Conditional and Unconditional Components of Post-Shock Freezing

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Abstract—Rats received shocks in one apparatus, and post-shock "freezing" was then assessed in that apparatus or in a different one. The assessment of freezing was made immediately after shock or after a 24-hour delay. Post-shock freezing was reduced when the animals were tested in a different apparatus from that in which shocks had been administered. No reduction in freezing was caused by the 24-hour delay. All the post-shock freezing was therefore attributable to contextual cues and to generalization between contexts. This pattern of results suggests that post-shock freezing is entirely produced by conditioned fear elicited by cues associated with shock and that no part of post-shock freezing is an unconditional response (UR) directly elicited by shock.

WHEN A RAT RECEIVES occasional electric shocks in some situation, ongoing behaviors are suppressed (Myer 1971), the rat becomes immobile or "freezes," (Fanselow and Bolles 1979a, Miller and Weiss 1969) and it tends to assume a characteristic "crouching" posture (Blanchard and Blanchard 1969). This immobility may be functional in a natural setting, inasmuch as a predatory cat is much less likely to attack a small motionless rodent than one that is moving (Hirsch 1977). Freezing may be thought of as one of the rat's species-specific defense reactions (Bolles 1970).

What is the immediate source of freezing behavior? Is it an unconditional response (UR) to the unconditional stimulus (US) of the shock? This idea would require some adjustment of the usual view of a UR as an immediate, short-lived reflex, because freezing is not like the usual sudden jerk UR that is invariably elicited by shock. The occurrence of freezing is probabilistic, freezing has a delayed onset, and it occurs in prolonged bouts lasting several minutes (Bolles and Riley 1973, Fanselow and Bolles 1979a). These probabilistic and temporal properties set the freezing response somewhat apart from the usual UR.

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Another alternative is that freezing is an instrumental response controlled by reinforcing consequences (*i.e.*, freezing somehow modifies the shock's delivery or impact). This possibility seems unlikely. Bolles and Riley (1973) showed that freezing is controlled by the schedule of delivery of shock and not by the programmed contingencies between the behavior and the shock. Comparisons with results of tests with yoked controls showed that contingencies of avoidance and punishment affected freezing only to the extent that these contingencies altered the programmed delivery of shock. In addition, the finding that a modest amount of freezing follows even a single 0.75-second shock (Fanselow and Bolles 1979a) indicates that freezing is acquired too rapidly to be an instrumental response.

A third alternative is that freezing is a conditional response (CR) that is produced by fear elicited by cues that predict painful stimulation, even if it is not a UR to shock. This would require an adjustment in the commonly held view (Hilgard and Marquis 1940, Jenkins and Moore 1973, Pavlov 1927) that a CR is some replica or component of the UR. There is some evidence to justify this view. When rats are given shock in one situation and then observed in another situation where shock has never been presented, they evidence much less crouching and freezing than do animals that are shocked and then observed in the same situation (Blanchard and Blanchard 1969, Bolles and Collier 1976). Thus, it appears that an appreciable part of freezing behavior has the status of a CR, since it depends in an important way upon

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the presence of contextual cues that have been correlated with the delivery of shocks.

However, Bolles and Collier also found that rats shocked and then switched to a novel nonshock observational arena evidenced more freezing than did nonshocked controls, which suggests that at least some freezing may be a delayed and prolonged UR to shock. On the other hand, it is conceivable that the post-shock freezing reported by Bolles and Collier was not actually elicited by the prior shock and did not result from some aroused "state" of the rats, but was due to generalization. That is, it is possible that freezing was seen in the novel test situation because this situation was sufficiently similar to the shock situation to elicit freezing as a generalized CR. Bolles and Collier's finding that freezing was reduced but not eliminated by shifting the animals to an observational arena differs from the results of Blanchard and Blanchard who found that when previously shocked rats were observed in the nonshocked situation, freezing was reduced to the level for control animals that had never been shocked. If the results of Blanchard and Blanchard are considered in the light of Bolles and Collier's finding that the physical construction of the test situation is a determinant of the level of freezing, then Blanchard and Blanchard's failure to counterbalance their shock and nonshock chambers leaves it ambiguous whether the observed reduction in freezing was due to the absence of an unconditional component of freezing, to the absence of generalization of the conditional component of freezing, or to the fact that the wire-mesh nonshock observational arena offered less stimulus support for freezing.

Thus, the question of whether or not some component of freezing is a delayed UR to shock remains unsettled. The present experiment addressed this question in another way. In addition to switching some animals to a novel, nonshock-related context for testing, as Blanchard and Blanchard and Bolles and Collier had done, some animals were also tested for the incidence of freezing following a 24-hour interval after delivery of shock. This interval should be sufficiently long to permit the dissipation of all US-elicited effects and all aftereffects of US presentation without materially reducing the effectiveness of the CS in eliciting a CR. That is, following a 24-hour delay, URs should be lost, but CRs should not be. This manipulation of delay would allow an assay of the strength of the unconditional component of freezing (freezing directly elicited by shock) that was independent of the strength of the conditional component of freezing. To assure the generality of the findings, two different US intensities were used.

Methods

Subjects

Subjects were 48 female rats of Long Evans descent, raised in the University of Washington's Psychology Department colony. The rats were between 100 and 106 days old at the time of the experiment. The animals were each handled for 1 minute/day for 2–6 days before the experiment. Free access to food and to water was provided in the individual housing cages. The experiment was conducted during the light portion of a 12 hour: 12 hour day: night cycle.

Apparatus

Two observational chambers were used. *Chamber A* was 26 cm long, 23 cm wide, and 24 cm high. One side (26 cm wide) and the top were clear plastic. The remaining walls were stainless steel. The grid floor was made of 13 stainless steel rods, 1 cm in diameter and spaced 2 cm apart center-to-center. Illumination was provided by a 7.5-watt red light bulb suspended 5 cm above the ceiling. *Chamber A* and its catch tray, 4.5 cm below the grid floor, were cleaned with a solution of 2.5% detergent (Vestal, 1-Stroke Ves-Phen) and water between periods of housing rats.

Chamber B was 23.5 cm long, 23 cm deep, and 24 cm high. It also had a clear plastic wall (23.5 cm wide) and top, the remaining walls being aluminum. The floor was composed of 11 aluminum rods, 1 cm in diameter, placed 2 cm apart center-to-center. Illumination was provided by a 10-watt white light bulb placed 2 cm above the ceiling. *Chamber B* and its catch tray, 3.5 cm below the grid floor, were cleaned with a solution of 25% vinegar and water between periods of housing rats.

Both chambers were placed inside of sound-attenuating chambers on opposite sides of the experimental room. The most salient differences between the two chambers, then, were in illumination (the irradiance at the plane of measurement, when a detector with cosine-angle sensitivity was pointed at a metal sidewall adjacent to the clear plastic wall, was 8.37 erg/sec/cm^2 in *Chamber A* and 9.5 erg/sec/cm^2 in *Chamber B*), smell (due to different cleaning solutions), and spatial location in the testing room. Pilot work indicated that these two chambers were discriminable but provided comparable stimulus support for freezing (*cf.* Bolles and Collier 1976).

A 0.75-second electric shock was delivered by a Grason Stadler shock generator/scrambler wired to each rod of the grid floor. Resistance between rods was checked for each animal to

TABLE 1. Mean Percentages of Freezing and Standard Errors of the Means for Different Groups*

Intensity of Shock	*Place*	*Time*	*Mean*	*SEM*
0	Same	Immediately	1	1
		Delayed	1	0
	Different	Immediate	1	0
		Delayed	1	1
0.5 ma	Same	Immediate	29	6
		Delayed	32	4
	Different	Immediate	14	8
		Delayed	3	2
1 ma	Same	Immediate	41	11
		Delayed	63	4
	Different	Immediate	10	4
		Delayed	21	3

* As a function of intensity of shock, whether testing was done in the place of shock or in a different place, and whether testing followed immediately after shock or 24 hours later.

insure that the cleaning solution, feces, or urine did not short out any rod combinations.

Procedure

Four rats were assigned to each of the 12 cells of a 3 × 2 × 2 design, the factors being shock intensity (1.0 ma, 0.5 ma, or no shock), delay (whether observation was immediately after shock or after a 24-hour delay) and place (whether observation was made in the same apparatus as delivery of shock or in a different apparatus). For half the animals of each cell, the observation took place in *Chamber A*, and for the other half, the observation took place in *Chamber B*.

A rat was placed in one of the chambers and 2 minutes later was given four shocks at the appropriate intensity. The shocks were spaced 20 seconds apart. Thirty seconds after the final shock (or an equivalent waiting period in the no-shock groups), the rat was removed from the chamber. If it was to be tested that day, depending on its group assignment, it was either immediately returned to the chamber where the shocks had been given or put in the other chamber for the observational period. If the rat was to be tested the following day, it was returned to its home cage for 24 hours and then placed in the appropriate chamber for the observational period. The design is summarized in Table 1.

During the observational period, which lasted 8 minutes, no stimuli were presented. Behavior was recorded using a time-sampling procedure. Every 4.6 seconds, the behavior that the animal was currently engaged in was classified as either *freezing* or *activity*. Freezing was defined as the absence of all observable movement of the skeleton and the vibrissae, except for those related to respiration. All other behavior was scored as activity.

Results

For each animal, the percentage of behavioral samples judged as freezing was determined. An analysis of variance indicated that the mean percentage of freezing was similar for observations in *Chamber A* and in *Chamber B* ($F(1, 24) < 1$), so the data were collapsed across that variable. The mean percentage of freezing and the standard error of the mean for each group are presented in Table 1. These scores were subjected to a 3 × 2 × 2 factorial analysis of variance.

Conditional Component of Freezing

The rats froze more when tested in the same location where they had been shocked than when tested in a different place ($F(1, 36) = 45.0$, $P < 0.005$). This indicates that there was a reliable conditional component of freezing. As the intensity of shocks is increased, more fear should

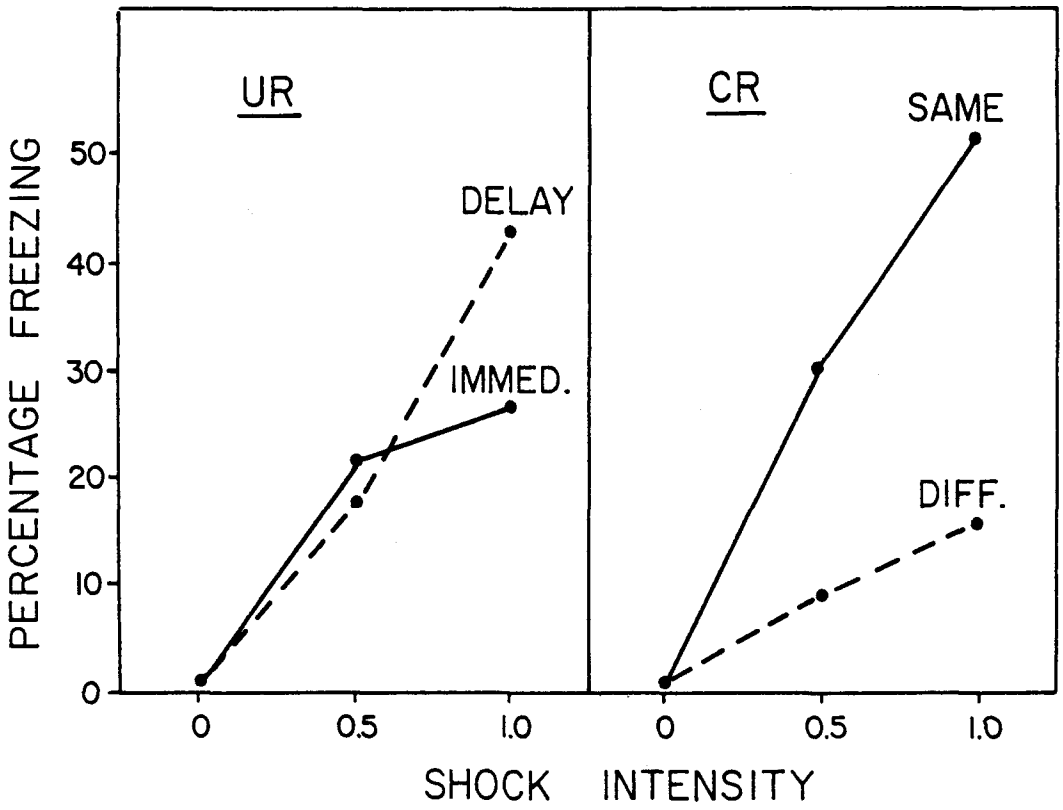

FIG. 1. Data from Table 1 sorted out to reveal the unconditional component of freezing (which would be displayed by a superiority of Immediate over Delayed scores) and the conditional component of freezing (which is shown by the superiority of Same over Different scores).

be conditioned to contextual stimuli, and this should produce more freezing. Therefore, we would expect a greater difference in responding between animals tested in the same versus a different place. A reliable place - by - intensity - of - shock interaction ($F(2, 36) = 14.0$, $P < 0.005$) indicated that this was the case. The data are summarized in the right panel of Figure 1.

Generalization

The above analysis indicates that freezing is a result of fear conditioned to contextual stimuli. It might then be expected that conditional fear would generalize to the different context to the extent that this context has features similar to those of the place where the rats have been shocked. Such a generalized fear component of freezing would be indicated by parallel functions of intensity of shock for freezing in the same and in different contexts. If generalized fear is associative, the amount of freezing due to generalized fear should show the same pattern in results for each group, regardless of whether tests were made immediately after shock or after the 24-hour delay. This pattern of results was indicated by a significant main effect for shock intensity ($F(2, 36) = 44.0$, $P < 0.005$) and by the absence of a place × delay × shock intensity interaction ($F(2, 36) < 1$).

Unconditional Component of Freezing

If shock elicited freezing directly, one would expect more freezing to occur immediately after shock than on the next day. As can be seen in the left panel of Figure 1, this was not the case. The main effect for delay was not reliable ($F(1, 36) = 2.0$, $P < 0.10$), and the trend was in the wrong direction.

As shock intensity increases, one might expect a greater unconditional component of freezing to occur. Therefore, greater differences in freezing behavior between animals tested immediately after shock and animals tested 24 hours after shock would be expected at higher intensities of shock. Although there was a reliable shock intensity × delay interaction ($F(2, 36) = 5.0$, $P <$

0.025), the pattern of results was opposite from what would be expected if there were an unconditional component of freezing. The rats that received the strongest shock froze more after the delay than immediately after shock ($F(1, 36) = 10.4$, $P < 0.005$).

Discussion

The present study provided no evidence that post-shock freezing is directly elicited by shock. Rather, the present data, like those of Blanchard and Blanchard (1969), indicate that post-shock freezing is a result of contextual stimuli that have been paired with shock. In the nonshock condition, the freezing that did occur appeared to have been due to generalization of fear between the contexts. Similar between-context generalization of fear was probably responsible for the findings of Bolles and Collier (1976) that more freezing is shown by animals that are moved to a different place after being shocked than by nonshocked controls.

If freezing is a conditional response to cues associated with shock, but is not an unconditional response to the shock itself, we need to explain why shock conditions freezing to these cues. The traditional views of Pavlovian conditioning, in which the CR is always considered to be some component or replica of the UR, will not do as an explanation of freezing, nor is this position any longer tenable for Pavlovian conditioning in general (Dickinson and Mackintosh 1978).

One alternative is that the UR does determine the nature of the CR, but that the effect of the CR is in the opposite direction to that of the UR (*e.g.*, Schull 1979, Siegel 1977). This view assumes that the CR is a compensatory response that serves to minimize deviations from some homeostatic norm (the UR) caused by the US. Support for this view comes predominantly from studies of morphine tolerance (*e.g.*, Siegel, Hinsen, and Crank 1978). The unconditional reaction to morphine is a loss of sensitivity to pain, but the conditional reaction to signals that predict administration of morphine is an increase in the sensitivity to pain (Siegel 1975). This compensatory-response view of Pavlovian conditioning has been very successful as a model of drug tolerance—tolerance is the cancelling of the UR produced by the compensatory CR (Siegel 1979). This model has also been successfully applied to other phenomena of aversive conditioning—shock causes pain, but the reaction to signals that predict shock appears to be an analgesia that minimizes the pain of the signaled shock (Fanselow 1979, Fanselow and Bolles 1979b). Consistent with such a view, shock elicits an increase in activity, whereas the CR to stimuli paired with shock is a decrease in activity (*i.e.*, increased freezing). Whether freezing is a compensatory response, in Siegel's sense that it minimizes the activity caused by shock, remains to be demonstrated. However, the finding that frightening stimuli (which presumably cause freezing) potentiate startle responses (Brown, Kalish, and Farber 1951) does not support the notion that freezing serves to diminish the activity caused by a subsequent US.

An alternative explanation of the production of freezing is offered by the Perceptual–Defensive–Recuperative model of fear and pain (Bolles and Fanselow 1980, Fanselow and Bolles 1979b). This model states that stimuli associated with noxious events will produce fear. Fear guarantees that the animal's behavioral repertoire will be limited to species-specific defense reactions (SSDRs), but fear itself does not determine which particular SSDR will occur. The selection of particular SSDRs is a function of the structure of the test environment. For example, contextual cues associated with shock will produce freezing in a small squarish chamber, like those used here, but when the same shock is given in an elongated chamber, much of the freezing will be replaced by attempts at flight (Bolles and Collier 1976). If shock is delivered through a localizable source (the CS is the source of shock) and there is bedding material available, the rat will neither freeze nor flee but will bury the source of the shock (Pinel and Treit 1979).

This Perceptual–Defensive–Recuperative model suggests that the CR bears no relationship to the UR and, in fact, serves an entirely different function (Bolles and Fanselow 1980). Whereas the CRs to stimuli which predict noxious events are integrated and complex defense behaviors (*i.e.*, SSDRs), the URs to the noxious stimulation itself are reflexes of withdrawal and, if there is some persistent tissue damage, recuperative behaviors which promote healing (Bolles and Fanselow 1980, Wall 1979).

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