Relaxation training inhibits fear and arousal during in vivo exposure to phobia-cue stimuli

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Abstract

Twenty carefully selected snake phobics were exposed to a caged snake for eight trials via a conveyor apparatus. During the first and eighth trials the subjects brought the snake toward themselves as closely as tolerable; records were kept of the end-of-trial distances remaining between the subject and the snake. For the six intervening trials the experimenter placed the snake a standard distance away; records were kept of the subjects' heart rates and skin-conductance levels before and during the exposures, and of their self-reported fear intensities after the exposures. Half of the subjects had received six sessions of progressive relaxation training before the exposures occurred. The results for subjects who had received relaxation training versus subjects who had not received relaxation training showed clearly that the training served to attenuate arousal and fear in the context of in vivo exposure. The results showed also that relaxation worked by lowering arousal throughout the course of exposure, not by hastening or facilitating arousal decrement during exposure. Some implications of the results are discussed. © 1999 Elsevier Science Ltd. All rights reserved.

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From the beginning of the behavior-therapy movement until the middle 1970s the treatment of choice for phobic disorders was Wolpe's (1958, 1973) technique known as systematic desensitization based on relaxation. Systematic desensitization involved training the patient to relax using an abbreviated form of Jacobson's (1938) progressive muscle relaxation, and enabling the patient to remain relaxed while imagining
increasingly fearsome phobia cues. According to the original explanatory theory (e.g. Wolpe, 1958) the parasympathetic neural substrate of muscular relaxation served to reciprocally inhibit (Sherrington, 1906) sympathetic arousal; the act of relaxing during imaginal exposure to fear cues rendered those cues conditioned inhibitors (Hull, 1943) of sympathetic activation.

During the late 1970s in vivo exposure (Marks, 1975, 1978) replaced systematic desensitization as the treatment of choice for phobic patients. In vivo exposure involves confronting the patient directly with phobia cues for as long as possible or until fear subsides. There are various post hoc theories that explain fear reduction from exposure (see Barlow, 1988).

One controversial question that emerged during the shift from systematic desensitization to exposure therapy is whether there is a place for relaxation training in treatment based on in vivo exposure. Wolpe argued steadfastly (1958, 1973, 1990) that reciprocal inhibition is necessary in successful anxiety treatment, and that muscular relaxation is an effective means of producing reciprocal inhibition. His argument rested on the clinical success of systematic desensitization based on relaxation, and on experimental results in which relaxation-trained subjects (Ss) showed lower arousal during fearsome imaging than did untrained Ss (e.g. Paul, 1969a, b; Wolpe & Flood, 1970). Marks (1976) argued to the contrary that there is no place for relaxation training in anxiety treatment based on in vivo exposure. His argument rests on a literature review about the clinical success of in vivo exposure (Marks, 1975) and on experimental results in which relaxation training did not influence imaginal fear reduction over and above effects from “imaginal exposure” (e.g. Waters, McDonald & Koresko, 1972).

There are two reports of experiments in which effects from in vivo exposure with and without relaxation training were compared. AuBuchon and Calhoun (1990) acquired passive-avoidance, self-report, and heart-rate measures of treated and untreated specific phobias before and after three variations of prolonged exposure in vivo: exposure alone, exposure after relaxation training, and exposure in the presence of a therapist. By contrast with subjects who received prolonged exposure alone, those who received relaxation training before exposure required fewer minutes of exposure to reach criterional fear reduction (defined as twice holding the feared stimulus without reports of undue fear). Relaxation-trained subjects also changed more than did subjects who received exposure alone on passive-avoidance and self-report measures of the generalization of treatment effects to untreated phobias.

McGlynn, Moore, Rose and Lazarte (1995) reported an experiment in which the effects of relaxation training on arousal and fear were monitored across repeated in vivo exposures. Eight yoked pairs of DSM-III-R (American Psychiatric Association, 1987) snake-phobic Ss were exposed to a caged snake while seated in front of a package-conveyor apparatus during eight 4-min trials. Heart rates and skin-conductance levels were recorded before and during each of the eight trials. Self-reports of fear were obtained after each trial. One subject in each yoked pair had received a regimen of group-administered progressive relaxation training beforehand. Subjects in each pair took turns controlling movement of the conveyor that presented
the caged snake. By contrast with unrelaxed Ss, those who had received relaxation training showed lower heart-rate change from baseline during the exposure trials, lower skin-conductance change from baseline during the exposure trials, and lower self-reports of fear after the exposure trials.

The results reported by McGlynn et al. (1995) are particularly intriguing because the effects of relaxation training during in vivo exposure were pronounced and were reliable across physiological and self-report measures. Relaxation-trained Ss also brought the caged snake closer to themselves than did untrained Ss during some exposures.

The experiment reported here was prompted by the results reported by McGlynn et al. (1995). One purpose was to replicate those results while improving the experiment in several ways. A second purpose of the work reported here was to describe empirically how a history of relaxation training affects measures of arousal during in vivo exposure. Theoretical interpretations of relaxation effects that are grounded in concepts such as reciprocal inhibition (Wolpe, 1958) afford the prediction that a history of relaxation training will suppress or attenuate, i.e. “inhibit” fear and arousal throughout a series of in vivo exposure trials. By contrast with data from unrelaxed Ss, measures of arousal and fear during repeated exposures among relaxation-trained Ss should be approximately parallel but uniformly lower. On the other hand, theoretical interpretations of relaxation effects that are grounded in concepts such as habituation (Watts, 1979) afford the prediction that a history of relaxation training will hasten the rate of arousal and fear diminution during a series of in vivo exposure trials. By contrast with data from unrelaxed Ss, measures of arousal and fear during repeated exposures among relaxation-trained Ss should be comparable early-on, but should decline or “habituate” more rapidly. In brief, the experiment reported here sought to replicate the reductions of arousal and fear during in vivo exposure that were associated with relaxation training in the earlier experiment; and it sought to characterize those arousal reductions as being consistent either with an “inhibition” (Wolpe, 1958, 1973, 1990) or with a “facilitated habituation” (Watts, 1979) conceptualization of relaxation effects during in vivo exposure.

1. Method

1.1. Subjects

The Ss were 20 female undergraduates who reported extreme fear of snakes. Each met the DSM-IV (American Psychiatric Association, 1994) criteria for specific phobia and showed no evidence of ongoing medical problems or of panic disorder. Ss received credit toward fulfillment of a class requirement.

1.2. Materials and apparatus

Potential Ss were selected with the Fear Survey Schedule-II (Geer, 1965), an omnibus questionnaire on which respondents rate from “none” to “terror” their fear
of 50 commonly encountered objects and events such as dogs, snakes, and thunderstorms. Tentatively chosen Ss were interviewed using sections of the Anxiety Disorders Interview Schedule-IV or ADIS-IV (Brown, DiNardo & Barlow, 1994), a structured interview guide for differential diagnosis of anxiety disorders according to the DSM-IV criteria.

Each S's self-assessments of fear intensity at eight points in the experiment were recorded on one response sheet. The sheet presented eight 10-cm lines anchored on the left by the words "no fear" and on the right by the word "terror". The S penciled a slash along one of the lines to report her current level of fear when prompted to do so.

Heart rate and skin conductance were measured using J & J Instruments Co. (22797 Holgar Ct. NE, Poulsbo, WA) #P-401 and #T-601 transducer modules, an I-330 interface, and USE software for a 386-25 IBM-compatible computer. Heart beats were recorded from pulse-volume changes beneath a sensor on the index finger of the motionless hand. Heart rates (based on heart periods in milliseconds) were calculated in beats per minute and stored at each second. Tonic electrodermal flow was monitored with pairs of gel-prepared silver/silver chloride electrodes placed on the fore and middle fingers of the motionless hand. Skin conductance from 0 to 100 µS was recorded using 0.166 applied volts DC and was averaged and stored at each second. Heart-rate and skin-conductance data were held in computer memory, then downloaded into separate files on high-density floppy disks.

Exposures to a caged, 3½-ft long, Ball Python snake were accomplished with a 10-ft long, motor-driven conveyor. A platform was fixed to the conveyor; a lidded Plexiglas cage that housed the snake was fixed to the platform. The conveyor motor was operated via a telegraph key and moved the cage toward the S at a speed of 2.5 in/s (see McGlynn, Rose & Lazarte, 1994).

Each S participated individually. She was seated upright in a standard classroom chair 1 ft from the end of the conveyor. The cage was presented at the S's approximate eye level.

1.3. Procedure

1.3.1. Subject selection

The FSS-II was administered in class to 264 female college students. Forty-two students who rated their fear of snakes as one of "terror" were contacted by telephone and interviewed briefly; 19 of the 42 students were either unwilling to participate or were judged as insufficiently fearful to do so. The 23 students who seemed appropriate during the telephone contact were then interviewed individually by the second author using the Specific Phobia section of the ADIS-IV. Of these 23 students 20 were selected who met the DSM-IV criteria for specific phobia, who did not have evidence of panic disorder, and who were without concurrent medication use.

1.3.2. Relaxation training

Ten of the 20 Ss were chosen randomly to receive relaxation training. Live relaxation training was conducted for six consecutive days. The first three training
sessions were group sessions conducted in a dimly lit room that was equipped with large, padded, armchairs. Each group session entailed 16 muscle-group training done by verbatim reading from pages 19-23 of the manual published by Bernstein and Borkovec (1973). Each session lasted 45 min. The next two sessions of relaxation training were conducted individually using large reclining chairs in a psychology clinic treatment room. These sessions entailed 4 muscle-group training according to pages 34-36 of the manual and lasted approximately 20 min. The sixth session used relaxation by recall; Ss were instructed to remember the sensations of tension and relaxation within 4 muscle groups and to “let go” any tension. The relaxation training was conducted by the second author who had no further role in conducting the experiment.

1.3.3. Pre-exposure assessment
Approximately two weeks after 10 Ss had received relaxation training all 20 Ss provided resting heart-rate and skin-conductance data during 10 min of quiet, unstructured relaxation in a standard classroom chair that later would be used during exposure sessions. The chair was positioned at the end of the conveyor as it would be positioned during the later exposure sessions. The S was seated and electrodes were attached after the skin had been cleansed with alcohol. The Ss were told that the snake was not in the building. Baseline recording was done by the 3rd and 4th authors who also conducted the subsequent behavioral testing and exposure phases of the experiment.

Shortly after baseline recording each S continued in the behavioral avoidance testing and exposure phases of the experiment. First the seated S was shown a telegraph key that would operate the conveyor during the behavioral avoidance test (BAT). Then the caged snake was placed manually on the opposite end of the 10-ft conveyor; the S was then instructed to bring it as close as possible and to concentrate on it for 5 s at its closest point. After the S complied with the instructions, the final placement of the cage was recorded in inches traveled, the caged snake was taken from the area, and the S was instructed to respond to the first of the visual analogue scales on her response sheet. The slash mark indicating her fear just after the trial was rounded-up to the nearest tenth of a centimeter.

1.3.4. Six standard exposure trials
After the behavioral test there were six 4-min exposure trials separated by 2-min intervals. Each exposure trial was initiated when the caged snake was brought into the room and the cage made contact with the conveyor surface 4 ft away from the S. Each exposure trial was terminated 4 min later when the experimenter lifted the cage from the conveyor surface and removed it from the area. At that point the S was prompted to rate her fear on one of the visual analogue scales on her response sheet; and to relax until the next exposure. Heart rate and skin conductance were recorded continuously throughout the 4-min exposures and 2-min intervening periods.

1.3.5. Post-exposure assessment
Two minutes after the sixth exposure trial the S participated in a BAT identical to the pre-treatment BAT. Afterward the S again rated her ongoing fear on one of the
visual analogue scales; progress of the cage during the test was again recorded in inches.

2. Results

2.1. Fear reporting

A report of ongoing fear (0–10 scale) was obtained from each of 20 Ss after each of six standard exposures. The means and standard deviations for the fear reports among 10 relaxation-trained versus 10 untrained Ss after each of the six exposures are shown in Fig. 1. The 120 fear ratings that produced the 12 data points in Fig. 1 were used in a 2 (relaxation conditions) × 6 (trials) mixed-model analysis of variance. (All statistical analyses were performed with SPSS, Inc. 1994 software.) The analysis yielded a significant main effect for relaxation condition, $F(1, 108) = 4.81, p < 0.030$. As is shown in Fig. 1 the fear reports of relaxation-trained Ss were uniformly low by contrast with the reports of untrained Ss. The analysis also yielded a significant main effect for trial, $F(5, 108) = 5.89, p < 0.000$. As is shown also in Fig. 1, Ss reported less fear across successive trials.

![Fig. 1. Mean SUDs (0-10) values for relaxation (n: 10) and no-relaxation (n: 10) groups. Error bars represent one standard deviation.](image)
2.2. Heart rate and skin conductance

The heart-rate and skin-conductance data were analyzed so as to answer two questions. Did a history of relaxation training produce relatively low arousal during exposure? Did a history of relaxation training produce relatively rapid arousal diminution during exposure? These questions were answered by comparing the data from relaxation-trained versus untrained Ss.

Heart rates expressed in beats-per-minute values were calculated from 20-s heart-period samples. A baseline heart rate for each S was based on a 4-min sample from the middle of the 10-min baseline recording period. The baseline heart rates for relaxed Ss ($M = 78.4$) and unrelaxed Ss ($M = 76.4$) were quite similar. Therefore, heart rates during exposure were calculated by subtracting the S's baseline heart rate from the heart rate during each 20-s interval within the six, 4-min exposure trials. Thus each S provided 72 heart-rate difference scores indicative of heart-rate responsivity during exposure.

Did relaxation training produce relatively low heart rates during exposure? In order to answer this question the 1440 heart-rate difference scores provided by the 20 Ss were used in a 2 (relaxation conditions) × 6 (trials) repeated-measures analysis of variance. The only significant effect yielded by the analysis was a main effect for relaxation condition, $F(1, 1428) = 13.69$, $p > 0.000$. The mean heart-rate change during exposure among Ss who had received relaxation training ($M = 0.87$, $SD = 2.86$) was lower than was the mean heart-rate change during exposure among Ss who had not received relaxation training ($M = 6.87$, $SD = 2.83$). Fig. 2 shows the mean heart-rate change among Ss in each relaxation condition during each exposure trial.

Did relaxation training produce relatively low skin-conductance change during exposure? Skin-conductance values in microsiemens were obtained and stored exactly as were the heart-rate values. The baseline values for relaxation-trained Ss (14.6) were the same as those for the untrained Ss (14.4). Therefore, the 1440 skin-conductance difference scores were analyzed as were the heart-rate values. The 2 (relaxation conditions) × 6 (trials) analysis of variance yielded significance only for the main effect of relaxation condition, $F(1, 1428) = 4.26$, $p < 0.04$. The mean skin-conductance change among Ss who had received relaxation training ($M = 0.89$, $SD = 1.24$) was lower than was the mean skin-conductance change among Ss who had not received relaxation training ($M = 3.87$, $SD = 1.09$). Fig. 3 shows the mean skin-conductance change among Ss in each relaxation condition during each exposure trial.

The analyses of variance within the heart-rate and skin-conductance difference scores showed that a history of relaxation training attenuated arousal during a series of six in vivo exposures. The failure to produce an effect for trials in either analysis showed that there was no habituation from trial to trial and, therefore, no opportunity for a history of relaxation training to affect the course of inter-trial habituation.

Did relaxation training affect the course of intra-trial habituation? That is, did heart rate or skin conductance during one or more of the six, 4-min exposure trials decline more rapidly among relaxation-trained Ss than among untrained Ss? To answer this question for heart rate each S's 12, 20-s, heart-rate difference-score values within each
Fig. 2. Mean heart rate change from baseline values in beats per minute for relaxation (n = 10) and no-relaxation (n = 10) groups across trials. Error bars represent one standard deviation.

Fig. 3. Mean skin conductance change from baseline values in microsiemens for relaxation (n = 10) and no-relaxation (n = 10) groups across trials. Error bars represent one standard deviation.
Table 1
Mean slopes for heart-rate change from baseline values

<table>
<thead>
<tr>
<th>Trial</th>
<th>Relaxation Mean</th>
<th>Relaxation SD</th>
<th>No relaxation Mean</th>
<th>No relaxation SD</th>
<th>p*</th>
</tr>
</thead>
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<tr>
<td>1</td>
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<td>0.081</td>
<td>0.043</td>
<td>0.172</td>
<td>0.076</td>
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<tr>
<td>2</td>
<td>0.100</td>
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<td>0.173</td>
<td>0.171</td>
</tr>
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<td>4</td>
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<td>-0.034</td>
<td>0.201</td>
<td>0.405</td>
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<td>-0.012</td>
<td>0.203</td>
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<td>0.100</td>
<td>0.205</td>
<td>0.686</td>
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</tbody>
</table>

Note: Negative slope values reflect a decrease in heart rate over time, and positive slope values reflect an increase in heart rate over time.

*p* = 2-tailed significance.

of the six 4-min exposure were plotted. The slopes of the best-fit lines for each S's six plots were then calculated. (Table 1 shows the six mean slope values for the 10 relaxation-trained Ss versus the 10 untrained Ss.) Finally the 60 heart-rate slope values for relaxation-trained Ss were contrasted with the 60 heart-rate slope values for untrained Ss using six, separate, independent-sample t-tests. None of the six t-tests produced a significant difference between the 10 slope values from relaxation-trained Ss and the 10 slope values from untrained Ss (p values are shown in Table 1). In brief, a history of relaxation training did not affect the rate of heart-rate decline during any of the six 4-min exposures.

Did skin conductance decline more rapidly within any of the six, 4-min exposures among relaxation-trained Ss than among untrained Ss? To answer this question the 20-s values for skin conductance in microsiemens were analyzed exactly as were the heart-rate data just described. The mean slopes from Ss in each condition at each exposure trial are reproduced in Table 2. Again none of the six independent-sample t-tests showed a significant difference between the slopes of relaxation-trained versus untrained Ss (p values are shown in Table 2). A history of relaxation training did not affect the slope of skin-conductance diminution during any of the six 4-min trials of in vivo exposure to the snake.

2.3. Fear behavior

During the first and eighth trials the 20 Ss conveyed the snake as close as possible. Records were kept in nearest inches of the 20 end-point distances between the S and the snake at each of the two trials. The first-trial mean end-point distance among relaxation-trained Ss was 81.4 in (SD = 20.1 in); the eighth-trial (post-exposure) mean end-point distance was 50.5 in (SD = 13.93). The first-trial mean end-point distance among Ss who did not receive relaxation training was 93.2 in (SD = 20.74); the eighth-trial (post-exposure) mean end-point distance was 72.0 (SD = 13.72). A 2 (relaxation
Mean slopes for skin-conductance change from baseline values

<table>
<thead>
<tr>
<th>Trial</th>
<th>Relaxation</th>
<th></th>
<th>No relaxation</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>p*</td>
</tr>
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<td>0.341</td>
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</tr>
<tr>
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<td>0.176</td>
<td>0.027</td>
<td>0.275</td>
<td>0.463</td>
</tr>
</tbody>
</table>

Note: Negative slope values reflect a decrease in skin conductance over time, and positive slope values reflect an increase in skin conductance over time.

*p* = 2-tailed significance.

conditions) × 2 (trials) analysis of variance produced significant effects for relaxation condition, \( F(1, 36) = 9.12, p < 0.005 \) and for trials, \( F(1, 36) = 22.32, p < 0.0001 \). After the six exposure trials Ss who had received relaxation training brought the snake nearly 2 ft closer to themselves than did Ss who had not received relaxation training. However, they had brought the snake nearly 1 ft closer before the exposure trials. Thus the analysis of variance produced a main effect for relaxation condition, not a relaxation condition × trials interaction.

3. Discussion

Does a history of relaxation training lower fear and arousal during in vivo exposures to a feared snake? McGlynn et al. (1995) produced strong effects from relaxation training on arousal and fear during in vivo exposures to a caged snake. In that experiment Ss were exposed to the snake in yoked pairs; members of each pair alternated control over a 10-ft long conveyor that was used to present the snake; one member of each pair had received relaxation training before the yoked exposure trials were begun. The experiment described here sought to replicate the effects of relaxation training on arousal and fear during in vivo exposure, and to do so with an experiment that differed in several ways from the experiment reported by McGlynn et al. Substantive replication was accomplished; relaxation training again reduced arousal and fear during in vivo exposures. Taken together, the two experiments provide noteworthy support for the empirical generalization that pre-exposure relaxation training can be used to reduce the levels of arousal and fear that would otherwise be occasioned by in vivo exposures to a feared stimulus. The relatively weaker effects for relaxation training reported by AuBuchon and Calhoun (1990) can be explained as reflecting the much less intensive single session of cue-controlled relaxation training (Paul, 1966) used in their experiment.
Does relaxation training suppress arousal and fear during in vivo exposure and/or does relaxation training hasten the diminution of arousal and fear during in vivo exposure? McGlynn et al. (1995) made no attempt to describe the effects of relaxation training on the course of arousal reduction or of fear reduction during the repeated exposure trials. In that experiment the timing and distances of successive exposures to the snake varied; thus there was no straightforward opportunity to compare the course of arousal reduction or fear reduction meaningfully within or across exposures. In the experiment reported here provision was made for repeated exposures using a standard exposure duration and a standard distance between the snake and the S. The experiment produced no evidence that a history of relaxation training affected the slopes of arousal diminution or fear diminution. Rather, a history of relaxation training suppressed arousal, i.e. relaxation training served to displace the arousal- and fear-diminution curves downward. In general, the uniform suppression of arousal throughout the in vivo exposure trials accords well with a reciprocal inhibition formulation of relaxation effects within exposure effects (Wolpe, 1976, 1989). Of course alternative conceptualizations are always possible (cf. Popper, 1959) and there are competing theories of exposure effects (e.g. Bandura, 1977; Thyer & Mathews, 1986; Weiss, Glazer & Pohorecky, 1976) that would call for competing conceptualizations of relaxation influences within exposure effects.

Recommendations regarding relaxation training as a component of exposure treatment await clinic data; for example, data related to effects from relaxation-assisted exposure versus comparable exposure without relaxation on end-state functioning, on follow-up outcome measures, on cost/benefit calculations, etc. Trends in the literature attest to the potential benefit of acquiring such clinic data. For the past 15–20 yr interoceptive cues have been acknowledged as controlling stimuli for panic onset (e.g. Hallam, 1978; Klein, 1981). More recently there has been interest in how interoceptive cues participate in fear-related disorders such as social phobia (Hope & Heimberg, 1993) and claustrophobia (Craske & Sipsas, 1992). Recognition of the roles played by somatic events in the ontogeny of anxiety disorders underscores the potential utility of relaxation training in exposure-based treatment because relaxation affects somatic phenomena. Jacobson (1939, 1940) demonstrated that progressive relaxation training can be used to reduce resting levels of heart rate, and blood pressure. Several investigators (Paul, 1969a,b; Paul & Trimble, 1970; Wolpe & Flood, 1970) have shown that relaxation training can reduce arousal associated with fearsome imaging. The experiments reported here and by McGlynn et al. (1995) show that relaxation training can suppress arousal substantially during in vivo exposures to feared stimuli.

The argument that clinic data are needed rests, in part, on challenges to the external validity of experiments that occur in non-clinic contexts. One challenge would focus on the fact that students, not clinic patients, were used as Ss (cf. Cooper, Furst & Bridger, 1969). In principle it is a non-trivial argument. The early behavior-therapy literature was inundated with reports of analogue-therapy research in which fearful college students had served as surrogates for phobic clinic patients (see especially Bernstein & Paul, 1971). There were no diagnostic criteria in the DSMs before 1980; the absence of diagnostic criteria left room for abuses in choosing research Ss; the
abuses occurred and led ultimately to widespread devaluation of the so-called ana-
logue therapy-outcome research (see McGlynn, Mealiea & Landau, 1981). Currently,
however, the DSM-IV (American Psychiatric Association, 1994) does provide diag-
nostic criteria for specific phobia. In addition the ADIS-IV (Brown, DiNardo &
Barlow, 1994) is a consensually endorsed structured-interview format for identifying
fearful persons who do and do not meet those criteria. The 20 snake-fearful Ss used
here met the consensual criteria for specific phobia according to the normative
procedure for making criterional judgements.

A second challenge to the external validity of generalizations based on non-clinic
experiments focuses on the artificiality of experimental events relative to their natural-
istic referents. The Ss in the present experiment did not handle the snake as would
patients in most variants of exposure therapy; rather they simply observed the snake.
Similarly the Ss did not show reduced arousal and fear during unpredictable encoun-
ters with a loose snake; rather they showed reduced arousal and fear during predict-
able encounters with a caged snake. Again such challenges are non-trivial (Lick
& Unger, 1977; Ost, 1989). The principle defense against criticism in terms of external
validity is that experimental research places a premium on internal validity. Attempts
to maximize internal validity often entail costs to external validity. The effort to
maximize internal validity here included using seated Ss so as to minimize “noise” in
psychophysiological recordings, and using standardized presentations of the snake so
as to eliminate trial-to-trial variability in feared stimulation.

In sum, data from two experiments support Wolpe’s (1976, 1989) argument that
there is a role for relaxation training in fear therapy based on in vivo exposure. Data
from one of the two experiments show also that pre-exposure relaxation training
serves to lower arousal and fear uniformly throughout exposure trials. Clinical
research is now needed to explore the possible contributions of relaxation training to
exposure therapy.

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