

# An Exploratory Study on the Effects of Tele-neurofeedback and Tele-biofeedback on Objective and Subjective Sleep in Patients with Primary Insomnia

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**Abstract** Insomnia is a sleeping disorder, usually studied from a behavioural perspective, with a focus on somatic and cognitive arousal. Recent studies have suggested that an impairment of information processes due to the presence of cortical hyperarousal might interfere with normal sleep onset and/or consolidation. As such, a treatment modality focussing on CNS arousal, and thus influencing information processing, might be of interest. Seventien insomnia patients were randomly assigned to either a tele-neurofeedback ( $n = 9$ ) or an electromyography tele-biofeedback ( $n = 8$ ) protocol. Twelve healthy controls were used to compare baseline sleep measures. A polysomnography was performed pre and post treatment. Total Sleep Time (TST), was considered as our primary outcome variable. Sleep latency decreased pre to post treatment in both groups, but a significant improvement in TST was found only after the neurofeedback (NFB) protocol. Furthermore, sleep logs at home showed an overall improvement only in the neurofeedback group, whereas the sleep logs in the lab remained the same pre to post training. Only NFB training resulted in an increase in TST. The mixed results concerning perception of sleep might be related to methodological issues,

such as the different locations of the training and sleep measurements.

**Keywords** Primary insomnia · Sleep disorders · Treatment · Neurofeedback · Biofeedback

## Introduction

One of the main characteristics of primary insomnia, a common sleep disorder in our society, is the presence of conditioned arousal, more specifically somatic, cognitive and/or cortical hyperarousal. The behavioural perspective (Spielman 1986), being one of the most dominant theoretical frameworks for the explanation of insomnia, posits that the presence of physiological or cognitive hyperarousal interferes with normal sleep-onset and –maintenance processes (Perlis et al. 1997). The physiological hyperarousal in patients with insomnia is reflected by elevated heart rate, body temperature, cortisol levels, and whole body metabolic rate (Bonnet and Arand 1995; Vgontzas et al. 2001; Rodenbeck et al. 2002). Cognitive hyperarousal, in turn, is characterised by intrusive and often negative cognitions close to bedtime (Robertson et al. 2007) and during sleep onset, as well as during periods of wakefulness during the night (Libman et al. 1997; Wicklow and Espie 2000; Harvey and Tang 2003). In this context, it has been observed that patients with insomnia perceive daily stressors and major life events as more stressful in comparison to healthy sleepers, resulting in higher presleep arousal at bedtime, which in turn is correlated with decreased sleep quality (Morin et al. 2003). The third arousal component has been introduced by Perlis et al. (1997) as part of a new perspective on insomnia, namely the neurocognitive model. This theoretical framework is an

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extension of the behavioural model, with an additional third arousal component, i.e. cortical arousal, reflected by heightened levels of high frequency EEG activity (beta and gamma power) during sleep onset (SO) and polysomnographic (PSG) sleep. Patients experiencing primary insomnia appear to exhibit higher levels of relative beta power during wakefulness in the sleep onset period (SOP; Lamarche and Ogilvie 1997), higher beta and gamma power during NREM sleep (Perlis et al. 2001; Krystal et al. 2002) especially during the second part of the night (Perlis et al. 2001), as well as during REM sleep (Merica et al. 1998). The neurocognitive perspective posits that the presence of these high EEG frequencies might explain the excessive discrepancies often seen in patients with insomnia between the subjective and objective sleep measurements. Standard Rechtschaffen and Kales (1968) sleep staging does not take into account the microanalysis of the sleep EEG and evaluation of high frequency activity such as beta and gamma power, which are correlated with sensory and information processes. Furthermore, in a preliminary study Perlis et al. (2001) showed that the meso-grade amnesia at sleep onset is attenuated in insomnia as reflected by a better retrieval of information from peri-sleep onset intervals. This cognitive process also appears to be correlated with relative beta power, as was shown by Wyatt et al. (1997) in healthy sleepers.

Besides the link between high beta EEG activity and cognitive processes, other studies have already shown the strong correlation between stress, cognitive and cortical arousal. Taken in to account that patients with insomnia report high stress levels during daytime functioning, and thus more cognitive arousal at bedtime, the increased high beta power during SO and sleep might also be related to the stress and anxiety. De Valck et al. (2004) showed that experimentally induced cognitive arousal in healthy sleepers resulted in increased subjective arousal, augmented cortical arousal reflected by high beta EEG power, as well as increased sleep latency.

Non-pharmacological treatment interventions for primary insomnia are based on the behavioural model, as such focussing on maladaptive sleeping habits, physiological and cognitive conditioned arousal and dysfunctional beliefs (Morin et al. 1999; Cervena et al. 2004). Cognitive Behavioural Therapy (CBT) has significant impact on both subjective and objective sleep parameters and is considered the treatment of choice for chronic primary insomnia (Morin et al. 1994; Murtagh and Greenwood 1995). However, CBT has limitations and is still open for enhancement. First of all, the average improvement after CBT does not bring the majority of patients experiencing insomnia in to the good sleeper range and observed effect sizes are smaller in comparison to the use of CBT in other disorders (Harvey 2002). Secondly, approximately 20% of

patients with insomnia do not respond to treatment (Morin et al. 1994; Harvey 2002). These findings suggest that the focus on cognitive and physiological arousal might not be enough, and a redirection to cortical arousal as an additional focus of treatment might be of interest (Cortoo et al. 2006). As such, EEG biofeedback or neurofeedback (NFB) can be an interesting tool in the non-pharmacological treatment of insomnia. NFB is a self-regulation method that makes use of operant conditioning, and intervenes on the level of the central nervous system (CNS). During training, the EEG is recorded and the patient receives instant feedback (auditory and/or visually) on the cortical activity of the brain. In a pioneering study, Serman et al. (1970) showed that a neurofeedback training of the sensorimotor rhythm (SMR, 12–14 Hz) in the wake EEG in cats resulted in a change in the sleep EEG. An increase in sleep spindle bursts and quiet sleep was observed, as well as a suppression of motor activity. Hauri (1981) tested the effect of SMR neurofeedback as well as theta neurofeedback + EMG frontalis biofeedback training, and EMG training only. Results showed no significant difference between all three training protocols. Post hoc analysis, however, revealed a correlation between baseline tension level and the outcome in both neurofeedback protocols. High baseline tension level resulted in better outcome after theta + EMG training, while low tension levels at baseline was an indication for SMR training. In a replication study Hauri et al. (1982) compared theta and SMR training and revealed the same results, suggesting that neurofeedback may have significant impact on patients with insomnia. However, he did not find a difference in the sleep diaries related to the appropriate feedback training. The studies by Hauri focussed primarily on one EEG frequency band during training and were concentrating on frequencies linked to sleep onset processes. In light of the neurocognitive perspective and the possible cortical hyperarousal related to information processes during sleep onset, an intervention on the level of information processes using neurofeedback training might be very promising. Indeed, training of SMR in combination with the inhibition of theta and high beta power in healthy controls has an impact on sensorimotor control resulting in improvement in attention processes (Hauri 1981; Hauri et al. 1982).

Summarizing, the presence of high frequency EEG activity during SO and sleep, may interfere with ongoing information processes (Perlis et al. 1997), which in return may partially explain the impairments in perception of wake and sleep in insomnia. Recent meta-analysis on CBT in patients with insomnia (Harvey 2002) suggest that a focus on cortical arousal might be of interest, since a substantial group of patients do not respond well enough to treatment focussing primarily on cognitive and physiological arousal components. In this single-blind exploratory

study we applied a neurofeedback protocol targeting several frequencies related to sleep and cognitive processes and compared this to an EMG biofeedback protocol. First of all, since the raise of theta power (4–8 Hz) is correlated with drowsiness (Ogilvie 2001) and low resting power is associated with good performance (Klimesch et al. 2007), we pursued the inhibition of this EEG frequency. Secondly, we reinforced SMR. Several studies have already shown its relationship with (optimal) cognitive performance (Vernon et al. 2003; Eegner and Gruzelier 2001, 2004; Sterman 1996; Hanslmayer et al. 2005) as well as sleep improvement and sleep spindle density (Sterman et al. 1970; Hauri 1981; Hauri et al. 1982; Berner et al. 2006). Thirdly, high beta power (20–30 Hz), which appears to be linked with cognitive processes (Basar-Eroglu et al. 1996; Jefferys et al. 1996; Makeig and Jung 1996) and arousal (De Valck et al. 2004), and is increased in patients experiencing insomnia during the SOP (Lamarche and Ogilvie 1997) and sleep (Perlis et al. 2001), was inhibited. The main goal was not to actively decrease the amount of high beta power, but stabilize the reactivity on arousal and stress reflected by the possible instability of this EEG frequency band. Serving as an active control group, a second group of patients with primary insomnia received EMG biofeedback. It has been shown that muscle relaxation alone (EMG biofeedback training or progressive relaxation training) can result in SO improvements, without being correlated with the actual decrease in EMG activity, suggesting that this resembles a placebo effect (Freedman and Papsdorf 1976; Nicassio et al. 1982). Innovating in this study is the use of tele-neurofeedback or tele-biofeedback: to diminish the commuting to/from the hospital—a minimum of 20 sessions/two or three times a week were recommended—the training was performed at home with an internet connection. To our knowledge, this is the first tele-neurofeedback study performed. It was hypothesized that the neurofeedback group will show greater improvement after training in comparison to the EMG biofeedback group on polysomnographic (objective) and sleep diary (subjective) data. The primary outcome variable was TST.

## Methods

### Subjects

Patients experiencing primary insomnia were recruited through clinical sleep centres and primary care physicians. First there was a short screening interview by phone to check for possible medical conditions, medication use, and the availability of a personal computer and internet connection. Afterwards possible candidates were invited for a full screening session. During an interview several

questionnaires were used: Pittsburgh Sleep Quality Index (PSQI; Buysse et al. 1989), Epworth Sleepiness Scale (ESS; Johns 1991) Athens Insomnia Scale (AIS; Soldatos et al. 2000), State Trait Anxiety Index (STAI; Spielberger 1983), Beck Depression Inventory (BDI; Beck et al. 1988), Presleep Arousal Scale (PSAS; Nicassio et al. 1985) with a somatic (PSAS SOM) and a cognitive (PSAS COG) subscale and the Mini International Neuropsychiatric Interview (MINI; Sheehan et al. 1988). Once enrolled in the study subjects received an explanation of the complete procedure and signed an informed consent. Of the 158 subjects with sleep complaints who wanted to join the study, 102 subjects were excluded (30 with comorbid medical complaints, 13 had new born children, 15 performed shift work, 9 patients were of age > 60, and 35 used medication on a daily basis). 56 subjects were invited for an interview, but 29 patients were excluded because of unwillingness to remain medication free during the entire study period (17), additional emotional or psychiatric problems (7), BMI > 30 (2), or self-induced sleep shortage (3). 27 patients with insomnia were invited for a polysomnography. 7 more patients were excluded because of excessive snoring (2), decreased REM latency (1), or no objective sleep disturbances (4). 20 patients with insomnia fulfilled the in- and exclusion criteria (see below). However, two subjects declined just before the start of the training because of sudden unavailability during the training period, and 1 participant had technical problems with her PC.

Seventeen patients with insomnia were randomly assigned to a neurofeedback (NFB;  $n = 9$ ) or biofeedback (BFB;  $n = 8$ ) group. The neurofeedback group consisted of three women and six men (mean age 41.5), the biofeedback group consisted of three women and five men (mean age 43.8). They were informed of the existence of two different training protocols but did not know to which one they were assigned.

Twelve healthy sleepers, 7 men and 5 women (mean age 44.4), participated in the study as a control group for baseline sleep comparisons. They were recruited through the university newsletter, or were friends from patients with insomnia who already joined the study, and underwent a similar screening session.

This study was evaluated and approved by the Medical Ethics Committee of the Brussels University Hospital.

### Inclusion and Exclusion Criteria

- Patients with insomnia between 18 and 60 years of age had to report either a sleep onset problem (latency >30 min), sleep maintenance problem (wake after sleep onset >30 min), sleep complaints minimum 3/week, and duration of insomnia >6 months. Impairment in

daytime functioning had to be present (first evaluated with PSQI and AIS, followed by a semi-structured psychiatric interview) and all participants had to be medication-free for at least 4 weeks before the start of the study, as well as during the whole study. All psychiatric or medical disorders were excluded, except for a positive response in the M.I.N.I. on dysthymia and/or generalized anxiety disorder when it was clearly related to their sleep complaints. Further exclusion criteria for all subjects: students, shift workers, pregnancy, consumption of more than two alcohol units/day for woman and three alcohol units/day for men, consumption of more than five caffeine beverages/day, phase delayed or phase advanced syndrome, abnormal bedtime hours (<09:30 pm) or irregular sleep-wake schedule, parents with newborns, excessive daytime sleepiness (ESS > 13 and subjective report of difficulty staying awake during the day), presence of other primary sleep disorders (RLL, PLM, sleep apnoea,...), BMI > 30.

### Sleep Diary and Actigraph

Two weeks before they came to the sleep laboratory, participants were asked to fill in a sleep diary and wear an actigraph during the night, to check for irregular sleep-wake schedules. The morning after the polysomnography, all participants were asked to fill in a morning questionnaire evaluating their night in the sleep lab, consisting of the Brussels Indices of Sleep Quality (BISQ) and a PSAS. At the beginning of training they were also asked to keep a sleep diary during the complete training protocol to monitor perception of sleep. Following variables were calculated from the sleep diary: Total Sleep Time (TST), Sleep Latency (SL), Wake after sleep onset (WASO), Sleep Efficiency (SE) and Time in Bed (TIB).

### Polysomnography

Sleep measurements were performed at the experimental sleep laboratory at the Vrije Universiteit Brussel. All participants underwent 1 polysomnography (PSG) before and after training. The recording montage consisted of 3 EEG electrodes referenced to a single mastoid (F3-A2, C4-A1, O1-A2), 2 EOG electrodes referenced to a single mastoid (LOC, ROC), a bipolar submentalis EMG, tibialis EMG, and EKG. A 32 channel Embla N7000 recording system was used (Medcare) with a DC offset of 500 mV max and a fixed DC low cut filter at 0.3 Hz. The signal was digitized at a sampling rate of 500 Hz using Somnologica Software. The EEG and EOG signals were high pass filtered at 0.5 Hz and low pass filtered at 40 Hz, EMG channels were high pass filtered at 5 Hz and low pass filtered at 70 Hz.

All data was scored in 30-s epochs according to the Rechtschaffen and Kales (1968) rules by a trained specialist, unaware of the training condition of the subjects. Outcome variables were Total Sleep Time (TST), Sleep Onset Latency (SOL), Wake After Sleep Onset (WASO), Sleep Efficiency (SE), % Slow Wave Sleep (SWS) of the Sleep Period Time (SPT), % REM sleep of the SPT, % Stage 1 sleep (S1) of the SPT and % Stage 2 sleep (S2) of the SPT. Additionally, the EMG level (Root Mean Square  $\mu$ V) of the first period of wakefulness during the sleep onset period was also analysed as a measure of baseline tension level. Movement artefacts were excluded from analysis.

### Neurofeedback/Biofeedback Training

Neurofeedback training sessions were performed at the homes of the subjects. A PC minimum Pentium IV with an internet connection and USB connection were required and Windows XP SP2 as operating system. Subjects used Ag/AgCl disposable snap-on electrodes located on Fpz—A2 and Cz-A2 with a ground electrode on A1. Training was performed using the Personal Efficiency Trainer (PET; Brainquiry, B.V.) which communicated through a wireless Bluetooth connection with SleepNF software (Brainquiry B.V.), a program designed specifically for this study. Sampling rate was 200 Hz and a D/C amplifier was used. The neurofeedback (NFB) group had to increase SMR (12–15 Hz) and inhibit theta power (4–8 Hz) and high beta power (20–30 Hz) at Cz. The biofeedback (BFB) group had to decrease EMG at Fpz which was equated with the reinforcement of relaxation. To avoid any visual differences between both groups the feedback screens always showed three bars, representing the amplitude of three different frequency bands. For the NFB group the bars represented, respectively, the amplitude of theta, SMR and high beta power. The BFB group received feedback on the 50 Hz frequency (as a measure of signal quality) of Cz, EMG power at Fpz (frontal EMG), and at Cz. The frontal EMG power was reversed when visualised in the bar on the screen, meaning that an increase was equal to a decrease in EMG. This resulted in identical screens for both conditions. The thresholds for the feedback were initially based on a baseline measurement at the first session. The following formula was used:

- *Reinforcers: mean amplitude + (standard deviation/4) : SMR and EMG*
- *Inhibitors: mean amplitude + (standard deviation/2) : theta, high beta power and 50 Hz*

For the following sessions, the threshold of the previous session was used. Each training session consisted of 4 times 5 min. In between subsessions the thresholds could be altered, depending on the % above or below threshold

(% of feedback). For the reinforcers, if feedback dropped below 20% or increased above 75%, the threshold was reset based on the mean and standard deviation values of the previous subsession. For the inhibitors the feedback range was set at 60–90%. Using a secured internet connection and VNC software (RealVNC Ltd.), the subjects logged on to the university computer system and the therapist gained control of their PC's. As such, it resembled as if the participants were at the sleep lab.

## Procedure

Subjects came to the lab for the baseline sleep measurement around 8:00 pm and received information on the procedure of the evening and purpose of the measurement. Around 8:30 pm the electrodes for the night were applied. Subjects went to bed between 10:30 pm and 12:00 pm, depending on their usual bedtime. Time in bed was approximately 7 h and 30 min and was kept stable for every subject. The next morning they were asked to fill in the Brussels Indices of Sleep Quality (BISQ) and PSAS to monitor their subjective sleep quality.

After the baseline sleep measurement, a training day was organised for all participants of the NFB/BFB protocol. Since the training sessions were at home, subjects were required to put on the electrodes themselves. They received an explanation and hands-on training on electrode placement and the use of the hard- and software. Afterwards every subject received a kit containing all the necessary products and installation disks for the software, as well as a detailed manual for the training procedures at home.

NFB/BFB training consisted of 20 sessions with an altering frequency of 2 or 3/week for a total period of 8 weeks. These two time groups were independent of training group and based on the individual preference of every subject. The first group started with 2/week during the first week, then 3/week during the second week, etc.... the second group used the opposite time table. Sessions were held at home between 6:00 pm and 10:00 pm. All subjects received a strict timetable, meaning that subjects received all their training sessions at the same time of day during the whole study. An average of 18 sessions was performed, with a minimum of 15 and a maximum of 21. The following procedure for the NFB/BFB sessions was used: (1) subjects placed the electrodes 10 min before their scheduled appointment, (2) the therapist called the subjects by phone and they were asked to log on to the university system (3) the therapist gained control over the PC of the subjects and the software program was initiated, (4) the signal quality was checked by ensuring the line noise (50 Hz) was below 10  $\mu$ V and if necessary electrodes were replaced, (5) subjects were asked if they had any questions, if not the phone connection was interrupted and the session

began. If no technical problems occurred, there was no more contact between the participant and the therapist.

Two weeks after the training was completed all participants returned to the sleep lab for their final PSG. The same procedure was used as described before.

## Statistical Analysis

All data were analysed using STATISTICA software. Baseline PSG variables were examined between the 2 insomnia groups and the controls using an Independent Samples *t*-test to check for difference in sleep variables between both groups. A Shapiro–Wilk *W* and a Levene's Test of homogeneity of variances were used to test the assumptions of parametric testing. If violations occurred, the non-parametric Man–Whitney *U* test was used. Pre- to post treatment changes in all sleep parameters were examined using a  $2 \times 2$  repeated measures ANOVA, with group (biofeedback, neurofeedback) as a between factor and time (pre-treatment, post treatment) as a within factor. If assumptions were violated, a non parametric Friedman ANOVA was used to assess pre to post treatment changes within one group. Significant level was set at  $\alpha = 0.05$ .

## Results

### Baseline Measurements

The clinical and demographic data of the subjects are displayed in Table 1. As mentioned before, variables that did not meet the assumptions for a parametric test, were analysed using a Mann–Whitney *U* test. No significant

**Table 1** Clinical and demographic data of the insomnia neurofeedback (NFB), biofeedback (BFB) groups, and controls

	Insomnia NFB ( <i>n</i> = 9)	Insomnia BFB ( <i>n</i> = 8)	Controls ( <i>n</i> = 12)
Duration insomnia (years)	10.7 (10.5)	14.3 (10)	–
Age	41.5 (9.5)	43.8 (9.5)	44.4 (7.8)
STAI 1	35.1 (3.8)*	33.0 (7.1)	27.7 (6.3)
STAI 2	43.2 (7.1)*	41.0 (7.5)	34.6 (9.0)
BECK	7.0 (5.5)	4.1 (4.7)	2.6 (3.0)
AIS	11.8 (3)*	12.6 (4.1)*	2.1 (1.5)
PSQI	11.7 (2.1)*	11.2 (1.9)*	4.0 (1.8)
PSAS SOM	12.8 (5.8)	9.7 (1.7)	9.5 (1.6)
PSAS COG	20.8 (8.7)*	20.8 (8.8)	11.75 (2.3)
ESS	8.2 (4.8)	7.0 (5.3)	7.1 (2.3)

Values in table are mean (SD)

NFB neurofeedback, BFB biofeedback

\* Indicates significant difference with control group ( $p < 0.05$ )

differences were found between both insomnia groups for all variables. Between the insomnia groups and controls no age difference was found. The control group had a significant lower score on the AIS (NFB:  $z = 3.87$ ;  $p < 0.0005$ ; BFB:  $z = 3.74$ ;  $p < 0.0005$ ) and PSQI (NFB:  $z = 3.85$ ;  $p < 0.0005$ ; BFB:  $z = 3.72$ ;  $p < 0.0005$ ) in comparison to both insomnia groups, but only a significant difference for the STAI 1 ( $t(19) = 3.11$ ;  $p < 0.01$ ), STAI 2 ( $t(19) = 2.36$ ;  $p < 0.05$ ) and PSAS COG ( $z = 1.97$ ;  $p < 0.05$ ) was found in comparison to the NFB group. No significant differences were found for the BECK, PSAS SOM and the ESS.

Regarding the polysomnographic data there were no significant differences between both insomnia groups (Table 2). The insomniacs had a significantly higher WASO (NFB:  $z = 2.42$ ;  $p < 0.05$ ; BFB:  $z = 2.39$ ;  $p < 0.05$ ), lower SE (NFB:  $z = -2.34$ ;  $p < 0.05$ ; BFB:  $z = -2.62$ ;  $p < 0.05$ ) and higher arousal index (NFB:  $z = 2.91$ ;  $p < 0.005$ ; BFB:  $z = 3.32$ ;  $p < 0.001$ ) in comparison to healthy controls. The BFB group had a significantly lower TST ( $t(17) = -2.38$ ;  $p < 0.05$ ), higher SL ( $z = 3.08$ ;  $p < 0.005$ ) and

lower percentage of REM sleep ( $t(18) = -2.67$ ;  $p < 0.05$ ) in comparison to healthy sleepers. No significant differences were found between all groups for the SWS, S1 and S2. Analysis on the morning questionnaires were performed using the nonparametric Man–Whitney  $U$ -test. Both insomnia groups also differed significantly on the reported SL (NFB:  $z = 3.41$ ;  $p < 0.001$ ; BFB:  $z = 3.51$ ;  $p < 0.0005$ ), WASO (NFB:  $z = 3.12$ ;  $p < 0.005$ ; BFB:  $z = 3.55$ ;  $p < 0.0005$ ), TST (NFB:  $z = -2.81$ ;  $p < 0.005$ ; BFB:  $z = -3.70$ ;  $p < 0.0005$ ), SE (NFB:  $z = -3.34$ ;  $p < 0.05$ ; BFB:  $z = -3.70$ ;  $p < 0.0005$ ) and PSAS COG (NFB:  $z = 2.57$ ;  $p < 0.01$ ; BFB:  $z = 2.04$ ;  $p < 0.05$ ) in comparison to the controls.

NFB/BFB Training Effects

A  $2 \times 2$  repeated measures ANOVA on the polysomnographic data revealed a significant main effect pre to post treatment for SL ( $F(1,15) = 10.56$ ;  $p < 0.01$ ;  $r = 0.41$ ; Fig. 1) and WASO ( $F(1,15) = 5.77$ ;  $p < 0.05$ ;  $r = 0.27$ ; Fig. 2). The NFB group showed a decrease of 39.7% on the SL and 53.6% on the WASO, while the BFB group decreased on these parameters with 44.9 and 13.2%, respectively. An interaction effect was found for TST ( $F(1,15) = 5.03$ ;  $p < 0.05$ ;  $r = 0.25$ ; Fig. 3). Post hoc Tukey analysis revealed a significant increase in TST only for the NFB group ( $p < 0.05$ ). Concerning sleep architecture, a significant increase in REM sleep ( $F(1,15) = 4.80$ ;  $p < 0.05$ ;  $r = 0.24$ ) was found (Fig. 4). No significant differences were found in the sleep diaries in the laboratory.

To test the post-hoc assumption formulated by Hauri (Murtagh and Greenwood 1995; Harvey 2002) regarding

**Table 2** Baseline polysomnographic and sleep diary data of the insomnia neurofeedback (NFB), biofeedback (BFB) groups, and controls

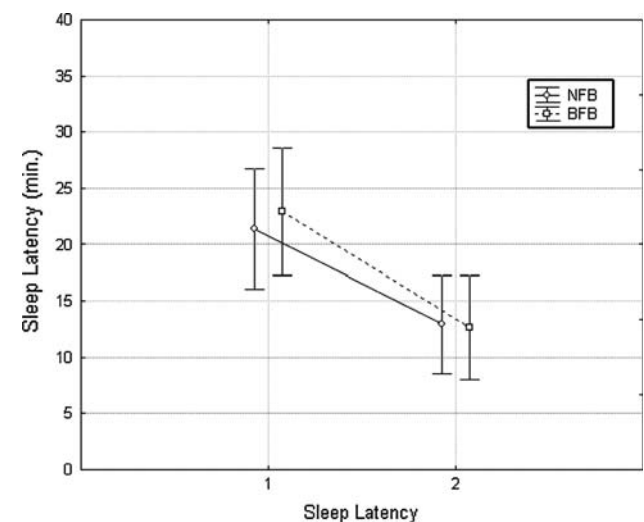
	Insomnia NFB ( <i>n</i> = 9)	Insomnia BFB ( <i>n</i> = 8)	Controls ( <i>n</i> = 12)
SL (min.)	21.4 (19.9)	22.9 (9.5)*	9.2 (4.5)
WASO (min.)	75.0 (42.9)*	82.2 (56.2)*	32.7 (21.5)
TST (min.)	380.6 (42.5)	364.4 (53.1)*	412.2 (31.5)
SE (%)	83.5 (9.1)*	81.8 (12.5)*	92.8 (4.7)
SWS (% SPT)	14.0 (4.2)	16.0 (6.1)	16.8 (8.2)
REM (% SPT)	19.3 (3.3)	17.2 (4.9)*	23.0 (4.7)
F1 (% SPT)	5.4 (2.5)	7.4 (3.7)	5.7 (1.6)
F2 (% SPT)	44.7 (5.6)	41.0 (10.8)	42.7 (13.4)
Awakenings	8.9 (6.1)	14.0 (8.5)	6.0 (2.7)
Arousal index	12.6 (4.8)*	16.2 (5.3)*	6.9 (2.9)
TIB (min.)	477.1 (15.9)	469.6 (27.5)	454.2 (38.8)
Sleep diary			
SL (min.)	43.9 (27.9)*	112.5 (133.9)*	10.7 (8.0)
WASO (min.)	90.0 (56.2)*	153.1 (117.0)*	18.7 (17.2)
TST (min.)	340 (82.1) <sup>#,*</sup>	255.0 (109.9) <sup>#,*</sup>	421.7 (32.5)
SE (%)	71.1 (17.3) <sup>#,*</sup>	54.6 (23.2) <sup>#,*</sup>	92.7 (5.3)
TIB (min.)	473.9 (16.2)	468.1 (23.7)	455.8 (40.1)
PSAS COG	15.7 (5)*	15.4 (5.9)*	10.5 (2.0)
PSAS SOM	10.0 (3.0)	9.4 (1.6)	8.6 (1.1)
Tension level			
EMG (RMS $\mu$ V)	6.18 (1.68)	7.44 (1.98)	6.10 (2.08)

Values in table are mean (SD)

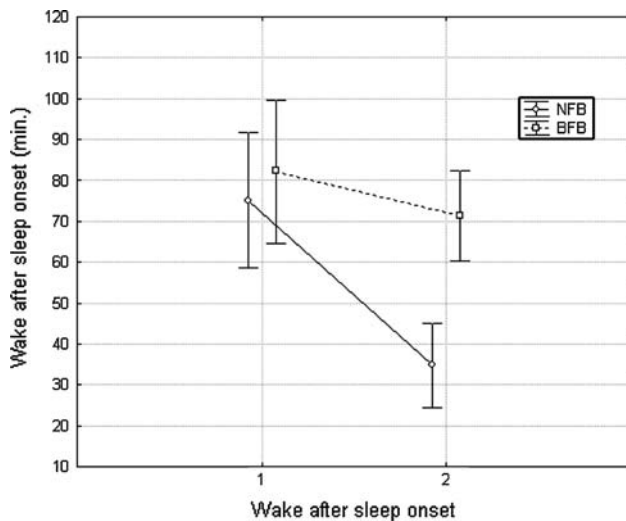
NFB neurofeedback, BFB biofeedback

\* Indicates significant difference with control group ( $p < 0.05$ );

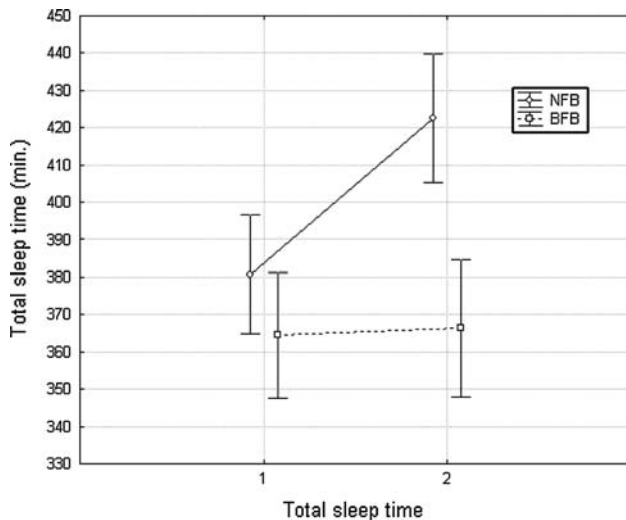
# indicates significant difference between both insomnia groups ( $p < 0.05$ )



**Fig. 1** Polysomnographically measured sleep latency (minutes) pre to post training. Vertical bars denote ( $\pm$ ) standard errors. SL sleep latency, NFB neurofeedback, BFB biofeedback



**Fig. 2** Polysomnographically measured WASO (minutes) pre to post training. Vertical bars denote ( $\pm$ ) standard errors. WASO wake after sleep onset, NFB neurofeedback, BFB biofeedback



**Fig. 3** Polysomnographically measured TST (minutes) pre to post training. Vertical bars denote ( $\pm$ ) standard errors. TST total sleep time, NFB neurofeedback, BFB biofeedback

the correlation between baseline tension level and training outcome, a Spearman Rank correlation was calculated between the baseline EMG level and improvement on the different sleep parameters for each training group, but no significant correlations were found.

Finally, we also compared the sleep logs at home 1 week before PSG measurement at the sleep laboratory pre to post treatment, using a Friedman ANOVA (Table 3). Results show a significant decrease in SOL ( $\chi^2 = 4.5; p < 0.05; r = 0.49$ ), WASO ( $\chi^2 = 4.5; p < 0.05; r = 0.59$ ), and an increase in TST ( $\chi^2 = 4.5; p < 0.05; r = 0.56$ ) and SE ( $\chi^2 = 8.0; p < 0.005; r = 0.63$ ) in the NFB group. Patients with insomnia receiving the EMG biofeedback training only improved in SE ( $\chi^2 = 4.5; p < 0.05; r = 0.63$ ).

### Discussion

#### Effects on Objective Sleep Parameters

Our study showed that a specific neurofeedback protocol targeting cognitive processing induced greater objective and subjective sleep changes in comparison to EMG biofeedback. An overall improvement in SL was observed, irrespective of training group, which is not surprising since our study sample dominantly comprised of patients experiencing sleep-maintenance insomnia. Only the insomnia group receiving a neurofeedback training focussing on inhibition of theta and high beta, as well as reinforcement of SMR, showed a significant increase in TST. Furthermore, the overall increase in REM sleep pre to post treatment can be seen as a direct result of the decreased WASO, since our patients with insomnia were dominantly sleep-maintenance insomniacs characterised by more wakefulness during the second part of the night.

The NFB protocol used in this study therefore appears to have a different impact on sleep than standard SMR training used by Hauri (1981) and Hauri et al. (1982). The post-hoc relation between baseline EMG tension level and

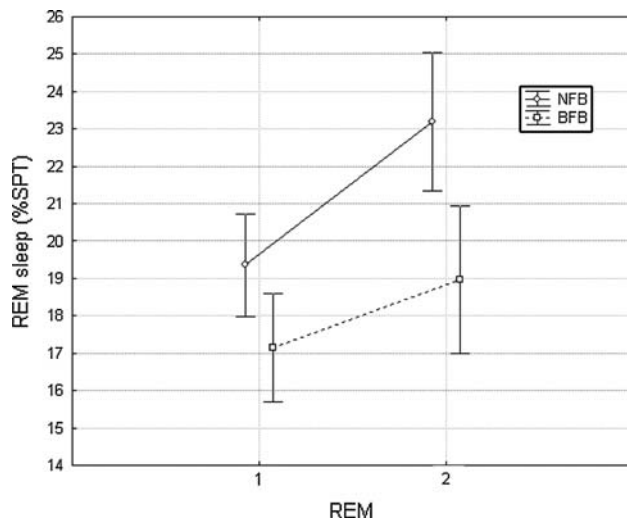
**Table 3** Sleep logs at home before and after training

Sleep logs at home	NFB Pre	Group (n = 8) Post	Effect (r)	BFB Pre	Group (n = 8) Post	Effect (r)
SL (min.)	44.0 (31.1)	31.0 (23.4)	0.49*	41.9 (23.9)	30.0 (15.0)	0.33
WASO (min.)	110.8 (63.4)	66.7 (36.3)	0.59*	136.8 (83.17)	103.5 (67.3)	0.42
TST (min.)	348.1 (67.2)	392.0 (66.0)	0.56*	322.3 (61.2)	359.5 (46.7)	0.38
SE (%)	70.0 (13.5)	79.7 (9.4)	0.63*	64.3 (12.8)	73.6 (12.0)	0.63*
TIB (min.)	498.8 (26.8)	490.3 (36.3)	0.14	501.1 (26.6)	493.2 (28.7)	0.21

Values in table are mean (SD)

NFB neurofeedback, BFB biofeedback, r effect size

\* (Group) indicates significant effect pre to post treatment ( $p < 0.05$ )



**Fig. 4** Polysomnographically measured REM (% SPT) pre to post training. Vertical bars denote ( $\pm$ ) standard errors. TST total sleep time, NFB neurofeedback, BFB biofeedback

treatment outcome that they found, was not apparent in our study sample. However, considering the fact that the patients with insomnia in this study did not show an increased amount of EMG tension, nor a higher reported somatic arousal at bedtime in comparison to healthy sleepers, suggest that somatic arousal was not dominantly present in our patient sample. Yet, baseline measures in our study did reveal the presence of more cognitive arousal during the SOP both at home as in the laboratory setting. On the one hand this corresponds to the study of Robertson et al. (2007) showing that around bedtime mostly cognitive arousal instead of somatic arousal is increased. On the other hand, it might just be a demonstration of the heterogeneous nature of the insomnia population when considering the different forms of arousal.

Several hypotheses are possible as an explanation for the presented effects of NFB on objective sleep parameters. First of all, Perlis et al. (1997) hypothesized that the presence of cortical hyperarousal during SO and sleep in patients experiencing insomnia, reflected by high frequency activity, may result in a disruption of ongoing information processes which in turn can interfere with normal sleep initiation and/or consolidation processes. Training in healthy subjects of the specific EEG frequencies we used in our study has previously resulted in an optimisation of attentional processes (Egner and Gruzelier 2001, 2004). Applying a NFB protocol intervening on the level of cognitive processing, may thus have had an influence on cortical arousal and information processing during sleep, resulting in an increase of TST. On a more concrete level, by inhibiting high beta (20–30 Hz) during NFB training we tried to intervene directly on the reactivity to stress and arousal. Furthermore, Serman et al. (1970)

showed that the increase of SMR resulted in a facilitation of sleep spindle bursts and quiet sleep. This in turn might have had an influence on the consolidation of sleep in our subjects and might explain the positive impact on sleep duration that we observed.

#### Effects on Subjective Sleep Parameters

Analyses of the sleep diaries at home, revealed an improvement on all sleep variables only in the NFB group, which is in contradiction with the results of Hauri et al. (1982). They found an improvement of subjective sleep irrespective of feedback protocol, suggesting that it was not the training itself causing this shift in perception. Furthermore, the sleep diaries at the laboratory did not change pre to post treatment, while the objectively measured sleep improved. Several annotations can be made to clarify these results. First of all, the perception of sleep appears to be a complex phenomenon, possibly related to more than just cortical arousal. The fact that the cognitive arousal subscale of the PSAS measured at the sleep laboratory did not show a decrease after treatment supports this hypothesis. Secondly, a discrepancy between objective and subjective measures is often reported in studies, and neurofeedback research is not an exception. This phenomenon has been reported by Egner et al. (2002) evaluating alpha/theta training and activation levels, as well as Egner and Gruzelier (2003) when examining the effect on performance anxiety. This change in perception often has been addressed as a feeling of relief or hope for change.

The intriguing difference in perception of sleep quality between the home and laboratory sleep logs might be related to methodological aspects. One major difference between all previous reported NFB/BFB studies and ours is the procedure of the training. Traditionally the training is performed in an experimental room with a therapist present, but our subjects performed the training at home without the physical presence of a therapist. This aspect potentially has a large impact on the results. Additional therapeutic effects caused by the direct interaction between patient and trainer are excluded, while at the same time transfer of the therapeutic results are optimized to the home environment. Since the sleep measurements took place at the laboratory, it is possible that the training effects were only transferred to their home sleep environment and not to the sleep laboratory bedrooms.

There are some limitations that need to be accounted for when interpreting these results. First of all, the sample sizes of our groups were small, affecting the power of our results. Secondly, the use of tele-neurofeedback holds a few disadvantages as well. Although it diminishes the time consuming aspects of training, the environment is not controlled for all participants. The way that participants

paid attention to the feedback and/or disruptive influences from distracting factors (noise, children, family) are not known in such a home protocol. Furthermore, only one PSG measurement was used, no waiting list was included for comparison of sleep changes over time, and no long term follow-up was performed. Moreover, due to technical difficulties with data storage of the training sessions, analysis of the possible changes in the EEG frequencies trained during the sessions was not possible in this exploratory study. Future studies comparing standard face-to-face neurofeedback with tele-neurofeedback are needed to clarify the possible outcome differences between both protocols. The use of our specific NFB protocol needs to be examined as well on the level of specific cognitive and information processes, such as ERP experiments before and during sleep, as well as microanalysis of the sleep EEG to clarify which mechanisms might have triggered the increase in TST. Finally, in our study we only used an inhibition paradigm for high beta EEG power focussing on the possible reactivity reflecting an increase in arousal and stress. In light of the reported literature on EEG and insomnia, future studies including a down training of high beta power, might be very interesting to validate the neurocognitive model of insomnia.

## Conclusion

The present study is innovating in two ways. First of all, our subjects performed the training at home through a secured internet connection, as such being less time-consuming than an in-hospital (EEG) biofeedback training (Cortooos et al. 2006). The fact that no drop-outs occurred, suggests that in this way it was possible to keep motivation sufficiently high in such an intense program (2–3 sessions a week). Secondly, we used a specific NFB protocol related to cognitive processes and associated EEG frequencies, which has never been applied in this patient population. Our analyses show that both EMG training as well as NFB training have a positive influence on the objective SL measured by a polysomnography, but an increase in TST was only obtained after the NFB training. Concerning the subjective report of sleep quality, the NFB training resulted in an overall improvement of subjectively measured sleep at home, but not in the lab, while the BFB group showed no improvement on the sleep logs in both locations. It is possible that the methodological differences in the procedure, such as the different locations for training and sleep measurements, are responsible for the discrepancy in perception of sleep improvement in the lab versus at home.

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