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The facilitative effects of heart-rate feedback in the emotional processing of claustrophobic fear

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Abstract

This study examines predictions derived from Foa and Kozak's theory of emotional processing. We hypothesized that the provision of heart-rate feedback would facilitate emotional processing through a fuller activation of the participant's fear structure, and by focusing participants' attention on information that is incompatible with the fear structure, i.e., the interoceptive pattern of habituation. Nonclinical students ($N = 54$) showing marked claustrophobic fear received 30 min of self-directed exposure to a claustrophobic chamber. Three exposure conditions (heart-rate feedback, paced-tone control, and exposure only control) were examined across six 5-min exposure trials. Participants receiving heart-rate feedback displayed greater between-trial habituation across treatment trials and lower levels of fear at post-treatment. Treatment process findings failed to support the fear activation hypothesis. Implications of the findings for theories of fear reduction are discussed.

1. The facilitative effects of heart-rate feedback in the emotional processing of claustrophobic fear

Evidence accumulated over several decades and numerous domains of situationally bound fear has demonstrated the potency of exposure-based methods in the treatment of phobic disorders (Marks, 1978; Rachman, 1978; Barlow, 1988). Nevertheless, considerable debate still exists regarding the mechanisms governing the reduction of pathological fear. Rachman (1980)

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proposed a theoretical account of fear reduction based on emotional processing. He defined emotional processing as the decline of emotional disturbance to the extent that other experiences and behaviors proceed without disruption, and as a process that was dependent upon direct experiencing of the emotional disturbance. Signs of incomplete processing include return of fear and disturbing dreams.

Based on Rachman's emotional processing theory and Lang's bioinformational theory of fear (Lang, 1979), Foa and Kozak (1986) outlined an emotional processing account of fear reduction that proposed two necessary conditions for emotional processing. First, the fear structure must be activated. The fear structure is hypothesized as a set of propositions about the stimulus, the response (including the physical, behavioral, and cognitive response systems), interpretive information about the meaning of the stimulus and the response. Activation of the fear structure is believed to occur by providing information that matches a part of the network, as would an accelerated heart rate match the response proposition of fear. Through generalization of activation, the other sections of the network are assumed to be activated, particularly in the cohesive networks representative of specific phobias.

According to Foa and Kozak (1986), a second necessary condition for emotional processing to occur is that information incompatible with elements of the fear structure must be made available and cognitively processed. Incompatible information is believed to emerge as a result of the experience of short-term, within-session physiological habituation. That is, reduction of arousal results in a disassociation between the stimulus and response propositions. As a result of repeated exposures, the perception of harm from the stimulus is lowered, as is the negative valence associated with the physiological responses to the feared stimulus. These cognitive changes accruing from repeated disconfirmatory experience result in less drive for preparatory arousal, in turn resulting in between-session habituation.

Accordingly, factors which inhibit initial fear activation, or which interfere with physiological habituation and cognitive change, should retard fear reduction. The factors identified by Foa and Kozak resemble those suggested by Rachman (1980) as potentially interfering with complete emotional processing. These include certain personality and stimulus factors that could impede emotional processing, with the latter category including concentration on a separate task and excessively brief presentations of the stimulus. To date, systematic investigations of these factors have been few and have exclusively focused on the role of distraction. Foa and Kozak (1986) suggested that distraction interferes with the activation of fear by disrupting the match between aspects of the stimulus setting and the fear structure. Moreover, distraction may also serve to block the adequate processing of corrective disconfirmatory information thus preventing the modification of the fear structure. Direct examinations of the effects of distraction on fear reduction have generally supported predictions from emotional processing theory (Grayson, Foa & Steketee, 1982, 1986; Sartory, Rachman & Grey, 1982; Craske, Street & Barlow, 1989; Telch, Ilai & Valentiner, 1990; Rodriguez & Craske, 1993). Interestingly, efforts to enhance emotional processing during exposure through manipulations designed to increase fear activation or increase the processing of corrective disconfirmatory information are lacking.

The aim of the present study was to test predictions derived from Foa and Kozak's emotional processing theory. Specifically, we sought to examine whether emotional processing of claustrophobic fear could be *facilitated* by a heart-rate feedback manipulation designed to

make fear-relevant information more available during exposure. It was hypothesized that providing heart-rate feedback during exposure would enhance the activation of the claustrophobia fear structure, and hence result in more complete within-trial habituation. Further, the provision of heart-rate feedback would enhance between-trial habituation by providing information incompatible with the fear structure (i.e., within-trial habituation of heart rate reactivity). These considerations led to the following four specific hypotheses: (a) claustrophobics receiving exposure with HR feedback would show significantly greater improvement on indices of subjective fear, heart-rate reactivity, and clinically significant improvement relative to claustrophobics who received exposure alone or exposure with paced-tone focusing (Hypothesis 1); (b) the provision of HR feedback will result in higher levels of initial fear activation during exposure (Hypothesis 2); (c) the provision of HR feedback will result in greater within-trial habituation across treatment trials (Hypothesis 3); and (d) the provision of HR feedback will result in greater between-trial habituation (Hypothesis 4).

2. Method

2.1. Study participants

Fifty-four University of Texas students displaying a marked fear of enclosed spaces took part in the experiment for which they received partial course credit. All participants were required to meet stringent behavioral criteria for claustrophobic fear (see below). The final sample of 54 participants was predominantly female (85.2%), with a mean age of 17.9 years (S.D. = 0.6).

Participants were selected from a large pool ($n > 5300$) of introductory psychology students through a two-stage screening procedure. Stage one consisted of a pencil and paper measure of claustrophobic fear administered during the second day of class. Those reporting a marked fear of enclosed spaces ($N = 138$) were then administered four consecutive behavioral challenge tests involving exposure to a claustrophobia chamber (see below). Of those, 44.3% ($N = 60$) met severity criteria for inclusion based on their performance on the BATs as defined below, and 90.0% ($N = 54$) agreed to participate in the treatment phase of the experiment.

2.2. Design

Participants were randomized to one of three 30-min exposure conditions: (a) Heart-rate feedback (HRF), (b) paced tone control (PTC), and (c) exposure only control (EOC). Tripartite assessments consisting of self-reported fear, behavioral approach, and HR reactivity were obtained at pretreatment, posttreatment, and three-week follow-up.

2.3. Procedure

2.3.1. Screening

During the first stage of screening, potential participants provided fear ratings to two screening questions: (a) overall fear of closed-in spaces; and (b) fear associated with entering a

very small, pitch-dark, narrow closet and remaining there for several minutes. Those students responding with a 3 (moderate fear) or higher to each question were invited for further screening.

During the second stage of screening, potential participants were administered a series of behavioral approach tests to enclosed spaces (see below). They were deemed eligible for participation if on two BATs they were unable to remain in the chamber for two minutes *or* reported a SUDS level of 50 (moderate anxiety) or greater.

2.3.2. Behavior approach tests (BATs)

Two separate BATs were administered at each of the three major outcome assessments (pre, post, and follow-up). The major aim of the BATs was to assess participants' subjective, behavioral, and psychophysiological reactions while exposed to two different enclosed test sites – a long narrow corridor (BAT 1) and a small elevator (BAT 2).

2.3.2.1. Bat 1. Participants were instructed to enter a long, dark, observation corridor measuring 11.40 m (length), 0.57 m (width), and 2.29 m (height). At one end of the corridor was an unlocked closed door. At the other end was a brick wall illuminated by a small flash-light. A 5 cm by 7.5 cm rectangle painted with a 'glow-in-the-dark' paint was placed on the exit door next to the handle to assist the subject in exiting the chamber.

Prior to completing BAT 1, participants completed the Anxiety Sensitivity Index and the State-Trait Anxiety Inventory. Each participant was then fitted with a heart-rate unit by a female undergraduate research assistant. The ambulatory heart-rate monitor (UNIQ Heartwatch Model 8799, Computer Instruments) consists of an electrode belt worn around the chest. This monitor transmits heart-rate signals to a wrist receiver that depicts and stores the participant's heart-rate data. The unit also has a built-in event marker to record when participants entered and exited the BAT chamber. Each participant was instructed to sit quietly for five-minutes while resting heart-rate data were collected.

Following the five-minutes resting baseline, the door of the chamber was partially opened (approximately 30°) and the participant was instructed to look inside for five seconds. The participant was then told that they would be asked to enter the chamber several times. They were informed that the door would remain unlocked and they would be free to leave the room at any time, however participants were encouraged to remain in the chamber for as long as possible and for at least two minutes.

Instructions outlining specific exit procedures were provided. Participants were told that once they left the designated location within the chamber, they were to continue without stopping to the exit door and leave, even if upon approaching the exit door their discomfort/anxiety was reduced to a manageable level. Moreover, participants were reminded that the experimenter would open the door to signal the end of the trial. Although participants were encouraged to stay for two minutes, they were not provided specific information on the duration of the exposure trial.

Upon completing baseline assessment, the participant was instructed to enter the chamber and to walk to the end without stopping or looking back. As the participant walked into the chamber, the experimenter pressed the marker button on the heart-rate unit to record the beginning of the trial. Upon reaching the end of the corridor, the participant was instructed to

remain standing there for as long as possible and was reminded that the exit door was unlocked.

If the participant remained in the chamber for the full two minutes, the experimenter opened the door and instructed the participant to exit. When the participant exited the chamber, the experimenter depressed the marker button on the heart-rate unit to record the end of the trial and recorded the time of exposure (in seconds). Immediately upon exiting, the participant rated their maximum subjective fear level during the trial.

2.3.2.2. Bat 2. The BAT 2 chamber consisted of a small, unlit elevator, measuring 1.00 m (depth) by 1.20 m (width) by 2.29 m (height). Upon completing BAT 1, the research assistant led the participant to the BAT 2 chamber (elevator) and directed the participant to enter and to ride down one floor. Following BAT 2, participants completed a postexposure questionnaires similar to the one used after exposure to the BAT 1 chamber.

2.4. Treatment procedures common to all exposure conditions

Eligible participants were scheduled for treatment appointments from one to three weeks following screening. Treatment consisted of 30 min of self-directed exposure to the BAT 1 chamber over a two-hour period. The procedure for treatment was identical to the procedure for the behavioral screening trials, with three important exceptions: (a) treatment exposure trials lasted a maximum of five minutes as opposed to two minutes, (b) during treatment exposure trials, participants were free to move to any location within the chamber, as opposed to the BAT's where participants were required to remain at the back of the corridor, and (c) participants were strongly encouraged to remain in the chamber for as long as possible. Following completion of the post-exposure questionnaire, participants were then directed to conduct the next treatment trial, and so on, until 30 min of exposure had elapsed. Between six and twelve treatment trials for all participants were conducted, with a majority of participants (72.2%) requiring only six trials to complete the 30 min of self-directed exposure.

2.4.1. Heart-rate feedback condition (HRF)

Participants assigned to the HRF exposure condition had a small electronic speaker attached to their ambulatory heart-rate monitor. This speaker device made audible tones that were synchronized with the participants heart-rate. Participants were informed that these tones reflected their heart-rate, and were instructed to concentrate on these sounds during the treatment trials. These instructions were repeated before each treatment trial.

2.4.2. Paced tone control condition (PTC)

Participants assigned to the PTC exposure condition also wore a small electronic speaker next to their heart-rate monitors. This speaker emitted tones at a constant rate of 90 min^{-1}. Participants were informed that these were paced tones, and were instructed to concentrate on these sounds during the treatment trials. These instructions were repeated before each treatment trial.

2.4.3. Exposure-only control condition (EOC)

Participants assigned to the EOC condition received the same 30 min of self-directed exposure as in the HRF condition. However, participants were not presented additional stimuli or instructions regarding their focus of attention.

2.5. Measures

2.5.1. Peak subjective fear

Immediately upon exiting the BAT chamber, participants rated the peak level of fear while in the chamber on a 0 (no fear) to 100 (very severe) scale. This scale was administered during the two pre-treatment BATs, each treatment trial, the two post-treatment BATs, and the two follow-up BATs.

2.5.2. Ending subjective fear

Immediately upon exiting the BAT 1 chamber, participants rated on a 0 (no fear) to 100 (very severe) scale, the level of anxiety they experienced at the end of their exposure in the BAT 1 chamber. This scale was administered at each of the treatment trials and was used in the calculation of within-trial habituation.

2.5.3. Heart-rate reactivity

Each participant's resting heart-rate was measured every fifteen seconds during the five-minutes resting period. Baseline heart-rate was computed by averaging the participants' HR over the 5-min resting period. Each participant's heart-rate was also measured every fifteen seconds during each exposure trial and averaged to produce a single index. Heart-Rate Reactivity was defined as heart-rate during exposure minus baseline heart-rate, with negative numbers recoded to zero. These measures were taken during the two pre-treatment BATs, each treatment trial, the two post-treatment BATs, and the two follow-up BATs.

2.5.4. Coping self-efficacy

Immediately prior to entering the claustrophobia chamber, participants rated each of the four coping self-efficacy items on a 0 (not confident at all) to 100 (extremely confident) scale. This four-item scale showed an internal consistency coefficient of 0.92 in an earlier study (Valentiner, Telch, Petruzzi & Bolte, 1996).

2.5.5. Threat expectancies

Immediately prior to entering the claustrophobia chamber, participants rated on a 0 (no concern) to 100 (extreme concern) scale, four suffocation, four entrapment concern, and one expected anxiety item. The two four-item entrapment and suffocation concern scales showed internal consistency coefficients of 0.93 and 0.91, respectively, in an earlier study (Valentiner et al., 1996).

3. Statistical analyses

To confirm that the randomization procedure resulted in comparable exposure condition groups, we examined group differences in pre-treatment levels of subjective fear and heart-rate reactivity using one-way ANOVAs. No significant differences between conditions were observed for subjective fear or heart-rate reactivity for either BAT 1 or BAT 2.

3.1. Treatment outcome

Planned multivariate contrasts (HRF vs. PTC and EOC) adjusting for pretreatment levels were performed on posttreatment fear indices (i.e., subjective fear and HR reactivity) to test the hypothesis that the HRF group would show greater fear reduction at posttreatment (Hypothesis 1) and less return of fear (Hypothesis 5) than either the EOC or PTC group. Considering the directional nature of our hypotheses, one-tailed significance tests were used in these analyses.

We also examined clinically significant change across exposure conditions. Participants were classified as achieving clinically significant change at posttreatment if they could remain in the corridor test chamber for two min with a SUDS level less than 50. A χ^2 test comparing the HRF group to the EOC and PTC groups was used as a further test of Hypothesis 1.

3.2. Treatment process

A growth curve approach (Francis, Fletcher, Stuebing, Davidson & Thompson, 1991; Willett, Ayoub & Robinson, 1991) was employed to test hypotheses concerning the differential effects of exposure condition on initial fear and fear change across treatment trials (i.e., Hypotheses 2 and 3). We refer to this approach as decay modeling to reflect the expected decrement in fear ratings during treatment trials. The first step of this approach involves modeling data within-subjects. Accordingly, a simple linear regression was calculated for each participant using subjective fear as the dependent variable, and treatment trial (numbered 0–5) as the independent variable. These analyses produced two parameters for each participant: (1) initial fear level, which corresponds to the intercept parameter in the within-subject regression model, and (2) fear change, which corresponds to the slope parameter in the within-subject regression model and is an estimate of the amount of change in subjective fear associated with each treatment trial.

The second step of the decay modeling approach involves testing hypotheses using these within-subject parameters as dependent variables in traditional between-subjects analyses. Thus, we performed an ANOVA using the initial fear parameter estimates from the within-subject analyses as the dependent variable, exposure condition as the group factor, and pre-treatment BAT 1 subjective fear as a covariate. Again, a planned contrast (i.e., HRF versus EOC and PTC) was performed to test the hypothesis that the provision of heart-rate feedback would result in higher levels of initial subjective fear (Hypothesis 2). This analysis was repeated using the fear change parameter estimates obtained from the within-subject modeling analyses in order to test the hypothesis that the provision of heart-rate feedback would results in greater change (i.e., reduction) in subjective fear across treatment trials (Hypothesis 3). Considering

the directional nature of our hypotheses, one-tailed significance tests were used in these analyses.

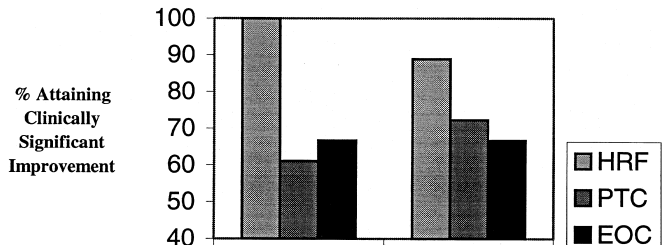
To examine changes in fear during treatment trials, twelve indices were calculated: the percent change in subjective fear during each treatment trial, and the percent change in heart-rate reactivity during each treatment trial. The percent change in subjective fear was defined as the difference between peak subjective fear and ending subjective fear, divided by maximum subjective fear. In the few instances when maximum subjective fear was zero, the percent change in subjective fear was coded to zero. The percent change in heart-rate was defined as the difference between heart-rate during the first and last minute of the treatment trials, divided by heart-rate during the first minute of the treatment trial. These indices were then entered as dependent variables in repeated-measure ANOVAs, with treatment trial (numbered 1–6) as the within-subject factor and exposure condition as the between-subjects factor. A planned contrast comparing the HRF to the EOC and PTC conditions was used to test the hypothesis that the provision of heart-rate feedback would result in greater within-trial habituation in subjective fear and heart-rate (Hypothesis 4).

4. Results

4.1. Effects at posttreatment

Means and standard deviations of subjective fear and HR reactivity at the posttreatment and follow-up assessments for the three exposure conditions are reported in Table 1. Consistent with prediction, the HRF condition showed significantly less subjective fear at posttreatment across the two BATs ($F_{(1,51)}=6.52$, $p < 0.01$). Subsequent univariate analyses for each of the two BATs were significant for BAT 1 ($F_{(1,51)}=11.80$, $p < 0.001$) but only approached significance for BAT 2 ($F_{(1,51)}=1.87$, $p < 0.09$). Analyses of heart-rate reactivity were in the predicted direction but were not significant for either BAT 1 or BAT 2.

Fig. 1 presents the percentage of participants in each of the exposure conditions who attained clinically significant improvement at post-treatment and follow-up assessments.



	Posttreatment	FU
HRF	100	88.9
PTC	61.1	72.2
EOC	66.7	66.7

Fig. 1. Percentage of participants attaining clinically significant improvement for the three exposure conditions.

Table 1
Group means and standard deviations for the primary outcome measures at pre, post, and follow-up assessments. BAT 1 was conducted in a long narrow corridor, BAT 2 was conducted in a small elevator. HRF = Heart-rate Feedback, EOC = Exposure Only Control, PTC = Paced Tone Control. Due to missing data, some cells are based on *N*'s as low as 17, and as high as 18

Measure	Exposure condition								
	Heart-rate feedback			Paced-tone control			Exposure only control		
	Pre	Post	FU	Pre	Post	FU	Pre	Post	FU
BAT 1 – Corridor									
Peak fear (0–100)									
Mean	73.3	19.4	18.8	71.1	34.4	34.4	67.2	36.1	33.3
S.D.	15.7	15.5	20.6	15.7	25.3	29.6	17.8	20.3	21.4
HR reactivity									
Mean	14.4	10.4	11.1	12.5	13.2	11.6	10.3	11.9	
S.D.	10.2	5.5	4.3	5.7	4.3	5.9	7.1	5.5	6.0
BAT 2 – Elevator									
Peak fear (0–100)									
Mean	43.9	17.8	12.2	55.6	28.3	27.1	49.9	32.2	22.8
S.D.	22.4	19.0	19.9	17.2	27.1	24.4	20.7	19.9	18.1
HR reactivity									
Mean	5.6	6.7	8.5	10.6	9.3	9.2	9.4	7.9	12.3
S.D.	5.3	6.3	7.9	5.9	5.5	4.6	5.3	4.4	5.6

Consistent with hypothesis 1, a higher percentage of participants in the HRF condition met criteria for clinically significant change at posttreatment relative to participants in the PTC and EOC conditions ($X^2(1) = 8.56$, $p < 0.01$).

4.2. Effects at follow-up

A similar pattern of findings emerged at the follow-up assessment. Participants in the HRF condition displayed significantly lower subjective fear ratings across the two BATs relative to the other two conditions $F_{(1,48)} = 7.25$, $p < 0.01$. Follow-up univariate contrasts performed on each BAT separately revealed a significant advantage for the HRF group on subjective fear on BAT 1 ($F_{(1,48)} = 10.28$, $p < 0.01$) and a non-significant trend in favor of the HRF group on BAT 2 ($F_{(1,49)} = 2.31$, $p < 0.09$). Analyses of heart-rate reactivity at follow-up were in the predicted direction but were not significant for either BAT 1 or BAT 2.

A higher percentage of participants in the HRF condition met criteria for clinically significant change at follow-up. However, this difference was not significant ($X^2(1) = 2.48$, $p < 0.06$).

4.3. Treatment process

4.3.1. Fear activation

Means and standard deviations of subjective fear for each exposure condition at each of the six exposure trials are presented in Table 2. Also presented in Table 2 are the means and standard deviations of the initial fear and fear change parameter estimates for the three exposure conditions. Decay lines based on the estimates of the within-subjects decay parameters for each of the three conditions are presented in Fig. 2.

The planned contrasts comparing the HRF to the EOC and PTC groups on initial subjective fear and heart-rate reactivity were not significant. These results fail to support the hypothesis that the provision of heart-rate feedback would result in higher levels of initial fear activation (Hypothesis 2).

To further examine the relationship between fear activation during treatment and treatment outcome, partial correlation analyses were conducted using HR reactivity during the first treatment trial as an index of fear activation. Contrary to prediction, HR reactivity during the first 5 min of treatment was associated with greater subjective fear during the posttreatment BAT 1 even after controlling for subjective fear at pretreatment (par $r = 0.36$; $p < 0.01$). A similar pattern was observed for BAT 2 posttreatment fear (par $r = 0.31$; $p < 0.05$).

4.3.2. Fear change within-trials

The means and standard deviations for within-trial changes in subjective fear during the six treatment trials are presented in Table 3. The planned contrasts comparing the HRF to the EOC and PTC conditions on within-trial change across the 30-mm of self-directed exposure in subjective fear and HR reactivity were not significant, although there was a non-significant

Table 2
Means and standard deviations of subjective peak fear indices and growth curve parameters across the six treatment trials. Each treatment trial consisted of 5 min of exposure to the test chamber

	Condition					
	Heart-rate feedback		Paced-tone control		Exposure only control	
	M	S.D.	M	S.D.	M	S.D.
Treatment trial						
1	53.9	17.5	56.1	25.7	63.9	10.9
2	48.3	19.5	51.7	27.3	55.0	14.7
3	40.0	20.0	44.4	30.7	50.0	20.0
4	27.2	21.1	40.0	34.3	46.1	20.0
5	20.0	20.0	40.0	35.8	36.5	21.2
6	15.0	19.5	35.0	33.8	32.4	20.8
Growth curve parameters						
Initial fear activation	55.0	17.2	54.9	26.6	63.0	14.3
Between-trial fear decline	8.4	4.5	4.1	5.8	6.2	4.1

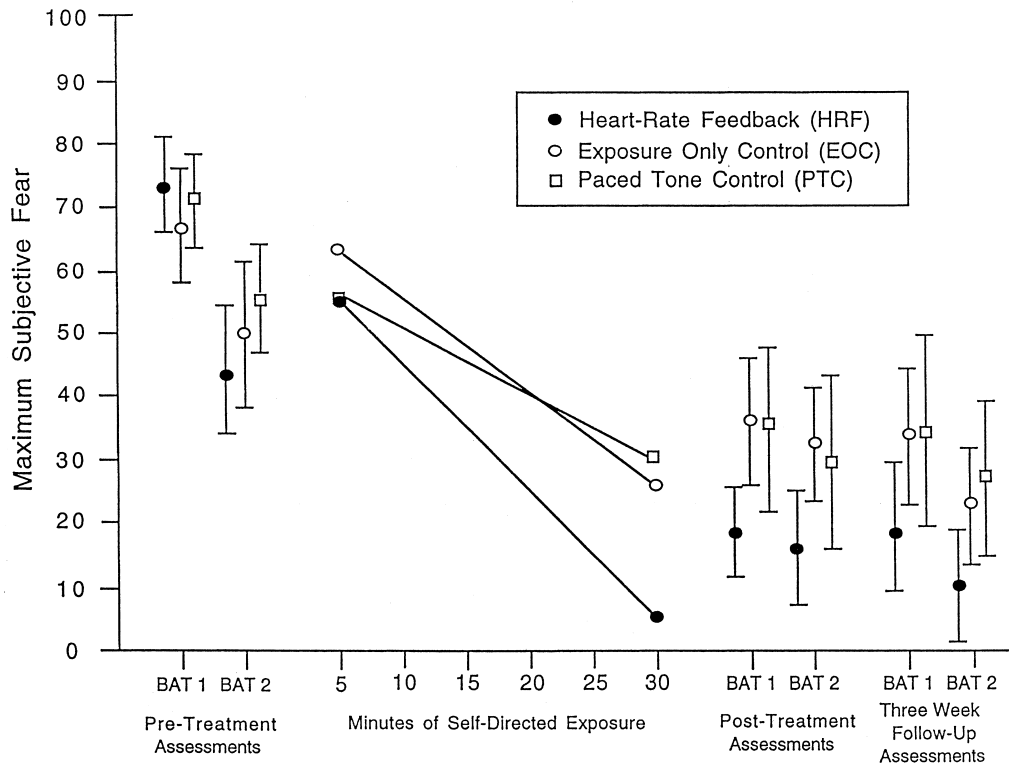


Fig. 2. Mean ratings of maximum subjective fear from two behavioral approach tests at pre-treatment, post-treatment, and three-week follow-up, and decay lines of maximum subjective fear during treatment for the three exposure conditions.

Table 3
Mean within-trial change in subjective peak fear across the six treatment trials. Each treatment trial consisted of 5 min of exposure to the test chamber. Percent change in subjective fear was defined as peak fear minus ending fear divided by peak fear, each of which were rated on a 100-point SUDS scale

Treatment trial	Condition					
	Heart-rate feedback		Paced-tone control		Exposure only control	
	M	S.D.	M	S.D.	M	S.D.
1	24.4	32.2	12.4	20.0	16.7	24.9
2	29.5	30.6	17.9	20.6	16.4	22.7
3	25.9	31.5	18.1	25.0	17.6	20.7
4	19.4	38.0	12.7	26.9	12.0	24.7
5	15.5	33.4	15.6	26.9	14.8	26.4
6	12.0	26.7	6.9	14.5	13.6	27.2

trend in the predicted direction for subjective fear ($p < 0.15$). However, closer inspection of the pattern of within trial changes in subjective fear showed significantly greater within-trial change during the first three treatment trials for the HRF condition relative to the EOC and PTC conditions $t(52) = 1.70$, $p < 0.05$. These results provide partial support for the hypothesis that the provision of heart-rate feedback would result in greater within-trial habituation (hypothesis 3).

4.3.3. Fear change across trials

Consistent with prediction, participants in the HRF condition showed significantly greater fear change across treatment trials relative to participants in the PTC and EOC conditions $t(50) = 2.25$, $p < 0.05$ (see Table 2 and Fig. 2). These results support the hypothesis that the provision of heart-rate feedback would result in greater between-trial habituation (Hypothesis 4).

5. Discussion

This experiment sought to examine several predictions derived from the Foa and Kozak (1986) emotional processing theory of fear reduction. Our general approach was to introduce an experimental manipulation designed to facilitate emotional processing by providing participants information incompatible with the response and meaning propositions presumed to make up the current claustrophobia fear structure. We selected heart-rate feedback as a suitable manipulation based on our previous work showing a consistent pattern of heart-rate decline during a 30-min period of self-guided exposure to a claustrophobia chamber. We reasoned that providing claustrophobics salient information concerning their heart-rate decline would in theory satisfy one of the necessary conditions for emotional processing proposed by Foa and Kozak, namely providing information *incompatible* with some of the propositions that make up the fear structure.

Results provided some support for the major predictions of the theory. First, the provision of heart-rate feedback during exposure led to significantly lower levels of claustrophobic fear at posttreatment and a significantly higher proportion of participants at posttreatment who met our criteria for clinically significant change. As expected, the facilitative effect of heart-rate feedback on fear reduction was more pronounced for the corridor (chamber where treatment occurred) than for the elevator (chamber for testing treatment generalization).

Our analyses provide some clues as to the theory-relevant process changes that occur during treatment. Foa and Kozak (1986) propose that activation of the fear structure is a necessary condition for emotional processing to occur. Our findings suggest that each of the three exposure conditions were able to activate participants' fears. To test whether the superior fear reduction achieved by the HRF group was due to greater fear activation during treatment, we examined the relationship between initial heart-rate reactivity and treatment outcome as well as the relationship between initial subjective fear and treatment outcome. We found no evidence to support the hypothesis that the greater fear-reduction evidenced by the HRF group was due to higher levels of initial fear activation. On the contrary, greater fear activation during the

first 5 min of treatment was associated with a poorer outcome (i.e., greater fear at posttreatment).

In addition to initial fear activation, we examined the degree of within-trial habituation as a function of treatment condition. Overall, participants receiving heart-rate feedback showed no greater level of within-trial habituation relative to those in the two control conditions ($p > 0.10$). However, closer inspection of the pattern of change did reveal greater within-trial change across the first 15-min (3 trials) of treatment among those receiving heart rate feedback. This finding suggests that the facilitative effect of heart rate feedback on within-trial fear reduction may occur when fear levels are still relatively high.

Treatment process analyses examining between-group differences in subjective fear *across* treatment trials revealed a pattern of findings suggesting superior between-trial habituation for participants receiving heart-rate feedback. How might the provision of heart-rate feedback during exposure facilitate between-trial habituation? Put in the framework of emotional processing theory, heart-rate feedback serves as incompatible information that would facilitate the dissociation between the stimulus elements (i.e., features of the claustrophobic chamber) and response elements (i.e., elevated heart rate). In addition, receiving information concerning heart-rate decline provides disconfirmatory evidence that the enclosed space poses a threat (since why would one's heart-rate decline if one were in danger). It also provides disconfirmatory information that one's fear while encountering the phobic situation is unmanageable and will persist indefinitely. To the extent that this disconfirming information is cognitively processed, one would expect greater between-trial habituation.

Other possible mechanisms for the facilitative effect of heart-rate feedback warrant consideration. One possibility is that the heart-rate feedback served as a cue for participants to engage in a self-guided 'biofeedback' process. To the extent that participants were successful in using the feedback to lower their heart-rate, the superior fear reduction achieved by this group could be explained by an enhanced lowering of their autonomic reaction relative to the other two groups. Our heart-rate data fail to support the hypothesis that participants used the heart-rate feedback to lower their heart-rate. Specifically, process analyses examining changes in heart-rate reactivity within treatment trials, showed negligible differences between the HRF group and the two exposure control conditions.

Alternatively, receiving heart-rate feedback may have enhanced participants' perceived efficacy to cope with their arousal in the chamber. We have recently shown that changes in coping self-efficacy during treatment predicts claustrophobia fear reduction even after controlling for danger expectancies and anxiety expectancies; whereas neither danger expectancies nor anxiety expectancies predict fear reduction after controlling for self-efficacy (Valentiner et al., 1996). It should be noted that the predictive significance of coping appraisals in fear reduction is not necessarily at odds with emotional processing theory. Perceptions of one's capacity to manage physiological arousal, fearful ideation, and behavioral action tendencies for escape may be construed as relevant 'meaning propositions' in the emotional processing framework. To test whether the differences between groups in between-trial habituation could be accounted for by differential changes in self-efficacy over the course of treatment, we subjected self-efficacy growth curve parameters to a between groups ANOVA. These analyses revealed similar changes in coping self-efficacy between the three exposure

conditions. Hence, these findings suggest that the greater fear reduction achieved in the HR feedback condition cannot be accounted for by differential changes in coping self-efficacy.

Alternatively, it is possible that the HR feedback achieved its facilitative effect on fear reduction by shifting participants attention away from the threatening elements of the chamber. There are several good reasons to suspect that this was not the case. First, studies that have examined the effects of distraction on fear reduction reveal an interference effect rather than a facilitative effect (Telch et al., 1990; Rodriguez & Craske, 1993). Second, to control for the effects of distraction, we included a paced tone control. If distraction was operating to facilitate fear reduction, participants instructed to focus on the tones would also be expected to show greater fear reduction.

These considerations lead us to the conclusion that HR feedback facilitates fear reduction by providing information that is incompatible with threat perception. This disconfirmation hypothesis should be viewed as tentative. Our experiment did not include a false heart-rate feedback condition. Thus, we cannot rule out the possibility that HR feedback facilitates fear reduction through some other mechanism other than threat disconfirmation. A more powerful test of the disconfirmation hypothesis would have been to also provide false feedback leading participants to believe that their HR was actually increasing over time. This added control would have provided useful information as to whether it was the nature of the HR feedback as opposed to the mere *focusing* on one's presumed heart rate that facilitates fear reduction during self-guided in vivo exposure.

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