THE EFFECT OF A PROGRAM OF OPERANT CONDITIONING
OF AUTONOMICALLY MEDIATED BEHAVIOR
ON MANIFEST ANXIETY

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The purpose of this experiment was to initiate research into the use of operant conditioning of autonomically mediated behavior (OCAM) in the modification of maladaptive behavior. Anxiety was chosen as a target behavior because of its apparent pervasiveness among many different maladaptive behaviors.

Initially, the theoretical background of OCAM was discussed. OCAM was probably late in its arrival as an experimental technique because of the many theoretical positions which maintained that OCAM was impossible. Only recently have there been appreciable experimental demonstrations of OCAM.

This thesis presented an experiment designed to test the efficacy of a particular OCAM Therapy program. Four hypotheses were proposed which could be summed up in one general statement: Subjects given training in control of Galvanic Skin Response reduction would show reductions in anxiety as measured by the Taylor Manifest Anxiety Scale.

Subjects appeared to demonstrate a reduction in Galvanic Skin Responses but instead of showing a reduction
in anxiety as predicted, a small increase in anxiety was observed.

In summary, two conclusions were made regarding this experiment: First, the slight increase in anxiety observed in the Experimental Subjects is not necessarily a sign that OCAM cannot be used therapeutically. On the contrary, the results are encouraging, since there appeared to be a change at all. It will be for future research to determine the appropriate OCAM program for producing a decrease in anxiety. Second, there may be adverse side-effects to some OCAM programs, and further research should also clarify this possibility.
THE EFFECT OF A PROGRAM OF OPERANT CONDITIONING
OF AUTONOMICALLY MEDIATED BEHAVIOR
ON MANIFEST ANXIETY

THESIS

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By

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Introduction

The problem of operant conditioning of autonomically mediated behavior (OCAM) has been a topic of recent controversy (Black, 1971; Miller, 1969; Katkin & Murray, 1968; Kimmel, 1967). This controversy is largely the result of certain theoretical positions concerning the conditionability of autonomically mediated responses. In tracing the history of this controversy, Miller (1969) notes that the autonomic nervous system has historically held an inferior position to the somatic nervous system, being equated with the emotions and other less than rational behaviors. In 1928, Pavlov stated that autonomic behavior was as conditionable as somatic behavior. Later, with the development of the two-factor learning theories, autonomically mediated behavior was generally excluded from the class of behaviors thought to be controllable through instrumental or operant conditioning. Researchers such as Kimble (1961), Mowrer (1947), Konorski and Miller (1937), Solomon and Wynne (1954), and Schlosberg (1937) denied or seriously questioned the possibility of OCAM. B. F. Skinner was uncertain in 1937 and skeptical in 1938 about the possibility of OCAM. However, in 1953, Skinner appeared certain that OCAM was impossible, and assumed the position that the occasional artifact of OCAM could be explained through
skeletal mediation. That is, operant skeletal behaviors provided conditional or unconditional stimulation which elicited the autonomically mediated behavior. Additionally, other possible explanations such as cognitive mediation have been proposed (Smith, 1954; Katkin & Murray, 1968).


However, some experiments controlling for skeletal activity reported skeletal activity preparatory to the autonomically mediated behavior (Gavalas, 1968; Shean, 1970). Furthermore, Belmaker has demonstrated that not all motor activity which is preparatory to autonomic activity is measurable by the physiological methods commonly used (1972). To avoid some of the theoretical problems associated with OCAM in this thesis, an operational definition was formulated: OCAM was defined as any empirical procedure where the probability of an autonomically mediated response is altered by response-contingent stimulation.

There is reason to believe that OCAM might prove to be a useful model in the development of a new technique in behavior modification. Ironically, an argument for this possibility was made by B. F. Skinner: "If some technique could be worked out to achieve this result [OCAM], it would be possible to train a child to control his emotions as readily as he controls the positions of his hands (1953, p. 114)." Skinner's statement is, of course, theoretical, but DiCara and Weiss described an experiment which appeared
to empirically demonstrate that OCAM could modify emotional behavior:

In the present experiment, we investigated how instrumentally learning to increase or decrease heart rate to avoid and/or escape electric shock would affect subsequent acquisition of one type of reaction, a skeletal-avoidance response. Rats were initially trained to increase or decrease their heart rate under curare, then tested in the free-moving state for transfer of heart-rate learning, and finally trained in a modified one-way avoidance situation (1969, p. 368). ... A striking aspect of the skeletal-avoidance learning was the rats' reaction to the shock. Fast heart-rate Ss were extremely reactive to the shock, generally jumping into the air, squealing, and turning toward their tail with each pulse of the shock, while between shocks they remained imobile or frozen. This sequence of responses greatly reduced their forward locomotion and prevented either escape or avoidance learning. Slow heart-rate Ss, in contrast, showed much more inhibited reactions to shock pulses, consisting of mild jerks forward, with slow walking between shocks.

These patterns of behavior suggested that fast heart-rate animals were hyperexcitable in comparison to Ss that had learned to slow their heart rate (1969, p. 371).

Others have noted therapeutic possibilities in OCAM (Ascough & Sipprelle, 1968; Miller, 1968, 1969; Murphy, 1970; Lang, 1970; DiCara, 1970; Shnidman, 1970; Budzynski & Stoyva, 1969; Belmaker, Proctor, & Feather, 1972; DiCara, 1972), but there has been very little experimental investigation of these possibilities. With one exception the few published papers on OCAM as a therapy (OCAM Therapy) have only dealt with psychophysiological disorders: vomiting (Lang & Melamed, 1969), spasmodic torticollis (Bernhardt, Hersen, & Barlow, 1972), hypertension (Benson, Shapiro, Tursky, & Schwartz, 1971), premature ventricular contractions (Weiss & Engle, 1971), and a case of essential blepharospasm (Ballard,
Doerr, & Varni, 1972). The one exception is an experiment with phobic subjects conducted by Shapiro et al. (Shapiro, Schwartz, Shnidman, Nelson, & Silverman, 1972) at Harvard Medical School. However, a recent correspondence indicated that this experiment has not yet been completed (Shapiro, 1972).

There is also a sparse but growing body of research parallel to OCAM Therapy in the realm of other bio-feedback approaches to behavior modification. Electroencephalographic feedback has been used in the modification of headache behavior (Gannon & Sternbach, 1971) and dyslexia (O'Malley & Conners, 1972). Electromyographic feedback has been used in the modification of tension headaches (Budzynski, Stoyva, & Adler, 1969) and involuntary motor behavior (Macpherson, 1967).

If Skinner is right in equating emotional self-control with OCAM, there may be very wide range of behaviors modifiable by appropriate OCAM programs. For example, if it is possible to learn emotional control through OCAM, then maladaptive emotions such as anxiety might be reduced through bio-feedback. Such anxiety might be made voluntary by instructing a Subject (S) in control of an autonomic function such as the Galvanic Skin Response (GSR). Since stress is typically accompanied by an increase in GSR amplitude, it might be possible to establish control over anxiety by establishing control over the GSR. This self-control, once established, should generalize to non-feedback situations, because anxiety reduction should be positively reinforcing and not dependent
on discriminative stimuli other than anxiety itself. Therefore, a person trained in control of GSR reduction should show a reduction in anxiety. This general statement may be reduced to the following empirical hypotheses:

1. Ss given GSR feedback and instructions to reduce GSR amplitude will show significant reductions in GSR amplitude relative to their base rate.

2. Ss given GSR feedback and instructions to reduce GSR amplitude will show significant reductions in GSR amplitude relative to a Control Group receiving false feedback and instructions to reduce GSR amplitude.

3. Ss receiving GSR feedback and instructions to reduce GSR amplitude will show significant reductions in anxiety as measured by the Taylor Manifest Anxiety Scale (TMAS), relative to a Control Group receiving false feedback and instructions to reduce GSR amplitude.

4. Ss receiving GSR feedback and instructions to reduce GSR amplitude will show significant reductions in anxiety as measured by the TMAS, relative to a No-Treatment Control Group.
Method

Subjects

The Ss were 166 volunteers from a freshman Psychology "subject pool" at North Texas State University, who were administered the TMAS. From this group of 166 Ss, 30 Ss were selected to form three matched groups on the basis of their having the distribution's highest matchable TMAS scores. Each of the three groups contained ten Ss with the following TMAS scores: 37, 34, 32, 30, 29, 29, 28, 27, 26, and 25. The three groups formed were an Experimental Group, a False-Feedback Control Group, and a No-Treatment Control Group.

Apparatus

The TMAS was used to assess and quantify anxiety. GSRs were recorded with a Lafayette #77010 single channel-recorder and psychogalvanometer. Lafayette #76602 chrome-plated electrodes were fixed to the Ss' right hands with Hewlitt-Packard Redux electrode paste. The electrodes were attached to the Ss' volar surfaces of the middle segments of their ring and index fingers. A current of .1067 amperes was passed through the electrodes and AC GRSs were recorded.
Procedure

Ss in the Experimental and False-Feedback Groups were monitored for base rate recordings of their resting GSR activity. Ss were instructed to sit as comfortably as possible during the recording and not to look at the psychogalvanometer, which was behind them. The Ss were further instructed that the recording sessions would last 20 minutes and that the Experimenter (E) would notify them when the time was up. After base rate GSRs were recorded, Ss in both groups were given three 20-minute sessions of training for control of their GSRs. Training sessions were conducted by giving the Ss GSR feedback and instructions to reduce the variability of their skin resistance as much as possible. Feedback was provided visually in the form of a printout line produced by the psychogalvanometer on a graphed paper roll. The Ss' task was to control the line produced on the paper roll so that it was straight down the middle of the roll, which was regarded as indicative of no appreciable change in skin resistance. Ss in both groups were given this OCAM Therapy program except that each S in the False-Feedback Group actually received the bio-feedback of his matched Experimental Group S. This was accomplished by removing the pen and rerolling the printout of the respective matched Experimental Group S and replacing it in the psychogalvanometer before giving each False-Feedback S his OCAM Therapy session.
After 3 OCCAM Therapy sessions for each Experimental and False-Feedback S, Ss in both groups were given a GSR recording without feedback. This final recording session was performed in the same manner as the initial, base rate session. Finally all Ss, including the No-Treatment Ss were readministered the TMAS.

Analysis of Data

GSR data were analysed by dividing each 20-minute session into 300 four-second segments and by defining a GSR as any four-second segment with a needle deflection of 2.5 millimeters or more from the middle line of a graphed paper roll. The number of GSRs per session was counted from each paper roll.
Results

All four hypotheses were separately tested with a t-test for differences in non-independent means, given as

\[ t = \sqrt{\frac{\frac{2o^2}{N} - (M_o)^2}{N-1}} \]

with df=9.

Table 1 depicts the GSR frequencies for the Experimental and False-Feedback Ss for the sessions one and five. The

<table>
<thead>
<tr>
<th>Experimental Group</th>
<th>False-Feedback Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ss</td>
<td>Session 1</td>
</tr>
<tr>
<td>-----</td>
<td>-----------</td>
</tr>
<tr>
<td>1</td>
<td>277</td>
</tr>
<tr>
<td>2</td>
<td>130</td>
</tr>
<tr>
<td>3</td>
<td>79</td>
</tr>
<tr>
<td>4</td>
<td>62</td>
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<td>5</td>
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<td>6</td>
<td>149</td>
</tr>
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<td>7</td>
<td>131</td>
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<td>8</td>
<td>36</td>
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<td>9</td>
<td>275</td>
</tr>
<tr>
<td>10</td>
<td>125</td>
</tr>
<tr>
<td>Sums</td>
<td>1495</td>
</tr>
<tr>
<td>Means</td>
<td>149.5</td>
</tr>
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</table>
Experimental Ss showed significant reductions in their GSR frequencies, with a mean GSR frequency of 149.5 in the first session and a mean GSR frequency of 33.2 in the fifth session. This difference supported hypothesis 1 with $t=4.01$ and $p<.01$. Hypothesis 2 was supported with the Experimental Group's mean GSR reduction of 116.3 over the False-Feedback Ss mean increase of 3.5 GSRs. As predicted, the Experimental Ss showed greater reductions in GSR frequency from sessions one to five over the False-Feedback Ss, with $t=10.57$ and $p<.001$. Table 2 shows the test and retest TMAS scores for all the Ss.

**TABLE II**

<table>
<thead>
<tr>
<th>Ss</th>
<th>Test</th>
<th>Retest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>for all Groups</td>
<td>Experimental Group</td>
</tr>
<tr>
<td>1</td>
<td>25</td>
<td>19</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>32</td>
</tr>
<tr>
<td>3</td>
<td>27</td>
<td>19</td>
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<tr>
<td>4</td>
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<td>30</td>
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<td>34</td>
<td>32</td>
</tr>
<tr>
<td>10</td>
<td>37</td>
<td>41</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Sums</th>
<th>300</th>
<th>252</th>
<th>279</th>
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</thead>
<tbody>
<tr>
<td>Means</td>
<td>29.7</td>
<td>30.0</td>
<td>25.2</td>
<td>27.9</td>
</tr>
</tbody>
</table>

Hypothesis 3 was not confirmed but was negatively significant. The False-Feedback Ss had a mean retest TMAS score of 25.2.
compared to the Experimental Ss mean TMAS score of 30. This
difference in anxiety reduction was significant, with $t=2.81$
and $p<.05$. Hypothesis 4 was not confirmed because the
results were not significant. The Experimental Ss did not
show significant reductions in anxiety compared with the
No-Treatment Ss with $t=.779$ and $p>.05$. 
Discussion

These results suggest that the GSR was brought under operant control, but the learned reduction in GSR activity did not bring about a measurable reduction in anxiety. The failure to bring about a reduction in anxiety may have occurred because the particular program used here modified resting GSRs rather than GSRs elicited by aversive stimulation. Research by L. S. Rubin (1964, 1965, 1970) has shown that the difference between neurotics and normals is not evident in their resting autonomic responding: "Although neurotics could not be differentiated from normals at rest or under stress, significant differences were observed in autonomic responsivity following the termination of aversive stimulation (1965, p. 572)." Rubin found that after the termination of aversive stimulation normals tended to return to a resting state of autonomic responding, but the neurotics tended to exhibit prolonged sympathetic activity after the termination of an aversive stimulus. Perhaps an OCAM Therapy program could be devised to provide bio-feedback to Ss after the termination of an aversive stimulus, and Ss could learn to reduce anxiety behavior by modifying their autonomic responding in the presence of this kind of bio-feedback.

Another point which should be raised here is that there may be negative side effects to some bio-feedback programs.
This point is illustrated by the superior anxiety reduction which occurred in the False-Feedback Group over the Experimental Group. The significant reductions in anxiety demonstrated by the False-Feedback Group might be the result of a placebo effect. However, one would expect this placebo effect to be operative in the Experimental Group because the experimental procedures were so similar. Furthermore, the non-significant difference between the Experimental and No-Treatment Ss' TMAS scores might be due to the possibility that the Experimental Group's actual control of the GSR had an effect to increase anxiety but was cancelled out by the placebo effect of anxiety reduction. The possibility that OCAM might have adverse side effects was noted by Adam (1967). Adam observed that autonomic functions were normally unconscious, but through bio-feedback and verbal labelling such autonomic functions could become conscious. Adam went on to say: "The basic prerequisite of the normal functioning of the organism seems to be that interoceptive impulses should remain unconscious (1967, p. 140)."

An alternative explanation of this phenomenon could be that the difference between the Experimental and False-Feedback Groups was small, i.e., p<.05, and could have occurred by chance. Actually, the t-score for the difference between the Experimental and False-Feedback Groups was much closer to the .02 level of chance expectancy because the t-score for the difference was 2.81 and a t-score of 2.821 was needed for demonstrating the difference at the .02 level.
Although these results are only suggestive, the possibility that some bio-feedback programs may have adverse side-effects deserves further attention. The recent explosion in bio-feedback research and the current availability of bio-feedback instruments to untrained individuals warrants further experimental investigation of this question. Nevertheless, the future for OCAM Therapy research should not appear gloomy. On the contrary, the apparent relationship between OCAM and anxiety is encouraging even though anxiety was not decreased in this experiment. It will be for future research to uncover the appropriate OCAM program for modifying anxiety in the appropriate direction.
References


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