Fixed-duration shock treatment: Pre- and posttreatment stimulation, activity, and skin resistance as predictors of escape performance

D. CHRIS ANDERSON, CHARLES R. CROWELL, and NEIL R. BOYD
University of Notre Dame, Notre Dame, Indiana

and

DONALD A. HANTULA
Temple University, Philadelphia, Pennsylvania

Movement rates and skin resistance levels were recorded during a series of fixed-duration treatment shocks (ST), some coupled with a feedback stimulus (FB), and others presented in the context of procedures that offset the effects of FB on ST. These measures also were recorded in an escape-training test situation, along with escape latencies. Major findings were (1) parallel decreases in both movement and skin resistance levels in response to ST, that (2) developed over blocks of treatment shocks, and that (3) were followed by attenuated movement in the test setting. These changes, in turn, (4) were associated with impaired test escape performances. However, (5) when a FB procedure was introduced that offset the development of these movement and skin resistance changes during ST, subjects moved and escaped the same during testing as non-ST controls. Finally, (6) two procedures designed to nullify the effects of FB, introduction (in various ways) of a brief shock following the FB event (Experiment 1) and repetitious pre-ST exposure to the FB stimulus (Experiment 2), were accompanied by reinstatement of attenuated movement rates and skin resistance levels during ST and of impaired test behaviors. These outcomes were consistent with current fear interpretations of inescapable shock-treatment effects, and with condition-inhibition but not disruption or dishabituation (antihypogestic) interpretations of FB effects.

Exposure to a series of unsignaled, fixed-duration, intense, and relatively prolonged shocks (ST) can affect performances in a variety of subsequent tests. ST has been shown to influence punishment learning (Anderson, Cole, & McVaugh, 1968; Walters & Rogers, 1963), nonaversively motivated behaviors (Anderson, Crowell, Koehn, & Lupo, 1976), and a number of physiochemical systems (Anisman & Sklar, 1979; Levine, Madden, Connor, Moskal, & Anderson, 1973; Maier, Davies, Grau, Jackson, Morrison, Moye, Madden, & Barchas, 1980; Weiss, Goodman, Lositi, Corrigan, Charry, & Bailey, 1981). Perhaps the most celebrated effect has been impairment of escape and/or avoidance performances as revealed in a variety of test situations (Crowell & Anderson, 1981; Maier, Albin, & Testa, 1973; Overmier & Seligman, 1967).

Much of the research investigating these escape/avoidance impairment effects has focused on the possibility of a cognitive and/or motivational deficit resulting from the lack of control putatively engendered by inescapable, but not escapable, treatment shocks (see Maier, 1990). However, other evidence also showed that noncognitive byprod-

ucts develop in the presence of inescapable, but not escapable, shocks that, if transferred to a test environment, could readily account for escape/avoidance impairment effects (Anderson, Crowell, Cunningham, & Lupo, 1979; Balleine & Job, 1991; Crowell & Anderson, 1979; Glazer & Weiss, 1976; Mineka, Cook, & Miller, 1984). The most recent of the theories focusing on the importance of these noncognitive byproducts in explaining ST, test-interference effects is founded on the view that inescapable ST should be considered as a "baseline" stress condition (Minor, Dess, & Overmier, 1991). By this view, introduction of an "escapable" contingency as part of an experimental design should be considered as but one of many possible extrinsic conditions that may "modulate" the behavioral and/or biophysiological consequences of this stressor.

Known byproducts of inescapable shock include attenuated intra- and post-ST movement patterns for subjects receiving either fixed- (Crowell & Anderson, 1981; Lawry, Lupo, Overmier, Kochevar, Hollis, & Anderson, 1978) or variable-duration, inescapable shocks (Anderson et al., 1979; Crowell & Anderson, 1979; Mineka et al., 1984) as their ST regimen. If transferred to an escape and/or avoidance test, these tendencies to remain immobile theoretically could account for impaired performances because of incompatibility with the response-activation demands of the test task. Support for this possibility is implicit in the research of Mineka et al. (1984) and Volpicelli, Ulm, and

Portions of this research were presented at the 1987 and 1991 meetings of the Psychonomic Society. Correspondence should be addressed to D. C. Anderson, Department of Psychology, 114 Haggard Hall, University of Notre Dame, Notre Dame, IN 46556.

Altener (1984). In these studies, one of several ST groups was exposed to a brief (5 sec) offset of a houselight (termed a feedback stimulus; FB) contiguous with termination of each treatment shock (i.e., ST-FB group). Mineka et al. (1984) showed that the movement rate during testing for this ST-FB group was indistinguishable from that of non-ST and escapable-ST groups, and was considerably higher than that of non-FB, ST subjects. Volpicelli et al. (1984) further showed that this FB procedure offset the impaired barpress escape performance evinced by ST-only subjects.

A prominent explanation of these results is that the FB event functions to curb the development of fear that otherwise may become associated with the static cues of the treatment chamber. Minor et al. (1991) embellished on this notion by proposing that in the absence of FB (or the stimulus consequences of an escape response) signaling the termination of each treatment shock (coupled with a relatively long shock-free period), anxiety may remain relatively high throughout the entire treatment session for inescapably shocked subjects. However, in being explicitly contiguous with the onset of the relatively long intershock periods in most ST procedures, the FB event may become a conditioned inhibitor (see Fowler, Kleiman, & Lysle, 1985; Minor, Trauner, Lee, & Dess, 1990; Rescorla & Holland, 1977). If so, the FB procedure may counter or offset the fear that otherwise putatively develops to chamber cues as a byproduct of ST.

Given that freezing is a major byproduct of acquired fear (Blanchard & Blanchard, 1969; Bouton & Bolles, 1980; Weiss, Kreikhaus, & Conte, 1968), the possible inhibitory role of FB could explain why ST subjects receiving this stimulus not only evince posttreatment movement rates comparable to those of non-ST subjects but also show unimpaired test escape performances. For ST subjects, fear acquired during treatment may, because of transsituational similarities (see Minor & LoLordo, 1984), transfer to the test situation, thereby producing the attenuated test activity (freezing) typically observed for this group (Mineka et al., 1984). Tendencies to freeze also, arguably, are incompatible with the activity required to make barpress escape responses during testing. This possibility thus may account for the impaired performances reported for these tests as well (including Volpicelli et al., 1984). Minor et al. (1991) have suggested alternative ways that fear could mediate ST effects. These include the possibility of ST "sensitization" expressed in the form of a neophobic reaction to the novel stimuli of the test situation, and/or as a possible source of "submission" behaviors that assumptively are rooted in the subject's genetic makeup. The FB event, in countering the fear that normally develops during ST, accordingly may eliminate fear-induced freezing (and/or neophobic/submission responses). If so, no fear would be available for transfer to the test setting for an ST-FB group. As a consequence, relatively "normal" movement and escape/avoidance performances would be expected for these subjects.

EXPERIMENT 1

Several testable hypotheses can be derived from this analysis. First, on the assumption that freezing and elevated autonomic activity (e.g., decreased skin resistance) are indicants of acquired fear, both should develop over a series of treatment shocks given to groups *not* receiving the FB stimulus. Second, following a speculation of Minor et al. (1991), this fear should remain relatively high throughout the treatment session. Third, and in contrast to the above, the emergence of these two indices of fear either should be retarded or even eliminated over the same series of shocks for subjects receiving FB. Fourth, the effectiveness of FB to offset fear should be nullified by conditions that prevent inhibitory properties from developing to this stimulus. Fifth, groups that develop fear during ST should show evidence of heightened emotionality in the test environment *prior* to the initial test shock. Finally, impaired performance during testing should occur only for groups showing evidence of transferred fear (from the treatment to the test situation).

An evaluation of these hypotheses required a procedure that allowed for modification of the inhibitory effectiveness of the FB event. Given that a necessary condition for FB effectiveness is a relatively long shock-free interval between the shocks of the ST regimen (see Rosellini, DeCola, & Warren, 1986), Experiment 1 entailed introduction of a brief shock in this interstimulus interval (ISI) for some but not other groups. Thus, along with groups that received ST, ST and FB, and no ST, Experiment 1 included 12 others in a factorial manipulation of the duration and location (in the ISI) of the short shock in order to determine whether the effect(s) of this procedure depended on critical parameters.

Direct evaluations of the above predictions required that measures reflecting fear be collected *both* during *treatment* and *escape-testing* phases. Movement and skin resistance levels accordingly were recorded during treatment, and data on pretest movement were collected in the test situation for all subjects as a measure of generalized fear. It was anticipated that any conditioning of fear to environmental cues during ST would be reflected by progressively lower movement rates and decreased resistance levels in the periods immediately preceding each ST shock. Accordingly, these pre-ST measures of movement and skin resistance could be taken as an indication of the *overall* anxiety level of each group throughout the ST session.

Method

Subjects

Subjects were 105 male, naive, singly caged, 90- to 100-day-old, Sprague-Dawley rats supplied by Harlan Sprague-Dawley, Inc. All were given continuous access to food and water throughout the experiment, and were weighed and gentled daily following a quarantine conducted in accord with campus stipulations governing animal care. A local veterinarian technician and licensed veterinarian supervised care of the subjects so as to conform to the standards of the American Association for Laboratory Animal Care and the Univer-

sity Animal Rights committee. Treatment and testing were initiated at 1:00 p.m. on each day, and lasted approximately 6 h. The various treatment and test conditions were equally represented and balanced across the time frames within these sessions.

Apparatus

The ST was given in a $25.3 \times 17.7 \times 17.7$ cm (length $\times$ width $\times$ height) chamber composed of electrically isolated stainless steel walls and grid flooring (0.63-cm diam; 1.5 cm between centers). The chamber was enclosed within a larger surrounding wooden box that fit tightly over a sawdust-covered floor. This environment in turn was located in an even larger sound- and light-controlled chamber equipped with a ventilation fan and a $7\frac{1}{2}$ -W chamber lamp, the offset of which served as the FB stimulus. Each wall and grid of the ST chamber was connected separately into a shock scrambler that in turn was wired through a device that, when switched by a computer, permitted an alternative wiring configuration between shock deliveries in order to record skin resistance. Treatment shocks were supplied by a dc, constant-current, tube-regulated source (Campbell & Teghtsoonian, 1958).

The test chamber, housed in a sound- and light-controlled (and ventilated) environment identical to that for the treatment environment, was a $21 \times 23 \times 20$ cm Gerbrands operant conditioning box equipped with a standard rat bar that protruded 1.5 cm from the front wall and 6.5 cm above the floor. The floor was composed of 18, 0.296-cm (diam) grids, spaced 1.0 cm apart (centers). The grids and two aluminum end walls (side walls were Plexiglas) were wired to a Gerbrands shock scrambler that, in turn, was connected to the switched output of a constant current, ac, Lafayette shock generator.

Gross bodily movement and skin resistance jointly served as measures of fear. Movement was recorded both in the treatment and test settings by flush-mounted ultrasonic transmitting and receiving transducers (centered and equidistant from the walls) in the ceilings of both enclosures. These, in turn, were connected to modified Alton, Model-B ultrasonic motion detectors. The 39-KHz sound emitted by these devices combined with that of the ventilation fans to provide a masking noise. Motion detectors were calibrated to equivalent sensitivities in both environments; that is, they responded only to gross bodily movements and *not* to minor actions such as vibrissae or head movements.

Skin resistance was recorded only during the ST session in the treatment chamber. This was accomplished by passing a $10\text{-}\mu\text{A}$ current produced by a Grass signal generator through the grid floor during the alternative wiring configuration between ST shock administrations. In this configuration, alternate bars in the grid floor were connected in series to form two electrodes in a manner similar to an arrangement described by Crowell and Frei (1972). Subject contact between two adjacent bars thus completed a circuit that served as input to a Grass preamplifier. The output of the preamplifier was fed to a lab computer and also was recorded as pen tracings on a Grass polygraph. A program on the computer sampled skin resistance changes at a rate of 100 times per second, thereby permitting the calculation of interpeak readings (as the subject "made" and "broke" grid contact). The lowest reading obtained during each 1-sec interval was recorded as the resistance level during that period.

All treatment and test shocks were set at an exact, unchanging 0.8 mA (measured with resistance settings substituted for the subject that ranged from 15 ohms to 0.5 M-ohms). Shock (and scrambler), FB stimulus, movement, skin resistance, and all test performance parameters were controlled and/or recorded by the lab computer, located in a different room.

Design

Fifteen groups of 7 rats each were formed. Of these, 12 were given ST in a basic three-way factorial design that involved the presence or absence of FB (Factor 1), and administration of either a

0.5-sec or a 2.0-sec brief shock (Factor 2) that was given 5 sec, 30 sec, or randomly (Factor 3) in the ISI (see description below). This portion of the study thus conformed to a 2 (FB vs. no FB) $\times 2$ (0.5-sec vs. 2.0-sec brief shock duration) $\times 3$ (onset of brief shock 5 sec, 30 sec, or randomly in the ISI) factorial model. Two of the remaining three groups also received ST, and one of these also was given FB. The remaining group was not given ST, FB, or short shocks. When appropriate, within-subject dimensions also were included in various analyses of the data in order to evaluate effects attributable to repetitious measurements (i.e., trials).

Procedure

Subjects were rank-ordered according to weight measured on the day before ST treatment. These weights were used in a randomized blocks procedure to form 15 groups of 7 rats each. On the following day, the subjects of each group were placed singly into the treatment chamber for about 50 min, during which 98 (14 groups of 7 each) were given a series of thirty 30-sec fixed-duration shocks. The ISI between these shocks averaged 60 sec, ± 15 sec, and the initial shock was given after a minimum 2-min wait. For groups also receiving FB, onset of this 5-sec stimulus (houselight offset) coincided with the offset (see Jackson & Minor, 1988) of each treatment shock.

Half of the 12 ST groups in the factorial portion of the experiment were given the FB event and the other half were not. The duration of the brief shock given respectively to sets of three ST and ST-FB groups was 0.5 sec, and was 2.0 sec for the remaining two sets of three in each of these treatment conditions. Finally, for the respective ST groups within each of the four conditions formed by the combinations of FB, no FB, and the two brief shock durations (accordingly designated ST-FB-0.5, ST-FB-2.0, ST-0.5, and ST-2.0 sec), onset of the brief shock was either 5 sec or 30 sec following offset of each treatment shock or was randomly given in the period beginning 5 sec following treatment-shock offset and 5 sec before the end of the ISI. For the remaining two ST groups, the subjects of one group also were given FB (respectively designated ST and ST-FB). Subjects in the untreated group received equal time in the treatment chamber but were not given ST, brief shocks, or FB (Group NST). A random assignment procedure was followed in choosing groups for the various treatment conditions.

For all subjects, the computer switched on the motion detector and skin resistance circuit for 5-sec periods prior to and following (as well as during, for the movement circuit) each shock of the ST regimen (timing periods were the same for NST subjects). Although full elimination of odors may not be possible in the absence of an odor-masking procedure, an attempt was made to at least control their contribution by thoroughly wiping and drying the ST and test chambers between each subject (see Minor & LoLordo, 1984; Minor et al., 1991, pp. 96-99).

Approximately 24 h (± 30 min) following the last treatment shock, subjects singly were tested using a delayed barpress escape task in the operant chamber. The motion detector was programmed to record movements on a second-by-second basis for the initial 5-min period following placement of each subject into the test box. The end of this period initiated the onset of test shock. Subjects could press the bar and terminate this shock, although shock termination was delayed for 3 sec (see Volpicelli et al., 1984). If a press did not occur, shock was terminated after 30 sec, and programmed to occur again about 60 sec later (variable-time, 60-sec schedule). A total of 30 of these trials were given in a single test session. Latency to press from onset of shock served as the dependent variable.

Results

Average movement rates were computed for each subject for the periods prior to (5 sec), during (30 sec), and following (5 sec) treatment shocks. Average skin resis-

tance levels were computed only for the periods prior to and following treatment shocks. These averages were then grouped into five blocks of six trials for each of the periods where collected. The test data were stabilized by averaging movement scores for the 5-min period prior to the first test trial (expressed as movements/second), and the escape latencies for each subject were averaged over the 30 escape test trials. Statistical outcomes were evaluated against the .05 significance level and, except for those entailing a repeated-measures variable, follow-up paired comparisons were conducted using the Newman-Keuls test.

Statistical Models

Analyses involving the factorial portion of the experiment provided the basis by which these and the remaining data were evaluated. These analyses included a 2 (FB vs. no FB) $\times$ 2 (brief-shock durations) $\times$ 3 (location within the ISI) $\times$ 3 (pre-, during-, and post-ST periods) $\times$ 5 (blocks of six shock trials) multivariate analysis of variance (MANOVA); the Wilks'-Lambda solution (O'Brien & Kaiser, 1985) was used for the movement data during treatment. A 2 $\times$ 2 $\times$ 3 $\times$ 2 (only pre- and posttreatment) $\times$ 5 MANOVA was utilized to analyze the skin resistance data collected during treatment. Because single averages were used to reflect test performances (no repeated measures), 2 (feedback) $\times$ 2 (brief shock duration) $\times$ 3 (ISI location) analyses of variance (ANOVAs) were applied to the movement and latency test data.

Wilks'-Lambda Rao R values associated with the periods and blocks effects were the only statistics that emerged as significant for the overall analyses of each *treatment* measure. However, the F values associated with the effects of FB [$F(1,72) = 3.5, p < .07$] ($X_s = 26.4$ sec and 27.5 sec for the FB and NFB conditions), and brief shock duration [$F(1,72) = 6.01$] ($X_s = 26.3$ sec and 27.6 sec for the 0.5-sec and 2.0-sec conditions) either approached or achieved significance for the escape latency (but not movement) test scores.

Because the F values reflecting the effects for ISI location did not approach standard significance levels for any of these (or any other) analyses, the data were pooled across the three values of this variable for all remaining statistical analyses. This resulted in four large groups ($ns = 21$), hereafter accordingly designated as ST-FB-0.5, ST-FB-2.0, ST-0.5, and ST-2.0. (Importantly, respective mean barpress escape latencies for these groups were 25.5, 27.4, 27.0, and 27.9 sec [all differences involving Group ST-FB-0.5 were significant; Newman-Keuls test].) The performances of these larger groups hereafter are compared with those of Groups ST, ST-FB, and NST ($ns = 7$) in order to reveal the major outcomes of Experiment 1. Additional justification for comparisons between different-sized groups was provided in part from heterogeneity (of variance) tests to determine the possibility of positive bias because of inflated variance estimates for the smaller groups (see Maxwell & Delaney, 1990, p. 698; all $F_{\max}$ values < 4.0 for all variance tests pertaining to the four dependent variables of Experiment 1; e.g., escape-latency variances for the four large groups ranged in value be-

tween 4.0 and 14.4, and between 4.1 and 13.6 for the smaller subsets). Indeed, the variances were, on the average, marginally smaller for the groups with 7 rats ($X = 6.17$) than for those with 21 subjects ($X = 7.42$).

Treatment Data

The panels of Figure 1 display movements per second, averaged by subjects and groups into blocks of six trials, for the 5-sec time frames prior to (Panel 1) and following (Panel 3), as well as for the 30-sec period during (Panel 2) treatment shocks. Figure 2 displays K-ohms of skin resistance for comparably blocked trials prior to (Panel 1) and following (Panel 2) treatment shocks. The initial points on the abscissas of Panel 1 of both figures are averages for all seven groups for the 5-sec period prior to the initial shock of the treatment series. These thus constitute pre-ST *baseline* (Bln) values for movement and for skin resistance. One-way ANOVAs failed to reveal any differences among groups for baseline movements [$F(6,98) < 1.0$], but showed that groups were different during the baseline skin resistance period [$F(6,98) = 3.24, p < .05$].

Movements. There are several noteworthy aspects of the movement patterns shown in Figure 1. In the pre-ST period (Panel 1), all ST groups except ST-FB were essentially "bottomed out" on the movement scale for this treatment period. In contrast, Group ST-FB showed a relative elevation in movement throughout this pre-ST apportionment. Moreover, all ST groups, including ST-FB, exhibited depressed movement during the pre-ST period with respect to their baseline levels and with respect to the group not receiving ST (NST).

During the shock-treatment phase (Panel 2), all ST groups exhibited markedly increased rates of movement compared with their pre-ST levels and compared with the movement of Group NST. The non-shock-treated (NST) subjects continued to move in this period at a level comparable to their pre-ST movement rates.

In the post-ST period (Panel 3), the movement rates of all ST groups declined to levels well below their baseline rates, but were still elevated with respect to pre-ST movement levels. Given that all ST groups in this period exhibited a downward trend of movement across trial blocks, it is reasonable to suppose that the even lower pre-ST levels are a reflection of the continuation of this trend for all ST groups. As for the post-ST period, the movement rate of Group ST-FB in this period again was elevated with respect to that of the other ST groups. Also, all ST groups moved less than Group NST during this period. Group NST, however, continued to move at about the same rate as in the previous periods.

These observations from Figure 1 were supported by results from the following statistical analyses. A significant R value [$R(12,194) = 2.48$] for the groups $\times$ periods interaction resulting from application of an overall 7 (groups) $\times$ 3 (periods) $\times$ 5 (blocks) MANOVA to these data necessitated separate analyses for each period. The 7 (groups) $\times$ 5 (blocks) MANOVA applied to the pre-ST data yielded a significant effect of groups [$F(6,98) = 16.73$] and a significant interaction between groups and blocks [$R(24,332)$]

Figure 1. Mean movement per second, averaged by subject and group into blocks of six trials for the 5-sec time frames prior to (left panel) and following (right panel) and for the 30-sec period during (center panel) treatment shocks. Legend refers to groups that received 30, 30-sec fixed-duration shocks (ST) or no shock treatment (NST), ST coupled with feedback (ST-FB), ST coupled with feedback that was followed by either 0.5-sec (ST-FB-0.5) or 2.0-sec (ST-FB-2.0) brief shocks, or ST followed by either 0.5-sec (ST-0.5) or 2.0-sec (ST-2.0) brief shocks.

Figure 2. Mean skin resistance, expressed in K ohms and averaged by subject and group into blocks of six trials for the 5-sec time frames prior to (left panel) and following (right panel) treatment shocks. Legend is the same as that in Figure 1.

= 1.93]. The effect of groups (all F s > 33.0) and the groups $\times$ blocks interactions [all R s(1,332) > 10.0] were statistically reliable for all pairwise follow-up comparisons involving Group NST and any other but Group ST-FB. The latter comparison produced a significant effect of groups [$F(1,98) = 4.5$], but not a significant interaction of groups $\times$ blocks ($R < 1.0$). Follow-up 2 (groups) $\times$ 5 (blocks) comparisons involving Group ST-FB and each of the remaining ST groups also provided statistically significant outcomes for groups effects (all F s > 12.0), although none of the groups $\times$ blocks interactions were significant. Parenthetically, when collapsed across blocks, all comparisons using the Newman-Keuls post hoc test that involved either Group NST or ST-FB were significant (all other comparisons, $p > .66$).

Although movement rates were lower for Group NST in the 30-sec periods otherwise occupied by shock exposures to the groups receiving ST, no other effects emerged as significant from the various analyses applied to these data. This was not the case, however, for analyses of the data collected in the post-ST period. The F and/or R values reflecting the effects of groups [$F(6,98) = 9.42$] and blocks [$R(4,95) = 25.2$] and the groups $\times$ blocks interaction [$R(24,332) = 1.97$] again exceeded statistically significant levels. Appropriate paired comparisons conducted for each block (Newman-Keuls tests) indicated that Group ST-FB engaged in significantly more movement than did the remaining ST groups for Trial-Blocks 1, 2, 3, and 5 of this post-ST period. In addition, movement rates for Groups NST and ST-FB did not differ for post-ST Blocks 1 and 2, but did for each of the remaining three trial sets.

Skin resistance. As shown in Figure 2, all groups, including NST, showed a precipitous drop in mean skin resistance from baseline levels throughout the pre- and post-ST periods. However, the decline for NST subjects was less marked and their resistance averages remained consistently higher than did those of the other groups for the blocks of each period. Also, as with the movement data, the resistance averages for Group ST-FB were intermediate to those of Group NST and the remaining ST conditions.

Although group differences between baseline resistance values were arithmetically modest, the fact that they were significant necessitated their inclusion as a covariate in further analyses of the skin resistance data. An overall 7 (groups) $\times$ 2 (periods) $\times$ 5 (blocks) MANCOVA yielded significant statistical values for the effects of groups [$F(6,97) = 5.56$] periods [$F(1,97) = 17.94$] and blocks [$R(4,91) = 36.4$], and for the periods $\times$ blocks interaction, but not for the groups $\times$ periods ($F < 1.0$), groups $\times$ blocks [$R(24,318) = 1.34$], or overall 3-way interactions. Relevant follow-up paired comparisons between Group NST and all other conditions yielded significant statistical values for all [F s(1,96) > 8.0] except Group ST-FB [$F(1,96) = 3.8$]. (The same pattern of probability values was obtained from post hoc Newman-Keuls tests.) All F values for pairwise comparisons involving Group ST-FB and each of the remaining subject sets that received treatment shock also exceeded standard significance levels (all F s > 18.0; the same pattern of probability values also occurred for the Newman-Keuls tests).

Blocks-within-groups analyses resulted in significant R values for each group that received ST (all d fs > 4, 23, all R s > 9.49) except for Group NST [$R(4,24) = 1.04$].

Figure 3. The left panel displays movements per second (averaged for the 300-sec period prior to escape testing) for the period preceding the initial test shock for each of the seven groups of Figures 1 and 2. The right panel shows barpress escape latencies (averaged over the 30 test trials) for each of the same groups. All but Group NST received treatment shocks (ST), and the x-axis depicts whether or not a feedback stimulus (FB) was given and/or the trailing brief shock duration (if given) for each group.

Testing

Pretest movement. Movements per second, averaged by group over the entire 300 sec prior to the initial test shock, are displayed as columns in Figure 3 (left panel) for the seven conditions. Groups NST and ST-FB did not differ and showed more activity overall than did subjects in any of the remaining conditions. These observations were supported by a simple between-groups ANOVA [$F(6,98) = 3.7$]. Follow-up pairwise comparisons (Newman-Keuls test) resulted in significant outcomes for all comparisons involving Groups ST-FB or NST and any of the other ST conditions, but not for the comparison involving these two.

Escape latency. Escape latencies were averaged over the 30 test trials for each subject and group and are also displayed as columns in Figure 3 (right panel) for the seven conditions. Groups NST and ST-FB performed about the same and evinced faster escape latency averages than did any of the other groups. These observations were supported by a simple between-groups ANOVA [$F(6,98) = 7.88$]. Follow-up paired comparisons (Newman-Keuls test) involving either Group NST or ST-FB and any other ST condition provided statistically significant outcomes. The comparison between Groups ST-FB and NST was not significant ($p = .443$). Finally, Group ST-FB-0.5 exhibited significantly faster escape latencies than did any of the other ST groups.

Correlations

A 24×24 correlation matrix was created in which respective baseline and trial-blocks data for pre- and post-ST movement (11 variables) and skin resistance (11 variables) scores and the two test indices (pretest movement; escape latencies) served as the measures [$r_{CV}(102) = .20$, $p = .05$]. Of the 276 resulting nonredundant coefficients, those reflecting on relationships between the various treatment and test measurements were considered most relevant to the hypotheses guiding this study. Parenthetically, it is noteworthy that baseline values neither correlated with each other, nor with measures derived from performances during treatment or testing.

Treatment-test/test-test coefficients. Both treatment measures accounted for significant amounts of variance in both test variables, although (as expected) the direction of these relationships differed. For example, in correlating the trial-block movement and skin resistance treatment measures with the averaged test movement scores, all coefficients were positive and 66% (approximately) of these exceeded the .05 significance level ($X_r = .61$). Additionally, *all* of the coefficients reflecting the relationship between these treatment measures and the averaged escape-latency scores were negative and exceeded normally accepted significance levels (averages ranged from $-.41$ to $-.48$). Thus, low movement rates and skin resistance levels during treatment were associated with low activity rates and with longer escape latencies during testing, and vice versa. Finally, the correlation [$r(102) = -.51$] between pretest movement and escape latency was also negative and highly significant.

Discussion

The hypotheses guiding this experiment generally were supported. In theorizing about the development and pervasiveness of fear during the various treatment conditions, the first three hypotheses received confirmation from (1) the decreases in both movement rates and skin resistance levels (presumed indicants of fear) that occurred over post-ST blocks during treatment for all ST but not for non-ST subjects, (2) the generally lower movement and skin-resistance values in the pre- than in the post-ST periods for these animals, and (3) the attenuation in these decrements exhibited the ST-FB animals. These outcomes are consistent with the presumption that ST promotes fear conditioning to contextual cues (first hypothesis) and that the FB event, without brief shocks following it, offsets the development of this association (third hypothesis). The second hypothesis, that chronic fear resulted from the fixed-duration treatment shocks, was confirmed by perseveration throughout the ISI of relatively low skin resistance values and movement rates both for Group ST and for the four groups that were given brief shocks. The fourth hypothesis was supported by the finding that a brief shock given following the FB event nullified the ability of feedback to offset ST-induced fear conditioning.

Hypothesis 5 related to the possibility that differences in fear development during treatment would be associated with differential transfer of fear from the treatment to the test environments. Under these circumstances, a correspondence between movement or skin resistance measures during treatment and the movement data collected immediately prior to escape testing was expected. Support for this hypothesis was obtained in showing that the groups exhibiting the least fear (relatively high movement and skin resistance levels) during the treatment session (e.g., Groups ST-FB and NST) also exhibited the highest rates of movement in the pre-escape-training period. Moreover, the high positive correlation coefficients between treatment (movement and skin resistance) and test (movement) measures provided convergent validation for this hypothesis.

The final hypothesis was confirmed by the relationship that emerged between pretest movement rate (judged to be an expression of transferred fear) and barpress escape test performances. Compared to NST controls, only those groups showing pretest fear (i.e., reduced movement) also showed impaired escape performances. This relation was further confirmed by the high negative correlations between movement and skin resistance scores collected during the ST session and the test escape latencies.

In developing an explanation for these relationships, a tenable conclusion is that, although perhaps necessary, ST itself cannot be viewed as a sufficient condition for attenuated escape responding during testing. This conclusion follows from the fact that Group ST-FB, although exposed to the same shock treatment as Group ST, did not show any evidence of impaired escape responding (compared with the performance of NST subjects). At the same time, however, it must also be concluded that the mere presence of a feedback stimulus following each treatment shock is not

sufficient to offset the debilitating effects of ST. Obviously, this is because not all ST groups receiving FB evidenced test performances similar to those of NST controls. Only one of the several groups given both ST and FB performed like this untreated group. In fact, the *only* factors in the present study consistently associated with impaired escape performance during testing were pronounced decreases in movement rates and skin resistance levels (collectively indicating fear) during the periods between treatment shocks and in the period immediately prior to escape testing. The tentative deduction from this finding is that the development of fear during treatment, along with its presence during testing, is the only factor that constitutes a sufficient condition for the occurrence of the ST-interference effects shown in Experiment 1.

This conclusion is consistent with certain aspects of the account offered by Minor et al. (1991). The present findings also are consistent with the conceptualization of Overmier (1988) regarding modulators of stress. Given that the fixed-duration character of the shocks used in the present study qualifies as inescapable and that, following Overmier (1988), this treatment represents a baseline-defining stress condition, the categorization of FB as a modulator variable was confirmed by showing that FB effectiveness was decreased and/or altogether eliminated by the trailing brief shocks.

One alternative interpretation of the present findings emerges from the possibility that the brief shock regimen(s) of Experiment 1 may have introduced uncontrolled complications. Although none of the movement or skin resistance measures in Experiment 1 supported the possibility that these shocks produced stresslike effects over and above those of ST, these regimens nonetheless added from 15 sec (0.5 sec) to 60 sec (2.0 sec) more shock to the overall ST total and, because of their inescapable (fixed-duration) nature, arguably could have supplied unexpected consequences of their own. (Even though escape performances were slightly faster for the ST subjects that received the 0.5-sec vs. 2.0-sec brief shock regimen, neither of these differed from the test performance of Group ST.) For example, when used in conjunction with the FB event, these shocks may have resulted in forward fear conditioning to this stimulus. (This possibility seems highly unlikely in view of the fact that we failed to find any significant differences due to the placement of the brief shock in the interstimulus interval. If fear conditioning to the FB stimulus were an operative factor, one could suppose that placement of the brief shock so as to coincide with FB offset [5-sec delay] would have different effects than a delay of 30 sec or random placement.)

Nonetheless, Experiment 2 examined the present fear interpretation of ST interference effects while avoiding possible complications introduced by a brief shock procedure.

EXPERIMENT 2

Prior exposure to a "neutral" stimulus can offset its ability to serve as a conditional stimulus (CS) in a standard learning procedure (see, e.g., Feldman, 1977; Lubow,

1973, 1989). On the assumption that FB acquires effectiveness because it functions as a CS in a contingent backward arrangement with the shocks of the ST (or, conversely, a "forward" arrangement with the shock-free periods following these shocks; see McAllister & McAllister, 1992), preexposure to this stimulus might offset its effectiveness in much the same way as occurs in standard conditioning paradigms. Thus, the exposure of one of two ST-FB groups to the FB event prior to ST might lead to the expectation that only the nonpreexposed FB group would exhibit fear attenuation during treatment and testing.

Our test procedure for this study was altered because of the finding by Whitehouse, Bersh, Blustein, and Troise (1988) that intragroup variability can be reduced for both NST and ST groups if the 3-sec delay in shock offset used in Experiment 1 is eliminated. Pilot research in our laboratory confirmed this possibility by showing that the individual performances of NST control subjects more closely paralleled those reflected by standard averaged learning curves when the 3-sec delay was omitted, that is, a decrease in escape latencies over trial blocks to an asymptotic minimum by the third or fourth block.

Method

Subjects and Apparatus

The subjects were 28 male, naive, albino rats of the same strain, age, and supplier as those in Experiment 1. Approvals, housing, and relevant experimental conditions were the same as those in Experiment 1. The treatment and test environments and equipment were also the same as those used in Experiment 1.

Design

Four groups of 7 rats each were formed. Three groups were given an ST identical to that of Experiment 1—that is, thirty 30-sec shocks spaced 60 sec apart (± 15 sec). Two of these three ST groups were given the same FB procedure as that used in Experiment 1. Of these two, one was additionally given thirty 5-sec exposures to the FB stimulus in the treatment chamber prior to beginning the ST (i.e., pre-ST). A fourth group (Group NST) was not shocked. Movement and skin resistance scores were collected during treatment as outlined in Experiment 1.

All subjects were given 30 barpress, (nondelayed) shock-escape test trials 24 h later. Preescape activity (5-min period) and shock-escape latencies were measured in the test setting as in Experiment 1. This resulted in 4 (groups) $\times$ 5 (minutes or trial blocks) designs pertaining to each of the test movement and escape-latency data sets.

Procedure

The same randomized-blocks procedure as that used in Experiment 1 (using weight as the index variable) was practiced in the present study to distribute the subjects to one of four groups. In order to equate groups for total chamber exposure, tenure in the treatment environment was double that of Experiment 1 for all subjects. During the initial 50 min of this period, Group pre-ST-FB was given 30 consecutive 5-sec presentations of the FB stimulus (offset of the houselight). Average ISI was 90 sec (± 20 sec). All other subjects simply resided in the chamber during this period. Thereafter, ST was given in a manner identical to that in Experiment 1 to all but Group NST. The subjects of Group pre-ST-FB and those of one of the other two ST groups (Group ST-FB) were given 5-sec exposures to offset of the houselight immediately upon termination of each shock of the ST. Subjects of the ST-only group were given treatment shocks without any embellishments. Movement and skin resistance were re-

coded in the same way as Experiment 1. Testing was identical to that in Experiment 1 with the single exception that no delay occurred between barpresses and shock offset.

Results

Unless otherwise indicated, the treatment and test data were averaged in same way as reported for Experiment 1.

Treatment

Movement and skin resistance. Movements per second for the pre-, during-, and post-ST treatment periods were averaged by groups into five blocks of six trials and are respectively displayed in the three panels of Figure 4. The skin resistance data were similarly averaged for the pre- and post-ST periods and are displayed in the two panels of Figure 5. The initial set of points in the left panels of each figure reflect baseline (Bln) values for each group that did not differ among themselves [all $F(3,24) < 1.0$].

The data shown in Figure 4 indicate that movement averages shown for the pre-ST period varied as a function of the ST, FB, and preexposure conditions. Whereas Groups NST and ST-FB performed about the same as their respective Bln movement levels (similar to those in Experiment 1), movement was obviously attenuated ("bottomed out") for Groups ST and pre-ST-FB. As expected on the basis of Experiment 1, the pattern of these differences changed in the period when ST was given (during-ST period). Whereas Group NST continued to move at the same rate as in the pre-ST period, those that received ST showed

marked increases in overall activity levels. Further, and consistent with their ordinal standing in Experiment 1, the Group ST-FB moved somewhat more than the others that were also given treatment shocks in this interval. Finally, movement levels subsided for the trial blocks of the post-ST period (except for Group NST, which did not change for any of the three time frames), although Group ST-FB again remained more active throughout this segment.

As shown in Figure 5, the pattern of findings for the skin resistance data was similar in both the pre-ST and post-ST periods (again like that of Experiment 1). In showing decreased resistance levels over trial blocks in both periods, the averages for Groups NST and ST-FB nonetheless were visibly higher throughout than those of Groups ST and pre-ST-FB.

The overall 4 (groups) $\times$ 3 (periods) $\times$ 5 (trial blocks) MANOVA applied to the movement data yielded significant statistical outcomes for effects associated with groups [$F(3,24) = 18.46$] and periods [$R(2,23) = 64.83$], and for the groups $\times$ periods interaction [$R(6,46) = 6.29$]. The only effect emerging as significant from a follow-up 4 (groups) $\times$ 5 (blocks) MANOVA applied to the pre-ST movement data was that of groups [$F(3,24) = 5.7$]. Follow-up paired comparisons (Newman-Keuls test) resulted in probability values of .026 or less for comparisons between Groups pre-ST-FB or ST and Groups ST-FB or NST (all other $ps > .67$).

The follow-up MANOVA applied to the data collected during the 30-sec shock exposures yielded significant sta-

Figure 4. Movement per second, averaged and partitioned in the same manner as in Figure 1 for the four groups of Experiment 2. Legend depicts groups that received treatment shocks (ST) or no treatment shocks (NST), ST coupled with a feedback stimulus (ST-FB), or for a group that was given FB both prior to and during ST (pre-ST-FB).

Figure 5. Mean skin resistance (in kilohms), averaged and partitioned the same as in Figure 2. The legend is the same as that in Figure 4.

tistical outcomes only for the groups [$F(3,24) = 14.5$] and blocks [$R(4,21) = 15.51$] effects. Appropriate pairwise follow-up comparisons (Newman-Keuls tests) yielded significant statistical outcomes respectively between the ST-FB and all other groups (all $ps < .05$) and Group NST and the two remaining ST groups, but not between Groups ST and pre-ST-FB.

The MANOVA applied to the post-ST movement scores resulted in a significant statistical value only for the groups variable [$F(3,24) = 10.01$]; the statistic pertaining to the groups $\times$ blocks interaction approached significance [$R(12,55) = 1.73$]. Follow-up pairwise comparisons (Newman-Keuls test) revealed that the differences between Group pre-ST-FB and each of the other three groups were reliable. The remaining groups did not differ from one another.

The overall 4 (groups) $\times$ 2 (pre- vs. post-ST periods) $\times$ 5 (blocks) MANOVA of the skin resistance data yielded significant F values for the effects of groups [$F(3,24) = 40.3$] and blocks [$R(4,21) = 114.0$], and for the periods $\times$ blocks interaction; follow-up 4 (groups) $\times$ 5 (blocks) MANOVAs for each period provided identical patterns of significant effects—that is, significant F ratios for groups [$F(3,24) > 20.97$] and blocks [$R(4,21) > 121.2$] effects, but not for the groups $\times$ blocks interaction. Follow-up tests involving paired comparisons (collapsed over periods; Newman-Keuls) revealed that the contrasts between Groups NST or ST-FB and Groups ST or pre-ST-FB were highly significant. Finally, whereas the comparison between Groups ST and pre-ST-FB did not result in a sig-

nificant outcome ($p = .189$), the contrast between Groups NST and ST-FB did ($p = .045$).

Testing

Preescape test movement (left panel, Figure 6) was averaged by subject and group into five, 60-sec time frames. Escape latencies (right panel, Figure 6) similarly were grouped into five blocks of six trials each. The orderly trial-block functions in these displays partly are the byproduct of less within-group variability than occurred for Experiment 1, and probably are due to the modified test procedure that was employed in the present study.

Movement. Although all groups decreased in movement rate over the 5-min period prior to escape testing, Groups NST and ST-FB did so more slowly than did Group ST. The movement rate for the pre-ST-FB condition was the lowest of any for the first minute, but was indistinguishable from that of the ST group for the final 4 min of this period. The 4 (groups) $\times$ 5 (minutes) MANOVA applied to these data yielded significant statistical outcomes for the effects of groups [$F(3,24) = 6.75$] and minutes [$R(4,13) = 18.54$]. The pattern of significant outcomes from follow-up pairwise comparisons (Newman-Keuls tests) is visually apparent from Figure 6; that is, differences between Groups ST-FB or NST and Groups pre-ST-FB or ST were significant ($ps < .05$), but those within each of these sets were not.

Escape latency. When compared with Groups ST-FB and NST (as revealed in the right panel of Figure 6), Groups ST and pre-ST-FB evinced both longer and somewhat

Figure 6. Mean movement prior to the first test shock (left panel) and escape latencies (right panel), expressed in seconds and averaged by subject and group into blocks of six trials. The legend is the same as that in Figure 4.

more erratic mean latencies across the trial blocks defining the escape test. Further, all groups evinced improvement over trial blocks in that the averaged latencies of each decreased from the first to the fifth trial set.

The 4 (groups) $\times$ 5 (trial blocks) MANOVA applied to these data revealed significant statistical outcomes for effects pertaining to groups [$F(3,24) = 9.21$] and to trial blocks [$R(4,21) = 2.85$], but not for the groups $\times$ blocks interaction. As occurred in connection with the test movement data, follow-up pairwise comparisons supported the observations (from Figure 6) that the escape performances of either Group ST or pre-ST-FB were significantly different from those of either Group ST-FB or NST ($p < .01$), but were basically the same within each of these two-group sets.

Correlations. The same correlational procedure as that outlined in Experiment 1 was followed for the present study [$r_{CV}(26) = .45$]. Correlations between either of the treatment measures and test activity [average $r(26) = .53$] and escape latencies [average $r(26) = -.51$], respectively, were relatively high and in the same directions as those reported for Experiment 1.

Discussion

A primary assumption guiding Experiment 2 was that arranging the onset of the FB event to coincide with the offset of each treatment shock (thereby also signaling the onset of the shock-free intertrial interval) would function as a CS for the development of conditioned inhibition. This inhibition, in turn, was thought to counter the fear that otherwise becomes associatively linked to contextual cues for ST subjects. The effectiveness of an FB procedure thus

was believed to depend on the presence of processes antagonistic to excitatory fear learning, the net consequence being to nullify the associative byproducts of ST that otherwise would be available for transfer to the test environment.

On the expectation that preconditioning exposure offsets the capacity of a stimulus to function as a CS (Lubow, 1989), this logic was tested in part by repeatedly presenting to one of two groups the same stimulus that later was arranged to function as an FB event during ST. If the FB stimulus served as a CS for inhibition, then this preexposure procedure should prevent it from entering into an inhibitory associative linkage (thus nullifying its subsequent ability to offset fear). The result, in turn, should be a full-blown ST effect for those subjects preexposed to FB (Group pre-ST-FB) but not for ST-FB subjects.

The outcomes of Experiment 2 were consistent with this theorizing. Subjects that repeatedly were given pre-ST exposures to the FB stimulus showed the same patterns of movement, skin resistance, and impaired escape performances as those that were given ST only. In contrast, none of these indications occurred for the nonpreexposed ST group that otherwise was given the same FB arrangement during ST. Pre-ST exposure to the FB event thus seems to have offset the ability of this stimulus to develop inhibitory properties. The result apparently was the absence, during ST, of a process that offsets the development (and hence transfer) of contextual fear.

Importantly, the outcomes of Experiment 2 were consistent with those of the first study in showing that neither fixed-duration ST per se nor ST coupled with FB is a sufficient condition for, respectively, promoting or eliminating the attenuation of movement or escape performances during testing. In terms of the sufficiency of ST, of the three

shock-treated groups in Experiment 2, only two evinced attenuated test activity and escape learning. The other ST group performed identically to NST controls. Moreover, because the possible complications of the brief shock procedures of Experiment 1 were avoided in Experiment 2, these performance differences could not be attributed to variations in overall amounts and/or numbers of shocks during the treatment phase.

In terms of the sufficiency of FB for nullifying ST effects, of the two groups receiving this procedure, only one performed the same as NST subjects. The other (because of pre-ST exposures to the FB event) performed identically to the ST-only group. This means that mere exposure to the FB procedure itself is not sufficient to offset ST effects during testing. Moreover, because other manipulations besides a feedback stimulus following treatment shocks have also been shown to nullify ST effects (see, e.g., Anderson, Elder, O'Brien, & Pearson, 1970; Lawry et al., 1978; Maier, 1990, Experiment 2; Merrill & Anderson, 1970; Payne, Anderson, & Murcurio, 1970), it is clear that the FB procedure is not a necessary condition for the amelioration of ST effects.

Instead, as in Experiment 1, the only feature in this study that distinguished the groups that showed impaired test behavior from those that did not was the distinctive pattern of movement and skin resistance changes suggestive of marked fear. Arriving at this finding with different procedures, this second study provided convergent validation for the assertion that only when fear develops during treatment and is also present during testing (as indicated by attenuated movement) are escape performances impaired. In contrast, when fear is prevented from developing during treatment and is essentially absent in the test environment, escape performances are not impaired.

Parenthetically, it is noteworthy that preexposed subjects did not respond differently from those in the ST-only group when given postshock presentations of the FB event, even though nonpreexposed subjects did. It is as if preexposed subjects did not "register" the FB event during ST. This finding is consistent with an opponent-process interpretation of the preexposure phenomenon (Solomon, 1980).

GENERAL DISCUSSION

The present studies documented changes in movement rates and skin resistance levels that were hypothesized to occur in connection with a series of fixed-duration treatment shocks (i.e., ST). These studies also showed how the movement and resistance measures were affected when ST was coupled with FB, and when two procedures, speculated to offset the effects of FB on ST, were employed. The relation of these changes during ST exposure to various performances measured in a different (test) setting also was evaluated. Major findings included (1) parallel decreases in both movement and skin resistance in response to ST, that (2) developed over blocks of treatment shocks, and that (3) were followed by attenuated movement in the test environment. The latter, in turn, (4) was consistently as-

sociated with impaired escape performances in the test setting.

However, (5) when an FB procedure was introduced that offset these changes in movement and skin resistance during ST, subjects moved and escaped the same during testing as non-ST controls. Finally, (6) procedures designed to nullify the effectiveness of FB were accompanied by the reinstatement of attenuated movement and skin resistance during ST and of impaired test behaviors. These procedures were (1) administration of a brief shock following the FB stimulus (Experiment 1) or (2) repetitious pre-ST exposure to the FB event (Experiment 2).

The correlational analyses in both studies between the treatment and test-performance data provided convergent confirmation that whenever movement rate and skin resistance levels were relatively low during treatment, movement rates during testing were also low and escape performances were impaired. In contrast, when movement rates and resistance levels were relatively high during treatment, as occurred for the ST subjects that received FB only (or NST groups), movement rates were also high during testing, and escape performances were "normal" or unimpaired. Noteworthy here is that relative movement rates measured during *either* treatment *or* testing served equally well as predictors of test escape performances.

The development in the present experiments of a distinctive pattern of behavior during treatment that appears later during testing is consistent with response-based theories of ST-interference effects (Anderson et al., 1968, 1979; Balleine & Job, 1991; Black, 1977; Brookshire, Littman, & Stewart, 1961; Levis, 1976). One such theory rests on the assumption that low movement levels during ST may be instrumentally reinforced by a partial reduction in pain resulting from adopting a favorable posture during ST. This immobility posture may become associatively linked with the contextual cues of the treatment setting and may, because of common trans-situational features, generalize to the test environment. In interacting with the demands of the test task, the presence of a generalized immobility tendency can be seen as incompatible with behaviors involved in acquiring an escape response, thus serving as a possible explanation of ST-interference effects such as those exemplified in the present research.

Another response-based interpretation of ST is that the source of attenuated movement associated with ST reflects the acquisition of conditioned fear during treatment that, because of trans-situational similarities, generalizes to the test environment. The presence of fear in the test situation could, among other things, result in freezing for ST subjects, a response that is also logically incompatible with acquisition of an active escape response (see Brookshire et al., 1961). Minor et al. (1991) have taken a less literal view in further suggesting possible nonresponse byproducts of transferred fear as sources of interference with escape learning.

The collection of both behavioral and physiological measures in the present research may be useful in choosing between these two different response-based theories of ST effects. Whereas autonomic concomitants would be ex-

pected to accompany the acquisition of fear, they would not do so in the case of reinforced postures. Accordingly, the correlation between lower skin resistance levels and impaired escape performances was more consistent with a fear than with an adventitious-reinforcement interpretation of ST effects.

Noteworthy here is a body of literature that seems to have provided outcomes that are contradictory to those of the present findings. Several published reports have failed to show orderly relations between movement patterns observed in various ways following inescapable shock treatments and deficits in escape test performances. However, the procedures employed in these studies differ markedly from those used in Experiments 1 and 2. Procedural differences include factors such as use of restraint during treatment; the procedures employed during testing, including the escape test requirements; different measurement procedures for movement; and use of potent pharmacological agents (Maier, 1990; Maier & Minor, 1993) and/or brain-lesioning techniques (Maier et al., 1993). Despite these differences, it is useful to examine certain of these conflicting reports more closely.

Consider first Experiment 2 of Lawry et al. (1978), who showed with rats that movement patterns engendered during exposures to different types of fixed-duration shocks (ac or dc shocks that either were or were not pulsed) did not correlate well with the subsequent occurrence or nonoccurrence of deficits in escape test performances. Although their subjects were restrained during ST, they nonetheless recorded movement *during* the treatment session, used a two-way shuttlebox escape procedure given 24 h after treatment, and did not employ drugs or brain lesions as a means of altering movement patterns. Importantly, the only group that evinced impaired escape performance in Lawry et al.'s study, also exhibited a pattern of decreased movement during treatment similar to that observed for subjects showing impairment in the present research. Of further interest is that the shock type in the present (scrambled dc given to free-moving rats) was perhaps most similar (physically) with that associated with impaired test performance in the Lawry et al. study (continuous ac shock given to restrained rats). Finally, none of the other groups in either of Lawry et al.'s experiments showed a decrease in movement level either within or across their treatment sessions that, in the present studies, was taken as support for the development of fear.

Comparisons are more problematic with the recent studies of Maier (1990); in Maier's study, procedural differences were even more pronounced than those of Lawry et al. (1978). This was most evident for Maier's Experiment 1, in which the inescapable shock regimen did not involve fixed durations, fecal and urine materials were permitted to accumulate during testing, and the test procedure involved a combination of initial shocks, followed by a relatively long rest, and then sequential exposure to a series of one- (FR-1) and two-way (FR-2) shuttle escape response contingencies. These procedural differences may explain Maier's (1990, Experiment 1) failure to find a correlation between observer-determined "freezing" in the

test setting (taken as a measure of fear) and impaired shuttle performances.

The procedures of Maier's (1990) Experiment 2, in which fixed-duration treatment shocks were employed and fecal and urine byproducts were cleansed from the test environment between subjects, were somewhat more similar to those of the present study. Here, freezing measured in the test setting 24 h following ST was not only higher than for controls, but was also associated with marked impairment by ST subjects of the FR-2 shuttlebox response component. Maier also reported that fear following (but not preceding) a single test shock seemed to be absent for subjects given ST 72 h previously. However, these subjects did not show a deficit in escape testing, a finding that may also be taken as consistent with present outcomes. Nonetheless, the absence of evidence either of fear or of impaired test performance 72 h following ST in Maier's study seems contradictory to findings from other reports. For example, Madden, Rollins, Anderson, Conner, and Levine (1971) showed both marked and high levels of fear (indexed by corticosterone levels) following a single test shock given 120 h following ST. Similarly, Levine et al. (1973) reported elevated fear as indexed by elevated corticosterone level and defecation rate and reduced ambulation in an open-field test conducted 288 h following ST. Crowell and Anderson (1981) showed marked impairment of one-way shuttlebox performances *both* 24 and 168 h following treatment. This latter finding was reported as consistent with the outcomes of Glazer and Weiss (1976) and of Overmier (1968).

It is noteworthy that some ST phenomena (e.g., interference effects 24 h following ST, but see Maier et al., 1973) seem to show up in spite of marked procedural variations whereas others do not (e.g., interference effects following much longer ST-test intervals). This may reflect on the differential importance of some of the procedural ingredients that characterize these different sets of studies. Allowing for this possibility, the present outcomes seem consistent with those of Maier (1990) in showing that fear may be both a necessary and sufficient condition for the occurrence of interference effects 24 h following ST.

Given the correlational outcomes of Madden et al. (1971) and Levine et al. (1973), the present research can be viewed as an extension combining a biophysiological with a behavioral measure of fear. Whereas Levine et al. were able to discern a relationship between the presence and absence of biophysiological byproducts of ST-induced fear and variations in subsequent test performances, the present outcomes occurred with manipulations that divorced *fear* from ST. Variations in test performances in the present studies (i.e., impaired or unimpaired escape learning) occurred only when fear (referenced by *both* attenuated movement *and* skin resistance level) was present for ST subjects. When fear was either eliminated or markedly reduced for ST groups, test performances were identical with those of non-ST subjects. This pattern of outcomes in the present research seems to imply that the presence of fear, not just decreased movement or mere exposure to treatment shock, is the single best predictor of impaired escape learning.

The present data may also reflect on the various ways that FB functions in offsetting the effects of ST on test escape performance (Mineka et al., 1984). One possibility is that FB simply disrupts rehearsal of the excitatory association formed by the contiguous pairing of contextual cues and each shock of the ST, thus precluding the development of fear during treatment. A related view derives from the possibility that the repetitious shocks of the ST may, through an opponent-processes-like mechanism (Solomon, 1980), promote habituation or hypoalgesia (Maier & Keith, 1987; although see Fanselow, DeCola, & Young, 1993, on the effects of spaced unsignaled shocks). Given that these processes may be conditionable (Solomon & Corbit, 1974), a basis exists for their generalization from the treatment environment to the physically similar test setting of the present studies (see Minor & LoLordo, 1984). A possible consequence of this would be reduced sensitivity (i.e., elevated threshold; see Anderson et al., 1968, Experiment 5) to test shock for ST relative to non-ST control subjects, and thus impaired performance for the former. If so, then the effect of FB might be understood as a source of dishabituation. In dishabituating the effects of ST, ST-FB subjects putatively would exhibit a sensitivity to test shock comparable to that of NST rats, thus explaining their lack of impaired escape performances.

These "disruption" interpretations of FB are not consistent with the present data. For example, it is not obvious how a trailing brief shock might offset the rehearsal-disrupting effect of FB on the formation of the ST-context association (thus preventing fear). Indeed, the brief shocks alone arguably could have served the same disruptive function as FB for such rehearsals. Thus, the fact that fear was both present and strong during treatment and testing for subjects receiving any of the brief shock regimens, whether or not FB also was involved, seems contrary to a rehearsal-disruption notion.

A possible counterargument here is that the brief shock procedure may have disrupted the rehearsal-disrupting or dishabituating influence of the FB event. Although conceivable, this argument would be difficult to reconcile with the fact that even the 0.5-sec brief shocks exerted the same influence whether they were given immediately or 25 sec following the offset of the FB event. If disruption of a rehearsal-disruption or dishabituation effect was involved, then FB should have been more effective in offsetting ST effects for the delayed than for the immediate brief shock condition.

Perhaps most problematic of all for a dishabituation-hypoalgesia interpretation is the fact that, although FB offset movement decreases during ST as well as during later testing, as would be expected given this view, an opposite effect should have occurred for skin resistance. In arguing that dishabituated ST subjects should remain *more* sensitive than habituated ones to shocks throughout both treatment and testing, the result should have been relatively greater decreases in skin resistance levels for ST-FB than for ST-only and related groups. Instead, decreases in skin resistance were offset by the FB event.

The notion of FB as a conditioned inhibitor (and perhaps the similar "unpredictability" interpretation of Rosellini, Warren, & DeCola, 1987) thus remains the more viable account of the present data. In repeatedly and consistently preceding relatively long shock-free periods, the FB event may have become associated with the unconditioned by-products (e.g., relief, relaxation, uncertainty reduction regarding the next occurrence of shock, etc.) of these exposures (Denny, 1971; Minor et al., 1991; Mowrer, 1960). On the premise that components of the emotional response following the initial shock of ST become conditioned to ambient contextual cues of the treatment chamber, the FB event may have functioned to offset fear through the presence of conditioned inhibitory processes. As noted, this interplay of circumstances respectively heralding shock presence (contextual cues) and then shock absence (FB) has been identified as a necessary condition for the development of conditioned inhibition (see Rescorla & Holland, 1977). If an additive relationship between the opposite processes of excitation and inhibition is assumed (Rescorla & Wagner, 1972), then the net effect over repeated shock trials in the present research should be only modest fear loading to the contextual cues. This interpretation is strengthened by the findings that when conditions were introduced that offset the capacity of the FB event to signal a shock-free period, contextual fear was reinstated.

An escapable ST condition was not included in the present studies for several reasons. In recent uses of this condition, researchers have attempted to equate shock parameters while comparing the role of an external FB stimulus with the FB-like role putatively served by a treatment-shock escape response. Because both FB and escapable groups have sometimes performed the same as NST subjects in subsequent tests (Mineka et al., 1984; Volpicelli et al., 1984), some have concluded that both manipulations serve similar fear-nullification functions (see also Cook, Mineka, & Trumble, 1987). However, Rosellini and his colleagues (Rosellini et al., 1986, 1987; see also Maier & Keith, 1987) have reported conditions (e.g., short intervals between treatment shocks) in which the effect of FB is eliminated even though the escapable condition remains effective in negating fear. Minor, Trauner, Lee, and Dess (1990; see also Jackson & Minor, 1988), in arguing that the standard FB procedure does not adequately model all of the stimulus features supplied by an escape response, showed equivalent fear-reducing effects for an escape response and an externally presented stimulus that was timed to occur (as does an escape response) immediately prior to the offset of treatment shocks of varying durations (i.e., a "cessation" stimulus).

Unfortunately, we have been unable to replicate this finding in our laboratory using fixed-duration treatment shocks (Torrez & Anderson, 1993). It thus may be that modulation of the stress effects of inescapable shock by a cessation (or FB) stimulus depends on how that event is dispensed (i.e., whether in a fixed- or variable-duration format). Although this seems consistent with attempts to model the effects of an escape response where variable

shock-exposure durations would be expected to occur as subjects mastered an escape contingency, such clearly was not the focus of the present research. Obviously, further research is needed concerning the circumstances that (1) optimize FB effectiveness and (2) compare FB procedures with conditions in which a response during treatment is made functionally effective in terminating ST.

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