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Biofeedback and Progressive Relaxation Treatment of Sleep-Onset Insomnia:

A Controlled, All-Night Investigation[1]

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Previous research suggests that self-defined insomniacs are distinguished from normals by high levels of anxiety and physiological arousal, which might be mitigated by muscle relaxation. This study assessed the relative effects of frontal EMG biofeedback, progressive relaxation, and a placebo set of "relaxation" exercises on the sleep of 18 onset insomniacs. Each subject was trained in one of these three methods for six half-hour sessions and slept in the laboratory for two consecutive nights before and after training. The experimental groups demonstrated significant decreases in physiological activity during training while changes in the control group were minimal. Reductions in sleep-onset time were: biofeedback group, 29.66 minutes; progressive relaxation group, 22.92 minutes; control group, 2.79 minutes. The experimental groups improved significantly ($p < .05$) more than the control group, but did not differ from each other. No significant relationships between physiological levels and sleep-onset time were found, which suggests that muscle relaxation alone was not responsible for subjects' improvements. Since 20 minutes of daily practice were required to achieve an approximate 30-minute decrease in sleep-onset time, the practical utility of the methods is questioned.

Chronic insomnia is a tenacious, debilitating malady suffered by an estimated 30 million Americans (Luce & Segal, 1969; Karacan, Williams, Littell, & Salis, 1973). Until recently, the treatment of insomnia has been

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exclusively pharmacological, employing sedatives, tranquillizers, and hypnotics. However, these drugs generally lose their effectiveness within two weeks and are fraught with potentially harmful side effects (Kales, 1971; Kales, Bixler, Tam, Scharf, & Kales, 1974).

In the past few years researchers have begun to examine the efficacy of nondrug treatments for insomnia, notably progressive relaxation and autogenic training. The experimental basis for this work is a study by Monroe (1967) which demonstrated that self-defined "poor sleepers" have higher levels of physiological arousal than "good sleepers" on the following measures: number of body movements per hour, number of peripheral vasoconstrictions, and mean rectal temperature. The poor sleepers also had significantly higher heart rates during a 30-minute presleep period, showed more psychopathology on the California Medical Index (CMI) and Minnesota Multiphasic Personality Inventory (MMPI), and grossly overestimated the amount of time it took them to fall asleep in the lab. Monroe's data is in part supported by subsequent studies. Johns, Gay, Masterson, and Bruce (1971) differentiated good and poor sleepers on the basis of a sleep habits questionnaire similar to Monroe's. The poor sleepers had higher daytime and nighttime levels of free cortisol, corticosterone, and 20 hydroxycortisol than did the good sleepers. The authors interpreted this increased adrenocortical activity as an indication of increased psychological stress. The poor sleepers also had significantly higher scores on the following MMPI scales: Hypochondriasis, Masculinity–Femininity, Conversion Reaction, and Manifest Anxiety. Goldstein, Graedon, Willard, Goldstein, and Smith (1970) compared the MMPIs of 10 laboratory selected insomniacs with those of Monroe's good sleepers. The insomniacs had significantly higher scores on Hypochondriasis (Hs), Depression (D), Masculinity–Femininity (Mf), and Social Introversion (Si).

Thus far the insomniac appears to be a psychologically disturbed, highly physiologically aroused person. However, there is some evidence to contradict this picture. While Haynes, Follingstad, and McGowan (1974) found manifest anxiety to be significantly correlated with reported sleep-onset time, they found no relationship between frontal muscle tension and sleep-onset time. The latter finding was replicated in an all-night study by Good (1975), who found a slight negative relationship ($r = -.23$) between frontal muscle tension and time to sleep onset in the laboratory. Gering and Mahrer (1972), in a factor analytic study of psychiatric patients, found that anxiety was not a factor related to difficulty falling asleep.

Despite these negative findings and the fact that no causal relationships have been established between arousal, psychological disturbance, and insomnia, the notion of alleviating insomnia by lowering physiological levels has been a particularly attractive one. The most common and perhaps

easiest way of doing this has been through muscle relaxation, effected either by drugs or by training. Paul (1969a, 1969b) and others (Johnson & Spielberger, 1968; Stoudenmire, 1972; and Edelman, 1970a) have shown that progressive relaxation training reliably lowers heart rate, systolic blood pressure, skin conductance, forearm EMG, respiratory rate, and state anxiety.

The first systematic study of the effects of relaxation training on insomnia was undertaken by Kahn, Baker, and Weiss (1968) using a method known as autogenic training. The authors claimed positive results but were later criticized for their lack of an independent control group and for failing to obtain behavioral or physiological measures to correct for possible biases in verbal report (Eisenman, 1970). Borkovec and Fowles (1973) compared the effects of progressive, hypnotic, and self-relaxation on a sample of insomniacs selected on the basis of questionnaire responses. Both progressive and hypnotic relaxation were more effective than no treatment in improving sleep-onset time, in reported feelings of rest, and in reported number of awakenings. However, reduction in physiological activity levels during therapy did not correlate with outcome, and the self-relaxation group improved almost as much as the other two treatment groups. The authors suggested that subjects may have been responding to demand characteristics or nonspecific therapeutic elements of the experiment and called for all-night studies to resolve these issues. In a similar experiment, Nicassio and Bootzin (1974) found progressive relaxation and autogenic training to be significantly better than self-relaxation or no treatment in reducing sleep-onset time and improving general satisfaction with sleep. While they provide some convergent data in the form of reports from other household members and a pupillography measure of drowsiness, these were obtained only for partial samples of the subjects. Furthermore, they do not deal with the important observations of Rechtschaffen (1968) and Monroe (1967) that insomniacs are notoriously unreliable reporters of sleep characteristics. A study of Steinmark and Borkovec (1974) is the most convincing to date since they demonstrated the effectiveness of progressive relaxation on insomnia in the face of counterdemand expectations. However, like all the previous investigators, they do not deal with the issue of the accuracy of self-report data.

Parallel to the research on relaxation training and insomnia has been a similar line of work involving biofeedback. In a study of frontal EMG biofeedback with ten chronically anxious patients, Raskin, Johnson, and Rondesvedt (1973) noted that the insomnia of five patients declined. While there was no formal control group in this study, all patients had been refractory to treatment with psychotherapy and medication for a period of two years. Peper (1972) anecdotally described a successful case of an insomniac treated

with EMG biofeedback. Sittenfeld (1972) and Budzynski (1973) employed a combination of EEG (theta) and frontal EMG biofeedback. Six out of eleven of Budzynski's patients improved by unspecified criteria, while four out of seven of Sittenfeld's subjects fell asleep twice in the lab within 20 minutes. These researchers both claim that EMG feedback alone is not sufficient to produce sleep in all insomniacs and that additional EEG conditioning is necessary. Research on biofeedback and insomnia has thus far been on the level of the pilot study and case report. Clearly, the same controls which are needed for the relaxation training studies are necessary for biofeedback research also.

The present study was designed to test the relative effectiveness of EMG biofeedback and progressive relaxation training on sleep-onset insomnia while controlling for as many variables as possible. The following questions were of interest:

1. Do EMG biofeedback and progressive relaxation training decrease sleep-onset time relative to the control condition?
2. Do they have differential effects on the stages of sleep?
3. Are the physiological changes which occur during training related to changes in sleep?
4. What are the patterns of these physiological changes? Does biofeedback of one muscle generalize to other muscles?
5. Do test-measured state anxiety and locus of control change with successful biofeedback or progressive relaxation?
6. How do self-report sleep data compare with all-night sleep data?

To answer these questions physiological recordings were obtained all-night before and after the training period and during each training session as well. Self-report and psychological test data were collected at various points during the study. Lastly, a plausible control procedure consisting of physical exercises which are actually unrelated to general relaxation was administered to a third of the subjects. The exercises were developed by Williams (1975), an orthopedic surgeon, to strengthen muscles related to low back pain.

METHOD

Subject Selection

Subjects were eight men and ten women ranging in age from 17 to 39 years (median = 23 years). They were recruited on a volunteer basis from

signs in the university community and newspaper ads, which explained that persons with difficulty falling asleep were needed for an experiment involving muscle relaxation. All respondents were first interviewed by the experimenter (the senior author) and were given the MMPI, Multiple Affect Adjective Check List—Today form (Zuckerman and Lubin, 1965) and the Rotter Locus of Control (IE) Scale (Rotter, 1966) for later analysis. A questionnaire assessing length of sleep-onset time, number of nighttime awakenings, subjective satisfaction with sleep, drug use, and history and causes of the insomnia was also given. The self-report criterion used to define insomnia was an inability to fall asleep within one hour at least four nights a week for a duration of at least six months. Subjects with significant medical or psychological disorders which might affect their sleep, such as hypertension, diabetes, manic depression, or schizophrenia were not used. All subjects were drug free with the exception of alcohol and marijuana for one month prior to their participation in the experiment. All drugs were prohibited during the two-week period during which each subject participated in the study.

Procedure

During the initial interview the experimenter explained the rationale and procedures of the study to the subject and obtained his informed consent. Subjects were told that we were investigating the hypothesis that reducing muscle tension might help insomniacs fall asleep faster. A brief description of each of the three relaxation procedures was then given. Subjects were told that they would be randomly assigned to one of the three conditions. When they invariably asked which procedure we thought was the best, we told them we did not know but might learn that as a result of the experiment.

Within two weeks of this interview, each subject slept in the laboratory for two consecutive nights from approximately 11:30 p.m. to 7:30 a.m. All-night recordings of occipital and central EEGs, infraorbital EOGs, and submental EMGs were obtained and scored according to the procedures described in the Rechtschaffen and Kales (1968) scoring manual. One hundred randomly selected pages of record were blindly scored by a professional electroencephalographer[3] and compared to the experimenter's scoring with a resulting interrater reliability of 93%. Data were recorded on the second night only, the first night being used to adapt to the laboratory environment. Subjects were unaware of this, however, as identical procedures, such as the attachment of electrodes, were followed on both nights.

[3]The authors wish to thank Dr. Paul Tucker of the EEG Laboratory, University of Michigan Hospital, for performing this service.

Subjects were then assigned to one of the three training procedures: frontal EMG biofeedback, progressive relaxation, or the Williams exercises (control group). There were six subjects in each group. The assignment to groups was random with the restriction that all three groups had roughly equal laboratory sleep-onset times (42–43 minutes). All subjects were run in pairs by the experimenter in the order in which they responded to the advertisements. Each received six half-hour training sessions in one of the three procedures spaced over a two-week period between the first two and last two laboratory sleeping nights. These sessions occurred between 10:00 a.m. and 5:00 p.m. and were arranged at the mutual convenience of subject and experimenter.

After entering the experimental room for the training session, the subject reclined on a bed with a pillow under his head. The experimenter attached the electrodes and informed the subject that he would return in five minutes after adjusting the equipment. The experimenter then went into the control room and waited five minutes before making a 1-minute baseline recording. He then returned to the experimental room and gave the subject the following instructions.

Biofeedback.

> Every muscle in your body generates electrical currents. In this experiment, the electrical currents in your forehead will be amplified by an electronic instrument in the control room and converted into an audible tone which you will hear coming out of the loudspeaker behind your head. Tensing your forehead will generate large currents which will raise the pitch of the tone. Relaxing your forehead will produce smaller currents which will lower the pitch of the tone.
>
> Your task is to make the pitch of the tone as low as possible. The tone is connected to your forehead because we believe the muscles there to be a good indicator of relaxation in the entire body. You should use whatever method works best for you in trying to lower the pitch of the tone and I will give you no further instructions about how to do this. I will turn the tone on when I return to the control room where I will remain until the end of the session.

The experimenter then returned to the control room and turned on the biofeedback unit. At 12-minute intervals, two more 1-minute recordings were made. The experimenter then unhooked the subject and scheduled the next training session.

Progressive Relaxation. After the 1-minute baseline recording, the experimenter returned to the subject room and personally administered instruction in progressive relaxation as described by Paul (1966). In the middle of the session, after giving instructions for the chin and throat, but before moving to the chest, the experimenter quietly withdrew to the control room. He made another 1-minute recording, returned, and completed the relaxation instructions. After making a final 1-minute recording, the experimenter unhooked the subject. The entire procedure lasted about 30 minutes, including the 5-minute adaptation period.

Williams Exercises. After the 1-minute baseline recording, the experimenter returned to the subject room and personally administered the following instructions:

> This group of exercises promotes relaxation throughout the entire body. You should repeat each exercise seven times and relax for two minutes between each exercise.
>
> Position for exercises 1–3: lying on your back with your knees bent and your feet flat on the bed.
> 1. Fold your arms across your chest and raise your head and shoulders off the bed.
> 2. With fingertips reaching toward your knees, sit up as far as you can.
> 3. Clasp your right knee in your right hand and your left knee in your left hand. Draw your knees to your chin, raising the end of your spine off the floor.
> 4. Position: sitting with both legs stretched out in front of you. Attempt to touch your toes without bending your legs.

During the 2-minute relaxation period following the fourth exercise, the experimenter left to make a 1-minute recording and returned.

> Position for exercises 5 and 6: back-lying at edge of bed.
> 5. Draw your right knee up to your chest and hold it with both hands. Drop your left leg over the side of the bed. Hold this position for five minutes.
> 6. Same as #5 except holding left knee and hanging right leg over side of bed.

The experimenter then made the last 1-minute recording and unhooked the subject.

Each subject was instructed to practice his relaxation method for 20 minutes each night before retiring and was requested to fill out a log sheet the following morning stating how long it took him to fall asleep, how long he practiced relaxation for, and what effects the relaxation had. Biofeedback subjects practiced at home without the use of a machine.

Two weeks after the second night in the laboratory, each subject slept for two more consecutive nights with data again being recorded on the second night only. All procedures were identical to those used on the first two laboratory nights except that each subject was given 20 minutes to practice his relaxation procedure before the polygraph was turned on and he was instructed to go to sleep. Biofeedback subjects again practiced without the use of the machine to avoid the discomfort of even more facial electrodes and to provide a more realistic test of the efficacy of the method: the typical insomniac after receiving his training from a practitioner would generally not have an instrument available for home use.

The morning following the last night in the laboratory, the Today form of the Multiple Affect Adjective Check List and the Rotter IE scale were readministered. The subject's sleep logs were collected, including those filled out for each laboratory night.

Approximately two months after the final laboratory night subjects were again interviewed by the experimenter to determine their satisfaction with the relaxation exercise, whether they were still using it, and how they thought it helped them. Many of the questions from the initial interview,

regarding sleep-onset time, number of awakenings, and satisfaction with sleep were readministered. Also, two scales designed to measure the credibility of the relaxation and control procedures were given. At the end of this follow-up interview, the experimenter attempted to answer any questions the subject had about the experiment. He also debriefed the control subjects and offered them the same biofeedback training that the experimental group received.

Apparatus

All subjects were run in two temperature-controlled, sound-deadened rooms within a laboratory suite in an isolated wing of a classroom building. A junction box located in each room was connected by shielded cable to the polygraph in an adjacent room. Subjects were grounded to the polygraph via the cable shield; the polygraph was in turn connected to an earth ground. No 60-Hz interference was detected during any recording session.

An eight-channel Beckman Type R dynagraph was used for all physiological recordings. Quarter-inch-diameter AgAgCl cup electrodes were used for all EEG, EKG, and ground leads, while quarter-inch AgAgCl recessed electrodes were used for EMG measures. Grass EC 2 electrode cream was used for all connections.

All-night recording of two EEG channels (C_3A_2,O_1A_2), one EOG channel, and a chin EMG channel were obtained and scored according to standard procedures (Rechtschaffen & Kales, 1968). The occipital (O_1A_2) EEG lead was substituted for the left eye lead to better distinguish the point of sleep onset, defined as the beginning of the first two consecutive minutes of stage 1 sleep.

During each daytime training session, simultaneous recordings of action potentials from the frontal, masseter, and forearm extensor muscles, as well as heart rate, were obtained in three 1-minute intervals from the beginning, middle, and end of each session, respectively. Standard EMG electrode placements as described in Lippold (1967) were used, as well as standard EKG lead I (right arm–left arm). The subject was grounded through a lead attached to the right ear lobe. Sites were scrubbed with alcohol before the application of electrodes, and resistances were maintained between 5 and 15 KΩ with negligible variance within each session.

The polygraph was run at 10 mm/sec for all recordings. A bandpass of 5–150 Hz was used for the masseter, forearm extensor, and EKG leads, while a 30–150-Hz bandpass, obtained by modifying the Beckman 9806A input coupler, was used on the frontal lead to eliminate possible EEG artifact. All EMGs were recorded at a gain of 20 μV/cm and were scored in the

following manner. Each chart page was divided into fifteen 2½-cm-wide vertical segments. Four segments out of each 1-minute recording interval (two pages) were randomly chosen. The maximum peak-to-peak amplitude of the raw EMG was determined for each of the four intervals by measuring with a millimeter ruler and magnifier. These numbers were then converted to microvolts and averaged together to give a single score for each 1-minute recording interval. Heart rate was calculated by counting the number of *R* waves per minute. All data were scored by the experimenter, who analyzed it on the University of Michigan IBM 370/168 computer.

The biofeedback signal was derived in the following manner. The output of the power amplifier of the frontal channel on the polygraph was further amplified, then rectified, filtered, and used to drive a voltage-controlled audio oscillator having a range of approximately 30 to 1000 Hz. The signal was heard by subject and experimenter through loudspeakers in their respective rooms. After the five-minute adaptation period the tone was turned on and its frequency adjusted to approximately 400 Hz. The subject then attempted to lower the pitch of the tone by relaxing the frontal muscles. No attempt was made to shape the subject's response within or across sessions by varying the gain of the feedback loop.

RESULTS

The all-night sleep data are presented in Table I. Results were analyzed using two-way repeated measures analyses of variance (treatment X time, pre–post), with the interaction effect being of major interest. Where initial levels differed appreciably, as in stages 3 and 4, analyses of covariance were performed, with post scores being covaried on pre scores. Values for stages 3 and 4 were also combined to give a total delta sleep measure and analyzed as described above.

The only significant treatment X time interaction effect was for the variable sleep-onset time ($F = 4.4981$, $df = 2/15$, $p < .03$). Specific comparisons between groups showed that both the biofeedback ($F = 8.3241$, $df = 1/15$, $p < .02$) and progressive relaxation ($F = 4.6335$, $df = 1/15$, $p < .05$) groups improved significantly more than the control groups. The biofeedback and progressive relaxation improvements were not significantly different from each other, however ($F = .5367$). Also, there were six subjects whose initial sleep onset times were less than 30 minutes and, therefore, might be classified as "pseudoinsomniacs." When these subjects were excluded from the above analyses, the results did not change.

EMG and heart rate data for the daytime training sessions are summarized within sessions in Figure 1. Detailed statistical analyses of these

Table I. Sleep Stages in Minutes

Sleep stage		Biofeedback		Progressive relaxation		Control	
		Pre	Post	Pre	Post	Pre	Post
Total Bedtime	Mean	452.83	442.67	460.67	426.33	453.25	453.25
	SD	34.48	42.73	21.02	37.20	20.10	22.06
Sleep Onset	Mean	42.33	12.67	43.25	20.33	43.12	40.33
	SD	21.93	14.77	23.46	15.65	41.02	28.26
Total stage W (includes sleep onset time)	Mean	70.75	18.17	75.42	37.75	79.17	58.91
	SD	36.17	20.56	51.89	30.45	69.00	41.51
Stage 1	Mean	43.25	38.00	35.33	41.33	44.75	52.60
	SD	39.76	19.68	22.76	25.55	16.00	19.46
Stage 2	Mean	207.33	228.92	224.83	204.00	205.42	221.83
	SD	41.98	9.15	67.92	36.64	67.64	69.42
Stage 3	Mean	28.33	36.33	35.00	24.67	25.67	26.67
	SD	17.99	19.14	20.21	4.90	11.41	23.66
Stage 4	Mean	8.33	5.25	4.83	17.25	6.50	2.58
	SD	9.88	6.99	5.37	21.41	11.68	5.84
REM	Mean	88.91	111.50	80.50	96.25	85.25	85.42
	SD	17.02	19.49	43.77	14.11	40.41	32.58
Movement time	Mean	5.89	4.47	4.81	5.10	6.58	5.15
	SD	1.79	2.61	2.37	3.25	4.83	1.79

data will be presented in a later paper. Briefly, the biofeedback and progressive relaxation groups demonstrated significant decreases in heart rate ($p < .01$) as well as frontal ($p < .01$), masseter ($p < .01$), and forearm extensor ($p < .05$) EMG, while changes in the control group were minimal. Biofeedback training of the frontal muscles generalized to the masseter muscle but not to the forearm extensors, while progressive relaxation training produced similar patterns of change in all three muscle groups.

To assess whether decreases in muscle tension were related to decreases in sleep-onset time, the following difference scores were computed for all subjects: (1) frontal level, beginning of session 1 minus frontal level, end of ession 6; (2) frontal level, end of session 1 minus frontal level, end of session 6; (3) sleep-onset time, pretreatment minus sleep-onset time, posttreatment. The Pearson product–moment correlation between measures 1 and 3 was .452 ($p < .06$) and between measures 2 and 3, .526 ($p < .05$). However, upon closer examination, these correlations proved to be statistical artifacts resulting in part from regression towards the mean. Initial frontal level correlated positively and significantly with difference score 1 ($r = .66, p < .01$), while initial sleep onset time correlated positively and significantly with difference score 3 ($r = .60, p < .01$). In other words, subjects

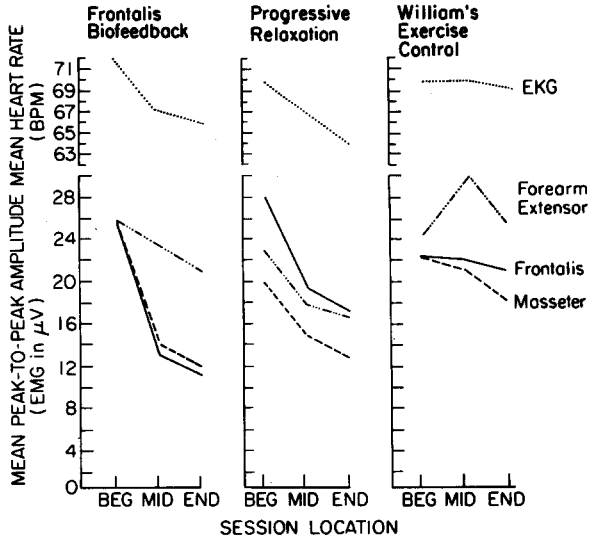

Fig. 1. Within-session physiological changes.

with the highest initial values were improving the most. The correlations between measures 1 and 3 and measures 2 and 3 were also strongest in the control group and either negative or nonsignificant in the experimental groups. Furthermore, there were no significant correlations between sleep-onset time and any of the baseline physiological measures. The correlation between initial frontal level and initial sleep latency was $r = -.27$, almost identical to that obtained by Good (1975). Thus, there appears to be no direct relationship between our physiological variables and sleep-onset time, whether expressed as absolute levels or as difference scores.

Intercorrelations between actual (EEG) sleep-onset time as obtained in the laboratory and reports of sleep-onset time in and out of the laboratory are presented in Table II. The highly significant correlation between EEG and sleep-onset pre- and posttreatment suggests that this is a fairly reliable measure for each subject. In contrast, the correlation between subjects' initial interview estimates of their typical sleep-onset time and their EEG sleep-onset time on the pretreatment laboratory night is small and negative. This confirms reports of Monroe (1967) and Rechtschaffen (1968) that insomniacs are poor estimators of how long it actually takes them to fall asleep, at least in the laboratory. Our subjects did not exaggerate as badly as Monroe's did, however. Our mean initial reported sleep-onset time was 81.3 minutes, while our mean pretreatment EEG sleep-onset time was 43.0 minutes. Monroe's subjects overestimated by a factor of 4. When our subjects made a specific estimate of how long it took them to fall asleep on

Table II. Correlations Between Actual and Reported Sleep Onset Time

	Actual – lab night 2 (pre)	Actual – lab night 4 (post)	Reported – initial interview	Reported – follow-up interview	Reported – lab night 2
Actual – lab night 4 (post)	.7438[a]				
Reported – initial interview	.0422	.1977			
Reported – follow-up interview	.0873	.0045	.3965		
Reported – lab night 2	.3908	.2671	.1300	.3168	
Reported – lab night 4	.4399[b]	.7506[a]	.2477	.0329	.5160[c]

[a] $p < .001$.
[b] $p < .1$.
[c] $p < .05$.

Table III. Follow-Up Data

		Biofeedback	Prog. rel.	Control
Estimated sleep onset time	Mean	34.16	59.03	45.00
	SD	23.96	52.93	40.90
Were you helped by the procedure?	Yes	5	5	4
	No	1	1	2
Number of subjects still practicing procedure	Regularly	2	0	2
	Occasionally	2	1	1
	Never	2	5	3
This relaxation treatment is a reasonable, logical, treatment for insomnia	Mean	2.16	1.83	2.83
	SD	.98	.41	1.47
I would recommend this treatment to a friend with insomnia	Mean	2.33	2.16	2.50
	SD	1.03	.98	1.52

their second night in the laboratory, the correlation with EEG time rose to .39 (N.S.). When they estimated their sleep-onset time for their last night in the laboratory, the correlation with the actual time was highly significant ($r = .75, p < .001$). This suggests that, as our subjects progressed through the study, they became more accurate estimators of how long it was actually taking them to fall asleep. It should be noted that subjects received no information about their actual sleep-onset times until the end of the study.

Our follow-up data, as shown in Table III, are much less clear. A one-way analysis of variance with specific comparisons of typical sleep-onset time as reported at the follow-up interview revealed no significant differences between the three groups. Most subjects reported that they were helped in some way by their relaxation procedure, while few continued to practice to any appreciable extent. At the follow-up interview all subjects were asked to respond to the following two statements to assess the credibility of the control procedure: "When the relaxation technique was first explained to me I thought it was a reasonable, logical approach to the treatment of insomnia." "I would be confident in recommending this treatment to a friend who had insomnia." Following the procedure of Borkovec and Nau (1972) subjects were asked to rate their agreement or disagreement with the statements on a 1–5 scale. One-way analyses of variance with specific comparisons revealed no significant differences between the three groups. In

addition, each control subject told the experimenter at the follow-up interview that he had been deceived by the control procedure.

Analyses of variance (treatment X time, pre–post) were carried out on the locus of control data (Rotter IE scale) and the anxiety and depression scales of the MAACL with no significant results. The MMPI data will be presented in a later paper.

DISCUSSION

It is clear from the above results that both the biofeedback and progressive relaxation groups decreased their sleep-onset times and several physiological measures relative to the control group. However, given the fact that none of the initial physiological measures correlated significantly with initial sleep-onset time, it is highly unlikely that the factor of lowering physiological arousal was alone responsible for decreasing the sleep-onset times of our insomniac subjects. It is also improbable that the lack of correspondence between physiological and sleep factors is spurious since the results have been replicated almost exactly by another laboratory (Good, 1975). Moreover, the same laboratory treated 10 sleep-onset insomniacs with a minimum of 13 sessions of frontalis EMG biofeedback with no improvement in onset time (Hauri & Good, 1975). What is the meaning of these discrepant results?

Let us consider the possibility that the improvements shown by our experimental subjects are an artifact of some aspect of the experiment. The sleep-onset times were scored by an objective standardized system and were checked by a blind independent scorer with a high degree of reliability. Thus, there is little chance of error in this part of the study. Looking at structural aspects of the experiment, it will be recalled that on the posttraining laboratory sleep nights the subjects were given 20 minutes to practice their respective procedures before being told to go to sleep. A comparable 20-minute period was not included on the pretraining night. It could be argued that these 20 minutes should be counted as time spent trying to go to sleep and therefore, should be added to the posttraining onset times. If this is done the times become 32, 40, and 60 minutes for the biofeedback, progressive relaxation, and control groups respectively. The significant decreases for the experimental groups disappear, but the control group shows a significant ($p < .05$) increase. Such an increase is an unlikely finding in light of the data of Karacan et al. (1973) and Karacan, Williams, Salis, and Hursch (1971), which show that untreated insomniacs tend to decrease their sleep latencies on successive nights in the laboratory. Is it possible that the control procedure made the subjects worse, since it did involve

some physical exercise? This is highly improbable in light of the following evidence. First, the procedure involved minimal physical effort and incorporated as much relaxation time as exercise time. Second, no physiological changes even approaching significance were shown by the subjects nor were any external signs of physical exertion detected by the experimenter. Lastly, Hauri (1969) has shown that subjects undergoing 6 hours of strenuous physical work took no longer to fall asleep than those who read and watched TV for 6 hours.

While the control task did not make the subjects worse, it is possible that it enforced wakefulness for the 20-minute practice period, while the experimental procedure permitted the subjects to fall asleep before this period ended. This, in fact, happened: two biofeedback subjects and one progressive relaxation subject fell asleep before the 20 minutes were up and their sleep-onset times were scored as zero. However, when these three subjects were excluded from the statistical analysis of sleep-onset time, the results did not change.

Lastly, the results could be explained by the control subjects being more psychologically disturbed or more physiologically aroused than the experimental subjects. Statistical analysis eliminates this possibility. One-way analyses of variance across treatment groups revealed no between-group differences on any personality measures and only one significant difference in initial levels of physiological measures: the biofeedback group had a significantly ($p < .05$) higher initial masseter level than either of the other two groups.

Given the fact that our experimental subjects clearly improved, how can we explain the fact that Hauri's (Hauri & Good, 1975) biofeedback-treated insomniacs did not? The most likely explanation is that Hauri's insomniacs represent a different population than ours. His subjects were referred to a sleep clinic from a wide geographic area, were largely non-students, and were older. Also, their cases were more chronic and more severe than our subjects, having had insomnia for at least two years and averaging 52.8 minutes to sleep-onset in the laboratory. However, the fact that extensive biofeedback training did not help these people raises serious questions about its usefulness with older, more severely afflicted subjects.

While we have thus far established the fact that our experimental insomniac subjects significantly reduced their sleep-onset times, we have shed no light on the mechanism by which this was accomplished. It has not really been established that insomniacs are more physiologically aroused than normals. We have no physiological data from a normal control group for comparison, and differences in populations and recording techniques render data from other laboratories relatively useless. Furthermore, the similarities between Monroe's (1967) highly aroused "poor sleepers" and

insomniacs are questionable. Monroe is clear in stating that his subjects were differentiated into good sleep and poor sleep groups on the basis of responses to a sleep habits questionnaire and were not selected on the basis of being insomniacs. Moreover, the sleep characteristics of his "poor sleepers" are vastly different than those of our insomniacs and those of other studies (Kales, 1969; Karacan et al., 1971; Kales et al., 1970). Monroe's subjects have a sleep latency of only 15.6 minutes, slightly increased delta sleep (28.2%), and decreased REM sleep (16.5%). Our subjects, similar to those in the Kales and Karacan studies, have a mean sleep latency of 43 minutes, increased stage 1 sleep (10.7%), decreased delta sleep (9.6%), and a normal amount of REM sleep (22.3%). Percentages here are expressed as fractions of total sleep time.

What then caused our subjects to improve? While our pre–post personality measures (locus of control, MAACL anxiety and depression) showed no changes, our subjects' reports of what had kept them awake at night and how the relaxation procedure had helped them proved to be of interest. At the initial interview the vast majority of our subjects claimed that repetitive cognition which they were unable to curtail kept them awake at night. In writing their sleep logs, several of the biofeedback subjects mentioned a process of mental clearing or focusing which helped them "slow down." Perhaps this process interrupted the repetitive cognition and fostered the regression and withdrawal from the external world which Vogel, Foulkes, and Trosman (1972) observed to be correlated with sleep onset.

Many of our biofeedback and progressive relaxation subjects complained that the procedures were repetitive and boring. It has been shown in several studies that monotonous auditory stimulation produces a habituation of the orienting reaction (Bohlin, 1974) and sleep (Bohlin, 1974; Oswald, 1960; Lovell & Morgan, 1942). Electrosleep therapy, involving the low-frequency electrical stimulation of the cranium, might be effective in the treatment of insomnia (Weiss, 1973; Rosenthal, 1972; Feighner, Brown, & Oliver, 1973). The use of a repetitive meditation-like technique might also be useful in treating insomnia (Benson, Beary, & Carol, 1974). While the results of the repetitive stimulation (Siddle, Smith, & Marcer, 1974) and electrosleep (Frankel, 1974) studies have been disputed, and while the similarities between these procedures and the relaxation techniques used in the present study are most unclear, the use of monotonous stimulation as a treatment for insomnia warrants further research. After all, counting sheep is probably one of the oldest "cures" for sleeplessness.

While our experiment demonstrates a method of helping sleep-onset insomniacs, the factors which actually caused them to improve are a mystery. This knowledge will remain obscure until we enlarge our meager pool of information regarding the etiology of the disorder. Increased autonomic arousal has been shown only in Monroe's (1967) study which

dealt with poor sleepers rather than with insomniacs. Increased anxiety was found in Monroe's study and that of Haynes et al., (1974), which did not use all-night recordings to verify the actual existence of insomnia in its subjects. The study of increased levels of stress-related hormones in insomniacs (Johns et al., 1971) has not been repeated. Only all-night sleep studies collecting multivariate psychological and physiological data and employing similar populations and techniques can ultimately further our knowledge of the origins of insomnia.

Lastly, the practical implications of our results must be considered, especially since the follow-up data are not encouraging. While it is not surprising that between-group differences in sleep-onset time would disappear since many subjets discontinued their relaxation practice, it is hard to know how accurate their time estimates are since there are no all-night data to compare them with. While it is true that subjects became better time estimators as they progressed through the study, this conclusion does not necessarily hold for general time estimates made outside the laboratory. Furthermore, it is interesting to examine why subjects decreased or curtailed practicing. Six subjects regarded themselves as cured, four said it was too much trouble, and four said it had never done them any good. The last four subjects (two control, one biofeedback, one progressive relaxation) said that, although the procedure helped them, they disliked it so much that they would rather stay awake. Although the six "cured" subjects (four biofeedback, one progressive relaxation, one control) reported substantially lower sleep-onset times than they had initially, it is difficult to believe that they achieved permanent mental or physical changes as a result of two weeks of relaxation training. It is possible that they developed a mental technique for falling asleep faster or ceased to worry about falling asleep.

Finally, it must be remembered that the most successful group of subjects, the biofeedback group, showed a mean decrease in sleep-onset time of approximately 30 minutes. While this difference is indeed statistically significant, it took these subjects a minimum of 20 minutes of daily practice to achieve it. One wonders if the average net daily gain of 10 minutes is worth this much trouble. The true test of the efficacy of these relaxation procedures will be on the severe insomniac who is taking well over an hour to fall asleep. If he can be helped by 20 minutes of daily practice, or if a less severe insomniac can benefit from less practice, then the benefits of the procedures may well outweigh the costs.

REFERENCES

Benson, H., Beary, J., & Carol, M. The relaxation response. *Psychiatry,* 1974, *37,* 37-45.

Bohlin, G. Monotonous stimulation, sleep onset and habituation of the orienting reaction. *Electroencephalography and Clinical Neurophysiology,* 1974, *31,* 593-601.

Borkovec, T., & Fowles, D. Controlled investigation of the effects of progressive and hypnotic relaxation on insomnia. *Journal of Abnormal Psychology,* 1973, *82,* 153-158.

Borkovec, R., & Nau, S. Credibility of analogue therapy rationales. *Journal of Behavior Therapy and Experimental Psychology,* 1972, *3,* 257-260.

Budzynski, T. Biofeedback procedures in the clinic. *Seminars in Psychiatry,* 1973, *5,* 537-547.

Edelman, R. Effects of progressive relaxation on autonomic processes. *Journal of Clinical Psychology,* 1970a, *26,* 421-425.

Edelman, R. Desensitization and physiological arousal. *Journal of Personality and Social Psychology,* 1970b, *17,* 259-266.

Eisenman, R. Critique of "Treatment of insomnia by relaxation training." *Journal of Abnormal Psychology,* 1970, *75,* 315-316.

Feighner, J., Brown, S., & Olivier, F. Electrosleep therapy: A controlled, double-blind study. *Journal of Nervous and Mental Disease,* 1973, *157,* 121-128.

Frankel, B. Research on cerebral electrotherapy (Electrosleep): Some suggestions. *American Journal of Psychiatry,* 1974, *131,* 95-98.

Gering, R., & Mahrer, A. Difficulty falling asleep. *Psychological Reports,* 1972, *30,* 523-528.

Goldstein, L., Graedon, J., Willard, D., Goldstein, F., & Smith, R. A Comparative study of the effects of methaqualone and glutethimide on sleep in male chronic insomniacs. *Journal of Clinical Pharmacology,* 1970, *10,* 258-264.

Good, R. Frontalis muscle tension and sleep latency. *Psychophysiology,* 1975, *12,* 465-467.

Hauri, P. The influence of evening activity on the onset of sleep. *Psychophysiology,* 1969, *5,* 426-430.

Hauri, P., & Good, R. Frontalis muscle tension and sleep-onset. Paper presented at the 15th Annual Meeting of the Association for the Psychophysiological Study of Sleep, Edinburgh, July, 1975.

Haynes, S., Follingstad, D., & McGowan, W. Insomnia: Sleep patterns and anxiety level. *Journal of Psychosomatic Research,* 1974, *18,* 69-74.

Johns, M., Gay, T., Masterson, J., & Bruce, D. Relationship between sleep habits, adrenocortical activity, and personality. *Psychosomatic Medicine,* 1971, *33,* 499-508.

Johnson, D., & Spielberger, C. The effects of relaxation training and the passage of time on measures of state and trait anxiety. *Journal of Clinical Psychology,* 1968, *24,* 20-23.

Kahn, M., Baker, B., & Weiss, J. Treatment of insomnia by relaxation training. *Journal of Abnormal Psychology,* 1968, *13,* 556-588.

Kales, A. Hypnotic drug abuse: Clinical and experimental aspects. *Medical Counterpoint,* 1969, *3,* (4), 13-17.

Kales, A., Allen, C., Scharf, M., & Kales, J. Hypnotic drugs and their effectiveness. *Archives of General Psychiatry,* 1970, *23,* 226-232.

Karacan, I., Williams, R. L., Littell, R. C., & Salis, P. J. Insomniacs: Unpredictable and idiosyncratic sleepers. In W. Koella, & P. Levin (Eds.), *Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications.* Basel: Karger, 1973.

Karacan, I., Williams, R., Salis, P., & Hursch, C. New approaches to the evaluation and treatment of insomnia. *Psychosomatics,* 1971, *12,* 81-88.

Lippold, O. Electromyography. In P. Venables, & I. Martin (Eds.), *A Manual of Psychophysiological Methods.* Amsterdam: North-Holland, 1967.

Lowell, G., & Morgan, J. Physiological and motor responses to a regularly recurring sound: A study in monotony. *Journal of Experimental Psychology,* 1942, *30,* 435-451.

Luce, G. G., & Segal, J. *Insomnia: The guide for troubled sleepers.* New York: Doubleday and Co., 1969.

Monroe, L. Psychological and physiological differences between good and poor sleepers. *Journal of Abnormal Psychology,* 1967, *72,* 255-264.

Nicassio, P., & Bootzin, R. A comparison of progressive relaxation and autogenic training as treatments for insomnia. *Journal of Abnormal Psychology,* 1974, *83,* 253-260.

Oswald, I. Falling asleep open-eyed during intense rhythmic stimulation. *British Medical Journal,* 1960, *1,* 1450-1455.

Paul, G. L. *Insight Versus Desensitization in Psychotherapy.* Stanford: Stanford University Press, 1966.

Paul, G. Inhibition of physiological response to stressful imagery by relaxation training and hypnotically suggested relaxation. *Behavior Research and Therapy,* 1969a, *1,* 249-256.

Paul, G. Physiological effects of relaxation training and hypnotic suggestions. *Journal of Abnormal Psychology,* 1969b, *74,* 425-437.

Peper, E. Frontiers of clinical biofeedback. In L. Birk (Ed.), *Seminars in psychiatry* (Vol. 5), New York: Grune & Stratton, 1973.

Raskin, M., Johnson, G., & Rondesvedt, J. Chronic anxiety treated by biofeedback-induced muscle relaxation. *Archives of General Psychology,* 1973, *28,* 263-267.

Rechtschaffen, A. Polygraphic aspects of insomnia. In H. Gastaut et al. (Eds.), *The Abnormalities of Sleep in Man.* Bologna: Aulo Gaggi Editore, 1968.

Rechtschaffen, A., & Kales, A. (Eds.). *A Manual for Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects.* Los Angeles: Brain Information Service, 1968.

Rechtschaffen, A., & Monroe, L. Laboratory studies of insomnia. In A. Kales (Ed.), *Sleep Physiology and Pathology: A Symposium.* Philadelphia: Lippincott, 1969.

Rosenthal, S. Electrosleep: a double-blind clinical study. *Biological Psychiatry,* 1972, *4,* 179-185.

Rotter, J. Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs,* 1966, *80,* 1-28.

Siddle, D., Smith, D., & Marcer, D. Effects of repetitive stimulation and reduced sensory input on cortical alertness. *Biological Psychiatry,* 1974, *1,* 181-188.

Sittenfeld, P. The control of the EEG theta rhythm. In D. Shapiro et al. (Eds.), *Biofeedback and Self Control.* Chicago: Aldine, 1972.

Steinmark, S., & Borkovec, T. Active and placebo treatment effects on moderate insomnia under counterdemand and positive demand instructions. *Journal of Abnormal Psychology,* 1975, *83,* 157-163.

Stoudenmire, J. Effects of muscle relaxation training on state and trait anxiety in introverts and extraverts. *Journal of Personality and Social Psychology,* 1972, *24,* 273-275.

Vogel, G., Foulkes, D., & Trosman, G. Ego functions and dreaming during sleep onset. In C. Tart (Ed.), *Altered States of Consciousness.* New York: Doubleday, 1972.

Weiss, M. The treatment of insomnia through the use of electrosleep: An EEG study. *Journal of Nervous and Mental Disease,* 1973, *157,* 108-120.

Williams, P. *The Lumbo-Sacral Spine: Emphasizing Conservative Management.* New York: McGraw-Hill, 1965.

Zuckerman, M., & Lubin, B. *Manual for the Multiple Affect Adjective Check List.* San Diego: Educational and Industrial Testing Service, 1965.

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