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# *RELAXATION TREATMENT OF PSEUDOINSOMNIA AND IDIOPATHIC INSOMNIA: AN ELECTROENCEPHALOGRAPHIC EVALUATION*

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Twenty-nine insomniacs underwent four consecutive sleep laboratory evaluations before and after receiving tension-release relaxation training, no-tension-release relaxation training, or no-treatment. On the basis of the discrepancy between subjective and EEG-defined measures of latency to sleep onset, subjects were classified as pseudoinsomniacs or idiopathic insomniacs. As predicted, tension-release relaxation was significantly more effective than the other two conditions on subjective sleep measures, regardless of insomnia subtype and on objective sleep measures only for idiopathic insomniacs. Subjective improvement was maintained at 12-month followup. Numerous differences between the two subtypes emerged on pretherapy and during-therapy measures distinct from the latency measures, but changes on those variables were unrelated to outcome improvement.

DESCRIPTORS: insomnia, pseudoinsomnia, treatment with progressive relaxation, humans

Our understanding of sleep-onset insomnia, the most common adult sleep disorder, has greatly increased during the last decade. Clinical sleep laboratories have identified numerous etiological factors (insomnia secondary to other psychological disturbances, Williams and Karacan, 1973; sleep apnea, Guilleminault, Eldridge, and Dement, 1973; restless leg syndrome, Frankel, Patten, and Gillin, 1974; nocturnal myoclonus, Lugaresi, Coccagna, and Cerni, 1969; drug dependence, Dement and Mitler, 1974; and circadian rhythm shifts, Hauri, Note 1). However, a substantial number of "residual" primary insomnias remain without identifiable cause. Theories advanced to explain these residual cases often emerge from research comparing insomniacs to good sleepers and reveal a variety of differences of potential etiological significance. For example, insomnia has been hypothesized to be a function of (a) anxiety (Haynes, Follingstad, and McGowan, 1974), (b) physiological overactivation (Johns, Gay, Masterson, and Bruce, 1971; Monroe, 1967), (c) presleep cognitive intrusion (Starker and Hasenfeld, 1976) and worry over sleep (Roth, Kramer, and Lutz, 1976), (d) variability in EEG sleep during successive nights (Karacan, Salis, and Williams, 1973), (e) less REM sleep (Rechtschaffen and Monroe, 1969) and/or less delta sleep (Frankel, Coursey, Gaarder, and Mott, Note 2), (f) personality differences (Kales, Caldwell, Preston, Healey, and Kales, 1976), and (g) conditioning of sleep-incompatible behaviors to bed-related stimuli (Bootzin and Nicassio, 1977).

Unfortunately, contrasting data exist indicating, for example, that the insomniac fails to show high frontalis EMG (Good, 1975; Haynes *et al.*, 1974), differences in personality (Rechtschaffen, 1969), or high frequency of sleep-incompatible behaviors in the bedroom (Haynes *et al.*, 1974) and does display *more* REM time (Othmer, 1966). Given the equivocal state of these data, basic research on insomnia has provided little conclusive evidence for any

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theoretical account of residual insomnia and little direction for the development of effective therapeutic strategies.

Regardless, the last 7 yr have also seen a growing trend toward behavioral treatment of insomnia. The short-lived effects of hypnotic medication with its associated tolerance, withdrawal, and addiction effects (Kales and Kales, 1973) no doubt has increased the urgency of identifying effective, nonpharmacological treatments. That trend has been reinforced by controlled investigations providing initial documentation of the efficacy of progressive relaxation as well as several other psychological interventions (*e.g.,* stimulus control, EMG biofeedback, sensory motor rhythm feedback, thought distraction, and numerous additional relaxation strategies including meditational, hypnotic, and autogenic procedures). In a recent review, Bootzin and Nicassio (1977) concluded that relaxation has been clearly demonstrated to result in moderate phenomenological improvement among mild to severe cases of insomnia. Unfortunately, as these authors pointed out, the majority of this evidence was based on self-report measures, which bear an unknown relationship to objective sleep indices. Furthermore, most of the studies simply compared treatment to placebo and no-treatment conditions on latency to sleep onset measures; few employed dependent measures and comparison conditions that would allow conclusions regarding either the maintaining factors of the disorder or the mechanisms of treatment by which improvement is established. Thus, applied research has similarly contributed little to our basic understanding of sleep and its disorders, even though no *a priori* reason prevents applied research designs from addressing such basic research questions.

We have perhaps made an even more serious error, however, in both previous basic and applied research efforts, one that threatens the validity of existing insomnia data accumulated during the past decade. Dement (1972) indicated that perhaps 50% of the primary insomniacs seen at the Stanford Sleep Clinic showed little evidence of sleep deficit according to EEG criteria. These "pseudoinsomniacs" reported requiring a long time to fall asleep at night and sleeping very little throughout the night, yet EEG revealed latency to sleep onset and total sleep time closer to normal limits than their reports suggested. The remaining cases showed clear sleep retardation and deficits, as measured by EEG, in accord with their subjective complaint and were labelled "idiopathic insomniacs". Other than Hauri's suggestion that idiopathic insomnia may be related to a weak sleep system exemplified by deficient sensory motor rhythm (Hauri, *in press*) and pseudoinsomnia to an overly active awakefulness system suggested by alpha-delta (nonrestorative) sleep (Hauri and Hawkins, 1973), no empirical comparisons between these two insomnia subtypes have been made. If Dement's (1972) estimates are correct, we can assume that samples from the sleep-disturbed population examined in both basic and therapy research have included both pseudoinsomniacs and idiopathic insomniacs in unknown numbers in any particular study and averaging 50% from each subtype over all studies.

The presence or absence of an objectively defined sleep deficit would seem to be of obvious and fundamental importance in insomnia research. Past failure to make that distinction leads to questioning the meaning of past research findings. For example, studies comparing insomniacs to good sleepers often, and not surprisingly, found the former group to "overestimate" latency to sleep onset relative to EEG measures (*e.g.,* Karacan *et al.,* 1973). Assuming equal representation of the two subtypes in such studies, the "overestimation" was quite likely a function of averaging both the self-reports and the EEG measures over the two insomnia subtypes, both reporting long latency to sleep onset, but only one group displaying objectively defined, severe retardation of sleep. Failure to obtain objective sleep measures in therapy research, combined with a failure to make the

subtype distinction, similarly leaves the behavioral intervention literature completely ambiguous. We know, for example, that relaxation resulted in subjective improvement (Bootzin and Nicassio, 1977). We do not know, however, whether the subjects had any initial, objective deficit at all or whether those who did were among the cases reporting phenomenological improvement after treatment.

Although two EEG studies of progressive relaxation (Borkovec and Weerts, 1976; Freedman and Papsdorf, 1976) provided tentative evidence that relaxation indeed produced objectively defined improvements in sleep, future research clearly must take the insomnia subtypes into account before meaningful conclusions regarding insomnia and its treatment can be drawn. Etiological and maintenance factors are quite likely different for the two disorders. Thus, appropriate interventions and/or the mechanisms by which target improvement is established within each disorder are probably different.

For the past 7 yr, the present author and his colleagues have been engaged in a component control analysis of the progressive relaxation treatment of insomnia. Progressive relaxation operationally involves (a) tension-release of muscle groups and (b) instructions to focus attention on the resulting physiological sensations. Presence or absence of each operation thus defines a $2 \times 2$ design for a complete component analysis of the active ingredients of the procedure. Briefly, the results of our investigations have indicated that tension-release relaxation with physiological attention-focusing (the progressive relaxation package) is superior in reducing subjective latency to sleep onset relative to (a) the control condition involving neither operation (Borkovec, Kaloupek, and Slama, 1975; Steinmark and Borkovec, 1974) and (b) no-tension-release relaxation with physiological attention-focusing (Borkovec *et al.*, 1975). However, tension-release relaxation *without* physiological attention-focusing has been found to be equivalent to progressive relaxation in inducing phenomenological improvement (Borkovec and Hennings, 1978), implicating muscle tension-release as the critical therapeutic ingredient. Replication of these studies has begun in our all-night lab series in order to assess relaxation effects on EEG-defined sleep. Some objective evidence for the effectiveness of progressive relaxation over the control condition involving neither operation has emerged (Borkovec and Weerts, 1976). All of these studies were conducted before we realized the importance of the pseudo- and idiopathic insomnia distinction, however.

Two of the primary goals of the present study were to continue our evaluation of the effects of relaxation and its components on EEG-defined and self-reported sleep onset, but more importantly, to assess differential treatment effects on the objective and subjective disturbance of pseudo- and idiopathic insomniacs. Given the lack of empirical data on the two subtypes, the study also afforded the valuable opportunity to compare the two groups on pretherapy EEG and self-report measures of potential descriptive and theoretical importance. Multidimensional assessments, including objective and subjective sleep measures before and after treatment and physiological and subjective measures during treatment, were obtained to provide evidence relating to some of the previously advanced theories of insomnia.

Conditions for the present study included tension-release relaxation with physiological attention-focusing (progressive relaxation), no-tension-release relaxation with physiological attention-focusing, and no-treatment. After completion of the study, subjects within each condition were categorized into pseudo- and idiopathic insomnia subtypes on the basis of the degree of discrepancy between pretherapy EEG and subjective measures of latency to sleep onset.

Our expectations of the outcome were straightforward:

(a) On the basis of our previous research, progressive relaxation was predicted to be su-

perior to the other two conditions in reducing subjective latency to sleep onset for both subtypes.

(b) Progressive relaxation should be superior to the other two conditions in reducing EEG-defined latency to sleep onset among idiopathic insomniacs. Since pseudoinsomniacs, by operational definition, display long subjective latency but short objective latency, no improvement in EEG-defined latency could occur, and therefore, no differences in objective outcome among the three treatment conditions would emerge.

## METHOD

### *Subjects*

At the beginning of the spring and fall semesters of 1976, a sleep questionnaire was administered to the introductory psychology class ($N = 1600$) at the University of Iowa. Potential subjects indicated that their typical latency to sleep onset was 60 min or longer, that they considered this latency to be a problem for them, and that they were willing to volunteer for a treatment study designed to help them overcome this problem. Further selection from this group was based on an initial interview designed to eliminate individuals who were currently receiving drug or psychological treatment of any kind or had obvious medical or psychological problems in addition, or contributing, to the sleep problem. The 30 subjects (15 from each semester) with the most severe latency problem were accepted into the study. The study's requirements were described in detail, and a commitment to participate for the sole benefit of receiving treatment for the problem was elicited before informing the subjects of monetary payment and research credit for their participation. They were informed that treatment would begin sometime within the next 12 weeks, although some assessment sessions would occur earlier. Within each sex, subjects were combined into groups of three on the basis of common bed time. Each triplet constituted one wave of subjects; subjects within each wave were randomly assigned to one of the two therapy conditions or to no-treatment. Each wave of subjects was given a packet of daily sleep questionnaires two weeks before their pretherapy lab nights and were instructed to complete one questionnaire each day, immediately upon awakening, during the entire duration of the study, until one week after the final treatment session. The questionnaire asked for latency to sleep onset and two nine-point ratings of degree of bodily tension and frequency of thoughts and images from the previous night's presleep period. One week before pretherapy lab nights, subjects completed the MMPI.

Subjects received $5.00 per all-night session, $2.00 per training session contingent on prompt attendance of scheduled sessions, and research participation credit in fulfillment of course requirements.

### *Procedure*

*All-night evaluations.* Subjects were evaluated in waves of three per week (one subject in each treatment condition) in the sleep lab for four consecutive nights (Monday to Thursday); each wave commenced the week after the preceding wave. The three subjects arrived 30 min before their typical bed time and were awakened in the morning at their typical awakening time. Electrode attachment and retiring procedures required 20 to 30 min. Monopolar EEG was recorded from C3 relative to a right mastoid neutral site (left mastoid being ground) using Beckman silver-silver chloride electrodes. Sites were abraded with Hewlett-Packard Redux abrasive gel to ensure low resistance (below 5000 ohms), and electrodes filled with EKG sol were firmly secured to the scalp by a gauze covering saturated in collodion. Recordings were made by a Beckman Type R611 Dynograph with 0.05 mV/cm sensitivity and a time constant of 0.3 sec. Chart speed was set at 10 mm per second. Each subject slept in an individual bedroom containing a bed, a small table, chair, and intercom. Upon awakening

in the morning, subjects completed a sleep questionnaire identical to the daily forms completed at home.

During the week following the eighth training session, subjects returned for posttherapy evaluations again involving four consecutive all-night sessions (Monday to Thursday) and following exactly the procedures employed for the pretherapy lab nights. The purpose of the second set of evaluation nights, as presented to the subjects, was for additional data collection regarding physiological aspects of sleep. Thus, no implication of "posttesting" or evaluation of therapy effects was made.

*Treatment.* Subjects assigned to therapy conditions returned each week for two individual training sessions per week for four consecutive weeks and one final session after the posttherapy lab nights. Subjects in the progressive relaxation condition received nine training sessions following the procedures of Bernstein and Borkovec (1973). In those procedures, 14 muscle groups are employed for the first three sessions, seven combined muscle groups for the next two sessions, four combined muscle groups for the next two sessions, and a four-group recall procedure for the last two sessions. Five to 7 sec of tension on each muscle group were followed by 30 sec of attending to the resulting relaxation sensations. Two such tension-release cycles were administered to each muscle group before proceeding to the next group. Additional cycles were provided if the subject reported remaining tension in the muscle group after two cycles. Training procedures for the no-tension-release relaxation condition (*cf.* Borkovec *et al.,* 1975) were identical to those employed in progressive relaxation in every respect, except that muscle tension-release was omitted. At the end of the first session, subjects completed a credibility questionnaire (Borkovec and Nau, 1972) designed to assess whether the two training conditions were equivalent in believability and inducement of an expectancy of ultimate therapeutic benefit (Kazdin and Wilcoxon, 1976). Subjects in both treated conditions received written instructions for twice-a-day relaxation practice between sessions, the last practice each day to occur upon retiring.

All training sessions were conducted by subject-controlled, automated, taped procedures (*cf.* Borkovec, Grayson, and Cooper, 1978). Two tapes (one containing two cycles on each muscle group and one containing repeated cycles) and a switching device activated by a button press allowed the subject to alternate tapes in response to questions regarding presence or absence of muscle tension. As such, the subject received training that was at his/her own pace and identical in procedure to how a therapist would conduct the session. Standardization of procedure and removal of a potentially confounding therapist factor were the major advantages of this method. Additionally, all other standard instructions and rationale statements for treated subjects and all communications with no-treatment subjects were made by tape-recorded messages. Two technicians intimately familiar with relaxation procedures and working with half of the subjects in each treatment condition did, however, answer questions and solve problems that the subject raised regarding the training and its application to his/her own situation.

Counterdemand instructions were presented during the initial interview, during the first session, and repeatedly in the form of a written message on the daily sleep questionnaire. This instruction informed the subject not to expect improvement until after the ninth session, because that many sessions and amount of practice time would be necessary to achieve a usable and effective level of relaxation skill. The purpose of this instruction was to reduce the influence of demand characteristics and expectancy effects on self-report measures. Our previous research using counterdemand has shown that it accomplishes this purpose, that is, placebo groups report no improvement while relaxation groups report significantly greater improvement during counterdemand (Borkovec *et al.,* 1975; Steinmark and Borkovec, 1974). The ninth ses-

sion occurred during the week after the posttherapy lab nights. No additional all-night evaluations were conducted, since counterdemand was no longer in effect after the ninth session; such data would be confounded by demand/expectancy effects.

*During-treatment physiological evaluations.* During Sessions 1, 7, and 9, physiological recordings (heart rate, respiration, frontalis EMG) were obtained during a 10-min adaptation period and throughout the training session. No-treatment subjects were brought into the lab at times corresponding to the first, seventh, and ninth sessions of their treatment counterparts for "physiological assessment sessions". They were told that these sessions were required, in conjunction with the sleep lab nights, for purposes of detailed assessment of their psychophysiological activity before initiation of the therapy, which would begin one week after the last set of lab nights. They were simply asked to sit quietly and relax themselves for a period of time matching the duration of the training sessions for treated subjects. All subjects completed presession and postsession Anxiety Differentials (Husek and Alexander, 1963). Heart rate was recorded via silver-silver chloride electrodes attached to the right and left lower ribs; mercury strain gauges attached to the upper and lower chest provided transduction for respiration; silver-silver chloride electrodes to the right and left frontal group provided EMG signals. All physiological recordings were obtained on the Beckman R611 Dynograph; chart speed was set at 5 mm per second.

Waiting-list no-treatment subjects received the full training sequence in progressive relaxation after the final posttherapy assessments.

*Followup.* Twelve months after the last treatment session, subjects were contacted and asked to estimate current, typical latency to sleep onset. They had not been informed previously that this phone contact would occur.

*Data reduction.* Each subject's daily sleep questionnaire data obtained at home were averaged over each of the seven total weeks of the study. Sleep questionnaire data obtained the mornings after sleep lab sessions were analyzed separately. EEG records were scored by Rechtschaffen and Kales' (1968) criteria for first occurrence of Stage I sleep, first occurrence of Stage II sleep, total number of minutes of Stage II, and total number of minutes of slow-wave sleep (Stages III and IV). Interrater correlations based on independent scoring of all records ($N = 228$) by two raters "blind" to condition status of the subjects were 0.85, 0.97, 0.82, and 0.62, respectively; average of the two raters was obtained for each subject's score and was employed in all subsequent analyses. All-night totals for Stage W (awake) and Stage I could not be validly obtained because one channel of EEG recording alone does not allow distinguishing these stages from Stage REM. Although first-night effects are frequently found in sleep research (*e.g.,* Scharf, Kales, and Bixler, 1975), initial analyses indicated no differences between Monday night data and the remaining nights; thus, subsequent analyses were based on records obtained from every evaluation night. During-treatment physiological records were scored for heart rate, respiration period, and frequency of EMG signals. Physiological samples were obtained during the last minute of adaptation, 1 min during the last minute of each third of the training session, and 1 min at the end of a 2-min "enjoyment period" subsequent to completing the formal relaxation training. Heart rate in beats per minute, respiration in seconds per cycle, and EMG signals of 50 $\mu$V amplitude or greater per minute were scored by raters from the paper records.

*Pseudoinsomnia and idiopathic insomnia categorization.* After completion of the study, a ratio (average self-reported latency to sleep onset during the four pretherapy lab nights to the average Stage I EEG latency to sleep onset) was calculated for each subject. Subjects whose ratio was greater than 1.5 were categorized as pseudoinsomniacs, whereas subjects whose ratio was less than 1.5 were categorized

as idiopathic insomniacs. Since no criteria for classification of insomnia subtypes have been established in the literature, we thus chose a face-valid criterion of 50% of self-report overestimation relative to EEG. This criterion resulted in dividing the subjects into pseudo- and idiopathic insomnia groups representing 60% and 40% of the sample, respectively. One subject in progressive relaxation sought professional help for severe obsessive-compulsive neurosis before the study was completed. With this subject excluded, nine progressive relaxation subjects (five pseudo- and four idiopathic insomniacs), 10 no-tension-release relaxation subjects (six pseudo- and four idiopathic insomniacs), and 10 no-treatment subjects (six pseudo- and four idiopathic insomniacs) completed the study. During-treatment physiological data on one of the six pseudoinsomniacs in the no-tension-release condition were lost due to recording distortions.

## RESULTS

### *Pretherapy Measures*

Initial analyses on pretherapy data tested (a) whether treatment conditions differed before therapy administration, in order to ensure pretherapy equivalence of the experimental groups, and (b) whether pseudo- and idiopathic insomniacs differed on sleep and nonsleep variables of descriptive importance.

*Self-report sleep measures.* Two-way analyses of variance (Subtype × Treatment) on pretherapy daily sleep questionnaire items completed at home revealed no significant effects. Subtype × Treatment × Night analyses of variance on the pretherapy sleep lab questionnaire data obtained in the morning after each all-night session found no differences associated with any factor on reported latency to sleep onset, frequency of cognitive activity, or number of awakenings. A significant Subtype × Night interaction, $F$ (3, 69) = 2.81, $p < 0.05$, however, indicated that idiopathic insomniacs reported greater presleep bodily tension early in the week (means over nights: 4.8, 3.8, 3.0, and 3.2) than pseudoinsomniacs (means: 3.4, 2.8, 3.2, and 3.5).

*EEG sleep measures.* Due to significant variance differences among the Subtype × Treatment cells on pretherapy Stage I and Stage II onset, nonparametric tests were employed on all EEG sleep onset data. Since idiopathic and pseudoinsomniacs differed on pretherapy EEG as assessed by Mann-Whitney $U$ test due to the criterion for subtype classification, $z = 2.94$ for Stage I and $z = 2.26$ for Stage II, treatment effects on these pretherapy measures were analyzed separately for each subtype. Kruskal-Wallis analysis of variance (Hays, 1963) revealed no pretherapy differences among pseudoinsomniacs on Stage I, $X^2 = 2.14$, $p > 0.30$, or on Stage II, $X^2 = 4.56$, $p > 0.10$. Although inspection of Figure 2 means to be presented below suggests that idiopathic subjects in progressive relaxation displayed much greater sleep retardation at pretherapy than did the idiopathic subjects in the other two treatment conditions, Kruskal-Wallis analysis indicated that the treatment difference did not approach significance, $X^2 = 2.88$, $p > 0.25$, for Stage I and $X^2 = 2.42$, $p > 0.30$, for Stage II. The above-chance differences, while not significant, do have interpretive implications for the later analysis of improvement effects. We will address these issues as they arise.

Subtype × Treatment × Night analyses of variance conducted on all-night totals revealed no effects on total number of minutes of Stage II sleep and a significant main effect of subtype on slow-wave sleep, $F$ (1, 23) = 11.81, $p < 0.003$: Pseudoinsomniacs obtained only two-thirds the amount of Stages III and IV sleep obtained by idiopathic insomniacs (means: 63.6 *versus* 92.0 min per night, respectively).

*Measures from first therapy session.* Subtype × Treatment analyses of variance on the adaptation period before the first training session indicated that treatment and subtype groups did not differ on resting heart rate, respiration

rate, or degree of frontalis EMG activity. The same analysis conducted on the credibility questionnaire data obtained after the first session from the two treated groups revealed no effects involving the treatment factor. Idiopathic insomniacs reported the training procedures to be significantly less logical as a treatment for insomnia, $F$ (1, 15) = 13.39, $p < 0.003$, and were significantly less confident that the treatment would successfully eliminate insomnia, $F$ (1, 15) = 7.60, $p < 0.02$, than the pseudoinsomnia group. The former subjects also required an average of 3.75 additional relaxation cycles during the first session to achieve a report of deep relaxation, compared to an average of 0.98 additional cycles for the latter group, $F(1, 15) = 4.68, p < 0.05$.

*MMPI.* Sixty-two per cent of the subjects produced MMPI profiles with one or more T-scores equal to or greater than 70. This percentage approximates that reported by Carskadon, Dement, Mitler, Guilleminault, Zarconi, and Spiegel (1976) for their drug-free insomniacs and is in contrast with the higher incidences found by Kales *et al.* (1976) and Roth *et al.* (1976). Pseudoinsomniacs showed higher mean T-scores on every scale than idiopathic insomniacs,[2] although the only difference approaching significance was on the Ma scale, $F$ (1, 26) = 3.10, $p < 0.10$. Profiles with the highest elevation for the total group involved *Pt, Sc,* and *Ma.* The usual dominance of the Depression scale found in previous insomnia investigations was not present in our sample.

[2]MMPI T-scores for total group and each insomnia subtype:

| *Insomniacs* | *MMPI Scale* | | | | |
|---|---|---|---|---|---|
| | *Hs* | *D* | *Hy* | *Pd* | *Mf* |
| | 56.7 | 60.6 | 58.6 | 62.1 | 56.3 |
| Pseudo | 57.1 | 61.0 | 58.7 | 65.1 | 57.7 |
| Idiopathic | 56.7 | 60.1 | 58.4 | 58.0 | 54.5 |
| | *Pa* | *Pt* | *Sc* | *Ma* | *Si* |
| | 59.2 | 65.0 | 68.1 | 66.4 | 53.7 |
| Pseudo | 60.2 | 67.1 | 68.6 | 69.3 | 55.0 |
| Idiopathic | 57.8 | 62.2 | 67.3 | 62.6 | 52.1 |

*Pretherapy/Posttherapy Change*

To test the specific predictions of the study, planned comparisons between progressive relaxation and the other two conditions were performed on the subjective latency measures averaged over the two subtype groups and on the EEG latency measures separately within each subtype. Figure 1 presents the average latency scores for the daily sleep questionnaires from the pretherapy week to the last week of the counterdemand period and for the sleep lab questionnaires from pretherapy to posttherapy lab nights. Figure 2 presents the mean Stage I and Stage II latencies to sleep onset obtained from the pretherapy and posttherapy lab nights. Each figure illustrates the pseudo- and idiopathic insomnia groups within each treatment condition.

*Daily self-report measures.* A planned comparison using the error term from a Subtype × Treatment × Week analysis of variance conducted on reported latency to sleep onset on daily questionnaires from the pretherapy week to the last week of the counterdemand period indicated that progressive relaxation produced a significantly greater reduction than did the combination of no-tension-release relaxation and no-treatment, $F$ (1, 23) = 6.82, $p < 0.025$). Test of the residual variance indicated no further significant effects, indicating that no-tension-release relaxation did not differ significantly from no-treatment. This latter finding replicates the results of the Borkovec *et al.* (1975) comparison of no-tension-release and no-treatment conditions.

Subtype × Treatment × Week analysis of variance indicated a significant Treatment × Week interaction on bodily tension ratings, $F$ (2, 23) = 5.72, $p < 0.01$. Progressive and no-tension-release relaxation groups reported reductions from pretherapy to the last week of the counterdemand period, while the no-treatment group reported an increase (means: 3.9 to 3.2, 3.7 to 2.7, and 3.0 to 3.7, respectively). The same analysis on the cognitive rating scale indicated that idiopathic insomniacs reported

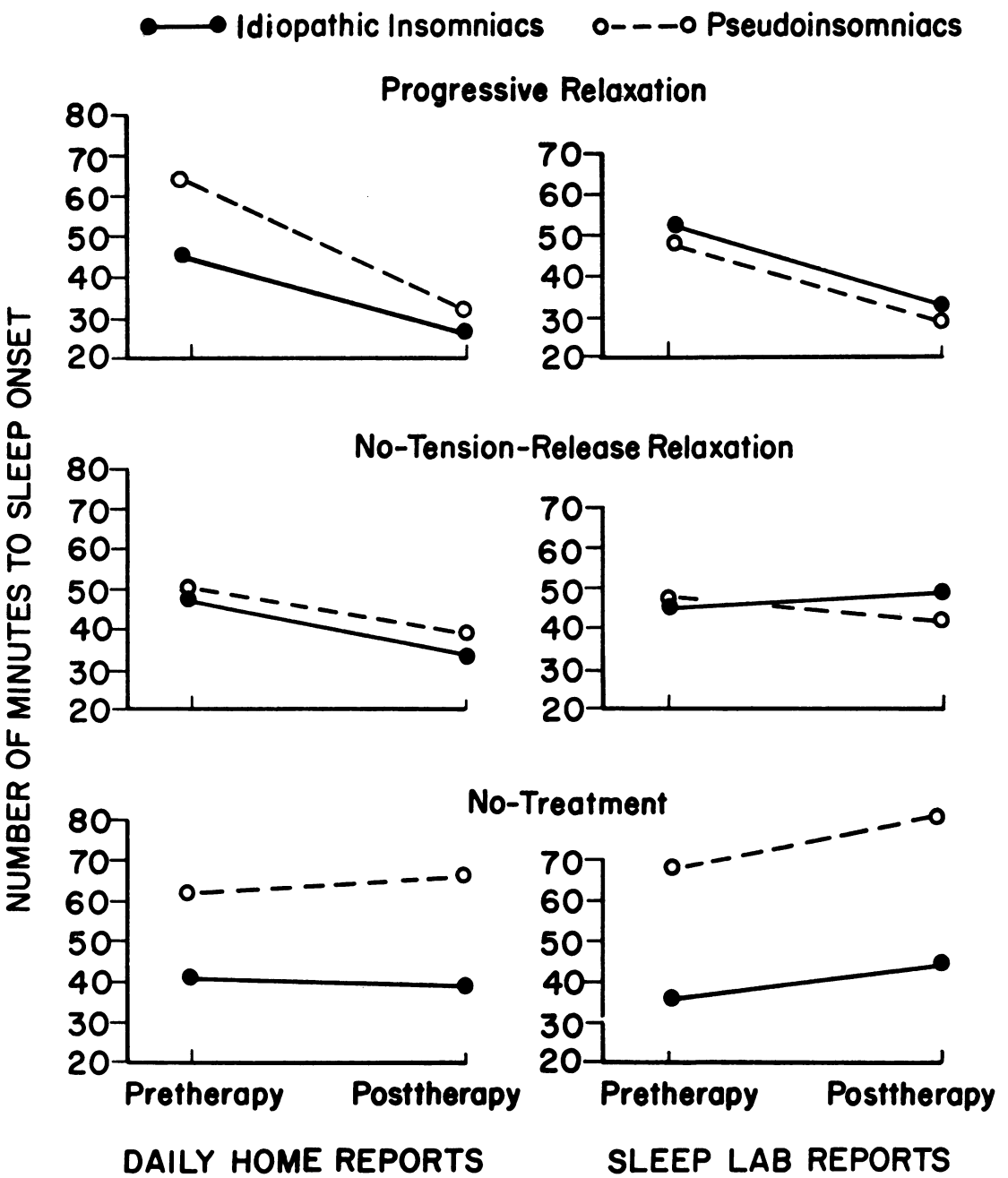


Fig. 1. Mean latency to sleep onset from daily home reports and sleep lab reports before and after treatment for idiopathic insomniacs and pseudoinsomniacs receiving progressive relaxation, no-tension-release relaxation, and no-treatment.

greater frequency of cognitive activity than pseudoinsomniacs, $F$ (1, 23) = 4.41, $p < 0.05$, means = 4.5 and 3.5, respectively. Treatment had no effect on the cognitive ratings. No effects emerged from analysis of number of awakenings.

*Sleep lab self-report measures.* A planned comparison using the error term from a Sub-

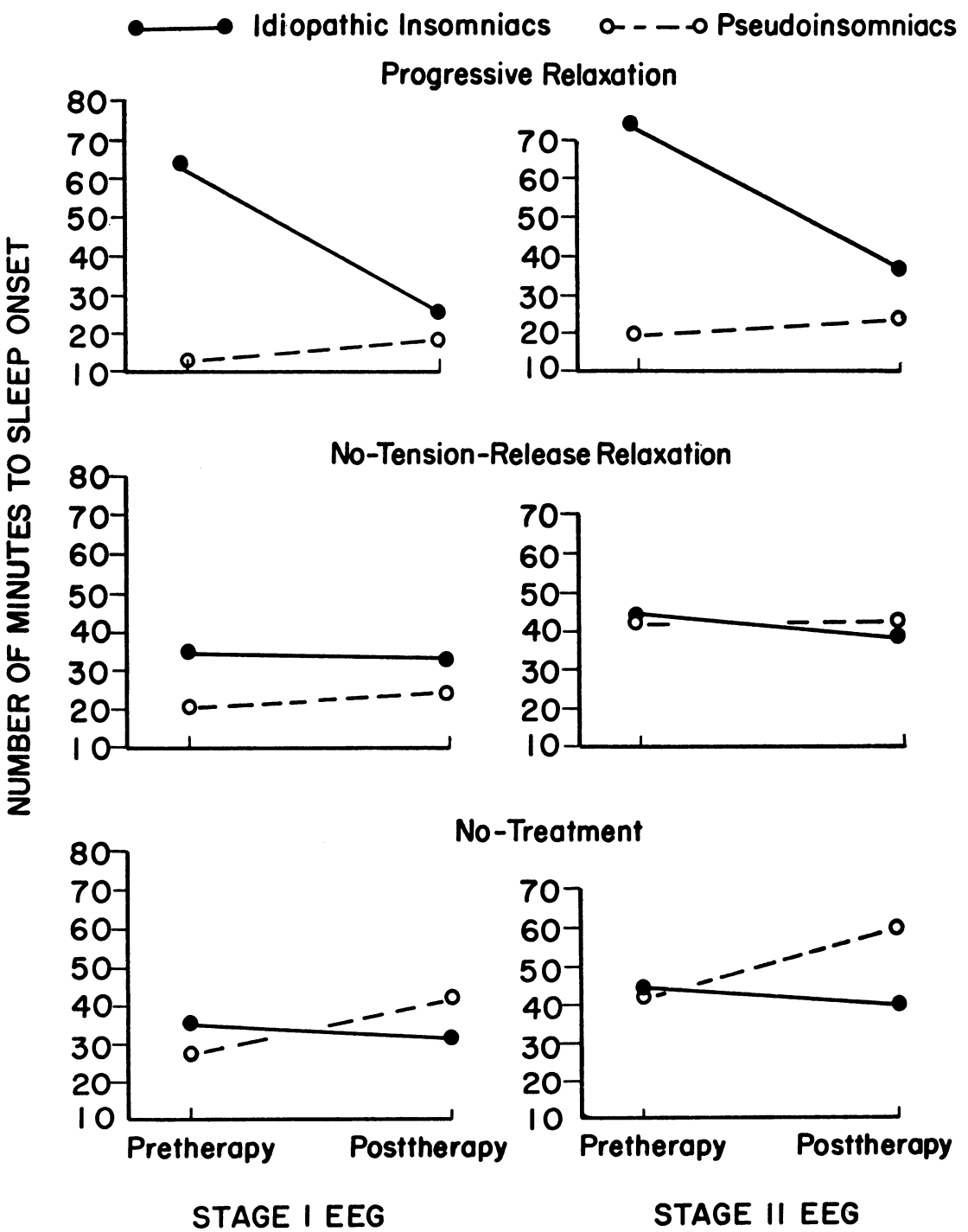


Fig. 2. Mean latency to sleep onset defined by Stage I and Stage II EEG before and after treatment for idiopathic insomniacs and pseudoinsomniacs receiving progressive relaxation, no-tension-release relaxation, and no-treatment.

type × Treatment × Pre/Posttherapy Week × Night analysis of variance conducted on reported latency to sleep onset on the sleep lab questionnaire from pre- to posttherapy lab nights revealed that progressive relaxation produced significantly greater reductions than the other two groups combined, $F$ (1, 23) = 7.55, $p < 0.025$. A test of the residual variance indicated no further significant effects, again indicating no difference between no-tension-release relaxation and no-treatment. Thus, principal analyses of both daily and sleep lab self-report measures confirmed our first hypothesis: progressive relaxation is superior to no-tension-release relaxation and no-treatment in reducing subjective latency of sleep onset, regardless of insomnia subtype.

Subtype × Treatment × Pre/Posttherapy Week × Night analyses of variance on the remaining sleep lab items found no significant effects on number of reported awakenings. Reflecting the same Subtype × Night interaction obtained on the pretherapy analyses, idiopathic insomniacs reported higher levels of bodily tension early in the week than the pseudoinsomniacs across pre-posttherapy nights, $F$ (3, 69) = 2.83, $p < 0.05$. Similar to the results of the daily questionnaires, idiopathic insomniacs reported higher levels of cognitive activity throughout sleep lab nights than the pseudoinsomniacs, $F$ (1, 23) = 5.03, $p < 0.04$, means = 5.1 and 3.8, respectively. The Treatment × Pre/Posttherapy interaction approached significance, $F$ (2, 23) = 2.25, $p < 0.13$, and is reported here only because the effect replicates the significant treatment influence on cognitive reports found in an earlier study (Borkovec and Hennings, 1978). Progressive relaxation was the only group to show reductions on the cognitive scale (means: 4.85 to 3.59) as compared to no-tension-release relaxation (means: 4.23 to 4.29) and no-treatment (means: 4.78 to 4.82).

*EEG latency to sleep onset.* Because pseudo- and idiopathic insomniacs differed significantly on pretherapy EEG latency measures, and because significant pretherapy variance differences existed among treatment conditions, nonparametric tests were applied separately with each subtype to the average reduction in EEG latency from pretherapy to posttherapy lab nights, *i.e.,* each subject's scores were averaged over the four pre- and four posttherapy nights, and pre-posttherapy change scores were then analyzed. For the idiopathic insomnia group, progressive relaxation was found by Mann-Whitney $U$ test (Siegel, 1956) to produce significantly greater improvement in both latency to Stage I onset, $U$ (4, 8) = 4, $p <$ 0.024, and latency to Stage II onset, $U$ (4, 8) = 4, $p < 0.024$, relative to the no-tension-release and no-treatment conditions combined.

Because of concern over the (nonsignificant) pretherapy differences among the idiopathic treatment groups, the individual Stage I and Stage II onset data are presented in Table 1. Inspection of these means indicates that progressive relaxation resulted in improvement in every case, regardless of initial level, while the most severe case in no-tension-release relaxation displayed increased retardation of sleep onset at posttherapy and two no-treatment subjects (one at the lowest pretherapy level and one at a higher level) also showed more delayed sleep onset during the posttherapy sleep lab week.

As expected, no significant treatment effects emerged from the same analysis of the pseudoinsomnia group. Thus, we felt our second hypothesis was confirmed.

Finally, since the criterion for subtype classification arbitrarily divided a continuous variable (ratio of reported to objective latency to sleep onset) into a dichotomy, the ratio scores were correlated by the Spearman rank method (Hays, 1963), with the number of minutes of improvement in Stage I onset from pre- to posttherapy nights within each treatment condition with the two subtypes combined. The correlation for the progressive relaxation condition was significant, $r$ (7) = −0.73, $p < 0.05$, whereas those for no-tension-release relaxation,

Table 1

Mean number of minutes to Stage I and Stage II sleep onset during pretherapy nights and posttherapy nights for each idiopathic subject receiving progressive relaxation, no-tension-release relaxation, and no-treatment.

| | *Progressive Relaxation* | | *No-Tension-Release Relaxation* | | *No-Treatment* | |
|---|---|---|---|---|---|---|
| | *Pretherapy* | *Posttherapy* | *Pretherapy* | *Posttherapy* | *Pretherapy* | *Posttherapy* |
| *Stage I* | | | | | | |
| S1 | 91.0 | 25.6 | 56.6 | 71.5 | 48.3 | 21.7 |
| S2 | 84.3 | 27.8 | 35.7 | 23.3 | 39.9 | 56.1 |
| S3 | 52.2 | 25.5 | 25.5 | 13.0 | 33.5 | 16.7 |
| S4 | 26.5 | 22.1 | 21.9 | 15.1 | 17.9 | 29.1 |
| *Stage II* | | | | | | |
| S1 | 110.0 | 31.8 | 61.0 | 74.9 | 54.1 | 36.6 |
| S2 | 88.9 | 61.1 | 43.7 | 29.0 | 44.2 | 59.8 |
| S3 | 58.7 | 30.7 | 36.4 | 38.1 | 44.0 | 22.7 |
| S4 | 35.0 | 28.1 | 30.2 | 19.3 | 34.3 | 41.4 |

$r$ (8) = +0.05, and no-treatment, $r$ (8) = −0.42, were not.

*All-night totals for Stage II and slow-wave sleep.* Subtype × Treatment × Pre/Posttherapy × Night analyses of variance were applied to the total number of minutes of Stage II and slow-wave sleep. Idiopathic insomniacs displayed a decline in amount of Stage II sleep from Monday to Thursday nights (means: 230.4, 204.7, 195.8, and 196.5 min), while pseudoinsomniacs began with less Stage II sleep and increased over nights (means: 202.0, 206.2, 220.6, and 222.2 min), $F$ (3, 69) = 3.68, $p < 0.02$. This effect was uninfluenced by either the pre-posttherapy factor or the treatment factor.

A main effect of Subtype, $F$ (1, 23) = 13.45, $p < 0.002$, indicated that pseudoinsomniacs showed less slow-wave sleep over all eight evaluation nights than idiopathic insomniacs (means: 58.6 and 85.4 min, respectively). While treatment did significantly influence the amount of slow-wave sleep from pre- to posttherapy, $F$ (2, 23) = 5.35, $p < 0.02$, that effect was primarily due to the greater decrease of the no-tension-release condition (means: 81.9 to 56.3 min) relative to the smaller declines of the progressive relaxation (means: 79.6 to 71.4 min) and no-treatment groups (means: 71.8 to 71.0 min).

*During-treatment measures.* No significant effects involving treatment or subtyped factors emerged from analysis of Anxiety Differential scores obtained from Sessions 1, 7, and 9. Subtype × Treatment × Session × Phase of Session analyses of variance revealed no effects involving subtype or treatment on the heart rate or EMG data. A significant Treatment × Session × Phase interaction, $F$ (16, 176) = 1.74, $p < 0.05$, emerged from analysis of respiration data. No-treatment subjects showed little variation over phases during all three sessions. Both treated groups displayed marked increases in respiration period during Session 1. During Session 7, no-tension-release relaxation resulted in little change, while progressive relaxation produced a substantial increase in period during the middle portion of the session. In Session 9, both treated groups showed increases in period, although no-tension-release subjects displayed a greater increase than progressive relaxation. While this effect suggests some minor physiological reduction effects of the two relaxation conditions, the size of the changes was not great and not wholly interpretable. In conjunction with the negative results of the other two measures, little evidence exists to support a physiological mediation hypothesis relative to the sleep onset improvement.

*Frequency of Relaxation Practice*

Treated subjects were instructed to practise relaxation twice a day. The overall mean number of daily practice sessions reported on the daily questionnaires was 1.44. Subtype × Treatment × Week analysis of variance of the practice reports resulted only in a significant three-way interaction. Among idiopathic insomniacs, no-tension-release subjects practiced more frequently than progressive relaxation subjects during the first three weeks, whereas both groups practiced equally often during the last three weeks. Among pseudoinsomniacs, progressive relaxation was practiced more frequently throughout the study than was the no-tension-release procedure. While this latter effect in itself might explain the failure of no-tension-release relaxation to influence the subjective sleep latency of the pseudoinsomniacs, no practice differences existed in a previous study of these two relaxation conditions, and yet progressive relaxation was superior to no-tension-release in reducing subjective latency (Borkovec *et al.*, 1975).

*Followup Self-Report*

Twelve months after the study concluded, we were able to contact six progressive relaxation, 10 no-tension-release relaxation, and seven no-treatment subjects. While interpretation of these data is unclear, since demand and other confounding variables are potentially operative and since differential attrition had occurred, the followup reports do provide some evidence regarding maintenance of treatment gains. All no-treatment subjects had received progressive relaxation training immediately at the conclusion of the study, thus providing a replication of relaxation effects on reported sleep disturbance.

Latency reports obtained during the final, positive demand week of the treatment study were subtracted from the followup latency reports. Both progressive and no-tension-release relaxation subjects essentially maintained whatever improvement had been achieved during the treatment phase (means = −0.2 and −5.8 min, respectively), a nonsignificant difference. No-treatment subjects reported reductions in sleep-onset latency (mean: −36.4) of a magnitude comparable to the improvement originally obtained by the treated groups.

## DISCUSSION

Two cautionary notes should be mentioned before offering our conclusions. First, there is concern over the pretherapy differences among the idiopathic treatment groups on the EEG sleep stage measures. Specifically, progressive relaxation displayed the greatest retardation. Since this group also showed the greatest decline subsequent to treatment to a posttherapy level not dramatically below the posttherapy levels of the other two conditions, one might well argue that simple regression accounts for the significant between-group effects. The authors feel this is not the case, for several reasons. First, it must be remembered that four consecutive nights of data were collected during pre- and posttherapy lab nights, providing a more adequate sample of the subject's sleep than is commonly employed in sleep research. Second, pretherapy differences did not approach significance. Third, variance tests confirm what inspection of Table 1 clearly suggests: significantly more homogeneous variance on posttherapy Stage I EEG for the idiopathic progressive relaxation group relative to every posttherapy *and* pretherapy variance of the other two conditions. On both stages for the former group, variance was dramatically reduced from pre- to posttherapy, while variance increased for the other two conditions. Finally, the first author has re-analyzed the Borkovec and Weerts (1976) data, employing the same criterion for subtype classification and has demonstrated results in complete accord with the present study (*cf.* Borkovec, *in press*). In that earlier investigation, progressive relaxation was compared

to pseudo-desensitization (involving neither muscle tension-release nor physiological attention-focusing), and waiting-list no-treatment. For idiopathic insomniacs, mean latencies to Stage I sleep onset (in minutes) from pretherapy to final posttherapy nights were 42.3 to 12.5 for progressive relaxation, 41.3 to 36.4 for placebo, and 43.8 to 37.2 for no-treatment. Again, progressive relaxation was found to be significantly superior to the other two groups, wholly without the presence of potentially confounding pretherapy differences. Furthermore, posttherapy variance was again significantly lower than posttherapy *and* pretherapy variances of the other two groups. Analysis of pseudoinsomniacs indicated no treatment effects (Stage I latency means from pretherapy to posttherapy: 10.2 to 7.2 for progressive relaxation, 13.0 to 9.2 for placebo, and 10.4 to 14.2 for no-treatment).

The second caveat is that the absolute amount of improvement on both subjective and objective measures was not dramatic. This is in agreement with Bootzin and Nicassio's (1977) conclusion that relaxation is only moderately effective as a treatment of insomnia. Progressive relaxation for the two subtypes in the present study resulted in a 50.0% reduction in reported latency at home and a 61.1% reduction in sleep lab reports. Among idiopathic insomniacs, the procedure produced reductions of 60.2% and 48.1% in the number of minutes required to achieve Stage I and Stage II sleep, respectively. In combination with the fact that the absolute posttherapy levels remained above latencies commonly reported by the vast majority of college students (0 to 20 min, Bernstein and Borkovec, 1973), these data suggest that we still have not identified the necessary or sufficient treatment conditions for either pseudo- or idiopathic insomnia. The gains reported are of some clinical significance, however, since a 50% problem reduction (on a carefully quantified behavior) is fairly substantial. Furthermore, our intention over the past 7 yr of research in this area has never been to develop an effective treatment for insomnia. Bootzin's stimulus control procedure (Bootzin and Nicassio, 1977) is far easier to implement and appears to have equivalent if not superior effects on subjective sleep disturbance. Our goal has been to investigate the mechanisms of progressive relaxation in the context of the insomnia problem and the mechanisms of insomnia itself.

To accomplish this goal, one must have a reliable phenomenon. The present study, we would argue, establishes that phenomenon fairly strongly. With these issues and arguments in hand, the discussion of the results follows below.

The moderate effectiveness of progressive relaxation training in the treatment of insomnia was supported by the above results. Furthermore, in replication of an earlier study, muscle tension-release was found to be crucial for producing immediate subjective improvement, while its role in producing objective improvement was documented for the first time. Most importantly, these effects were found to be specific to the specific deficits associated with each subtype of insomnia. For insomniacs who displayed an objective retardation of sleep onset in accord with their subjective complaint, tension-release relaxation resulted in both subjective and objective improvement. For insomniacs who reported a subjective disturbance greater than that revealed by EEG-defined sleep onset, tension-release relaxation was effective in reducing the subjective complaint. While the phenomenological improvement of the idiopathic insomniacs may well have been based on changes in objective sleep onset, the improvement among pseudoinsomniacs cannot be so attributed. In either case, however, short-term modification of the problem behavior was a function of the muscle tension-release ingredient of progressive relaxation.

The followup data indicated long-term maintenance of whatever subjective improvement was established immediately at the end of treatment. Our past studies have typically found

maintained improvement among subjects receiving progressive and no-tension-release relaxation training and loss of improvement among placebo conditions (*cf.* Borkovec and O'Brien, 1976). Our past placebo conditions represent the only comparison groups which we have had to rule out "spontaneous remission" at followup, as our no-treatment groups receive relaxation training at the end of each study. Two no-treatment subjects in the Borkovec and Weerts (1976) investigation, however, decided not to receive the training and displayed 12-month followup latencies equivalent to their original latencies. Thus, while the followup data are potentially confounded, the various lines of evidence from previous research support the maintenance of relaxation effects independent of placebo and "spontaneous remission" influences. Second, no-tension-release relaxation appears to produce long-term subjective improvement, although the gains achieved during the treatment period are inferior to those of progressive relaxation. Whether maintenance of objective gains occurs for either relaxation condition cannot be answered without sleep lab evaluation at followup.

In addition to the predicted tension-release effects on objective sleep onset, pseudo- and idiopathic insomniacs differed significantly in numerous respects, further confirming our suspicion that previous basic and applied research efforts have ignored a dimension of insomnia that is of potentially fundamental importance. The differences that were found may ultimately contribute to a clearer understanding of the etiological and maintaining factors of each subtype. However, the fact that no treatment effects occurred on any of the measures discriminating the two groups suggests that modification of the processes reflected by those measures is unnecessary for the reduction of the basic sleep problem:

(1) Frankel *et al.* (Note 2) have reported less delta sleep among insomniacs. In the present study, pseudoinsomniacs in particular displayed less slow-wave sleep. Lighter sleep could provide the basis for their phenomenological complaints in terms of the quality of nighttime sleep as well as tiredness during the subsequent day. However, this would not account for their reports of delayed, initial sleep onset. Furthermore, the absence of any increase in slow-wave sleep after treatment suggests that their subjective onset improvement could not have been mediated by a greater amount of deep sleep.

(2) Idiopathic insomniacs obtained more Stage II sleep early and less Stage II sleep late in the week. Again, however, treatment had no effect on total amount of Stage II sleep. Thus, latency improvement was not associated with changes in absolute quantity of Stage II.

In any case, both of the above subtype differences must be viewed with caution. Because we had no valid means of determining total amount of sleep obtained, percentage of time spent in Stages II, III, and IV may have been identical for the two subtypes, even though absolute number of minutes differed significantly.

(3) The insomniac sample as a group displayed deviant MMPI profiles, a finding that led Kales *et al.* (1976) to argue that obsessive worry mediates insomnia. Our pseudoinsomniacs showed higher elevations than the idiopathic insomniacs, but none of the scale differences were significant.

(4) On a perhaps related measure, Slama (*cf.* Borkovec, *in press*) recently found in a pilot study in our laboratory that chronic insomniacs report higher frequencies of presleep cognitive activity relative to good sleepers. Starker and Hasenfeld (1976) found evidence of a similar phenomenon among their poor sleepers. In the present study, however, idiopathic insomniacs displayed higher levels of such activity than the pseudoinsomniacs both at home and in the sleep lab throughout the study. Together, these studies suggest that cognitive intrusions may be associated with the objective retardation of sleep of the idiopathic insomniac. Such activity may be the basis of the "racing mind" experience often reported by sleep-disturbed patients. Whether

such activity is simply a byproduct of being awake longer in the bed or is a cause of sleep retardation remains undetermined. In a previous study (Borkovec and Hennings, 1978), reductions in reported cognitive activity paralleled improvement in reported onset latency subsequent to treatment. The effect of treatment on cognitive activity in the present study approached significance. Thus, reduction of that activity may be causally related to either the subjective or objective sleep onset improvement demonstrated by the current sample receiving progressive relaxation, or it may be epiphenomenal.

(5) Idiopathic insomniacs reported greater presleep bodily tension during initial sleep lab nights at both pre- and posttherapy periods and required a greater number of relaxation cycles during the first training session. Thus, this subtype may be associated with higher levels of experienced tension. No subtype effects emerged from analysis of the during-treatment physiological measures to support that claim objectively, and treatment effects on the physiological measures were minimal. Furthermore, no treatment effects were found on the Anxiety Differential, while both of the treated groups reported declines in presleep bodily tension relative to no-treatment. Consequently, whatever the basis or role of subjective tension among idiopathic insomniacs, the phenomenon appears to be unrelated to physiological activity or outcome improvement.

(6) Finally, idiopathic insomniacs were more skeptical than pseudoinsomniacs regarding the potential usefulness of the treatment procedures. Because the former group showed significant improvement on both subjective and objective latency measures subsequent to progressive relaxation training, credibility, expectancy, and demand characteristics can be ruled out as viable explanations of the outcome results. The occurrence of self-report and EEG-defined improvement during a counterdemand period further mitigates the validity of a demand/expectancy interpretation.

Thus, while the combination of results argues in favor of the moderate effectiveness of relaxation training in the treatment of both pseudoinsomnia and idiopathic insomnia, they do not elaborate the mechanisms by which improvement is established. Several theories of insomnia discussed earlier find partial support in the present data, although some of the hypothesized insomnia characteristics appear to be associated more with one subtype than the other. Furthermore, numerous physiological and subjective measures distinct from the latency measures reflected few treatment effects. In cases where treatment was influential, the changes generally did not parallel the treatment effects on the latency measures (*e.g.*, both relaxation conditions reduced respiration and presleep bodily tension ratings; no-tension-release produced a possibly deleterious reduction in slow-wave sleep). Thus, it is unlikely that latency improvement was due to changes in these hypothesized mediators of insomnia. A variety of findings from our laboratory and those of others does lead to some compelling arguments regarding the nature and treatment mechanisms of the two insomnia subtypes. Unfortunately, that elaboration is beyond the scope of the present discussion and will be presented elsewhere (Borkovec, *in press*). We are left, however, with at least one major hypothesis to be pursued in future research: *muscle tension-release is the critical ingredient mediating the changes in insomnia induced via progressive relaxation and its effect on one or both insomnia subtypes may be due to self-generated monotonous stimulation* (Borkovec and O'Brien, 1976). A variety of research over several decades has documented the soporific effects of repetitive, nonsignal stimuli including visual, auditory, and tactile modalities (*cf.* Nau, Note 3; and Bohlin, 1971, 1972, 1973) has provided several well-controlled investigations examining the parameters of variable-interval monotonous stimulation. Progressive relaxation inherently involves monotonous, variable-interval, tactile stimulation. We hypothesize that these self-generated

events provide the focal stimuli for directed attention. The direct effect of such attention-focusing is the establishment of a soporific monotonous stimulation paradigm, while preclusion of cognitive activity occurs as an indirect effect. Pseudoinsomnia may be due to heightened, affectively laden, presleep cognitive material that is less distinguishable from NREM imagery experience; tension-release relaxation may thus create presleep experience more distinguishable from sleep experience via a reduction in affective cognitions. The presleep cognitive activity of the idiopathic insomniac, on the other hand, does not involve affectively laden material, is more distinguishable from NREM mentation, and is perhaps simply a byproduct of retarded sleep onset due to a dysfunctional sleep system (Hauri, *in press*); the direct, soporific effects of progressive relaxation may counteract the weak sleep system. Since traditional hypotheses regarding the maintenance of insomnia have not been strongly supported by the authors' research or that of others, the monotonous stimulation notion appears to be at least heuristc and will be the focus of our own research in the future. Once again, science may ultimately demonstrate the well-accepted: counting sheep puts people to sleep.

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