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Topographic EEG Mapping of the Relaxation Response

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The purpose of this study was to assess the central nervous system effects of the relaxation response (RR) in novice subjects using a controlled, within- subjects design and topographic EEG mapping as the dependent measure. Twenty subjects listened to a RR and control audiotape presented in a counterbalanced order while EEG was recorded from 14 scalp locations. The RR *condition produced greater* ($p < .0164$) *reductions in frontal EEG beta activity relative to the control condition. No significant differences were observed for any other frequency band or scalp region. These findings suggest that elicitation of the RR produces significant reductions in cortical activation in anterior brain regions in novice subjects.*

KEY WORDS: relaxation response; topographic EEG mapping; central nervous system; peripheral nervous system; anterior cortex.

The relaxation response (RR) is a set of integrated physiological changes that are elicited when a subject engages in a repetitive mental activity and passively ignores distracting thoughts (Wallace & Benson, 1972). The response is associated with physiological changes that include decreased oxygen consumption, heart rate, arterial blood pressure, respiratory rate, arterial blood lactate (Wallace & Benson, 1972), and reduced responsibility to norepinephrine (Hoffman et al., 1982).

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The RR is now widely used to treat a wide variety of medical disorders that are caused or exacerbated by sympathetic nervous system arousal, including hypertension (Benson, Rosner, Marzetta, & Klemchuk, 1974), headaches (Benson, Klemchuk, & Graham, 1974), anxiety and pain in patients undergoing stressful medical procedures (Mandle et al., 1990), premenstrual syndrome (Goodale, Domar, & Benson, 1990), insomnia (Jacobs, Benson, & Friedman, 1993), and infertility (Domar, Seibel, & Benson, 1990). Recent reports also indicate a high degree of acceptance and widespread use of the RR among patient populations and also by the medical community (Eisenberg et al., 1993; Friedman, Zuttermeister, & Benson, 1993). The growing acceptance and use of the RR makes a more complete understanding of its physiological effects particularly important at the present time.

The peripheral nervous system effects of the RR have been relatively well defined and include reductions in sympathetic nervous system and skeletal muscle activity (Lehrer, Carr, Sargunaraj, & Woolfolk, 1994). Despite this focus on peripheral physiological alterations, there is an implicit assumption that any relaxation-mediated change in systemic physiology is secondary to alterations within the central nervous system (CNS). Unfortunately, comparatively little empirical research has addressed the CNS effects of the RR.

The research on the CNS effects of the RR that has been done suggests that the RR results in reductions in CNS arousal as measured by increases in alpha or theta EEG activity (Delmonte, 1984; West, 1980). However, this research has been hampered by a number of methodological problems, including lack of empirically sound experimental designs (self-selection bias, nonrandom assignment, no pretesting prior to training), reliance on visual analyses of a few channels of EEG activity, and failure to assess CNS effects in novice subjects to determine if the CNS effects of the RR are immediate.

The purpose of the present investigation was to assess the CNS effects of the RR in novice subjects using a controlled, within-subjects design and topographic electroencephalographic (EEG) mapping as the dependent measure.

METHOD

Subjects

Twenty male ($n = 8$) and female ($n = 12$) subjects, aged 18-49, with no history of neurological disorders, no history of RR training, and no current use of CNS-acting medications, were recruited through a newspaper advertisement. Subjects were not compensated monetarily but were given a relaxation book and tape for their participation in the study.

Procedure

Informed consent was obtained from all subjects after the nature and possible consequences of the study were explained. While seated in a reclining chair in a quiet room, each subject listened to two 15-minute audio tapes in a randomized, counterbalanced order. One tape contained guided instructions for repetitive muscle relaxation and abdominal breathing techniques (RR condition). The other tape, using the voice of the person who made the RR tape, involved a discussion of the RR and its benefits, but did not provide instruction on how to elicit the RR (control condition). Subjects were instructed to passively ignore distracting thoughts that might occur during each tape. Thus, both conditions contained one element of the RR — the passive disregard of distracting thoughts. The second element — engagement in a repetitive mental activity — was experimentally manipulated.

The control condition used in this study was carefully considered. It has been hypothesized that the active ingredient of the RR is the focusing of attention on a repetitive mental stimulus (Benson, 1975; Borkovec, 1982). Relaxation studies have employed a variety of control conditions, including "just relax," music, audiotape novels, reading quietly, etc. The control condition used in the present study was chosen for several reasons. First, an audiotape involving a discussion of the RR and its benefits, but not providing instruction on how to elicit the RR, is a credible control for expectancy. Secondly, the control condition was identical to the RR condition with the exception of a repetitive mental focus.

EEG Recording and Quantification

EEG was recorded from 14 scalp locations (F3/4, F7/8, T3/4, T5/6, C3/4, P3/P4, O1/O2 of the International 10/20 system) using a Lycra cap (manufactured by Electro-Cap International, Inc., Eaton, OH) that was positioned on the subject's head using known anatomical landmarks (Bloom & Anneveldt, 1982). During recordings, all sites were referenced to linked ears (A1 + A2). All electrode impedances were below 3 Kiloohms. One electrooculogram (EOG) channel was recorded to facilitate eye movement artifact scoring of the EEG.

The EEG was amplified with a Grass polygraph model 8-16C electroencephalograph (60 Hz notch filter in and low- and high-frequency filters set at 1 and 35 Hz, respectively). The amplified signal was digitized using a Stellate Systems RHYTHM software program at a rate of 256 samples per second per channel. The digitized signal was then digitally filtered with a 15-point finite impulse response filter having a cutoff of 50 Hz.

The EEG data were displayed on computer and were visually and blindly scored to identify portions of the data to be deleted due to eye movements, muscle movements, and other sources of artifact. When artifact occurred on a given channel, data from all channels were removed.

Fifty-microvolt, 10-Hz sine waves were recorded on each channel and used to calibrate the digitized EEG. All artifact-free chunks four seconds in duration were tapered through a cosine window and subjected to the fast Fourier transformation (the highest component of the Fourier transformation that is retained by the RHYTHM program is 31.75 Hz). The fast Fourier transformation was used to derive estimates of the relative spectral power of the digitized EEG in the beta (13-32 Hz), alpha (8-12.75 Hz), and theta (4-7.75 Hz) frequency bands. (Since it is difficult to accurately separate eye movements from delta activity, delta power was not included in the analyses.) To normalize the data, relative power values were log-transformed.

EEG log power was computed for three time periods for both the RR and control taped instructions: beginning (minutes 1-5), middle (minutes 6-10), and end (minutes 11-15). For each of the three time periods, mean EEG log power for the 14 channels was averaged to derive grand means for the five independent cortical regions (frontal, temporal, central, parietal and occipital). This resulted in three (beta, alpha, and beta frequency bands) 2 × 3 (group × time) ANOVAS that were Bonferroni-adjusted ($p \leq .0166$) for inflation of type 1 error for each of the five independent cortical regions.

RESULTS

Frontal Scalp Region

Although the RR and control conditions exhibited similar beta log power values in the frontal scalp region during the beginning and middle of the taped instructions, the RR condition exhibited a reduction in beta log power, while the control condition exhibited an increase in beta power in the frontal scalp region at the end of the taped instructions (see Table I; more positive log values indicate greater beta power). This resulted in a significant Group × Time interaction for frontal beta log power, $F(2, 76) = 4.34$, $p < .0164$; the RR condition resulted in a greater decrease in beta log power relative to the control condition (see Figure 1).

No significant main effects were observed for frontal beta log power. Furthermore, planned comparisons indicated no significant between- or within-group differences on this variable.

Table I. Mean (*SD*) Frontal Beta, Alpha, and Theta Log Power Values for the RR and Control Conditions

	Control	RR
Beta		
Beginning	-1.6 (.7)	-1.6 (.6)
Midddle	-1.6 (.5)	-1.6 (.6)
End	-1.5 (.4)	-1.7 (.6)
Alpha		
Beginning	-.4 (.8)	-.4 (.7)
Midddle	-.4 (.7)	-.5 (.8)
End	-.5 (.7)	-.5 (.8)
Theta		
Beginning	-1.6 (.5)	-1.6 (.4)
Midddle	-1.6 (.4)	-1.5 (.5)
End	-1.5 (.4)	-1.4 (.5)

The significant but small reduction in frontal beta relative log power for the RR condition, coupled with the relatively small contribution of relative beta power to total EEG power, was associated with small and insignificant effects on frontal alpha and theta relative log power (see Table I).

Temporal, Central, Parietal, and Occipital Scalp Regions

No significant interaction effects were observed for any frequency band for either the temporal, central, parietal, or occipital scalp regions, although there was a consistent trend for the RR condition to exhibit greater reductions in beta log power from beginning to end of the taped instructions that diminished from the temporal ($p < .06$) to central ($p < .15$) to parietal ($p < .23$) to occipital ($p < .36$) scalp region.

DISCUSSION

Since beta EEG power is an indicator of cortical activation (Markand, 1990; Oken & Salinsky, 1992), these data suggest that elicitation of the RR produces significant reductions in cortical activation in anterior brain regions. Furthermore, the results suggest that these CNS effects are present the first time a subject follows taped RR instructions and that a repetitive mental focus significantly reduces anterior cortical activation in anterior brain regions. Furthermore, the results suggest that these CNS effects are

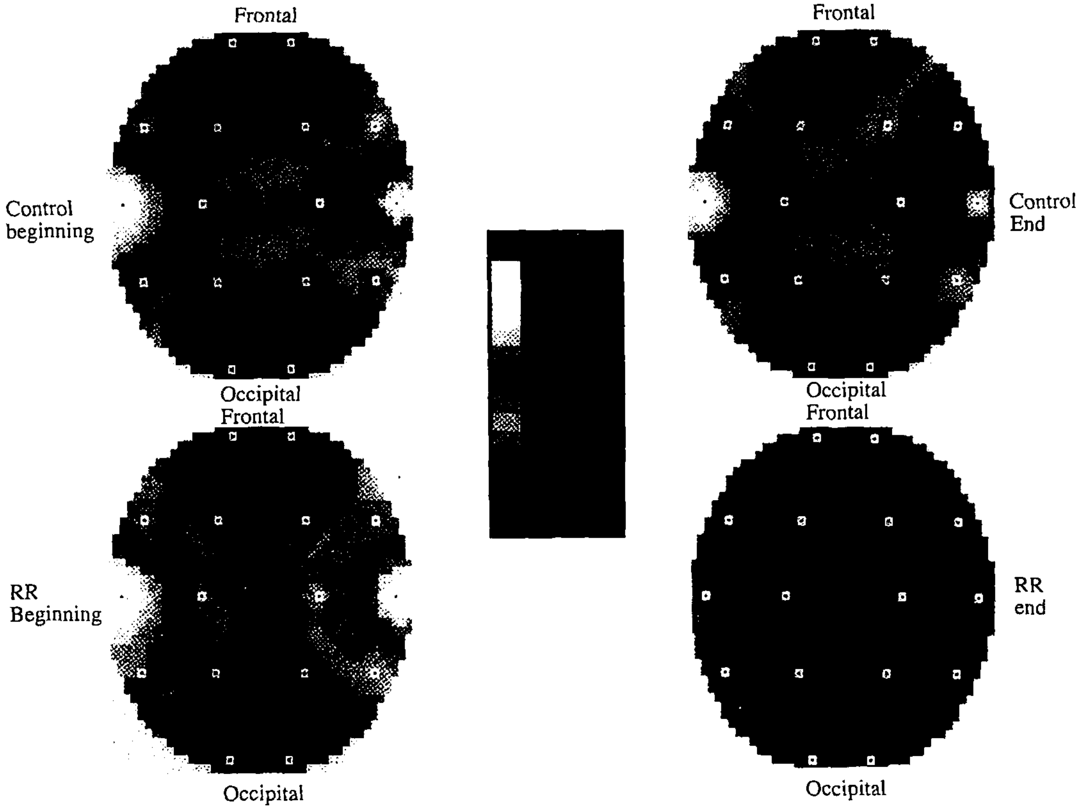

Fig. 1. Beta relative power for control and RR conditions. Vertical color bar indicates beta relative power (white highest, black lowest). Topographic maps are displayed in relative spectral power for greater resolution. Note: At RR end (lower right), beta relative power is significantly ($p < .0164$) decreased in frontal areas.

present the first time a subject follows taped RR instructions and that a repetitive mental focus significantly reduces anterior cortical activation in novice subjects. However, because only muscle relaxation and abdominal breathing techniques were employed in this study, additional research is needed to determine whether the present findings are generalizable to other relaxation response techniques such as autogenic training, imagery techniques, etc.

Although the EEG data were carefully and blindly edited for artifact, it is possible that the observed differences in frontal beta power were confounded by muscle artifact. However, a number of factors argue against this possibility. One is that muscle artifact is most likely to confound beta EEG in the temporal regions, not the frontal regions. Second, as part of a study on the peripheral nervous system effects of the RR in novice subjects that will be reported on separately, we recorded cervical electromyogram (EMG) simultaneously with EEG during both the relaxation and control condition in the present study. Analyses of these cervical EMG data indicated no relationship between reductions in EMG and the observed reduction in beta EEG power. Specifically, although beta EEG power decreased from beginning (minutes 1-5) to end (minutes 11-15) of the relaxation condition, cervical EMG was low and *constant* during these time periods (.9 microvolts). Additionally, beta EEG power showed a nonsignificant increase from beginning to end of the control condition, while cervical EMG *decreased* during these time periods (1.1 and .9 microvolts, respectively). Third, the reduction in frontal beta power during the RR condition was accompanied by a nonsignificant increase in frontal theta power (the reduction in frontal alpha power during the RR condition, which was contrary to earlier studies reporting increased alpha activity during relaxation, was consistent with more recent studies using power spectral analyses that found reductions in alpha power and increases in theta power during relaxation, e.g., Jacobs & Lubar, 1989; Pagano & Warrenburg, 1983). In summary, although it is possible that the reduction in frontal beta power was confounded by muscle artifact, these three factors argue against this possibly.

The anterior cortical regions have been associated with emotional self-regulation via connections with subcortical structures such as the limbic system and hypothalamus (Nauta, 1971). These subcortical structures are intimately involved in the sympathetically mediated fight-or-flight response. The therapeutic effects of elicitation of the RR have traditionally been attributed to self-regulation of the sympathetic nervous system. The current results suggest that the RR may exert its initial effects through the self-regulation of activation in frontal cortical and subcortical structures.

The majority of previous studies on the CNS effects of the RR, which did not employ topographic EEG mapping or novice subjects, reported that the CNS effects of the RR involved increases in alpha or theta EEG activity. The present study, employing both topographic EEG mapping and novice subjects, found that the CNS effects of the RR involved reductions in beta EEG power the first time subjects elicit the RR. In order to more fully elucidate the CNS effects of the RR, future research will need to study novice subjects in a controlled, randomized longitudinal design in which topographic EEG mapping of all the cortical regions is performed the first time subjects elicit the RR and then at repeated time periods as the RR is practiced over time. This approach will help to clarify the changes in EEG power in different frequency bands and cortical regions as a function of practice of the RR.

REFERENCES

Benson, H. (1975). *The relaxation response.* New York: Avon.

Benson, H., Klemchuk, H. P., & Graham, J. R. (1974). The usefulness of the relaxation response in the therapy of headache. *Headache, 14,* 49-52.

Benson, H., Rosner, B. A., Marzetta, B. R., & Klemchuk, H. M. (1974). Decreased blood pressure in pharmacologically treated hypertensive patients who regularly elicited the relaxation response. *Lancet, 1*(852), 289-291.

Bloom, J. L., & Anneveldt, M. (1982). An electrode cap tested. *Electroencephalography and Clinical Neurophysiology, 54,* 591-594.

Borkovec, T. D. (1982). Insomnia. *Journal of Consulting and Clinical Psychology, 50,* 880-895.

Delmonte, M. M. (1984). Electrocortical activity and related phenomenon associated with mediation practice; A literature review. *International Journal of Neuroscience, 24,* 217-231.

Domar, A. D., Seibel, M., & Benson, H. (1990). The mind/body program for infertility: A new behavioral treatment approach for women with infertility. *Fertility and Sterility, 53,* 246-249.

Eisenberg, D. M., Kessler, R. C., Foster, C., Norlock, F. E., Calkins, D. R., & DelBanco, T. L. (1993). Unconventional medicine in the United States: Prevalence, costs and patterns of use. *New England Journal of Medicine, 328,* 246-252.

Freidman, R., Zuttermeister, T., & Benson, H. (1993). (Letter to the editor). *New England Journal of Medicine, 329,* 1201.

Goodale, I. L., Domar, A. D., & Benson, H. (1990). Alleviation of premenstrual syndrome symptoms with the relaxation response. *Obstetrics and Gynecology, 75,* 649-655.

Hoffman, J., Benson, H., Arns, P., Stainbrock, G. L., Landsberb, L., Young, J. B., & Gill, A. (1982). Reduction sympathetic nervous system responsivity associated with the relaxation response. *Science, 215,* 190-192.

Jacobs, G. D., & Lubar, J. F. (1989). Spectral analyses of the central nervous system effects of the relaxation response elicited by autogenic training. *Behavioral Medicine, 15,* 125-132.

Jacobs, G. D., Benson, H., & Friedman, R. (1993). Home-based central nervous system assessment of a multifactor behavioral intervention for chronic sleep-onset insomnia. *Behavior Therapy, 24,* 159-174.

Lehrer, P. M., Carr, R., Sargunaraj, D., & Woolfolk, R. (1994). Stress management techniques: Are they equivalent, or do they have specific effects? *Biofeedback and Self-Regulation, 19,* 353-401.

Mandle, C. L., Domar, A. D., Harrington, D. P., Leserman, J., Bozadjian, E., Friedman, R., & Benson, H. (1990). The relaxation response in femoral arteriography. *Radiology, 174,* 737-739.

Markand, O. N. (1990). Alpha rhythms. *Journal of Clinical Neurophysiology, 7,* 163-189.

Nauta, W. J. H. (1971). The problem of the frontal lobe: A reinterpretation. *Journal of Psychiatric Research, 8,* 167-187.

Oken, B. S., & Salinsky, M. (1992). Alertness and attention: Basic science and electrophysiological correlates. *Journal of Clinical Neurophysiology, 9,* 480-494.

Pagano, R. R., & Warrenberg, S. (1983). Meditation: In search of a unique effect. In R. Davidson, G. Schwartz, & D. Shapiro (Eds.), *Consciousness and self-regulation.* New York: Plenum.

Wallace, R. K., & Benson, H. (1972). The physiology of meditation. *Scientific American, 226,* 84-90.

West, M. A. (1980). Mediation and the EEG. *Psychological Medicine, 10,* 369-375.