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Countercontrol Treatment of Sleep-Maintenance Insomnia in Relation to Age

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We administered countercontrol behavioral therapy for sleep-maintenance insomnia to 34 insomniacs—ranging in age from 35 to 78 years—in small groups. Twenty-two subjects received immediate and 12 received delayed treatment. Three self-report measures of sleep disruption were collected on daily sleep diaries at baseline, termination of treatment, 1-month follow-up, and 12-month follow-up. Although amount of time awake at night was correlated with age ($r = .50$), response to treatment was not. Even though older people experienced more time awake after sleep onset prior to treatment, they were able to profit from therapy as well as the younger insomniacs. Countercontrol therapy reduced the sleep complaint for the total group by about 30% at the end of treatment, with gradual improvement continuing through a 4-week follow-up. Nevertheless, it appears that sleep-maintenance insomnia may be more difficult to treat than sleep-onset problems.

Older adults have a high rate of sleep-related complaints (Miles & Dement, 1980; Webb, 1983), which are most commonly treated with hypnotics and other sedating drugs. Use of sleeping pills is very common among older adults, even though they are known to have only short-term effectiveness and many detrimental side effects. The latter include development of tolerance, next-day drowsiness and lowered performance, toxic drug interactions, compromised cortical activity, and the possibility of impaired respiratory, hepatic, or renal functioning because of drug accumulation (Bootzin, Engle-Friedman, & Hazelwood, 1983; Miles & Dement, 1980; Regestein, 1980). Many studies have explored the usefulness of a variety of nondrug, behavioral treatments for insomnia. Borkovec (1982) concluded that stimulus control strategies have demonstrated the highest overall success for young and middle-aged adults. Only one study has examined the effectiveness of the stimulus control treatment with older sleep-onset insomniacs (Puder, Lacks, Bertelson, & Storandt, 1983). Older adults showed improvements that were equivalent or slightly better than those obtained by individuals under age 60 in a previous study (Lacks, Bertelson, Gans, & Kunkel, 1983).

Almost all of the studies of the behavioral treatment of insomnia have focused on sleep-onset difficulties (i.e., problems in getting to sleep). Older adults, however, more frequently suffer from sleep-maintenance insomnia (more wake time after sleep onset and more arousals; Webb & Campbell, 1980). Very little has been published on the treatment of sleep-maintenance insomnia for any age group (for several case studies, see Coates & Thoresen, 1979; Thoresen, Coates, Kirmil-Gray, & Rosekind, 1981). The one controlled group study of younger individuals with sleep-maintenance problems showed that stimulus control reduced wakefulness significantly, but no more so than did a credible placebo (Lacks, Bertelson, Sugerman, & Kunkel, 1983). Stimulus control treatment consists of using the bed only for sleep and establishing a regular sleep schedule. Many of the subjects in that study complained about the stimulus control requirement of getting out of bed after every 10-min period of sleeplessness during the night. It is possible that this requirement may have adversely influenced the sleep-maintenance insomniacs' response to treatment.

In looking more closely at the stimulus control procedure, several researchers (e.g., Borkovec, 1982) suggested that it may owe most of its effectiveness to the disruption of sleep-incompatible behaviors such as cognitive activity or restless tossing. If the crucial component to stimulus control is the disruption of these activities rather than reestablishing the bed as a discriminative stimulus for sleep, then the requirement to leave the bed and bedroom should be largely superfluous. By removing the out-of-bed requirement, perhaps effectiveness would be improved for the more-difficult-to-treat sleep-maintenance problem. A treatment that can be done in bed may also be more feasible for the sometimes less ambulatory older adult.

An intervention developed by Zwart and Lisman (1979) seems suited for this goal. The treatment, called *countercontrol,* was designed to disrupt sleep-incompatible activities without introducing novel procedures (such as leaving the bed) that might be responsible for any observed improvement. Participants were instructed to deliberately engage in a nonarousing activity (e.g., dull reading) in bed whenever they were unable to

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sleep. The administration of the countercontrol procedure is identical to that of stimulus control, excluding departure from the bedroom when awake and temporal instructions (like napping restrictions or a consistent time of morning arousal). To preclude an explanation of the bed as a discriminative stimulus for sleep, each individual also spends 30 min per day engaging in nonsleep activities while in bed. In their study of young sleep-onset insomniacs, countercontrol and stimulus control were found to be equally effective. For these reasons, we chose to explore the efficacy of the countercontrol procedure in this study of sleep-maintenance insomnia in relation to age.

Method

Participants

Recruitment of research volunteers was through media announcements and current waiting lists. Participants were 34 adults with sleep-maintenance insomnia of at least 6 months duration. Sleep-maintenance insomnia was defined as wake time after sleep onset (WASO) of at least 30 min per night at least 1 night per week, as corroborated through sleep diaries for 7 baseline days. Individuals with concomitant sleep-onset insomnia were included if they considered their sleep-maintenance difficulty to be the primary problem. Participants had to be in the age range of from 20 to 80 years and could not have received previous behavior therapy for insomnia. They could not be taking sleep medication, or they had to be willing to discontinue hypnotics under a physician's care prior to treatment. A standard protocol (available from the authors)—including responses to extensive sleep questionnaires, the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1943), the Zung (1965) Self-Rating Depression Scale, and personal interviews—was used to eliminate anyone whose insomnia was considered secondary to serious, painful medical conditions (e.g., arthritis), psychopathology, sleep apnea, or nocturnal myoclonus. The spouse's perception of the subject's sleep problem was elicited when necessary.

A total of 202 individuals were contacted about participating in the study. After a brief telephone screening, 94 individuals were scheduled for more extensive evaluation. Of these, 18 did not attend the first appointment, 21 were rejected because they did not meet inclusion criteria, and 55 were accepted and assigned randomly to either immediate or delayed treatment groups. Of the accepted subjects 12 chose not to begin treatment. After treatment began, 9 more were dropped from the study: 1 for medication use during treatment, 5 for noncompliance with daily return procedure of diary data, and 3 by personal choice. In total, 34 people completed treatment, 22 in the immediate group and 12 in the delayed group. The 18 men and 16 women ranged in age from 35 to 78 years (M = 58.59; SD = 10.98) and had 12 to 21 years of education (M = 15.62; SD = 2.67). The sample sizes by decade were as follows: 31–40, n = 2; 41–50, n = 6; 51–60, n = 10; 61–70, n = 12; 71–80, n = 4.

Comparisons of immediate and delayed treatment groups revealed no significant differences between the two groups with respect to age, education, sex, occupational or socioeconomic status, duration of insomnia, or the constancy of the problem. Mean baseline WASO was 79.91 min, with an average of 13.41 years duration in the immediate treatment group, and 73.92 min, with an average of 18.00 years duration in the delayed treatment group.

Measures

The primary dependent variable from the daily sleep diaries was wake time after sleep onset. Of secondary interest were total number of arousals and number of arousals exceeding 10 min. The daily sleep diary is the most frequently used outcome measure in insomnia treatment research. Although insomniacs' estimates of sleep parameters are significantly different from those obtained from all-night sleep recordings, their self-reports have been shown to vary from EEG findings in a consistent way. Therefore, self-reports can provide a reliable and valid *relative* index of insomnia. The sleep diary, when used with insomniacs, has been shown to have high test–retest reliability and substantial correlations with EEG estimates (Bootzin & Engle-Friedman, 1981; Coates, Killen, George, Marchini, Silverman, & Thoresen, 1982; Frankel, Coursey, Buchbinder, & Snyder, 1976; Lichstein, Nickel, Hoelscher, & Kelley, 1982). To ensure monitoring on a daily basis and to eliminate the possibility of retrospective estimates, participants were required to complete the diary each morning upon awakening and to mail it to the experimenters every day.

Treatment

During baseline periods and the 4 weeks of treatment, participants were asked to refrain from the use of sedative medications, caffeinated foods and beverages within 6 hr of bedtime, and vigorous exercise or alcohol consumption within 2 hr of bedtime. This restriction was used to minimize the chance of drug-related sleep disturbances confounding baseline measurements or the occurrence of treatment effects.

The countercontrol treatment was closely modeled after the procedures of Zwart and Lisman (1979). The purpose of the treatment was to prevent subjects from engaging in cognitive arousal or any other sleep-incompatible behaviors by introducing structured noncognitive activities. During the night, if the subjects were unable to fall back to sleep within 10 min of waking up, they were to sit up in bed and engage in some noncognitive activity until drowsy. This activity consisted of such things as reading dull material, watching brief television shows, or listening to the radio. Choosing a noncognitive activity had the dual purpose of breaking the contingency between waking up and engaging in the former sleep-incompatible behaviors (such as tossing and turning) and of preventing cognitive arousal. Participants were free to select their own activity, as long as it was not cognitively stimulating and could be done in the bed. This procedure was repeated each time the person could not return to sleep within 10 min of waking up. In order to preclude explanations of treatment effects based on the bed as a discriminative stimulus for wakefulness, participants also were instructed to engage in similar noncognitive activities in bed for 30 min every day.

Procedure

Therapy began after a 2-week baseline sleep-monitoring period. The sleep diary ratings for the first week of the baseline period were discarded to minimize self-monitoring adjustment effects. The sleep diary was also completed daily during the 4 weeks of treatment, and again for 7 days at a follow-up 4 weeks after treatment. Those in the delayed treatment group completed the same diaries, with the addition of a second baseline after their waiting period. Individuals in the delayed treatment group were told that they were on a waiting list and would begin treatment in about a month, but were asked to begin sleep hygiene practices during this waiting period.

Treatment was conducted by two female clinical psychology graduate students, trained in the techniques by an experienced female clinician. A detailed treatment manual and weekly supervision sessions were used to standardize therapy procedures between the two therapists, each of whom treated equal numbers of groups. No therapist effects were found in treatment outcome. The first session consisted of explaining the treatment rationale and procedures. Subsequent sessions focused on discussing problems in implementation encountered during the week and generating ways of improving therapy adherence. Therapy was done in small groups ranging from three to seven members and was adminis-

Table 1
Means of Three Indices of Insomnia for Two Treatment Groups Throughout the Course of the Study

Treatment group	Time of measurement					
	Week 0	Week 3	Week 4	Week 7	Week 8	Week 12
	Minutes awake after sleep onset (WASO)					
Immediate						
M	79.91	65.91	58.00		54.90[a]	
SD	48.17	50.40	48.69		45.31	
Delayed						
M	73.92		78.42	48.42	54.75	31.64[a]
SD	42.68		34.45	51.27	54.69	34.21
	Average no. of awakenings per night					
Immediate						
M	2.54	1.66	1.82		1.92	
SD	1.02	0.80	0.89		0.77	
Delayed						
M	1.27		1.37	1.32	1.22	1.04
SD	0.53		0.83	1.05	0.66	0.81
	Average no. of awakenings of 10 min or more					
Immediate						
M	2.11	1.50	1.59		1.68	
SD	0.84	0.84	0.90		0.86	
Delayed						
M	1.09		1.31	1.06	1.04	0.81
SD	0.61		0.86	0.77	0.58	0.84

Note. Treatment occurred between Week 0 and Week 4 for the immediate group (n = 22) and between Week 4 and Week 8 for the delayed group (n = 12).
[a] These follow-up data are based on 19 people in the immediate group and 11 in the delayed group.

tered in sessions held once per week and averaging 1 hour each. In an attempt to reduce demand effects, participants were given counterdemand instructions (i.e., they were told not to expect any improvement in their sleep until the 4th week of treatment). Pre- and posttreatment ratings of therapy credibility by participants (on five 7-point scales) showed generally favorable responses to this experience.

Results

Average Time Awake After Sleep Onset

To determine the effectiveness of the countercontrol treatment procedure for sleep-maintenance insomnia, the immediate treatment was compared with the control (delayed treatment) group by means of two-way analysis of variance with one between-subjects variable (group) and one within-subjects independent variable (pre- vs. posttreatment WASO). Two such analyses were conducted; the first used Week 3 WASO for the immediate treatment group and the second baseline WASO for the control group as the posttreatment values. This analysis allowed assessment of treatment impact under conditions of counterdemand. The second analysis used the positive demand Week 4, or end of treatment, WASO for the immediate treatment group as the posttreatment value.

In the first analysis, no significant effects were obtained (ps > .20). In the second, there was a significant Group × Time interaction, $F(1, 32) = 5.15, p < .05$; neither the main effect of group nor time was significant. As can be seen in Table 1, the two groups were not significantly different at the first baseline, $t(32) = 0.36, p > .70$. Also, the first and second baseline WASOs (Weeks 0 and 4) for the control group were not significantly different by correlated t test; no reductions occurred in WASO during the 4-week waiting period. The correlated t test of the baseline (Week 0) and Week 4 WASOs for the immediate treatment group, however, was significant $t(21) = 3.19, p = .004$. These individuals reported approximately 22 min less time awake after sleep onset at the end of treatment than they had prior to treatment.

For ethical reasons the control group was offered the same treatment after they provided the second baseline data. Correlated t tests revealed significant decreases in WASO both at the third, $t(11) = 2.43, p < .05$, and fourth, $t(11) = 2.22, p < .05$, weeks after treatment onset (Weeks 7 and 8 in Table 1), in comparison with second baseline (Week 4) WASO values. The decrease in WASO in this partial replication of the treatment study was approximately 30 and 23 min for the third and fourth weeks, respectively.

To determine the stability of the treatment effect, sleep diaries were obtained from 30 individuals 4 weeks after the end of treatment (3 people in the immediate and one in the delayed group declined to provide diaries at 1-month follow-up). The immediate and delayed treatment groups were combined for this analysis because both had received treatment prior to follow-up. WASO at the end of treatment and 1 month later were compared by correlated t tests and revealed continued improvement, $t(29) = 2.15, p < .05$. The average decrease in WASO

from the baseline immediately prior to treatment onset (M = 77.79) to end of treatment (M = 56.85) to follow-up 4 weeks later (M = 46.37) was 31 min for the combined treated groups.

Average Number of Awakenings Per Night

Two two-way analyses of variance, one for Week 3 and one for Week 4, were used to determine the impact of treatment on the average number of awakenings per night, as was done with WASO. In the analysis that used the average number of awakenings of the immediate treatment group at Week 3 and second baseline for the control group as the values obtained at the second time of measurement, both the main effect of group, $F(1, 32) = 10.70$, $p < .01$, and time, $F(1, 32) = 4.25$, $p < .05$, were significant. More important, the Group × Time interaction was also significant, $F(1, 32) = 6.72$, $p < .02$. Comparable results were obtained when the Week 4 average number of awakenings was used as the measure for the second time of assessment for the immediate treatment group: Group, $F(1, 32) = 8.64$, $p < .01$; time, $F(1, 32) = 7.26$, $p < .02$; and Group × Time, $F(1, 32) = 12.76$, $p < .0011$. Table 1 shows the average number of awakenings for the immediate treatment and control (delayed treatment) groups. Although the control group did not differ significantly at their first and second baseline measurements, correlated $t(11) = -0.65$, $p > .50$, the immediate treatment group declined significantly both by Week 3, $t(21) = 3.32$, $p < .01$, and Week 4 $t(21) = 4.91$, $p < .0001$, in comparison with their average number of awakenings at baseline (Week 0).

Although the immediate treatment and control groups were formed through random assignment and their WASOs did not differ significantly at baseline (see previous section), the two groups did differ significantly in average number of awakenings at baseline, $t(32) = 4.00$, $p < .0001$. The immediate treatment group awoke, on average, twice as often as did the control group (2.54 vs. 1.27). Even though the average number of awakenings per night decreased significantly in the immediate treatment group, by Week 4 they still experienced more awakenings than did the control group at their second baseline measurement; this difference, however, was not statistically significant.

Given that the delayed treatment group began the study with a smaller number of awakenings per night, it is not surprising that they demonstrated no significant decline on this variable when they received treatment. Comparison of the average number of awakenings per night at end of treatment (1.69) with awakenings at follow-up (1.61) for the combined groups revealed no further changes in this variable 4 weeks after the end of treatment.

Table 1 also indicates our findings for longer awakenings. When the computation of average number of awakenings per night was restricted to those of 10 min or greater duration, the results were essentially identical to those obtained for average number of awakenings of any length.

Relation of Age to Treatment Effects

Significant correlations between age and WASO just prior to treatment (r = .50) and at the end of treatment (r = .41) were obtained in the combined groups. For example, at baseline, mean WASO for those age 60 and under was 70.7 min (SD = 44.2) and for those 61 and older was 82.4 min (SD = 46.26). Age, however, was not related to success of treatment. This finding was revealed in a hierarchical multiple regression analysis for the combined groups, in which WASO at the end of treatment was regressed on WASO at baseline, immediately prior to countercontrol treatment and age, in that order. The R was .75 with $F(1, 31) = 42.12$, $p < .0001$, and did not change when age was added to the equation. The partial correlation between age and posttreatment WASO, controlling for baseline WASO and treatment, was .06. Thus, age was not related to treatment efficacy once the initial magnitude of the insomnia was controlled. The results were the same when WASO at the third week of treatment was used as the dependent variable.

Although older people experienced more time awake after sleep onset prior to treatment, age was not significantly correlated with the average number of awakenings at the beginning of the study (r = .15, n = 34). Age also was not related to success of treatment when number of awakenings and awakenings of 10 min or greater were subjected to hierarchical multiple regression analyses analogous to those described for WASO.

One-Year Follow-Up

One year after treatment, we were able to obtain 1 week of diaries for 16 of the people in the study and brief questionnaire responses from 19. Although all three indices increased slightly in this subset of the sample, none of these differences were significant when compared by correlated t test with the values obtained from the same 16 people at 1 month after treatment. The means for these 16 participants at 1-month and 1-year posttreatment, respectively, were 45.88 and 55.06 min for WASO, 1.48 and 1.60 for numbers of awakenings per night, and 1.16 and 1.36 for number of awakenings of 10 min or greater duration.

Discussion

In this study we corroborated the widely held view that sleep deteriorates as an individual grows older. Although age was uncorrelated with the number of times people reported awakening during the night, older people remained awake for longer periods of time. That is, they found it more difficult to return to sleep after awakening. Nevertheless, even though they began and ended treatment with more sleep disturbance, the older adults were able to gain from treatment, in that response to treatment was unrelated to age of the insomnia sufferer.

The generalizability of these results is confined to older adults who are independent and self-sufficient, as well as relatively well educated. Future researchers will need to explore the effectiveness of behavioral techniques in ameliorating sleep complaints of the institutionalized older person. The majority of older adults, however, are ambulatory; thus clinicians can use behavioral strategies for the insomnia of older adults, confident that they will respond as well as do younger persons. This result constitutes good news, indeed, because insomnia is a major health problem for older people, and the typical treatment with sleep medication carries special additional risks with increasing age.

A second finding of this study is that the countercontrol treatment was successful in reducing the sleep-maintenance com-

plaints of this group of volunteers. After only 4 weeks of this behavioral intervention, 74% of the participants decreased their WASO by at least 20%, with an average WASO reduction of 30%. Number of arousals was also reduced by about 20%. Number of nights of insomnia per week was reduced by 1 night. Although sleep-onset latency was not a target of the therapy, this complaint was also reduced from a nightly mean of 30 min to 16 min. These patterns were basically the same regardless of the age of the volunteer. Gradual improvements continued through the 4-week follow-up period, and these reductions were essentially maintained a year later. At the 1-year follow-up, 6 of 19 participants claimed their insomnia was "cured" (i.e., they no longer considered themselves to have insomnia). Many of the others commented on enhanced feelings of self-efficacy or ability to control their sleep and reduced performance anxiety over sleep. Only 6 of the 19 reported a return to sleeping medication.

Although improvement using the countercontrol procedure was demonstrated, it did not provide the magnitude of results reported by users of stimulus control for both sleep-onset (Lacks, Bertelson, Gans, & Kunkel, 1983) and sleep-maintenance insomnia. For example, stimulus control treatment reduced WASO time by 48% in an earlier study by Lacks, Bertelson, Sugerman, and Kunkel (1983), whereas countercontrol in this study reduced WASO by 30%. Also, 1 year after treatment, 40% of the 16 participants who responded reported some worsening of their problem. Confirmation of this pattern will await a direct comparison of the two therapies.

One explanation for this difference is that in order to keep the treatment relatively pure, we did not include any temporal control components, which are part of the standard stimulus control package. Having a person arise at a consistent hour and avoid all daytime naps may serve to stabilize the individual's sleeping cycle. This stabilization may be of particular importance in treating sleep-maintenance problems, which disrupt the sleeping cycle throughout the night. There is also evidence that older adults may be susceptible to desynchronization of internal circadian rhythms (Regestein, 1980). This change may be due to a generalized deterioration of regulatory mechanisms associated with the normal aging process or due to the breakdown of normal daily routines (i.e., absence of regular activities; Shock, 1977; Wever, 1975). Thus, desynchronization of the sleep pattern may be a contribution to insomnia in older adults, and the temporal element of the treatment procedure may be of importance for this age group.

It is also possible that the inclusion of individuals suffering from early awakening problems may have confounded our treatment results. Informal observations during treatment suggested that early awakening problems did not seem to respond readily to countercontrol treatment. Traditionally researchers have failed to distinguish between types of sleep-maintenance insomnia. Just as it is important to differentiate sleep-onset insomnia from sleep-maintenance insomnia (the two may occur for different reasons and be amenable to different types of treatment), so it may be important to differentiate various kinds of sleep-maintenance insomnia.

One of our reasons for using the countercontrol technique was that our previous research participants complained about the necessity in stimulus control therapy for leaving the bed when they could not fall asleep. Unfortunately, just as much resistance was voiced in this study to turning on the lights in the middle of the night and engaging in some activity.

Everyone wakes up during the night. The real issue is how quickly do they fall back asleep? The key to successful treatment for those who do not readily and rapidly return to sleep (i.e., insomniacs) may lie in determining how they differ in their cognitive or emotional responses to such awakenings. The success of the countercontrol treatment lends credence to current beliefs that the active ingredient in the stimulus control treatment is the provision of activities that can disrupt the cognitive arousal about which many insomniacs complain. Both of these behavior therapies require the insomniac to interrupt his or her period of sleeplessness with some activity, either in bed (countercontrol) or out of the bedroom (stimulus control). This interruption, which is filled with some dull but attention-holding activity (usually reading or watching television), makes it more difficult for the insomnia sufferer to engage in the typical pattern of increasingly racing thoughts and worry about lack of sleep.

The area of disruption of cognitive arousal in the treatment of insomnia merits further research. It may be that both of these treatments indirectly reduce this problem. Perhaps a treatment, such as cognitive refocusing or meditation, that addresses cognitive arousal more directly would have greater success as a treatment for insomnia. One researcher (de la Pena, 1978) suggested that increased nighttime cognitive arousal may be due to the daytime physical hypoactivity of insomniacs who have a more sedentary lifestyle. This factor is particularly pertinent to the older adult. Perhaps the development of strategies to alter daytime behaviors rather than concentrating on nighttime activities would prove a promising avenue in the treatment of insomnia.

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