Reducing EMG and Cardiovascular Reactivity with Cue-Controlled Relaxation

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The present study compared cue-controlled relaxation (CCR) to progressive muscle relaxation (PMR) as possible methods of reducing reactivity to active stressors (math and anagram tasks). The CCR training entailed practicing relaxation during exposure to the active stressors, while the PMR training did not. Ten cardiac rehabilitation patients served in an experiment which used a repeated-measures ANOVA to assess changes in frontal EMG, skin conductance level (SCL), systolic (SBP) and diastolic (DBP) blood pressure, and pulse-rate reactivity across three experimental conditions. These were: baseline, progressive muscle relaxation, and cue-controlled relaxation. The results indicated that CCR led to reduced EMG, SBP, and pulse-rate (ps < .05) reactivity. The stressors maintained their ability to elicit reactivity over repeated (6-13) experimental sessions, while no significant changes occurred in task performance. Neither Type A behavior nor subjective anxiety changed as a result of training.
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ACKNOWLEDGEMENTS

For my parents--Kenner and Kay--in gratitude for their encouragement.

Janel Kay Harris
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CHAPTER I

INTRODUCTION

Cardiovascular reactivity to stress has recently been discussed as a possible risk factor for the development of coronary heart disease (CHD) (Matthews, Weiss, & Detre, 1984). These authors defined reactivity as the deviation from a comparison or control value resulting from a response to a discrete environmental stimulus which could be primarily physical or psychological in nature. Reactivity is relatively stable over time (Manuck & Garland, 1980), and at least three studies have confirmed an association of reactivity with the later development of cardiovascular disease. Keys et al. (1971) demonstrated that diastolic blood pressure reactivity to a cold-pressor test predicted the occurrence of CHD at a 23-year follow-up. Falkner, Onesti, and Hamstra (1981) observed a significant correlation of blood pressure (BP) reactivity to stress in borderline hypertensive adolescents and the later development of fixed hypertension. Kaplan et al. (1983) found greater coronary artery atherosclerosis in monkeys who displayed enhanced reactivity to social stress, as compared to monkeys which were less reactive.

Reactivity has also been circumstantially linked to CHD because of its association with several known CHD risk factors. These include Type A behavior, family history of hypertension and/or CHD, and elevated blood lipid levels (Dembroski, 1984; McKinney,
In addition, reactivity is frequently more pronounced in hypertensive, as compared to normotensive, individuals (Falkner, 1984; Obrist, Light, Hastrup, & Langer, 1981) and in coronary patients, as compared to matched controls (Corse, Manuck, Cantwell, Giordani, & Matthews, 1982; Dembroski, MacDougall, & Lushene, 1979; Sime, Buell, & Eliot, 1980).

While the mechanisms which may relate reactivity to CHD are unclear (Herd, 1984; Matthews et al., 1984), efforts to reduce reactivity with behavioral techniques such as cognitive self-control, relaxation training, or biofeedback have already begun (see review by Jacob & Chesney, 1984). These efforts were begun in the context of findings which suggested that behavioral techniques in general, and relaxation training in particular, were effective in controlling resting levels of BP in Type A individuals and hypertensives (see reviews by Benson, 1982; Suinn, 1982).

Many early studies of reactivity management employed passive stressors. As defined in this paper, these are reactivity-eliciting stimuli which do not evoke active coping responses of the type described by Obrist et al. (1981). Among passive stressors in common use were electric shock, films or slides of accidents or medical procedures, visualization of feared stimuli, or loud tones. Studies using passive stressors have produced mixed results. Some have reported reduced cardiovascular reactivity to stress, while others have not (see review by Jacob & Chesney, 1984). Further, the work
of Obrist and his colleagues (described in Obrist et al., 1981) suggests that passive stressors are less clearly implicated in the development of essential hypertension and lead to less pronounced cardiovascular reactivity than is found for active stressors. Thus, the use of active stressors may be more appropriate to the study of reactivity.

Active stressors are reactivity-eliciting stimuli which evoke active coping responses. Among active stressors in common use in studies of reactivity management were mental arithmetic, visual puzzles, reaction time tasks, and letter naming sequence tasks.

Researchers have used a variety of interventions in an attempt to reduce reactivity to active stressors. For example, Steptoe (1977) reported significantly reduced pulse transit time (TT) (an indirect measure of blood pressure) reactivity to an auditory choice reaction time task following biofeedback training. The results were somewhat ambiguous, however, because the biofeedback group had greater initial reactivity to the stressors. Thus, greater potential for reactivity attenuation existed in the biofeedback, as compared to the instructed relaxation, group. In addition, performance accuracy on the reaction time task increased as the study progressed. This suggests that the tasks had become less difficult—and possible less stressful—as the study progressed.

A later study (Falkowski & Steptoe, 1983) compared SCL biofeedback with simple instructions to relax. Biofeedback did not reduce SCL or HR reactivity to active (visual puzzle tasks) or
passive (industrial accident film) stressors.

In the same year, English and Baker (1983) reported that both progressive relaxation training and a transcendental meditation analogue reduced BP reactivity to a reaction time task. Subjects in a wait-list control showed no attenuation of reactivity. It is notable, however, that reactivity, as measured from a running baseline, was not reduced. Instead, these researchers observed tonic BP reductions throughout the baseline, relaxation, stress-induction, and recovery periods for subjects in both of the active treatment conditions. Further, since wait-list control subjects received less exposure to the experimental setting, diminished adaptation to setting variables cannot be ruled out as a possible reason for the obtained group differences.

Each of the studies cited above provided training in a calm environment and exposed subjects to stressors at a later time when reactivity was assessed. The studies described below provided some form of training during the time that subjects were exposed to stressors.

Several researchers have provided training in the presence of passive stressors. Burish and Schwartz (1980), for example, provided frontal EMG biofeedback in conjunction with shock-threat. They reported that EMG reactivity was reduced in groups trained with or without threat-of-shock. Cardiovascular reactivity, however, was unaffected.

A second study which provided training in conjunction with
shock-threat (Holmes, Frost, Bennett, Nielson, and Lutz, 1981) found that instructed relaxation reduced skin resistance (SR) reactivity to a greater degree than did SR feedback. However, since neither treatment was presented in the absence of shock-threat, a comparison of training with or without shock-threat was precluded.

In the first study to present active stressors during training, Steptoe (1978) reported reduced TT reactivity to a reaction time task for subjects receiving TT feedback during exposure to the tasks. Unfortunately, a comparison of training with concurrent stressors to training in the absence of stressors was again precluded, as all subjects were exposed to alternating periods of both types of training.

A study by Benthem and Glaros (1982) was the first to provide a direct test of the possible advantage of presenting active stressors in conjunction with training. Significantly, they reported the virtual elimination of TT reactivity by subjects receiving TT feedback in conjunction with mental arithmetic tasks, whereas TT reactivity was unaffected for subjects receiving biofeedback under nonstressful conditions, false feedback, or repeated exposure to the tasks.

This finding raises questions about the advisability of providing training in a calm environment when the training objective is to reduce reactivity in stressful environments. Since stimulus control may affect generalization of training effects to nontraining conditions (Skinner, 1953), the logic of presenting
reactivity-eliciting stimuli during training for reactivity management is compelling. Yet, few researchers have trained individuals to relax when stressed—the very stimulus conditions under which the ability to relax is valued.

The present study, therefore, compared cue-controlled relaxation (CCR) to progressive muscle relaxation (PMR) as possible methods of reducing reactivity to active stressors (math and anagram tasks). The CCR training entailed practicing relaxation during exposure to active stressors, while the PMR training entailed practicing relaxation under calm conditions. Since reactivity management may be particularly relevant in patient groups known to display enhanced reactivity, cardiac rehabilitation patients having coronary disease and/or hypertension were recruited for this study.

This study extended the work of Steptoe (1978) and of Benthem and Claros (1982) by its use of relaxation, rather than TT biofeedback, training in conjunction with active stressors. It was also the first study of reactivity management which used a clinically relevant subject population—cardiac rehabilitation patients—as subjects. All previous studies used normotensive subjects reporting no history of coronary disease.
CHAPTER II

METHOD

Subjects

Ten cardiac rehabilitation patients (9 men, 1 woman), aged 36-73, volunteered for this study. All presented with a diagnosis of heart disease and/or hypertension and were in the maintenance phase of a cardiac rehabilitation program. No major changes in diet, exercise, or medication had occurred over the previous one year period. Maintenance dosages of beta-blocking medication were taken by 5 of the 10 subjects on a regular basis throughout the study. In addition, all subjects engaged in supervised aerobic exercise one to three times weekly. And, this was supplemented by home exercise. All had served in a prior study comparing the effects of different psychological stress conditions (Maldonado et al., 1984). Six of the subjects were classed Type A and four were classed Type B according to the Framingham Type A Scale (Haynes, Feinleib, & Kannel, 1980).

Setting

The study was conducted at the Cardiac Rehabilitation Institute of the Borgess Medical Center (Kalamazoo, Michigan). A graduated aerobic exercise program was a major component of the rehabilitation program. Patients were treated by an interdisciplinary team of cardiologists, exercise physiologists, nurses, dietitians,
psychologists, and exercise leaders.

Apparatus

Psychophysiological Measures

**Frontal EMG.** Activity of the frontal musculature was detected by a J & J EMG (Model M-52) with the frequency bandpass set at 100-200 Hz and was recorded from a J & J Digital Integrator (Model D-200) which displayed mean integrated EMG from successive 1 min periods of integration on a continuous basis throughout the study. Frontal EMG records were obtained by applying silver-silver chloride electrodes to the frontalis muscle in the manner described by Lippold (1967). The electrodes were applied after filling the electrode cups with conductance gel and cleansing the subject's forehead with a mild abrasive followed by an alcohol wipe.

**Skin conductance level.** Detection of skin conductance level (SCL) was accomplished with a J & J EDG (Model R-72) with the high sensitivity range set at ± 2 μmho/mV full scale and was recorded from a J & J Digital Integrator (Model D-200) which displayed mean integrated SCL over successive 1 min periods on a continuous basis throughout the study. Monitoring of SCL was done by applying silver-silver chloride finger electrodes to the volar surface of the first and third distal phalanxes of the left hand after cleansing the surfaces with a mild soap.

**Cardiovascular measures.** Systolic (SBP) and diastolic (DBP)
blood pressure and pulse were electronically recorded and displayed by an Astropulse 88 (Marshall Electronics, Inc.) microphone-triggered sphygmomanometer positioned on the subject's left arm. Measurements were taken by activating an automatically inflatable BP cuff to a pre-set level during the final minute of each rest and stress period.

**Behavioral Measures**

**Task Performance.** Subject responses to computer presented math and anagram tasks were recorded and analyzed via a Commodore VIC-20 microcomputer. This yielded measures of the total number of problems attempted, the percentage of correct responses, the average response latency, and the average trial duration for individual sets of tasks from each stress period.

**Anxiety ratings.** At the end of each rest and stress period, subjects rated their anxiety levels on a 7-point scale. A rating of 1 corresponded to feeling very calm, as calm as I have ever felt, while a rating of 7 corresponded to feeling extremely tense, as tense as I have ever felt.

**Type A behavior.** Subjects completed the Framingham Type A Scale (Haynes et al., 1980) at the beginning and end of this study. Data for one subject were incomplete, however, and were excluded from the analysis. The scale, consisting of 10 items with two to four alternatives each, is predictive of CHD.
Stress Induction

Reactivity was experimentally induced by exposing subjects to microcomputer controlled, time-limited trials of math and anagram tasks. Time limitations for task presentation were individually set at values of 10, 12, 15, or 20 sec such that all subjects attained initial baseline performance rates of approximately 60% correct, with a range of 50-71% across all baseline sessions. When tasks were displayed on the television monitor, subjects responded by pressing a sequence of keys, representing letters or numerals. Responses were made with the right hand since the left arm and hand were immobilized for physiological recording.

In an effort to decrease adaptation to the reactivity-induction procedure, new sets of tasks were devised for each experimental session. The stimulus conditions and response contingencies under which the tasks were presented are described below.

Easy problems. These were three-to-five-letter anagrams randomly alternated with two-operation arithmetic problems having three-digit answers (e.g., TORIHSY = , 61 + 85 - 29 = ). Subjects were told that the problems were hard and that they would earn points when correct or hear a loud buzz when incorrect.

During these two conditions, correct responses resulted in a display of points earned, whereas incorrect or incomplete answers resulted in a loud buzz. The microcomputer controlled delivery of the buzz through the television monitor speakers at a volume
represented by the midpoint of the volume-control knob.

At the end of each session, points earned were exchanged for small sums of money on the basis of percentage of correct responses. For example, when subjects scored 60% correct, they earned the average award of $2.00 per session. Awards ranged from $0.60 to $4.85 across subjects and conditions, but did not differ in any systematic manner.¹

Experimental Conditions

Subjects sat in a chair facing a Commodore VIC-20 microcomputer and television monitor. All physiological measures, except frontal EMG, were obtained from the subject's left arm and hand. Recording equipment was positioned on a counter to the left of the subject such that only a trainer who was present could observe the digital displays. Sessions were held on an individual basis and were conducted according to a standardized protocol by one of three doctoral-level clinical psychology students who were experienced in providing relaxation training.

In this study, all subjects were exposed to each of three

¹During the baseline phase, subjects were exposed to a forced failure and an extinction condition. Both were subsequently dropped from the experimental procedure because of their relatively diminished ability to produce physiological response. Data from these two conditions were, therefore, exempted from analysis. The two remaining stress conditions—easy problems and hard problems—were each presented in random order during all remaining experimental sessions. This was done as a precautionary measure only, as the two conditions did not appear to produce differential physiological response.
experimental conditions. These are described below.

**Baseline**

After a 10 min adaptation period, subjects were exposed to 4 cycles of 5 min rest and 3 min stress. Data from two of these cycles were discarded (see Footnote 1). A trainer, who was present during baseline (BL) assessment sessions, monitored the digital displays of physiological response and provided instructions for the rest and stress periods.

The baseline phase consisted of three to five (M = 3.6) sessions per subject. In the BL phase—as in all subsequent phases—subjects were exposed to the experimental conditions until stability of EMG measures was observed across a minimum of three experimental sessions. Stability of EMG measures was judged on an individual basis by a visual analysis of plotted EMG data. (The EMG data were selected for visual analysis because of an a priori assumption that the planned treatment—relaxation training—would have a direct effect on EMG, but not on cardiovascular or SCL measures.) The EMG data were analyzed on a daily basis. The analysis which was applied was a within-subject, multiple baseline across subjects experimental analysis. Data from this analysis appears in a later section of this paper.

**Progressive Muscle Relaxation**

After a 10 min adaptation period, subjects practiced progressive
muscle relaxation (PMR) for 18 min in the absence of stressors. They were then exposed to 2 cycles of 5 min rest and 3 min stress. The rationale given to subjects for providing PMR training was to produce generalized muscular relaxation as a means of inhibiting reactivity to stress.

During these sessions, a trainer provided instructions and corrective feedback on the tensing and relaxing of specific muscle groups, using the Bernstein and Borkovec (1973) manual as a general guide. This training was conducted under calm conditions in the absence of stressors, as is recommended for PMR training. The initial session included verbal instructions, modeling of the tense-relax sequence, and corrective feedback prior to activation of tape-recorded relaxation instructions. The audiotapes were pre-recorded by one of the authors and included the 16-muscle group, tense-relax sequence described by Bernstein and Borkovec. All subsequent PMR sessions began with the activation of tape-recorded relaxation instructions.

While the tapes were activated and the subjects practiced PMR, a trainer monitored EMG levels and immediately informed the subjects that the EMG levels were increasing/decreasing, whenever the levels increased/decreased by about 2-3 µV (except in the obvious cases where EMG levels increased by more than 2-3 µV in response to tape-instructed muscular contraction). As the subjects became skilled in PMR practice, trainer intervention was discontinued—usually within two or three sessions.
In a procedural variation of PMR training, subjects verbalized the word "relax" immediately before relaxing each muscle group. The purpose of this procedure was to establish a relaxation response to the subject's verbalization, rather than to the taped instructions. During the next phase, subjects applied these self-instructions to relax during exposure to active stressors.

The PMR phase ranged in length from 3 to 10 (M = 6.3) sessions per subject. Subjects remained in this condition until stability of EMG measures was observed, as explained above.

**Home practice.** This consisted of daily use of PMR tapes and self-monitoring of PMR practice. Adherence was roughly assessed during this and the following phase by examination of the self-monitoring records.

**Cue-Controlled Relaxation**

After a 10 min adaptation period, subjects were exposed to 4 cycles of 5 min rest and 3 min stress. During the stress periods, subjects practiced CCR via self-instructions to relax at the beginning of each math and anagram trial and at times of increased EMG activity (e.g., immediately before pressing the response keys). The rationale given to subjects for CCR training was to enable relaxation during exposure to stress.

During relaxation (and exposure to stressors) in the CCR condition, a trainer monitored EMG levels and immediately informed the subjects that the EMG levels were increasing/decreasing.
whenever the EMG levels increased/decreased by about 2-3 uV. This form of verbal feedback was provided throughout the PMR and CCR conditions whenever the subjects were practicing relaxation. During the CCR condition, however, when the EMG levels increased, the trainer told the subjects to say the word "relax" to themselves. If this procedure failed to produce decreased levels of EMG activity, the trainer instructed the subjects to momentarily discontinue responding to the problems. Subjects maintained their visual focus on the problems while attempting self-relaxation. After a brief period of less than 30 sec, subjects returned to active task performance, as before. As the subjects became skilled in this procedure, trainer prompts were discontinued—usually within two or three sessions.

The CCR phase contained from four to nine (M = 6.5) sessions per subject. And, the phase was not terminated until stability of EMG measures was observed across at least three experimental sessions.

**Home practice.** This consisted of the daily practice of PMR and the application of CCR when feeling stressed. Subjects self-monitored both PMR and CCR home practice.

**Data Reduction**

For EMG and SCL, scores from the last 3 min of each rest period were averaged to yield a rest score for each session. Likewise, scores from each 3 min stress period were averaged to yield a
stress score for each session.

For SBP, DBP, and pulse, scores collected during the final minute of each rest or stress period were averaged to yield a rest score and a stress score for each session. The same procedure was followed for ratings of subjective anxiety which were obtained immediately following each rest and stress period. Change scores were then calculated by subtracting the average rest score from the average stress score for each session.

Experimental Design

After calculating change scores for EMG, SCL, SBP, DBP, and pulse, data from the last three sessions of each condition were subjected to a repeated measures analysis of variance (ANOVA) using the BMDP2V (Jennrich & Sampson, 1979) computer program in a 10 x 3 x 3 (subjects x sessions x condition) experimental design. (Data from the last three sessions of each condition were selected for this analysis because a post-hoc visual analysis of the EMG data suggested that steady-state responding was most likely to have occurred during the last three sessions of each condition.)

Whenever a significant ($p < .05$) F-ratio was obtained in these ANOVAs, as determined by the conservative Greenhouse-Geisser probability generated by the program, subsequent comparisons among means were performed using the Tukey HSD test (Hopkins & Glass, 1978, pp. 358-367). Performance data and averaged anxiety change scores were subjected to the same analysis. (In each of the above
analyses, the experimental comparison of PMR and CCR is additive, rather than balanced, since subjects must learn to relax the muscles in some manner as a prerequisite to practicing CCR.

Pretest and posttest scores from the Framingham Type A Scale were subjected to a \( t \)-test for dependent observations to determine whether or not changes had occurred in Type A behavior as a result of training.
CHAPTER III

RESULTS

Psychophysiological Measures

Repeated-Measures Analysis of Variance

The results of this analysis indicated that CCR led to diminished EMG and SBP reactivity, as compared to PMR and BL, and to diminished pulse-rate reactivity, as compared to PMR (see Table 1 and Figure 1). Significant main effects for condition were obtained for EMG, SBP, and pulse ($F_s(2,18) \geq 4.16, ps < .05$). Subsequent comparisons revealed decreased reactivity to the tasks during CCR, as compared to PMR and BL, for EMG ($Ms = 4.67, 10.13, \text{ and } 10.76 \mu V, ps < .05$) and SBP ($Ms = 5.20, 10.88, \text{ and } 10.20 \text{ mmHg, } ps < .05$). Decreased reactivity was also found during CCR, as compared to PMR, for pulse ($M_s = 2.62 \text{ vs. } 6.85 \text{ bpm, } p < .05$). An examination of mean rest and stress period scores across the three experimental conditions further suggests that the observed reductions in reactivity were due to decreased physiological reactivity during stress periods rather than to increased activity during rest periods (see Table 1). Although session effects were otherwise absent, a significant effect of this type was obtained for EMG ($F(2,18) = 4.15, p < .05$). A subsequent comparison revealed that Session 1 was associated with significantly more reactivity than Session 2 ($M_s = 9.23 \text{ vs. } 7.96, p < .05$). As seen in Figure 1,
Table 1

Mean Rest, Stress, and Change Scores during Baseline (BL), Progressive Muscle Relaxation (PMR), and Cue-Controlled Relaxation (CCR).

<table>
<thead>
<tr>
<th></th>
<th>BL</th>
<th>PMR</th>
<th>CCR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EMG (μV)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>5.5</td>
<td>5.2</td>
<td>4.3</td>
</tr>
<tr>
<td>Stress</td>
<td>16.3</td>
<td>15.3</td>
<td>9.0</td>
</tr>
<tr>
<td>Change Score</td>
<td>10.8</td>
<td>10.1</td>
<td>4.7</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>11.3</td>
<td>10.9</td>
<td>5.1</td>
</tr>
<tr>
<td><strong>SCL (μhos)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>5.4</td>
<td>6.0</td>
<td>4.9</td>
</tr>
<tr>
<td>Stress</td>
<td>7.2</td>
<td>8.3</td>
<td>6.4</td>
</tr>
<tr>
<td>Change Score</td>
<td>1.8</td>
<td>2.3</td>
<td>1.5</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>1.8</td>
<td>2.5</td>
<td>1.2</td>
</tr>
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Table 1 - continued

<table>
<thead>
<tr>
<th></th>
<th>BL</th>
<th>PMR</th>
<th>CCR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SBP (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>123.6</td>
<td>122.4</td>
<td>124.3</td>
</tr>
<tr>
<td>Stress</td>
<td>133.8</td>
<td>133.3</td>
<td>129.5</td>
</tr>
<tr>
<td>Change Score</td>
<td>10.2</td>
<td>10.9</td>
<td>5.2</td>
</tr>
<tr>
<td>SD</td>
<td>13.4</td>
<td>12.0</td>
<td>10.0</td>
</tr>
<tr>
<td>*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>DBP (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>70.7</td>
<td>71.5</td>
<td>71.5</td>
</tr>
<tr>
<td>Stress</td>
<td>76.3</td>
<td>76.8</td>
<td>75.7</td>
</tr>
<tr>
<td>Change Score</td>
<td>5.6</td>
<td>5.3</td>
<td>4.2</td>
</tr>
<tr>
<td>SD</td>
<td>8.1</td>
<td>7.3</td>
<td>8.1</td>
</tr>
<tr>
<td>*</td>
<td></td>
<td></td>
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<tr>
<td><strong>HR (bpm)</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Rest</td>
<td>59.9</td>
<td>58.9</td>
<td>60.4</td>
</tr>
<tr>
<td>Stress</td>
<td>65.3</td>
<td>65.4</td>
<td>63.4</td>
</tr>
<tr>
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<td>5.4</td>
<td>6.5</td>
<td>3.0</td>
</tr>
<tr>
<td>SD</td>
<td>6.8</td>
<td>8.7</td>
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* p < .05

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Figure 1. Mean change (△) scores for EMG (μV), SCL (μhos), SBP (mmHg), DBP (mmHg), and pulse (bpm) during the last three sessions of each condition.
a pattern of reduced reactivity during CCR, as compared to BL and PMR, was obtained for all physiological variables. However, the obtained mean differences during BL, PMR, and CCR for DBP (Ms = 5.60, 5.35, and 4.13 mmHg) and SCL (Ms = 1.76, 2.31, and 1.45 μmhos) failed to reach significance (Fs(2, 18) ≤ 1.75).

*With-Subject, Multiple Baseline Across Subjects Analysis of EMG Data*

Figures 2 and 3 illustrate session-by-session changes in EMG levels in representative subjects. The data indicated that practicing PMR had no effect on EMG levels during stress periods (see Figure 2). In contrast, practicing CCR during the stress periods reduced stress-period EMG levels in Subjects 1-8 (see representative Subjects 1 and 3 in Figure 1) from the EMG levels which were observed in the BL and PMR phases.

The absence of trends during the BL and PMR phases further indicated that reduced stress-period EMG levels during the CCR phase were unrelated to the simple passage of time or repeated exposure to the stressors (see Figures 2 and 3). In fact, stable stress-period EMG levels were typically maintained until introduction of the CCR training after a total of 6-13 sessions.

The observed effect of CCR training in Subjects 1-8 was not reproduced in Subjects 9-10 (see representative Subject 10 in Figure 1), however. In these two subjects, neither PMR nor CCR appeared to affect EMG activity during the stress periods despite a second exposure to both the PMR and CCR conditions. Further, the
Figure 2. Frontal EMG (µV) across the baseline (BL), progressive muscle relaxation (PMR), and cue-controlled relaxation (CCR) conditions in three representative subjects.
Figure 3. Frontal EMG (µV) across the baseline (BL), progressive muscle relaxation (PMR), and cue-controlled relaxation (CCR) conditions.
failure to obtain reduced EMG activity in Subjects 9-10 appeared to be unrelated to low initial values of EMG activity, as Subjects 4 and 8 were able to reduce EMG activity during stress periods despite similarly low initial levels of EMG activity (see representative Subject 8 in Figure 3).

Throughout the study, the rest-period levels of EMG were substantially unchanged. In fact, only Subject 3 (see Figure 2) showed evidence of changed rest-period levels of EMG. In this subject, CCR—as compared to BL and PMR—led to reduced EMG activity during the rest periods. Further, this subject was the only subject who consistently displayed rest-period EMG levels which were in excess of 10 μV.

Behavioral Measures

Task Performance

There were no significant changes in any of four performance measures across the three phases of this study (Fs(2,18) ≤ 3.57). Thus, the total number of problems attempted, the percentage of correct responses, the average response latency, and the average trial duration remained stable throughout the study. The observed reductions in EMG, SBP, and pulse-rate reactivity were, therefore, unrelated to performance variables.

Type A Behavior and Subjective Anxiety

No changes in either Type A behavior or subjective anxiety
were observed in the participants of this study. A t-test for dependent observations failed to reveal significant differences in a pretest-posttest analysis of scores obtained from the Framingham Type A Scale ($t(8) = 1.22$, $M_s = 49.3$ vs. 35.8). Though lacking statistical significance, this observed reduction does denote a change from the Type A (37-100) to the Type B (0-37) range of scores.

A repeated measures ANOVA of subjective anxiety change scores failed to reveal significant differences across the BL, PMR, and CCR conditions ($F(2,18) \leq 1.62$, $M_s = .86$, 1.05, and .74). Thus, decreased reactivity in EMG, SBP, and pulse-rate were not accompanied by statistically significant changes in subjective anxiety or the Type A behavior pattern.
CHAPTER IV

DISCUSSION

In this study, CCR led to reduced EMG and cardiovascular reactivity in cardiac rehabilitation patients given prior exposure to PMR training. In contrast, the PMR training alone had no effect on the EMG or cardiovascular reactivity of these subjects. Further, the reactivity-reducing effects of the CCR training were independent of task performance. This finding was revealed by a repeated measures ANOVA which showed that no changes in task performance had occurred across the BL, PMR, and CCR phases of this study. And, it was unlikely that reduced reactivity during the CCR phase was a probable effect of habituation to the stressors. This finding was revealed by a within-subject, multiple baseline across subjects analysis which showed that EMG reactivity remained stable across 6-13 sessions—until introduction of the CCR training. A repeated measures ANOVA also indicated that EMG, SCL, and cardiovascular measures showed no significant change across the BL and PMR phases. Thus, the reduced EMG and cardiovascular reactivity which were observed after introduction of the CCR phase appear to be the primary result of the CCR training.

Methodologically, this study contrasted with some earlier studies (e.g., Steptoe, 1978) in that the stress-induction technique produced stable levels of reactivity across repeated experimental sessions. This suggests that the stress-induction technique used...
in this study may be of use to future studies in which stability of EMG and cardiovascular reactions to stress is desired.

The results of this study suggest that relaxation training which is provided in conjunction with exposure to stressors, as in the CCR phase, leads to reduced reactivity. In contrast, relaxation training which is not provided in conjunction with exposure to stressors, as in the PMR phase, does not lead to reduced reactivity. Overall, these results confirm and extend the findings of Benthem and Glaros (1982) who found that biofeedback training was effective in reducing reactivity only when training was provided in conjunction with exposure to stressors.

Although reactivity has been associated with the Type A behavior pattern (see reviews by Dembroski, 1981; Holmes, 1983; Houston, 1983; Krantz, Glass, Schaeffer, & Davia, 1982), reductions in reactivity—as seen in this study—were not accompanied by significant reductions in either the Type A behavior pattern or measures of subjective anxiety. This suggests that reductions in EMG and cardiovascular reactivity which are of the magnitude of those found in this study may not lead to reductions in Type A behavior or subjective anxiety. And, it is unknown whether reductions in reactivity which are of a larger magnitude or which are induced by other methods (e.g., aerobic exercise) may lead to reductions in Type A behavior.

Because this study was implemented in a cardiac rehabilitation setting, the presence of variables—other than PMR and CCR—
which may affect reactivity to stress was inevitable. For example, all subjects participated in regular aerobic exercise as part of their rehabilitation program. And, recent evidence (e.g., Hull, Young, & Ziegler, 1984) suggests that aerobic exercise reduces cardiovascular reactivity to stress.

In addition, half of the subjects in this study received maintenance dosages of beta-blocking medication throughout the study. And, preliminary evidence (e.g., Schmeider, Friedrich, Neus, Rüdel, & Von Eiff, 1983) suggests that beta-blockers also reduce reactivity to stressors. Thus, one might expect that the addition of a relaxation therapy to a regimen which already includes regular aerobic exercise and/or beta-blocking medication, may have little added effect.

In the present study, however, CCR did lead to reduced reactivity in the cardiac rehabilitation patients who were maintained on stable regimens of aerobic exercise and/or beta-blocking medication. Thus, it may be tentatively concluded that CCR does contribute to the management of reactivity in addition to whatever effects may be produced by aerobic exercise and beta-blocking medication. However, efforts to determine the independent effects of relaxation training, aerobic exercise, and beta-blocking medication—when present in a cardiac rehabilitation population—are clearly desired.

In addition to determining the independent effects of relaxation training, aerobic exercise, and beta-blocking medication on
reactivity, a further analysis of the behavioral and physiological mechanisms whereby these variables affect reactivity is warranted.

For example, the present study suggests that combining relaxation and exposure to stress may be a possible behavioral mechanism which leads to reduced reactivity. Thus, the utility of combining relaxation and exposure to stress deserves a closer analysis.

A microanalysis of reactivity to active stressors suggests the probability that a single stimulus—the active stressor—has at least three potential effects. One effect is the elicitation of physiological response—probably through respondent conditioning. Another effect is the evocation of a problem-solving response—probably through operant conditioning. A third effect (produced as a side-effect of the evocative properties of the stimulus) occurs when the subject who is presented with the task is engaging in relaxation, and that effect is to disrupt the ongoing relaxation. Thus, when the active stressor is presented to the subject, it elicits respondently conditioned physiological reactivity, evokes operantly conditioned problem-solving behavior, and disrupts the ongoing practice of relaxation (if relaxation is being practiced at the time that the stressor is presented).

Thus, in reactivity management, the need arises for weakening the eliciting and disruptive effects of the active stressor, while leaving the evocative effects intact. Based on the findings of the present study, it appears that CCR (when preceded by PMR)
accomplishes these aims. The result is a subject who displays decreased physiological reactivity, while operant problem-solving (task performance) behaviors remain undisturbed. The precise mechanisms by which these results may be produced remain speculative, however, and are deserving of further study.

Despite the positive features of this study, several cautionary notes are in order. First, although CCR led to reduced reactivity in this study, this observation may be limited to the clinical population—consisting of cardiac rehabilitation patients—in which it was observed. And, in any event, the clinical significance of the reduced EMG and cardiovascular reactivity in this group of patients is unknown. However, since cardiovascular reactivity to stress is being explored as a possible risk factor for developing CHD (Matthews et al., 1984), further study of reactivity management in clinical populations seems reasonable.

Second, interpretation of the results of this study is limited to a comparison of the additive effects of PMR and CCR. And, a more direct analysis of the effects of different temporal combinations of relaxation training and exposure to stress awaits further study.

Third, because reactivity to naturalistic stressors was not assessed, the degree to which CCR may impact reactivity during exposure to naturalistic stressors remains unclear. It is reasonable to assume, however, that to be effective in changing any CHD-risk which may attend enhanced cardiovascular reactivity, that the control of reactivity must be extended to naturalistic stressors.
Of less concern, perhaps, is the possibility that the word "relax" which was used during the CCR condition as a means of self-relaxation, may have served more than one function. It may have served as a distractor from the active stressors and/or as an evocative stimulus for relaxation. Since this was not examined in the present study, further analysis is needed.

Finally, although a repeated-measures ANOVA suggested that CCR—as compared to PMR and BL—resulted in reduced reactivity, the inclusion of a placebo control group would have permitted a more convincing demonstration of the effectiveness of the CCR training. This limitation, however, was overcome in part by the inclusion of the within-subject, multiple baseline across subjects analysis of the EMG data. The latter rather clearly suggested that CCR produced greater decreases in EMG reactivity than were found for PMR.

In conclusion, this study presents evidence that CCR (when preceded by PMR) reduces EMG and cardiovascular reactivity to laboratory analogue stressors of the type designated as active stressors. This finding confirmed the hypothesis that a relaxation training program which includes the presentation of active stressors during the practice of relaxation results in superior control of reactivity, as compared to relaxation training which is provided under calm conditions. Further, these effects were independent of task performance variables and cannot be explained by decreased effectiveness of the stressors in eliciting physiological reactivity.

These results are consistent with a stimulus control analysis.
which suggests that reactivity to stress may best be controlled by providing relaxation training in the context of stress rather than in an environment characterized by peace and calm. Because of the possible status of reactivity as a CHD risk factor, further study of the effects of CCR in reactivity management is indicated.
BIBLIOGRAPHY


