*. Darien, IL: Author.

Buysse, D. J., Germain, A., Hall, M. L., Moul, D. E., Nofzinger, E. A., Begley, A., … Kupfer, D. J. (2008). EEG spectral analysis in primary insomnia: NREM period effects and sex differences. *Sleep, 31*(12), 1673–1682.

Cortoos, A., Valck, E. D., Arns, M., Breteler, M. H., & Cluydts, R. (2010). An exploratory study on the effects of tele-neurofeedback and tele-biofeedback on objective and subjective sleep in patients with primary insomnia. *Applied Psychophysiology and Biofeedback, 35*(2), 125–134.

Cortoos, A., Verstraeten, E., & Cluydts, R. (2006). Neurophysiological aspects of primary insomnia: Implications for its treatment. *Sleep Med Reviews, 10*(4), 255–266.

Hauri, P. (1981). Treating psychophysiologic insomnia with biofeedback. *Archives of General Psychiatry, 38*(7), 752–758.

Hauri, P. J., Percy, L., Hellekson, C., Hartmann, E., & Russ, D. (1982), The treatment of psychophysiologic insomnia with biofeedback: A replication study. *Biofeedback and Self-Regulation, 7*(2), 223–235.

Hoedlmoser, K., Pecherstorfer, T., Gruber, G., Anderer, P., Doppelmayr, M., Klimesch, W., Schabus, M. (2008). Instrumental conditioning of human sensorimotor rhythm (12–15 Hz) and its impact on sleep as well as declarative learning. *Sleep, 31*(10), 1401–1408.

Kay-Stacey, M., & Attarian H. (2016). Advances in the management of chronic insomnia. *The BMJ*, 354, i2123.

Laudon, M. & Frydman-Marom, A. (2014). Therapeutic effects of melatonin receptor agonists on sleep and comorbid disorders. *International Journal of Molecular Sciences, 15*(9), 15924–15950.

Perlis, M. L., Giles, D. E., Mendelson, W. B., Bootzin, R. R., & Wyatt, J. K. (1997). Psychophysiological insomnia: The behavioural model and a neurocognitive perspective. *Journal of Sleep Research, 6*(3) 179–188.

Schabus, M., Heib, D. P., Lechinger, J., Griessenberger, H., Klimesch, W., Pawlizki, A., … Hoedlmoser, K. (2014). Enhancing sleep quality and memory in insomnia using instrumental sensorimotor rhythm conditioning. *Biological Psychology, 95*, 126–134.

Sterman, M. B., Howe, R. C., & Macdonald, L. R. (1970). Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. *Science. 167*(3921), 1146–1148.

Shona L. Halson

Correspondence: Shona L. Halson, PhD, Department of Physiology, AIS, P.O. Box 176, Belconnen, ACT 2617, Australia, email: shona.halson@ausport.gov.au.