

Analysis of Stimulus Control Treatment of Sleep-Onset Insomnia

Cheryl A. Zwart and Stephen A. Lisman
State University of New York at Binghamton

This study was undertaken to examine the components responsible for the efficacy of the stimulus control treatment of sleep-onset insomnia. Forty-seven college students with sleep-onset insomnia were assigned to one of five treatment conditions. Subjects were instructed not to expect improvement until after the fourth treatment session. Only stimulus control and a treatment violating the associative aspects of stimulus control produced significantly greater improvement in reported latency to sleep onset than the waiting-list condition. Results are discussed in terms of several possible explanations for improvement due to so-called stimulus control treatment for insomnia.

The explanation typically offered for the reported efficacy of the stimulus control treatment of sleep-onset insomnia is that cues associated with falling asleep are separated from activities incompatible with sleeping. The bed becomes a discriminative stimulus (S^D) for falling asleep (Bootzin, 1972).

Several studies have indicated that this treatment may lead to greater reductions in sleep latency than progressive relaxation, and that both treatments are superior to high-demand placebo conditions (Bootzin, Note 1; Lawrence & Tokarz, Note 2). However, these findings do not necessarily support stimulus control theory or the applicability of the term *stimulus control* to this treatment. Indeed, the results of some recent studies suggest other factors may be

responsible for the effectiveness of the so-called "stimulus control" procedures.

Tokarz and Lawrence (Note 3) attempted to separate stimulus control components from temporal control components in Bootzin's procedures. They found that both sets of procedures resulted in reports of reduced latencies to sleep onset, whereas placebo and no treatment did not. However, their temporal control condition involved a component not present in Bootzin's procedures. Therefore, it is not clear that the more temporal components of Bootzin's procedures alone would result in reduced latencies. This study is also limited by a failure to assess the credibility of the treatments used.

Haynes, Follingstad, and McGowan (1974) investigated the sleep patterns of insomniacs and noninsomniacs and found no significant differences between the frequencies of sleep-incompatible behaviors associated with the bed and bedroom by these two groups. These findings make the need for further investigation even more compelling.

The present study was undertaken to examine the components responsible for the efficacy of the stimulus control procedure and to assess the applicability of the term *stimulus control* to this procedure. Toward this end, we isolated some of the apparent

This report is based on a master's thesis completed by the first author under the direction of the second author. Parts of this research were presented at the meeting of the Association for Advancement of Behavior Therapy, New York City, December 1976.

We are grateful to C. James Scheirer and Lisa A. Lisman for their insightful comments and criticisms during the design and completion of this work.

Requests for reprints should be sent to Stephen A. Lisman, Department of Psychology, State University of New York, Binghamton, New York 13901.

contingent and associative components of the stimulus control treatment.

Method

Subjects

We recruited undergraduates from psychology classes who indicated on a general sleep questionnaire that they had problems falling asleep, usually took longer than 30 minutes to fall asleep, and were interested in participating in a treatment study of sleep disturbance. Students reporting that their sleep disturbance was due to external noise, that they were currently taking drugs, or that they were receiving other treatment for their sleep disturbance were excluded from the study.

At an introductory meeting, retained subjects ($N = 47$; 22 females and 25 males) were told that treatment would involve four additional group meetings and were asked to sign a consent form indicating they understood that their participation was completely voluntary. Then, they were given a packet of seven daily sleep questionnaires to fill out on awakening each morning in the following week (baseline week). Subjects were asked not to drink alcohol or take medication for 3 hours prior to retiring to sleep for the duration of the study.

Treatment

Daily reports of sleep-onset latency during the baseline week enabled us to rank subjects and then randomly assign them within levels of severity to one of the following five conditions:

Stimulus control. As described by Bootzin, subjects were told: (1) Lie down to sleep only when sleepy. (2) If unable to fall asleep after 10 minutes, get up out of bed and engage in some activity (read, eat, watch television, etc.). Return to bed only when sleepy, and repeat this entire procedure as often as necessary. (3) Use the bed only for sleeping and/or sexual activities. (4) Do not take any naps. (5) Arise by alarm at the same time each morning.

Noncontingent control. The theoretically critical contingency aspect of the treatment was removed. Thus, subjects were told to arise a fixed number of times within 20 minutes of retiring. Since this fixed number was determined by the number of arisings in the stimulus control group, this group functioned almost like a yoked control.

Countercontrol. If unable to fall asleep within 10 minutes of retiring, subjects were to remain in bed, sit up, and engage in some activity (read, eat, watch television, etc.). This procedure was to be repeated as often as necessary. Subjects were also instructed to engage in some activity in bed for at least $\frac{1}{2}$ hour every day. If successful, this procedure would reflect the efficacy of con-

tingent disruption of the difficult sleep-onset period, but would preclude explanations based on Bootzin's notion of the bed as an S^P.

Temporal control. Subjects were told Instructions 1, 4, and 5 provided to the stimulus control group to control for the possibility that treatment success is due primarily to temporal components.

Waiting list. Subjects were told they would receive treatment later in the semester and were requested to complete all daily sleep reports similar to the other four groups until that date.

At the beginning of treatment, all treated subjects were given counterdemand and positive demand instructions to the effect that improvement was likely only after the fourth treatment session. Thus, the demand characteristic during the first 3 weeks of treatment were in the direction of eliciting reports of nonimprovement. Critical statistical comparisons were made prior to the fourth treatment week.

Except for the waiting-list group, each group met once weekly for 30 minutes for 4 weeks beyond baseline. All subjects were contacted 4 weeks after the end of the treatment period and asked to complete an additional set of questionnaires in the following week.

Results

Subject Attrition

Because of failure to cooperate with procedural requirements, 6 subjects were excluded from the study prior to the fourth week. This resulted in 41 subjects (8 stimulus control, 8 temporal control, 9 noncontingent control, 9 counter control, and 7 waiting list) being included in the analysis of data from baseline through Treatment Week 3. Questionnaires from 1 temporal control subject and 2 countercontrol subjects were missing for Treatment Week 4 due to subject illness and loss of questionnaire in the mail. Finally, 33 subjects completed the daily questionnaire during the follow-up week (6-7 subjects in each group).

Daily Sleep Questionnaires

The daily sleep questionnaires assessed five aspects of sleep disturbance: (a) latency to sleep onset on the previous night (in minutes), (b) ratings on a 6-point scale of difficulty falling asleep, (c) the number of times the subject awoke during the night,

(d) the number of times the subject awoke and had difficulty falling back to sleep, and (e) ratings on a 4-point scale of restfulness on awakening in the morning. Sleep latency was calculated from the time the subjects reported first going to bed intending to sleep until the time the subjects reported falling asleep.

One-way analyses of variance indicated the groups did not differ on any of the questionnaire items during baseline week, with the exception of degree of restfulness on awakening. To partially control for these initial differences, analyses of covariance were performed on the restfulness item. To help account for the large within-groups variance on sleep latency created by the procedure used in assigning subjects to groups, analyses of covariance also were performed on the sleep latency scores. F s for homogeneity of regression were run for all comparisons performed on the degree-of-restfulness and sleep latency items. None approached significance. One-way analyses of variance were performed on the rest of the items for each week of the study. As sleep latency was the variable of most interest, Table 1 presents the means for each of the five groups on the sleep latency item

of the questionnaire for the baseline week, Therapy Week 3 (counterdemand), Therapy Week 4 (positive demand), and follow-up.

Counterdemand period. Treated subjects were instructed not to expect improvement during the counterdemand period. Analyses of data from Therapy Week 3 (the last week of the counterdemand period) revealed overall significant differences between groups on the sleep latency variable, $F(4, 35) = 4.19$, $p < .01$, and differences approaching significance on the ratings of difficulty falling asleep, $F(4, 36) = 2.28$, $p < .10$. Considering the investigative nature of this study, it was of interest to determine if the stimulus control group improved significantly over any of the control groups and of equal interest to determine if any of the control groups improved significantly over the waiting-list group. As the overall F s for sleep latency for Therapy Weeks 3 and 4 were significant, least significant differences tests were conducted on these variables, and parallel comparisons were run on the other items on the questionnaire (Carmer & Swanson, 1973).

Planned comparisons revealed that both stimulus control and countercontrol groups

Table 1
Unadjusted Mean Sleep Latency (in Minutes) for Groups at Baseline Week, Therapy Week 3 (Counterdemand), and Therapy Week 4 (Positive Demand)

Week	Group				
	Stimulus control	Temporal control	Noncontingent control	Counter-control	Waiting list
Baseline week					
<i>M</i>	46.12	40.50	43.22	49.11	46.00
<i>SD</i>	20.82	13.93	17.81	29.42	18.97
Therapy Week 3 (Counterdemand)					
<i>M</i>	26.00	30.50	45.33	29.22	45.57
<i>SD</i>	16.38	12.39	18.37	17.03	8.38
Therapy Week 4 (Positive demand)					
<i>M</i>	26.37	19.00	34.22	25.22	39.41
<i>SD</i>	11.65	10.18	8.05	11.78	15.46
Follow-up					
<i>M</i>	25.16	22.71	27.00	18.42	41.57
<i>SD</i>	10.28	19.04	20.56	5.28	16.40

reported shorter latencies to sleep onset than the waiting-list group: $F(1, 35) = 8.48$, $p < .01$, and $F(1, 35) = 6.84$, $p < .025$, respectively. Stimulus control and counter-control groups also reported less difficulty in falling asleep than the waiting-list group: $F(1, 36) = 6.60$, $p < .025$, and $F(1, 36) = 4.94$, $p < .05$, respectively. There were no significant differences between the stimulus control and countercontrol groups on these or any other variables. The stimulus control group also reported shorter latencies to sleep onset than the noncontingent control group, $F(1, 35) = 9.28$, $p < .01$. No significant effects emerged from analysis of the number of times subjects awakened, the number of times subjects had difficulty returning to sleep, or rated restfulness on awakening.

In general, these comparisons suggest that during the counterdemand period, the stimulus control and countercontrol procedures led to equivalent and significant improvement on several variables, whereas temporal control and noncontingent control procedures did not.

Positive demand period. The same analyses that were performed on data from each item for Therapy Week 3 were also performed on each item for Therapy Week 4. Subjects had been told to expect improvement during this week. Analyses here revealed significant differences between groups on the sleep latency variable, $F(4, 32) = 3.89$, $p < .025$, and on the restfulness-awakening variable, $F(4, 32) = 7.35$, $p < .001$. During the positive demand period, all treated groups showed significant improvement over the waiting list group on at least three items of the questionnaire (all at least $p < .05$).

Follow-Up

Subjects were contacted 4 weeks after the conclusion of treatment and asked to complete daily questionnaires for 1 additional week. Table 1 indicates that at follow-up, both stimulus control and counter-control groups maintained or further enhanced the gains reported during counter-

demand conditions. Yet no significant differences among groups emerged for sleep-onset latency or any other measure. We suspect that the disappearance of earlier differences was due both to the reported improvement of the temporal and noncontingent control groups (a possible carry-over of positive demand?) and some loss of subjects ($N = 33$ for the follow-up). Except for the waiting-list group in which no subjects were lost, attrition ranged from one to three subjects per group.

Credibility

A frequent criticism of analogue therapy research is that placebo groups may not adequately control for expectancy of improvement (Borkovec & Nau, 1972). Therefore, we asked an independent group of undergraduates ($N = 56$) to read one of the four treatment rationales and sets of instructions and rate it on four 6-point scales assessing how logical the treatment was, how well it generated expectancy for improvement, and if the subject would be willing to recommend the treatment to a friend or undergo the treatment him- or herself. One-way analyses of variance indicated that the four treatments did not differ on any of these scales.

Discussion

The results of the present study both demonstrate the effectiveness of the stimulus control procedure in the treatment of moderate insomnia and suggest that such improvement could not be attributed to the idea that the bed had become an S^D for sleep. Furthermore, the responses of our various treatment and control groups appear to rule out explanations of such differential improvement based on fatigue, regular scheduling of retirement, and credibility or placebo (demand) effects.

We believe that three hypotheses best account for the comparable improvement of our stimulus control and countercontrol groups. First, both groups involve contingent disruption (and concomitant limita-

tion?) of bed and bedtime as cues for the arousal possibly associated with worrying, tossing, and turning in bed often characteristic of insomniacs (Freedman & Papsdorf, 1976; Monroe, 1967).

Second, as suggested by Bootzin (1972), our subjects may have experienced contingent punishment of continued wakefulness, since having to sit up, get out of bed, and so on may have been seen as aversive. Although we did not systematically attempt to gather such information, several subjects in both the stimulus and countercontrol groups commented on the discomfort of the procedures.

A third hypothesis derives from self-perception theory (Bem, 1967). It is readily observed that active attention or engaging in unfamiliar activity decreases the apparent duration of an interval (Loehlin, 1959). Thus, it is possible that improvement in stimulus control and countercontrol groups reflects a change in subjects' estimations of time during the critical period between first getting into bed intending to sleep and first falling asleep. Indeed, reports that poor sleepers or insomniacs tend to overestimate the amount of time it takes them to fall asleep (Carskadon et al., 1976; Frankel, Coursey, Buchbinder, & Snyder, 1976; Monroe, 1967) point to the possibility that this overestimation itself should be considered part of the disturbance. Self-perception theory would suggest that more accurate (i.e., decreased) estimations of time to fall asleep would be inconsistent with labeling oneself or perceiving oneself as an insomniac. Resolution of this inconsistency by removing the label should, in turn, lead to diminished worry, concern, and more rapid sleep onset.

Each of the treated groups improved significantly over the waiting-list group on at least three aspects of sleep disturbance assessed by the daily sleep questionnaire during the positive demand week. However, because subjects were told to expect improvement during this week, their reported improvement may represent the confluence of many factors. As noted before, although no differences between groups were found

at follow-up assessment, this may have been due to loss of subjects. Treatment gains were clearly maintained in stimulus control and countercontrol groups, which is consistent with other studies noted previously that have indicated continued improvement in subjects treated with stimulus control procedures.

In light of the above considerations, we believe that the effectiveness of the stimulus control treatment is most parsimoniously explained by its ability to contingently disrupt sleep-incompatible activities and/or cognitions that occur during the difficult period prior to sleep onset. The findings of this study call into question the applicability of the term *stimulus control* to this treatment, and our alternative hypotheses suggest questions for future research.

Reference Notes

1. Bootzin, R. *Stimulus control of insomnia*. Paper presented at the meeting of the American Psychological Association, Montreal, September 1973.
2. Lawrence, P. S., & Tokarz, T. *A comparison of relaxation training and stimulus control*. Paper presented at the meeting of the Association for the Advancement of Behavior Therapy, New York City, December 1976.
3. Tokarz, T. P., & Lawrence, P. S. *An analysis of temporal and stimulus factors in the treatment of insomnia*. Paper presented at the meeting of the Association for the Advancement of Behavior Therapy, Chicago, November 1974.

References

- Bem, D. J. Self perception: An alternative interpretation of cognitive dissonance phenomena. *Psychological Review*, 1967, 74, 183-200.
- Bootzin, R. A stimulus control treatment for insomnia. *Proceedings of the 80th Annual Convention of the American Psychological Association*, 1972, 395-396.
- Borkovec, T. D., & Nau, S. D. Credibility of analogue therapy rationales. *Journal of Behavior Therapy and Experimental Psychiatry*, 1972, 3, 257-260.
- Carmer, S. G., & Swanson, M. R. An evaluation of ten pairwise multiple comparison procedures by Monte Carlo methods. *Journal of the American Statistical Association*, 1973, 68, 66-74.
- Carskadon, M. A., et al. Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *American Journal of Psychiatry*, 1976, 133, 1382-1388.

- Frankel, B. L., Coursey, R. D., Buchbinder, R., & Snyder, F. Recorded and reported sleep in chronic primary insomnia. *Archives of General Psychiatry*, 1976, 33, 615-623.
- Freedman, R., & Papsdorf, J. D. Biofeedback and progressive relaxation treatment of sleep-onset insomnia: A controlled, all-night investigation. *Biofeedback and Self-Regulation*, 1976, 1, 253-271.
- Haynes, S. N., Follingstad, D. R., & McGowan, W. T. Insomnia: Sleep patterns and anxiety level. *Journal of Psychosomatic Research*, 1974, 18, 69-74.
- Loehlin, J. C. The influence of different activities upon the apparent length of time. *Psychological Monographs: General and Applied*, 1959, 73(4, Whole No. 474).
- Monroe, L. J. Psychological and physiological differences between good and poor sleepers. *Journal of Abnormal Psychology*, 1967, 72, 255-264.

Received April 14, 1978 ■