American Journal of Clinical Hypnosis

Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/ujhy20

EMG Biofeedback, Attained Relaxation and Hypnotic Susceptibility: Is There a Relationship?

Nicholas P. Spanos & Lorne D. Bertrand M.S.

Carleton University, USA


To cite this article: Nicholas P. Spanos & Lorne D. Bertrand M.S. (1985): EMG Biofeedback, Attained Relaxation and Hypnotic Susceptibility: Is There a Relationship?, American Journal of Clinical Hypnosis, 27:4, 219-225

To link to this article: http://dx.doi.org/10.1080/00029157.1985.10402611

PLEASE SCROLL DOWN FOR ARTICLE

Full terms and conditions of use: http://www.tandfonline.com/page/terms-and-conditions

This article may be used for research, teaching, and private study purposes. Any substantial or systematic reproduction, redistribution, reselling, loan, sub-licensing, systematic supply, or distribution in any form to anyone is expressly forbidden.

The publisher does not give any warranty express or implied or make any representation that the contents will be complete or accurate or up to date. The accuracy of any instructions, formulae, and drug doses should be independently verified with primary sources. The publisher shall not be liable for any loss, actions, claims, proceedings, demand, or costs or damages whatsoever or howsoever caused arising directly or indirectly in connection with or arising out of the use of this material.
EMG Biofeedback, Attained Relaxation and Hypnotic Susceptibility: Is There a Relationship?1

NICHOLAS P. SPANOS AND LORNE D. BERTRAND
Carleton University

Subjects low in hypnotic susceptibility were assigned to three treatments. Those in one group received two EMG biofeedback training sessions and were post-tested for susceptibility at the end of their second training session (i.e., while still experiencing high levels of training induced relaxation). Those in a second group also received two EMG training sessions but were posttested for susceptibility in a separate session. Controls were posttested without intervening training. None of the groups showed significant baseline to posttest increments in susceptibility. Nevertheless, subjects in the two training groups showed significant decrements in muscle tension and significant increments in rated relaxation across the training sessions. Moreover, subjects posttested at the end of their training session began hypnotic posttesting with significantly lower muscle tension and significantly higher rated relaxation than subjects in the other two groups. Contrary to the relaxation hypothesis muscle tension was positively (rather than negatively) correlated with posttest susceptibility.

Several investigators (Simon & Salzberg, 1981; Wickramasekera, 1972, 1973) have reported that electromyographic (EMG) biofeedback training increases hypnotic susceptibility. On the other hand, no significant increase in susceptibility occurred with false EMG biofeedback (Wickramasekera, 1973) or with progressive relaxation procedures (Simon & Salzberg, 1981). Unfortunately, these investigators did not provide data concerning their subjects’ baseline EMG levels or treatment-induced EMG changes. Thus, it is not clear whether the increments in susceptibility obtained in those studies resulted from treatment-induced relaxation. Radtke, Spanos, Armstrong, Dillman and Boisvenue (1983) were unable to replicate these earlier findings. However, despite their failure to find training-induced increments in susceptibility, Radtke et al. (1983) did note that both EMG and progressive relaxation training produced a significant decrease in their subjects’ EMG levels.

Wickramasekera (1972, 1973) and Simon and Salzberg (1981) posttested subjects for susceptibility at the end of their last relaxation training session. On the other hand, Radtke et al. (1983) posttested subjects for susceptibility in a later session. This procedural variation between studies may have led to discrepant results for at least two different reasons. EMG biofeedback,
even if successful, may not enable subjects to relax during a later hypnotic session. On the other hand, subjects who are relaxed at the end of an EMG biofeedback session may maintain their high levels of relaxation and immediately following the hypnotic procedure. In short, Wickramasekera (1972, 1973) and Simon and Salzberg (1981) may have obtained large susceptibility increments because their subjects were highly relaxed before and during hypnotic testing. Radtke et al.’s (1983) subjects may have learned to relax during their training sessions, but may have been unable to generalize this learning to the hypnotic situation.

The present study was designed to test this hypothesis. Subjects who obtained low scores on hypnotic susceptibility were divided into three treatment groups. These subjects were used in order to reduce the possibility of ceiling effects on increases in susceptibility, as well as to provide a stable baseline against which posttest scores could be compared. Those in one treatment group (EMG-immediate posttest) were administered two EMG biofeedback training sessions and, at the end of the second training period, were posttested on susceptibility. Those in a second treatment group (EMG-delayed posttest) were also administered two EMG training sessions, but were posttested for susceptibility in a separate session. Control subjects underwent susceptibility posttesting without any intervening treatment. EMG levels and verbal ratings of relaxation were obtained from all subjects immediately before and after their posttest hypnotic induction procedure (i.e., before administration of the susceptibility test suggestions). Moreover, for subjects in the two training groups, EMG levels and relaxation ratings were also obtained at several points during each training session.

It is important to note that the available data concerning a possible relationship between hypnotic responding and relaxation are not consistent. For example, Barber (1969) reviewed a large body of evidence indicating that short instructions designed to increase subjects’ alertness and motivation are as effective in producing increments in suggestibility as traditional hypnotic induction procedures that emphasize relaxation. Similarly, Banyai and Hilgard (1976) and Spanos, Radtke-Bodorik and Stam (1980) reported that hypnotic instructions aimed at enhancing alertness and arousal, and traditional relaxation inducements produced equivalent levels of responsiveness to suggestions. For this reason, it was possible that the EMG relaxation conditions used in the present study could fail to enhance susceptibility.

**Method**

**Subjects**

Thirty introductory psychology students (20 females, 10 males, mean age 21.4 years) who obtained scores ranging from zero to three on the objective dimension of the Carleton University Responsiveness to Suggestion Scale (CURSS) (Spanos, Radtke, Hodgins, Stam & Bertrand, 1983a) volunteered to participate in a two- or three-session experiment on hypnosis and relaxation. All subjects received course credit for their participation.

**Apparatus**

**EMG Recording.** Frontalis EMG was recorded using surface electrodes placed approximately 2.54 cm above the center of each eyebrow. The ground was located in the center of the forehead, and electrode resistance was maintained at less than 10,000 ohms.

A Biofeedback Information Feedback System, Model B-2, and accompanying Pre-Amplifier, Model PA-2M, measured frontalis muscle tension and provided feedback. Signals were integrated over 1-minute intervals, and the integrated values
in peak-to-peak mv's were recorded manually. The feedback signal was a pure, variable frequency tone delivered over stereo headphones. Relatively high frequency corresponded to relatively high muscle tension.

**Hypnotic susceptibility.** The CURSS yields three hypnotic suggestibility scores for each subject. CURSS:O (objective) scores reflect the number of suggestions to which the subject made the appropriate overt response and range from 0 (no suggestions passed) to 7 (all suggestions passed). CURSS:S (subjective) scores reflect the extent to which the subject experienced the subjective events called for by suggestions and range from 0 to 21. CURSS:O-I (objective-involuntary) scores reflect the number of suggestions to which the appropriate response was experienced as an involuntary occurrence. These scores ranged from 0 to 7.

During pretesting, the CURSS was administered via tape recordings to subjects in groups of 5 to 10. During posttesting the CURSS was administered via tape recording to each subject individually.

**Procedure**

Subjects who met the pretest criterion were randomly assigned to the three treatment groups with the restriction of an equal number of subjects (n = 10) in each group: EMG-immediate posttest (8 females, 2 males), EMG-delayed posttest (7 females, 3 males), No EMG controls (5 females, 5 males).

**EMG Training.** Subjects in the two experimental groups underwent two 25-minute EMG training sessions separated by a 3-7 day interval. Subjects were seated in a comfortable reclining chair in a dimly lit sound-attenuated chamber. EMG was recorded every 5 min of each session. Following electrode attachment, subjects were instructed to close their eyes and sit quietly for five minutes. The feedback tone was not presented during this baseline interval. EMG training lasted for 20 minutes. EMG levels were assessed every 5 minutes of each session, providing one baseline and four test recordings for each session. An EMG value integrated over the last minute of each 5-minute period was recorded. At the end of the 5-minute baseline period and again at the end of the EMG training period subjects reported a number ranging from 0 (not at all relaxed) to 20 (the most relaxed I have ever felt) that reflected their current level of relaxation.

**Posttesting**

Subjects in all treatment groups were individually posttested on the CURSS in the same chamber used for biofeedback training. Before CURSS administration EMG electrodes were attached to subjects in the EMG-delay and control groups (electrodes were already in place for EMG-immediate subjects). Subjects in all groups sat quietly with eyes closed for a 5-minute baseline period and an EMG reading integrated over the last minute was recorded. An EMG reading was also taken immediately after the 5-minute hypnotic induction procedure (before administration of the suggestions). Immediately after taking each EMG recording, subjects were signaled to rate their level of relaxation.

**RESULTS**

**Susceptibility**

Separate 3 (groups) × 2 (pretest/posttest) split plot analyses of variance (ANOVA's) were conducted on each of the three CURSS dimensions. These means are presented in Table 1. None of the main
effects or interactions in any of the analyses attained significance. In other words, there were no significant changes in susceptibility from pretest to posttest in any of the groups. The data were analyzed further by classifying subjects in each treatment into those who showed an increase, a decrease, or no change in susceptibility on each CURSS dimension. These frequency counts are presented in Table 2. Separate chi-square analyses on each CURSS dimension indicated no significant treatment differences in the likelihood of subjects changing in susceptibility from pretest to posttest. Examination of the raw data with respect to the CURSS:O dimension indicated that 80% of the subjects in each group showed CURSS:O increases of two points or less between pre- and posttesting.

Training Effects on EMG Score and Relaxation Ratings

The effects of biofeedback training on EMG scores were assessed with a 2 (groups) x 2 (training sessions) x 5 (recording intervals) split plot ANOVA. Groups (EMG-immediate/delay) was a between-subjects factor, while sessions and intervals were within-subjects factors. The main effect for sessions was significant, F(1,18) = 4.52, p < .05. Subjects showed significantly lower EMG levels in the second training session, M = 5.52, than in the first, M = 7.84. No other effects approached significance (all ps > .10).

The effects of training on relaxation ratings were assessed with a 2 (groups) x 2 (sessions) x 2 (rating intervals) split plot ANOVA. The intervals factor was highly significant, F(1,18) = 51.60, p < .001. Subjects rated themselves as more relaxed at the end of sessions, M = 13.33, than at the beginning, M = 9.09. None of the main effects or interactions for the sessions variable, and none of the interactions for the groups variable approached significance (all ps > .10). However, the main effect for groups was significant, F(1,18) = 4.97, p < .05. EMG-immediate subjects, M = 12.27, reported higher relaxation than EMG-delay subjects, M = 10.14, throughout the two training periods. It is important to keep in mind that the failure of the groups factor to interact significantly with the remaining two factors means that the two groups (a) differed in rated relaxation before beginning training, and (b) showed the same degree of change in rate relaxation across sessions.

### EMG and Relaxation Ratings on Posttesting

Treatment effects on EMG levels before and after the posttest hypnotic induction procedure were assessed with a 3 (EMG-

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>EMG-immediate</td>
<td>1.50 (1.18)</td>
<td>0.90 (1.20)</td>
<td>4.70 (2.75)</td>
<td>2.80 (2.49)</td>
<td>0.20 (0.42)</td>
<td>0.20 (0.63)</td>
</tr>
<tr>
<td>EMG-delay</td>
<td>1.80 (0.92)</td>
<td>2.20 (1.99)</td>
<td>5.90 (1.80)</td>
<td>6.80 (3.46)</td>
<td>0.90 (0.88)</td>
<td>0.80 (1.32)</td>
</tr>
<tr>
<td>Control</td>
<td>1.40 (1.08)</td>
<td>1.80 (1.75)</td>
<td>4.30 (2.21)</td>
<td>5.00 (3.50)</td>
<td>0.40 (0.70)</td>
<td>0.70 (0.82)</td>
</tr>
</tbody>
</table>

Note: Numbers in parentheses are standard deviations. For all groups, n = 10.
TABLE 2
NUMBER OF SUBJECTS WITHIN EACH GROUP INCREASING, DECREASING OR
NOT CHANGING SUSCEPTIBILITY SCORES FROM PRE- TO POSTTEST

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>EMG-immediate</td>
<td>1</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>7</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>EMG-delay</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Control</td>
<td>4</td>
<td>2</td>
<td>4</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>7</td>
</tr>
</tbody>
</table>

immediate/EMG-delay/control) × 2 (Before/After Induction) split plot ANOVA.
The interaction was significant, F (2,27) = 6.13, p < .01; and the relevant means are shown in Table 3.

Subsequent simple main effects analyses on the within-subjects means indicated that EMG-delay subjects showed significantly lower EMG levels after the hypnotic induction procedure than before it; F (1,27) = 9.30; p < .01. Neither EMG-immediate nor control subjects showed significant changes in EMG level across the induction procedure. Between-subjects simple main effect analyses on EMG scores indicated a significant effect for the three groups before the induction period; F (2,54) = 15.60, p < .01. Subsequent Newman-Keuls post hoc comparisons on these means indicated that EMG-immediate subjects had significantly lower EMG levels before the induction than did either EMG-delay subjects or controls. The latter two groups failed to differ on this variable before the induction.

After the induction none of the groups differed significantly from one another on EMG level.

A Groups × Before/After induction split plot ANOVA on relaxation ratings yielded a significant interaction, F (2,27) = 6.26, p < .01; and the relevant means are given in Table 3. Subsequent simple main effect analyses indicated that EMG-delay subjects, F (1,27) = 23.87, p < .01, and also control subjects, F (1,27) = 25.39, p < .01, reported significant increments in relaxation from before to after the induction. EMG-immediate subjects reported no significant changes in relaxation between these periods. Between subjects simple main effect analyses indicated that, before the induction, the groups differed on rated relaxation. Subsequent Newman-Keuls analyses indicated that EMG-immediate subjects reported significantly greater relaxation than either EMG-delay subjects. The latter two groups failed to differ significantly from one another. After the in-

TABLE 3
MEAN EMG SCORES AND MEAN RELAXATION SCORES
BEFORE AND AFTER POSTTEST HYPNOTIC INDUCTION

<table>
<thead>
<tr>
<th>Group</th>
<th>EMG Means Before</th>
<th>EMG Means After</th>
<th>Relaxation Means Before</th>
<th>Relaxation Means After</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMG-immediate</td>
<td>3.27</td>
<td>4.35</td>
<td>14.5</td>
<td>14.9</td>
</tr>
<tr>
<td></td>
<td>(1.65)</td>
<td>(1.22)</td>
<td>(2.51)</td>
<td>(3.00)</td>
</tr>
<tr>
<td>EMG-delay</td>
<td>8.16</td>
<td>6.33</td>
<td>9.5</td>
<td>12.6</td>
</tr>
<tr>
<td></td>
<td>(2.79)</td>
<td>(1.89)</td>
<td>(2.95)</td>
<td>(4.03)</td>
</tr>
<tr>
<td>Control</td>
<td>6.57</td>
<td>6.20</td>
<td>9.5</td>
<td>12.7</td>
</tr>
<tr>
<td></td>
<td>(1.98)</td>
<td>(2.05)</td>
<td>(2.32)</td>
<td>(2.71)</td>
</tr>
</tbody>
</table>

Note: Numbers in parentheses are standard deviations.
For all groups, n = 10
duction procedure, none of the groups differed significantly on rated relaxation.

**Correlations**

There were no significant correlations between subjective ratings of relaxation following the posttest induction and posttest scores on the CURSS:O, CURSS:S, and CURSS:O-I. Moreover, correlations between EMG scores following the induction and posttest susceptibility scores were significant but in the direction opposite to that predicted by the relaxation hypothesis. Thus, EMG scores correlated positively (i.e., high EMG score signifying high muscle tension) with posttest CURSS:O, \( r(28) = .55, p < .05 \); CURSS:S, \( r(28) = .55, < .05 \); and CURSS:O-I, \( r(28) = .59, p < .05 \); scores.

**DISCUSSION**

Unlike Wickramasekera (1972, 1973) and Simon and Salzberg (1981), we found no significant increments in susceptibility following EMG biofeedback training even when susceptibility posttesting occurred immediately after the last EMG training period. Our negative findings were not due to a failure of our EMG training to induce relaxation. Subjects in both biofeedback groups showed significantly less muscle tension in the second training session than in the first and, within each session, reported significantly greater relaxation at the end of the session than at the beginning. During posttesting both the EMG-delay and the control subjects showed significant EMG decreases and significant increases in rated relaxation from the beginning of the hypnotic induction procedure to its termination. EMG-immediate subjects showed low EMG levels and reported high levels of relaxation before the posttest induction procedure and after the induction they remained as low as the EMG levels of subjects in the remaining groups. Contrary to the relaxation hypothesis, posttest susceptibility was positively (rather than negatively) correlated with degree of post-induction muscle tension.

Our negative findings replicate those of Radtke et al. (1983) and are consistent with a large body of other data indicating little or no relationship between relaxation and susceptibility. For instance, a substantial number of studies (reviewed by Barber, 1969; Diamond, 1974) indicate that brief task motivation instructions that do not include a relaxation component are as effective at increasing responsiveness to suggestion as hypnotic induction procedures that emphasize relaxation. Similarly, Springer, Sachs, and Morrow (1977) reported that six one-hour relaxation training sessions enhanced susceptibility by less than two points on a twelve-point scale, and Katz (1979) found that progressive relaxation training combined with social learning training did not increase susceptibility over and above the improvements produced by social learning training alone.

The reasons for the discrepancies between the Wickramasekera (1972, 1973) and the Simon and Salzberg (1981) results on the one hand and the present findings and those of Radtke et al. (1983) on the other remains unclear. It is unlikely that these discrepancies resulted from the use of the CURSS in the present study and the Harvard Group Scale of Hypnotic Susceptibility: Form A (HGSHS:A) (Shor & Orne, 1962) and the Stanford Hypnotic Susceptibility Scale: Form C (SHSS:C (Weitzenhoffer & Hilgard, 1962) in previous experiments. The CURSS correlates as highly with the HGSHS:A and the SHSS:C as these scales correlate with one another (Spanos et al., 1983b). Furthermore, the present findings are consistent with those of Radtke et al. (1983), who employed scores on both the HGSHS:A and the SHSS:C as subject selection criteria. On the whole the findings in this area do not support the idea of a close relationship be-
tween subjects’ level of relaxation and their hypnotic susceptibility.

REFERENCES


