The Use of EEG Theta Biofeedback in the Treatment of a Patient with Sleep-Onset Insomnia

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In this report, the treatment of a 42-year-old female with a complaint of chronic sleep-onset insomnia is described. Following the unsuccessful use of relaxation training, treatment consisted of 11 sessions of EEG theta rhythm (4–7 Hz) biofeedback. Theta density and five sleep indices were monitored throughout baseline, placebo, and treatment sessions. A significant increase in theta density was accompanied by reports of a decrease in sleep latency and an increase in total sleep time. This improvement was maintained after withdrawal of medication and at 3-month follow-up.

It is estimated that between one-sixth and one-fourth of the adult population is affected by sleep problems (Shepherd, Cooper, Brown, & Kalton, 1966; Dunnell & Cartwright, 1972). The most prevalent sleep disorder is insomnia, and it is one of the commonest problems encountered in medical practice (Johns, 1972). Hammond and Garfinkel (1969), in perhaps the largest survey to date, noted that in their sample, 13% of men and 26% of women over the age of 30 complained of insomnia. A wide variety of patient complaints and EEG disturbances are found (Shimizu, Shiotsuki, & Ichino, 1970), suggesting that there are probably several different subtypes of insomnia. Night to night variability is also a characteristic of the sleep of insomniacs (Frankel, Buchbinder, Coursey, & Snyder, 1973).

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Treatment of insomnia has been primarily pharmacologically based, and over half of those individuals complaining of insomnia are thought likely to be using one or more sedative drugs on a regular basis (Guilleminault, Spiegel, & Dement). Dunlop (1970) estimated that in Great Britain 1 night's sleep in 10 is induced by hypnotic drugs. The use of such drugs to treat insomnia has been criticized by Ribordy and Denny (1977) because of (a) tolerance effects, (b) carry-over effects, (c) alterations in sleep pattern, (d) rebound effects, and (e) attributional effects.

Given these limitations, it is not surprising that psychological approaches have been employed as alternatives to drug therapy. Behavioral treatments are summarized by Montgomery, Perkin, and Wise (1975) and by Ribordy and Denny (1977). The majority of these are relaxation-based, on the assumption that heightened physiological arousal is a feature of insomniacs and that behavioral techniques derived for the reduction of arousal would therefore be appropriate. Such techniques have included applied relaxation, systematic desensitization, EMG biofeedback, and electrosleep. Although some success has been claimed for all of these, firm evidence that insomniacs are more physiologically aroused than normals is lacking (Hauri, Phelps, & Jordan, 1976). Alternative techniques, deriving from other views of the etiology and/or maintenance of insomnia, include stimulus control procedures (Bootzin, 1972), cognitive control procedures (Mitchell & White, 1977), and attribution therapies (Storms & Nisbett, 1970).

A recent development has been the use of EEG theta biofeedback (Budzynski, 1973). Budzynski contends that muscular relaxation may not necessarily lead to brain-wave patterns characteristic of presleep drowsy states. This period is characterized by the breakup of alpha rhythms and the emergence of low-amplitude theta frequency (4–7 Hz) activity. Using an elaborate treatment sequence comprising frontalis EMG training, alpha EEG training, and EEG theta training, Budzynski has reported improvements in sleep-onset latencies for 6 out of 11 chronic insomniac clients, but inadequate information is given regarding assessment procedures and training, and no controls were used. Furthermore, it is impossible to determine the crucial elements of the procedure.

**CASE HISTORY**

The patient was a 42-year-old, married, female secretary. Her difficulty in falling asleep began in 1968 and was of gradual onset. No precipitants could be discerned and she was in good general health, with no history of psychiatric illness. Her only other complaint was of occasional migraine,
occurring usually premenstrually and at a frequency of approximately once a month. This was treated symptomatically with Ergotamine tartrate 2 mg, Cyclizine Hydrochlor 50 mg, and caffeine 100 mg, and the patient expressed satisfaction with this treatment. For the last 5 years, she had taken Nitrazepam 5 mg/nocte to facilitate falling asleep. No other medication was regularly taken. Despite her medication, the patient still complained of difficulty in falling asleep initially and she expressed fears that she had become “addicted” to the drug. She had made three attempts to discontinue the medication in the last 2 years, but the failure of these attempts had reinforced her belief that she was now entirely dependent on it.

METHOD

Procedure

The design adopted in this study was the systematic case study (Hersen & Barlow, 1976).

Data on five self-report sleep indices (time of settling down to sleep, presleep intrusive cognitions, estimated sleep latency, number of awakenings, and total sleep time) were obtained throughout the baseline, relaxation, placebo, biofeedback, and follow-up phases of treatment. This was done by asking the patient to complete a sleep log on waking each morning. EEG theta activity was measured during the baseline, placebo, and feedback phases.

Baseline I. The patient was instructed to keep a daily sleep record for 2 weeks.

Relaxation. The patient was instructed in progressive relaxation, using a modified version of Jacobsen’s (1938) technique. In addition to four sessions in the clinic, the patient was given instructions on home relaxation practice, to be carried out daily. This stage lasted 4 weeks. It was anticipated that this procedure would be of some benefit; however, in view of the lack of improvement an alternative approach, that of EEG biofeedback, was employed.

Baseline II. Two baseline sessions were held, during which EEG theta activity was monitored.

Placebo. Two placebo sessions followed. During these, white noise was played through headphones to the patient. They were otherwise identical to the baseline sessions. It was suggested to the patient that the monotonous noise would help her fall asleep.

Actual question: “Before falling asleep, was your mind overactive or preoccupied with certain thoughts or worries?” This was rated on a 5-point scale; 0 = not at all, 5 = continuously.
EEG Feedback. During treatment sessions, the presence of EEG theta activity was monitored and fed back to the patient over headphones in the form of a continuous tone. Only a dichotomous feedback signal and scoring index were available. This phase comprised 11 sessions.

Finally, medication was withdrawn by being reduced to 2.5 mg/nocte for 2 weeks and then discontinued.

Apparatus

A Biofeedback Systems EEG 90 amplified the theta activity, the threshold being set at approximately 25 µV throughout the treatment sessions. Electrodes were placed at sites T3 and P3, with a reference electrode over the ipsilateral mastoid bone. The apparatus converted the signal into a continuous tone when the fixed threshold was exceeded, and also triggered a cumulative digital timer. The patient was instructed to produce the tone, thereby increasing theta density.

Each session was of 20 minutes' duration and was preceded by 5 minutes in which the patient accustomed herself to the electrodes. The patient reclined with eyes closed throughout each session. The sessions were held once per week, always at the same time of day (6:00 p.m.). The patient was instructed to practice at home, when attempting to fall asleep, the same strategy used in the treatment sessions to produce theta activity.

RESULTS

The data presented in Table I and Figure 1 suggest that feedback was effective in reducing the target variable, sleep latency. Mean sleep latency showed a decrease of approximately 31 minutes from baseline sessions to the last two treatment sessions, and a further reduction of approximately 9 minutes had taken place at 3-month follow-up. Moreover, these gains were maintained during subsequent withdrawal of medication. The mean pretreatment theta density of 8.95% showed a corresponding increase to 17.19% by the final two treatment sessions.

Estimated total time asleep increased by approximately 1 hour following treatment. Presleep intrusive cognitions showed an increase during relaxation and placebo periods, but remained stable during the feedback period at close to baseline levels. A marked decrease was reported at 3-month follow-up, however. The mean frequency of reported awakenings from sleep showed an increase from .43 (baseline) to 1.08 (last two sessions of feedback). Time of settling down to sleep showed no change.
<table>
<thead>
<tr>
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<th>Baseline I (2 weeks)</th>
<th>Relaxation (last 2 weeks)</th>
<th>Placebo (2 weeks)</th>
<th>Treatment I (first 2 weeks)</th>
<th>Treatment II (last 2 weeks)</th>
<th>Follow-up (2 weeks at 3 months)</th>
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</thead>
<tbody>
<tr>
<td>Time of settling down to sleep</td>
<td>12.08</td>
<td>12.30</td>
<td>12.02</td>
<td>12.34</td>
<td>12.32</td>
<td>12.14</td>
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<tr>
<td>Presleep intrusive cognitions</td>
<td>.84</td>
<td>1.79</td>
<td>1.99</td>
<td>1.14</td>
<td>1.14</td>
<td>.40</td>
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<td>Sleep latency (mins.)</td>
<td>54.29</td>
<td>45.82</td>
<td>48.89</td>
<td>44.45</td>
<td>23.07</td>
<td>16.43</td>
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<tr>
<td>N of awakenings</td>
<td>.43</td>
<td>.48</td>
<td>.8</td>
<td>.64</td>
<td>1.08</td>
<td>1.19</td>
</tr>
<tr>
<td>Total sleep time (hours)</td>
<td>4.73</td>
<td>4.39</td>
<td>4.73</td>
<td>4.65</td>
<td>5.71</td>
<td>5.67</td>
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</table>
A short (30-item) version of the General Health Questionnaire (Goldberg, 1972), which was designed to identify psychiatric illness in general practice patients, was administered as a measure of emotional upset. This showed a drop in score from 17 (pretreatment) to 1 by the termination of treatment, representing a change in classification from "probable case" to "probable normal," and supports the patient's report of greatly reduced distress.

Drug withdrawal was carried out in two 2-week phases. For the 1st week of each phase, sleep latency and number of awakenings increased, while total sleep time decreased. These findings are, of course, in keeping with well-known rebound drug-withdrawal effects. These measures returned to end-of-treatment levels in the 2nd week of each phase.

The frequency of the patient's migraine headaches showed no change during the entire period of intervention.

**DISCUSSION**

The above results suggest that treatment intervention was highly effective in reducing the time taken to fall asleep. Although Raskin, John-
son, and Rondestvedt (1973) reported that improvements in sleep latencies did not extend to other sleep measures, in this study total sleep time did improve along with latency. Despite an increase in the number of wakenings from sleep, the patient was very satisfied with this sleep, and reported that such wakenings did not distress her, since they were of very short duration and she could easily return to sleep within a few minutes. No obvious explanation exists for this increase, however.

Although a substantial increase in theta density occurred over treatment sessions, this effect could conceivably be due to increased drowsiness as a consequence of adaptation. No firm conclusions can therefore be drawn as to whether theta activity was in fact brought under the control of the patient. Nevertheless, increased theta density during sessions was accompanied by a reported decrease in sleep latency at home.

Repetitive cognitions showed no change from baseline to end of treatment, despite greater reported ease of falling asleep. Thus the "mental focusing" described by some of the subjects of Freedman and Papsdorf (1976) would not appear to have been the mechanism of change in this case. The decrease in the intensity of presleep intrusive cognitions reported by the patient at follow-up would seem simply to reflect the patient's sense of well-being.

Clearly the reliance on self-report measures is a major problem in this study, since it is known that insomniac patients overestimate sleep latency and underestimate total sleep time (Carskadon, Dement, Mittler, Guillemi- nault, Zarcone, & Spiegel, 1976). Nevertheless, although direct measurement of sleep is undoubtedly invaluable, it has been found that insomniac patients are able to describe their sleep more accurately on a night-to-night basis than on a global rating (Freedman & Papsdorf, 1976). Daily rating was required by this study. The results of Frankel et al. (1973), Kales (1969), and Karacan, Williams, and Salis (1971) further suggest that although the patient may be inaccurate in estimating the absolute amount of difficulty, sleep EEG records generally confirm subjective assessments. Bixler, Kales, Leo, and Slye (1973) therefore conclude that the physician can rely in great part on the patient's complaint of disturbed sleep as being valid.

Finally, a cautionary note is perhaps necessary, since Hauri et al. (1976), using EMG biofeedback, found that although their subjects claimed to sleep better after feedback, this was not confirmed in the laboratory. Furthermore, it would be misleading to think of any treatment as an overall cure for "insomnia" in view of the wide range of clinical manifestations of this problem. Nevertheless, in the case reported here, theta feedback does seem to have been effective in reducing sleep latency, although nonspecific therapy effects cannot be ruled out. Thus this procedure would seem to warrant further investigation in the laboratory.
REFERENCES


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