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Trait anxiety and sleep-onset insomnia Evaluation of treatment using anxiety management training

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Abstract

Objectives: This study was initially designed to test the notion that generalized anxiety is a predominant factor in the maintenance of psychologically determined sleep-onset insomnia and that a trait anxiety reducing technique can provide significant therapeutic gains. **Methods:** Twenty participants (age 19–63) with moderate to severe sleep-onset chronic insomnia were first asked to monitor their sleep-onset latency (SOL) for a 3-week baseline period at home using a SOL clock device. Then, 10 received anxiety management training (AMT) for 9 weeks, while the remaining 10 were trained in the use of progressive relaxation (PR). All participants were measured before and after therapy using sleep laboratory recordings (three nights each), the Spielberger Trait Anxiety Inventory and the Beck Depression Inventory. Daily home sleep-onset measures with the SOL clock device were also taken during therapy. **Results:** There was no change in SOL over the 3-week baseline period. However, both groups experienced a significant improvement in SOL from pretreatment (end of baseline) to posttreatment periods. In the laboratory, both groups experienced a reduction in Stage 1 sleep as well as an increase in slow wave sleep (SWS) and sleep satisfaction. On the personality measures, both groups experienced a significant reduction in trait anxiety and a decrease in depression. Overall, there was no indication that one of the therapies was significantly better than the other in effecting changes. **Conclusion:** These results suggest that both PR and AMT are efficient therapies for sleep onset insomnia and overall sleep quality. Improvements in the application of the AMT technique are proposed to maximize its usefulness.

Keywords: Insomnia; Anxiety management training; Anxiety; Relaxation

Introduction

Research on the nonpharmacological treatment of insomnia has confirmed the short and long term benefits of several behavioural techniques. Such therapies have taken a wide variety of forms. Indeed, progressive and autogenic relaxation [1–3], systematic desensitization [4], paradoxical intention [5–7], sleep restriction therapy [8–10], stimulus control [11,12] and biofeedback [13,14] are all examples of successful techniques to alleviate symptoms of sleep-onset insomnia. They have become preferred alternatives to drug therapies [15,16]. Several reviews are available (e.g., Refs. [17,18]). More recently, their combination with new hypnotics appears most successful [19]. These approaches focus on eliminating competing behaviours and reducing levels of anxiety present at bedtime.

Several studies have linked insomnia with the presence of increased general trait anxiety [20–22]. It appears that, at least in certain conditions, life-stress factors are prevalent and cause or exacerbate chronic insomnia. Learning to cope with these life-stress events would contribute to a more global solution for the treatment of insomnia. More specifically, it would follow that treatment, which includes a reduction of trait anxiety, would be even more efficacious in alleviating symptoms associated with sleep-onset insomnia than therapies that focus only on bedtime conditions. In this study, such a treatment, anxiety management training (AMT) [23,24] which has been shown to reduce trait anxiety, was compared to Jacobson's [25] progressive relaxation (PR). It was predicted that AMT would lead to a greater reduction in trait anxiety, sleep-onset latency (SOL) and negative psychological correlates of insomnia than PR.

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Method

Participants

Following invitations on radio, television and local newspapers, over 200 individuals responded to our call for participants in the study. Of these, 47 fulfilled the preliminary requirements (as determined in a phone interview) and were subsequently invited for a personal assessment interview. It was important to exclude individuals whose insomnia was not strictly psychological. As a first step, a thorough sleep behaviour questionnaire combined with an in-depth interview was used in an attempt to assess the specific cause of the sleep disorder. This allowed a first screening out of a history of many types of sleep-onset problems caused by physiological deficiencies (i.e., sleep apneas, periodic leg movements during sleep (PLMS) and so on). Twenty subjects were retained and completed the MMPI in order to further help to exclude persons presenting signs of severe psychological and psychiatric disorders. More specifically, candidates presenting abnormal values on the scales other than anxiety were excluded. Those exhibiting overuse of drugs and/or alcohol were not retained. Potential candidates using sleeping medication were required to withdraw from their medication for at least a month prior to the experiment. Finally, in order to ensure that participants were experiencing a relatively high level of trait anxiety, a score situated above the 60th percentile on the trait dimension of Spielberger's State-Trait Anxiety Inventory (STAI) was required. This brought down the number of subjects to 27.

Sleep-onset insomnia was considered at least moderate if there was a SOL greater than 45 min at least four nights per week. To provide a preliminary assessment of sleep latency, a special switch-activated clock [26] was used by the participants at home for 1 week prior to final selection. Each daily SOL was recorded and returned to the experimenter for analysis. It was expected that participants with moderate to severe insomnia would present not only sleep-onset delays but also frequent nocturnal awakenings. This, in turn, warranted the use of polysomnographic measures in order to assess the level of sleep efficiency at the different stages of the study. Four subjects were excluded because they presented more than five PLMS.

Applying the above criteria 23 subjects were invited to participate in the study. Two dropped out before the end of the experiment and one was dropped for use of hypnotics. The remaining 20 subjects who complied with all the components of the study ranged in age between 19 and 63 years old. When asked during their personal interview "how long does it take you to fall asleep?," the average response was 92 min (S.D. = 45.5). In addition, these insomniacs averaged around 5.5 h (S.D. = 1.05) in total sleeping time as assessed subjectively at home. Finally, when queried about the last time when they had a good night sleep, eight respondents stated that they did not remember, while the remaining participants estimated an average of 20 days.

Procedure

In order to minimize a priori differences, participants were matched for sex, age, level of insomnia and anxiety on assignment to one of two treatment groups (10 in each). The anxiety management group had four males and six females (mean age = 35.7), and the PR group, two males and eight females (mean age = 36.1). This matched assignment was carried out by an independent specialist who was unaware of the hypothesis of the study. Participants were then brought into the laboratory for one night to obtain the polysomnographic screening measures.

The selected participants were told that a waiting period of approximately 3 weeks was necessary. During that period, they were instructed to keep a daily log of their sleep activities and their varying levels of sleep satisfaction using a short self-report questionnaire in addition to the SOL clock. After the 3-week period, they returned to the lab for psychological and polysomnographic baseline measures for three consecutive nights. The MMPI and the STAI were completed on the eve of the first night, which also served as habituation to the laboratory. Polysomnographic data was collected during the next two nights. Following this, the two experimental groups received their respective treatment program (details presented below).

Throughout the baseline and treatment periods, the participants were seen once every 2 weeks in order to encourage compliance and collect data. Finally, the two groups returned to the laboratory for three nights for the final psychological and polysomnographic measures.

Treatments

One male and one female doctoral students in their late 20s and nearing the end of their PhD program acted as therapists in this study. They were trained in the administration of the two therapeutic approaches and were randomly assigned participants to whom they applied the treatment. Thirteen participants (seven PR and six AMT) were treated by the male therapist, while the remaining seven (three PR and four AMT) were seen by the female therapist. The therapists had strict instructions not to overstep the boundaries prescribed by the technique itself. Thus, their role was to simply reiterate the instructions contained in the original cassette used during the initial session. One of the therapists was aware of the details of the study and the other one was not. No therapeutic effect was observed between the two therapists.

Progressive relaxation

The members of the first treatment group were individually taught PR using live instructions from one of two participating therapists. Then, for a period of about 9 weeks, they were asked to use a tape-recorded version of the first relaxation session, which lasted close to 30 min. This tape was to be practised twice daily, once during the day and the

second time while the participant was in bed and ready to fall asleep. During this home treatment period, participants were asked to fill out and submit a special sleep questionnaire on a biweekly basis to allow monitoring of treatment effectiveness and compliance. In addition, the SOL clock was used at home to provide continuous daily measures.

Anxiety management training

The second treatment group was taught AMT [24]. This procedure requires three basic steps:

1. A half-hour training in deep muscle relaxation using tape-recorded instruction.
2. A 1-h training session (with a therapist) in visualization of an anxiety-arousing scene, then visualization of a scene reinstalling competency or successful response, and finally visualization of a scene associated with relaxation.
3. A 1-h take-home tape-recorded version of the second step where anxiety is aroused, followed by either competency or relaxation. The participants listened to this tape at least once at the end of each day. As with the PR treatment, the AMT treatment lasted 9 weeks. Also, the same measures of anxiety and SOL as for the other group were used.

Measurements

(1) Polysomnography: The participants slept alone in a relatively soundproof room where temperature was maintained between 21° and 22° for a total of seven nights. Standard electroencephalogram (EEC) (C4/Al, C3/Al), electrooculogram (EOG) and electromyogram (EMG) were monitored as prescribed by Rechschaffen and Kales [27]. In addition, respiration and lower limb muscle activity were monitored for the first baseline period to detect sleep apnea and PLMS. In order to ensure comparability with previous studies, the criterion used for sleep onset was 5 min of Stage 2. In the case of female participants, attention was given not to schedule recording nights during the premenstrual and menstrual phases of the menstrual cycle. Recordings were scheduled during intermediate phases. The polysomnographic data was scored and compared in an epoch by epoch fashion by three independent judges. Inter-scorer agreements were above 80% of epochs for the three nights compared per participant.

(2) SOL monitor: An improved version of Franklin's [28] SOL monitor was developed in our laboratory to accurately measure SOL in the home environment. It consists of a time base counter and display module [26]. The participant is required to press the button of a hand-held switch with the thumb. The initial contact causes the display to go blank so that the time cannot be viewed by the subject. Releases of the button within a preset interval (for example, we use 5 min) are ignored by the device. Should a release exceed this period, the display is reactivated and shows the SOL time in a coded format. SOL is therefore measured from the moment the button is pressed to the time when the button has been released for a period exceeding 5 min. In the morning, the participant simply writes down the coded number on a special form. Only the experimenter can decode the number displayed and translate it to the proper SOL figure [26]. This device was validated in our laboratory against electrophysiological measures of SOL [26]. Results showed high correlations between onset of Stage 2 (as measured polysomnographically) and the SOL monitor readings. It was concluded that the SOL monitor would be a very useful tool in this study, providing a reliable yet inexpensive measure of SOL in the home environment.

(3) Sleep satisfaction: Participants also reported their level of sleep satisfaction each morning on a scale from 1 to 4 (*much* = 1, *some* = 2, *little* = 3, *no* = 4).

(4) Psychological measures: *Personality*: Given the extensive use of the MMPI in studies of insomniacs [29], this test was selected to detect severe pathologies commonly associated with insomnia and as a measure of depression (see below). *Anxiety*: Generalized anxiety was measured using the STAI [30]. *Depression*: In addition to the depression scale of the MMPI, the Beck Depression Inventory was also used as a quick and efficient instrument in measuring varying levels of depression.

Results

Preliminary analyses indicated no differential therapeutic effect across therapists so the data were pooled for further analyses.

Most analyses consisted of two-way ANOVAs with a between-subjects factor (two treatments) and a within-subjects factor (time periods). The hypothesis of overall changes induced by treatments was tested by the within group main effects, whereas the superiority of AMT over PR was tested by the interaction term. For the most important dependent variable, SOL, a separate analysis was conducted for the 3-week baseline period for which measures were also available.

Personality measures

Table 1 presents the means and standard errors (S.E.M.) on the pre- and posttreatment measures in personality dimensions and sleep variables. In the case of sleep measures, the pretreatment means are averages of nights 3 and 4, and the posttreatment means are averages of nights 5 and 6. The initial expectation of this study was that AMT would significantly reduce trait anxiety, which, in turn, would make it a better treatment of sleep onset insomnia than simple relaxation, particularly in this highly anxious sample. A 2 Treatment Groups × 2 Time Periods analysis of variance on the STAI data did reveal a significant overall reduction in trait anxiety [time period: $F(1,18) = 14.45$,

Table 1
Means and S.E.M. for the personality and sleep measures (PR and AMT)

		Before		After		
	Group	Mean	S.E.M.	Mean	S.E.M.	TE[a]
STAI	PR	88.2	3.11	70.0	5.90	**
	AMT	89.8	3.01	65.2	10.56	
Beck	PR	8.77	1.45	6.44	1.19	**
	AMT	12.5	2.91	6.70	2.65	
SOL clock	PR	68.25	13.71	42.85	10.80	**
	AMT	81.06	8.26	45.72	13.58	
SOL poly.	PR	37.75	7.02	34.78	11.86	ns
	AMT	47.80	22.28	21.22	3.54	
Duration	PR	389.10	27.79	390.75	18.30	ns
	AMT	348.33	27.79	387.40	18.30	
% Stage 1	PR	6%	1%	3%	1%	*
	AMT	13%	5%	4%	1%	
% Stage 2	PR	52%	2%	52%	4%	ns
	AMT	56%	4%	57%	3%	
% Stage 3	PR	9%	1%	10%	2%	ns
	AMT	8%	1%	8%	2%	
% Stage 4	PR	8%	2%	11%	2%	ns
	AMT	2%	1%	6%	2%	
Delta	PR	18%	2%	22%	3%	*
	AMT	10%	2%	14%	3%	
REM	PR	23%	1%	21%	2%	ns
	AMT	18%	2%	23%	2%	
Sleep efficiency	PR	0.94	0.02	0.94	0.01	ns
	AMT	0.89	0.06	0.91	0.03	
Sleep satisfaction	PR	2.32	0.21	2.10	0.22	**
	AMT	2.59	0.13	2.12	0.18	

ns: $P > .05$.

[a] TE = within group treatment main effects; there were no between group effects nor any significant interactions.

* $P < .05$.

** $P < .01$.

$P < .001$], but, contrary to expectation, no interaction between treatments and time periods, indicating that AMT and PR were equally successful in reducing trait anxiety. The large posttreatment S.E.M. in STAI is due to the fact that four out of 10 participants experienced a very large decrease (>50%) in anxiety, whereas the remaining participants did not. Similar results were observed on the Beck Depression Inventory [time period: $F(1,18) = 10.53$, $P < .001$] and the depression scale of the MMPI [time period: $F(1,18) = 8.21$, $P < .01$]. The fact that both groups experienced a significant reduction in anxiety decreases the likelihood of differential treatment effects on insomnia but is consistent with an overall improvement of sleep. This is what the remainder of the results show.

Sleep-onset latency

The self-report results on SOL were analysed first. Over the 11 weeks of data collection, participants failed to self-report 10% of the time in the AMT group and 15% in the PR group. In a manner similar to clinical drug trials [31], values were interpolated where information was available before and after the missing point, and replaced by the end-point score if values were missing until the end of the treatment period. The data was then condensed in blocks of 3 weeks (except the last block, which has only 2 weeks) for statistical analyses.

As the top panel of Fig. 1 shows, self-reported SOL decreased markedly in the two treatment groups immediately after the baseline period and remained shorter during the 9 weeks of treatment. This is confirmed by the statistical analyses. A 2 Groups × 4 Periods split-plot ANOVA indicated a significant change over time periods [$F(3,45) = 4.02$, $P < .03$], but no group by period interaction and no difference between AMT and PR. Bonferroni post-hoc analyses showed a significant improvement in SOL from baseline to treatment period 1 [$F(1,15) = 13.72$, $P < .02$] and no other change from periods 1 to 2 or from 2 to 3. To further ascertain that changes over time were due to the treatments and not simply to time passage, the baseline data was examined with a 2 Groups × 3 Weeks split-plot ANOVA. No significant effects were found.

A similar picture emerges from the more objective SOL clock data, as depicted in the bottom panel of Fig. 1. Again, missing values were interpolated for 6% of the original data points in the AMT group and 3% in the PR group. A 2

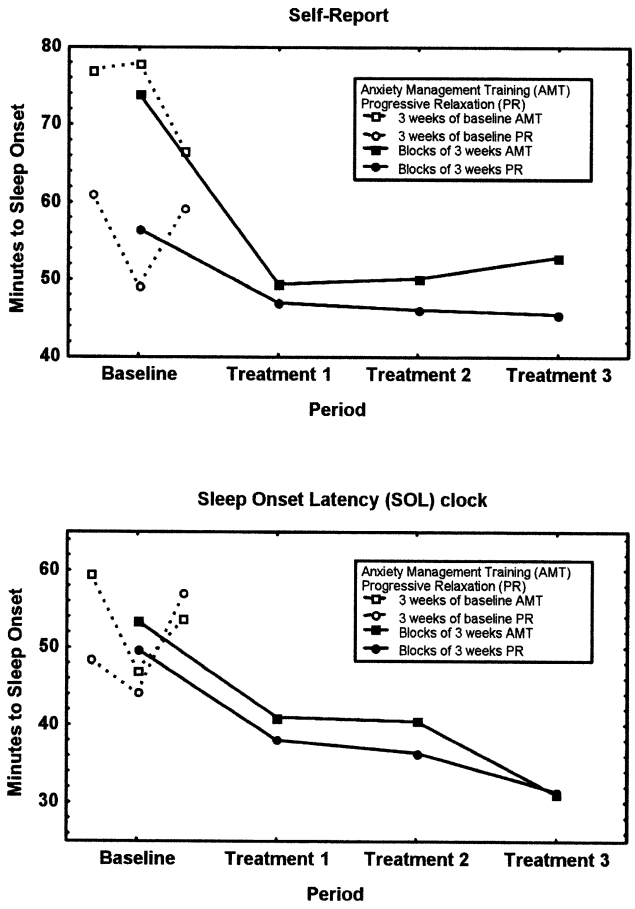

Fig. 1. SOLs during treatment as self-reported and as measured mechanically.

Groups × 3 Weeks split-plot ANOVA of the baseline data revealed no significant differences. However, the 2 Groups × 4 Periods split-plot ANOVA indicated a significant reduction of SOL in the two treatment groups [$F(3,48) = 8.31$, $P < .001$], but no difference across treatments nor any Treatment × Time interaction. Bonferroni post-hoc tests located the significant latency reduction in the change from baseline to treatment period 1 [$F(1,16) = 9.47$, $P < .05$], with no other change from periods 1 to 2 or from 2 to 3.

A within-subject measures (2: Self-Report and SOL) × Periods (4) ANOVA was performed in order to assess the correspondence between the self-report data and the objective measure provided by the SOL clock. The pooling of treatment groups was justified by the lack of significant differences in the previous analyses. The periods are included to eliminate their contribution to the variance. Although the SOL clock data are consistently lower than the self-report, as can be seen in Fig. 1, the analysis revealed no significant difference across measures. The reliability of the two types of measures was further contrasted by comparing the frequencies of missing values in each data set. The SOL clock method of measurement contained significantly less missing values than the self-reports (McNemar's $c^2 = 124.15$, $P < .01$).

Finally, as Table 1 reveals, there was no significant decline in SOL as measured with polysomnography in the laboratory. However, it should be noted that during laboratory recordings, there was a high correlation between SOL from the monitor and the Stage 2 latencies ($r = .72$, $P < .001$) with an average overestimation of only 5 min with the monitor. At the descriptive level, it is of interest to note that, in each group, 7 out of the 10 subjects had a decrease in SOL in the laboratory. Furthermore, of the six subjects who had an increase in laboratory SOL, five had baseline laboratory SOL below 30 min (range: 10–26 min). Overall, 10 of the 20 subjects had baseline levels of laboratory measured SOL below 30 min, whereas only 1 had a baseline home measured SOL below 30 min (actually 26 min).

Sleep in the laboratory

Split-plot analyses of variance comparing AMT and PR on sleep stages showed within group differences indicating a significant decrease in percentage of Stage 1 [$F(1,18) = 6.38$, $P < .02$] and an increase in delta sleep percentage [$F(1,18) = 4.87$, $P < .04$]. In addition, there was a main effect improvement in self-rated sleep satisfaction [$F(1,18) = 11.53$, $P < .001$]. However, no interactions were noticeable for these sleep measures. Thus, both treatments affected the sleep stages similarly. In addition, there was a marginally significant main effect increase in the percentage of REM after treatment [$F(1,18) = 3.97$, $P < .06$].

Unlike the SOL clock data, the analysis of polysomnographic data revealed no significant difference in mean latency to Stage 2. Since Table 1 reveals an unusually large pretreatment variability in the AMT group (S.E.M. = 22.28), we verified that this lack of difference was not an artefact. A deleted residual analysis identified one participant as an outlier with a pretreatment average latency of 223 min. Re-analyzing without this participant did not change the conclusions.

One aspect of the data that deserves further comment. With only 20 participants, statistical power is limited. For instance, the observed power for the pre–post main effect in the percentage of time spent in Stage 1 sleep was 63%. However, the crucial hypothesis of treatment differences in this split-plot design is tested by the interaction term, which uses the same degrees of freedom and error term as the pre–post main effect and, thus, would have the same power for a constant effect size. Since we have found pre–post main effects, we would have been able to detect similar effects with similar power if they had been located in one treatment group but not in the other. Visual inspection of means also does not suggest that there were sizeable interactions going undetected.

Discussion

These results further confirm that behavioural approaches based on relaxation are efficient in the treatment of sleep onset insomnia. The data clearly indicates an overall improvement in the time elapsed before falling asleep. Because the experiment was costly and time consuming, its design did not include a no-intervention comparison group. The lack of no-treatment control group for the entire period covered by the study limits its claim. It is possible that the attention given to the subjects during treatments could have had its own beneficial effect. However, three aspects of the results concur to reinforce the conclusion that the improvement is not simply due to the passage of time, but rather is caused by the two treatments. First, no significant change occurred during the 3 weeks of the baseline period. Second, latencies are reduced in the two groups as soon and only as soon as each treatment is applied. Third, the improvement remains for as long as the treatments are in force. Thus, both AMT and PR are effective in reducing SOL.

Improvements were not only seen in the latency to sleep onset at home but also on some laboratory polysomnographic measures. Perhaps, as important, key psychological dimensions such as anxiety and depression were significantly improved. Again, the overall results of the analyses indicate that both treatment approaches were successful, with no significant added advantage for AMT. It should be noted however that our application of AMT (as prescribed by the authors) may have been less than optimal in facilitating a positive outcome for insomniacs. Although AMT was administered using a proven cassette format, some participants found it difficult to listen to the final AMT cassette at bedtime. These individuals were required to heighten their anxiety several times during the exercise.

Some complained of still feeling somewhat anxious even after the completion of the nightly session. They reported that this voluntary increase in amount of anxiety at bedtime hindered their sleep-onset. Certain individuals thought it was counterproductive to raise anxiety at a time when it should be at its lowest. This probably explained the large discrepancy between subjects in the AMT group on the posttreatment levels of anxiety as mentioned above. One way of bypassing this obstacle would be to instruct participants to listen to the AMT tape during the day while practising only the deep muscle relaxation component of the treatment at bedtime. With these instructions, the individual would profit fully from a technique which otherwise may not be tailored to the special needs of insomniacs. Such an application may indeed prove even more useful than other therapies with highly anxious insomniacs.

Our results provide new information about the effects of relaxation on insomnia. As pointed out by Lacks [32], the past success of PR in dealing with sleep-onset insomnia was largely based on subjective reports from the participants. To our knowledge, the current research is the first to objectively demonstrate with the SOL device that PR improves sleep-onset latencies at home.

One important finding was the nearly complete absence of polysomnographically measurable sleep disturbances in the laboratory except for long SOL (only in half the subjects). This in itself strongly supports Jacobs et al. [33] and Haynes et al.'s [34] conclusion that, as predicted by the stimulus control paradigm, insomniacs in the sleep laboratory take less time to fall asleep than the usual latencies reported at home. The same authors attributed this to the fact that stimuli associated with wakefulness at home are no longer present in the sleep laboratory.

The overall increase in slow wave sleep (SWS) observed following treatment and the possible increase in REM sleep percentage are optimistic signs that sleep architecture is improved by those simple techniques.

The simultaneous collection of self-reported and objective data on sleep onset latencies allowed for an interesting methodological comparison. Overall, both measures lead to the same conclusion as to the pattern of improvement. In addition, the size of the mean difference between measures is not large enough to reach significance. This suggests that other studies which relied exclusively on self-report of SOL can be trusted to some extent, although the objective measure has the advantage of being more complete (less missing values), and hence more representative of what is really happening. Even if just for that reason alone, its usage is recommended for future studies.

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