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ALTERATION OF CLASSICALLY CONDITIONED HEART RATE BY OPERANT REINFORCEMENT IN MONKEYS[1]

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Monkeys were run alternately on classical conditioning and on operant heart rate training schedules. The classical unconditioned stimulus was identical to the operant negative reinforcement. After operant training, some subjects changed their heart rate responses to the classically conditioned stimulus. When both the operant and the classical schedules were in force simultaneously, all subjects changed their previous heart rate responses to the classically conditioned stimulus without significantly changing their blood pressure responses to this stimulus. The changes in heart rate response to the conditioned stimulus sometimes persisted long after the operant schedules were no longer in force. These results show that a classically conditioned response can be altered by operant reinforcement, and they suggest that the classical unconditioned stimulus actually may be an operant reinforcer.

Most definitions of classical conditioning state that the conditioned response (CR) is innately determined by the nature of the unconditioned stimulus (US). Although most authors have abandoned literal response-substitution theories because of the differences between the CR and the unconditioned response (UR), they still imply that each CR has a one-to-one relationship with each corresponding US (Kimble, 1961, pp. 78–108; Rescorla & Solomon, 1967, p. 156 ff.). Perkins (1955) has suggested that CRs are governed, like operant responses, by the differential reinforcement that follows them, a hypothesis also implicit in Zener's view (1937) that CRs are attempts to prepare for the US. If this last view is true, it should be possible to abolish CRs or substitute new ones by offering differential reinforcements greater than those inherent in the original US. However, the lack of responses that could be brought under the control of both operant reinforcement and USs has tended to restrict research on this question to indirect methods (e.g., Tighe, Graves, & Riley, 1968).

An exception has been the conditioning of skeletal muscle responses, such as eyeblink or limb withdrawal, to cues predicting noxious stimuli. This has been held to be true classical conditioning because the CRs have different time courses from analogous voluntary responses (Spence & Ross, 1959). Instructions not to respond, which presumably act through some kind of operant process, reduce the incidence and amplitude and increase the latency of these responses to some extent but do not abolish them (Hilgard & Humphreys, 1938; Nicholls & Kimble, 1964). Punishment for making the CR has little effect (Norris & Grant, 1948). The small effect of operant reinforcement reported in these experiments may be due to an unlucky choice of the time interval between the conditioned stimulus (CS) and US. Ominsky (1968) has shown that the amount of voluntary inhibition obtained can be augmented greatly by increasing the time interval between the CS and US.

A greater difficulty with these experiments is that they may not pit the operant and classical learning processes against each other. Where the incentive to modify the CR can be specified at all it is a new kind of stimulus, which might act or fail to

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act because of its own classical conditioning effect rather than because of operant reinforcement as in Norris and Grant (1948). Furthermore, the distinction between conditioned and voluntary eye blink responses drawn by Spence and Ross (1959) does not seem to be a hard and fast one. Although instructions to subjects not to respond "voluntarily" increase the latency of responses compared with instructions to avoid the US, response latencies produced by these 2 kinds of instructions overlap (Gormezano & Moore, 1962). The long-latency as well as short-latency responses may be attempts to avoid the noxious US and, therefore, are not CRs as usually defined. Thus, it remains a question whether CRs can be modified by operant reinforcement.

The recent discovery that animals and humans can learn to emit autonomically mediated responses as operants (Engel, 1972; Miller, 1969) both increases the significance of the question and provides a means to answer it. But to our knowledge, there has been no direct attempt to alter conditioned autonomic responses by operant reinforcement.

This study asks whether operant learning could modify monkeys' cardiac responses to impending electric shock. This response has generally been reported to be an increase in heart rate (HR), often followed by a decrease which may reach or go below baseline level as shock approaches (Smith & Stebbins, 1965; Snapper, Pomerleau, & Schoenfeld, 1969; Stebbins & Smith, 1964). In humans this decrease is nearly abolished by breath holding, which has led to the suggestion that it may be mediated by respiratory movements (Smith, 1966). Miller and Caul (1969) reported that this decrease may not be seen. They also said that using 2 different, successive CSs nearly abolishes the initial acceleration to the CS, and produces a pronounced deceleration as shock approaches (their Figure 2). There has been little conditioning of other cardiovascular responses in monkeys. Blood flow follows a biphasic curve similar to the one for HR (Smith & Stebbins, 1965). Blood pressure has not been studied in monkeys, but in baboons, Ferreira, Gollub, and Vane (1969) report increases in systolic and diastolic pressure in the presence of CSs to electric shock.

METHOD

Subjects

Subjects were 6 adolescent male monkeys (*Macaca mulatta*), weighing from 3 to 4 kg. at the beginning of the study. Five were experimentally naive; one had previously been trained to slow its HR to avoid electric shock.

Apparatus

Polyethylene catheters were implanted permanently in one of each subject's external iliac arteries by the method of Forsyth and Rosenblum (1964). The catheters were kept open by a constant infusion of about 1.1 ml/hr of 20 U of heparin per milliliter in physiological saline. Blood pressure (BP) was detected by a Statham P23Db pressure transducer. Heart rate and BP were measured continuously on a beat-by-beat basis through an on-line Raytheon 704 computer, which also controlled the various experimental procedures. Subjects lived in restraining chairs[4] in individual booths that were nearly soundproof when the doors were closed. Facing each subject were a loudspeaker and a row of 3 different colored lights.

Design

Experimental sessions consisted of 20 min. of rest with the booth door closed followed by a 512-sec. baseline period and a 2,048-sec. experimental period. The experimental period was divided into 16 continuous 128-sec. segments. Usually 4 sessions were run each weekday. Each subject was exposed to classical conditioning and operant learning schedules, and combinations of both in sequences that will be presented below.

Initially subjects were given 40 sessions in each condition. However, because behavior often did not seem to have stabilized at the end of 40 sessions, it was decided to continue each remaining condition until inspection of the data showed stability.

Classical Conditioning

Subjects were exposed to 2 auditory stimuli: 128 sec. of 2/sec clicks, which occupied the first, fifth, ninth, and thirteenth segment of each experimental session, and 128 sec. of 20/sec clicks, which occupied the third, seventh, eleventh, and fifteenth segments of each session (Figure 1). After it was determined that there were no differences in HR in response to the 2 click frequencies, one

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[4] Animals adapt to the chairs readily and do not develop any significant medical complications such as decubiti (Engel & Gottlieb, 1970; Forsyth & Rosenblum, 1964.)

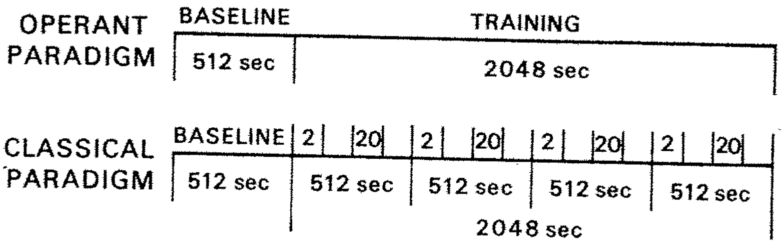

FIGURE 1. Diagram of the 2 kinds of experimental sessions. (During operant training cue lights gave subjects feedback about their HR performance. During classical conditioning, 128-sec. periods of 2/sec and 20/sec clicks occurred as indicated. One of these click patterns was always followed by unavoidable shock. During the combined condition the 2 paradigms were superimposed.)

click pattern was designated as the warning cue for each animal; the other pattern became the neutral cue. At the end of each warning cue, the subject received a .5 sec., 10-ma. shock to the tail.

Operant Training

The method of training was adapted from Engel and Gottlieb (1970). During slowing training, a red light on the right side of the booth door was lit during the entire experimental session; during speeding training a green light on the left side of the booth was lit during the experimental session. The red and green lights functioned as discriminative stimuli for the 2 operant contingencies. During both kinds of training sessions, a middle white light was lit whenever the interval between the last 2 heart beats (i.e., the heart period) met a criterion. The criterion was an interval corresponding to an HR less than the subject's mean resting HR + 20 beats during slowing training, and more than the subject's resting HR — 20 beats during speeding training. Mean resting HR was determined in the immediately preceding baseline period. A free-running clock produced an electrical impulse every eighth second, which caused the animal to get a .5-sec., 10-ma. shock to its tail if the middle light was not on when the impulse occurred. In effect, subjects whose HR did not meet the criteria got an initial shock on a variable interval of 8 sec. schedule, followed by shock on a fixed interval of 8 sec. schedule as long as their HRs were outside the criteria. While the subjects' operant performance was first being shaped, the experimenter administered additional shocks by hand or withheld programmed shocks to facilitate learning. Once a subject showed evidence of having learned the tasks this was never done, and data from the shaping sessions are not included in any of the reported results. Subjects were protected from excessive shock by a program that automatically made the criterion 5 beats/min easier whenever the subject received more than 10 shocks in the preceding 128-sec. interval. The criterion automatically was made 5 beats/min harder whenever no shocks had occurred in the preceding 128-sec. interval and the subject's HR during that period was faster (during slowing training) or slower (during speeding training) than its baseline HR.

Combined Situation

The classical conditioning and operant rate training schedules described above were in effect concurrently. Subjects now received tail shocks when they failed to keep their HRs within the operant criterion, and they received unavoidable shocks at the end of each warning cue.

Sequence of Training

Training was given in 3 different sequences, 2 monkeys receiving each sequence. After each kind of training the subjects' responses to cues warning of impending shock were tested to see whether any modifications induced by this training would extinguish. Table 1 outlines the training sequences for each animal. Animal A developed a dysentery and died before it could complete the study. Animal F outgrew the restraining chair and was euthanized before it completed the study.

Method of Analysis

During each 2,048-sec. session, mean HR and systolic and diastolic blood pressures (SBP and DBP, respectively) to the warning and neutral clicks were compared. Throughout the experiment DBP varied closely with SBP. Thus they will be spoken of together as blood pressure (BP), but they are reported separately. All of the statistical

TABLE 1

SEQUENCE OF TRAINING FOR EACH SUBJECT

Animal	Initial Training	Retraining
A	C S C C + S C	
B	C S C C + S C	F C C + F C
C	C F C C + F C	S C C + S C
D	C F C C + F C	S C C + S C
E	— S C C + S C	F C C + F C
F	— S C C + S C	

Note. Abbreviations: C = classical conditioning; S = operant slowing; F = operant speeding.

analyses were done on individual animals. Most of the hypotheses were tested by making comparisons within specific phases of the study (e.g., response to neutral cue vs. response to warning cue), therefore paired t tests were utilized.

RESULTS

Classical Conditioning prior to Operant Training

Three of the 4 subjects which underwent classical conditioning prior to operant training (A, B, C, and D) significantly increased their HRs to the warning cue relative to the neutral cue. Animal B's HR showed no evidence of discrimination between the 2 cues. All 4 animals' BP responses showed significant evidence of discrimination between the warning cue and the neutral cue. The average responses (warning cue − neutral cue) for the 4 animals were 9.0 beats/min (HR), 5.8 mm. Hg (SBP), and 5.7 mm. Hg (DBP). The average number of sessions was 35. There were 4 presentations of the warning cue and 4 presentations of the neutral cue during each session.

Effect of Operant Conditioning To Slow Heart Rate on the Conditioned Responses

Training in operant heart rate slowing. Four animals (A, B, E, and F) were trained first to slow their HRs operantly. All 4 animals slowed HR significantly from baseline. Blood pressure responses were variable: 2 animals significantly increased SBP, one animal did not change its SBP significantly; one animal significantly increased its DBP and one animal significantly decreased its DBP, whereas, the other animals did not change their DBPs. The average responses (baseline − training) were −15.0 beats/min (HR), −.8 mm. Hg (SBP), and −.8 mm. Hg (DBP).

Conditioned responses following operant training to slow heart rate. Figure 2a presents the HR and SBP responses of the animals to the CS (response to warning cue − response to neutral cue) during classical conditioning following operant training (average number of sessions was 88). Diastolic pressures have been left out of this figure and Figure 3 since DBP responses were so similar to SBP. The HR and BP responses of Animals A and B were similar to their responses prior to operant training. During sessions in which operant HR slowing and classical conditioning were combined (average number of sessions was 105), 3 of the animals significantly *slowed* their HRs to the warning cue relative to the neutral cue, and all 4 animals slowed their HRs to the warning cue of these combined sessions relative to the warning cue of the previous postoperant slowing, uncombined sessions. All 4 animals continued to increase their SBPs (and DBPs) to the warning cue relative to the neutral cue. Thus, the effect of combined training in operant HR slowing and classical conditioning was to reverse the classical HR response from a tachycardia to a bradycardia without reversing the direction of the classical pressor response.

During a subsequent series of sessions of classical conditioning alone (average number was 73), 2 of the animals persisted in slowing HR to the warning cue, and all 4 slowed HR relative to the precombined warning cue. All 4 animals continued to increase SBP (and DBP) to the warning cue relative to the neutral cue.

Effect of Operant Conditioning To Speed Heart Rate on the Conditioned Responses

Training in operant heart rate speeding. Two animals (C and D) were trained first to speed their HRs operantly. Both animals speeded HR significantly from baseline. Blood pressure responses usually increased: One animal increased its SBP significantly and both animals increased their DBPs significantly. The average responses (baseline − training) were 17.6 beats/min (HR), 2.1 mm. Hg (SBP), and 3.8 mm. Hg (DBP).

Conditioned responses following operant training to speed heart rate. Figure 2b presents the HR and SBP responses of the animals to the cues during classical conditioning following operant training (average number of sessions was 40). The HR and BP responses of these animals were increased relative to their responses prior to operant training. This is in contrast to the

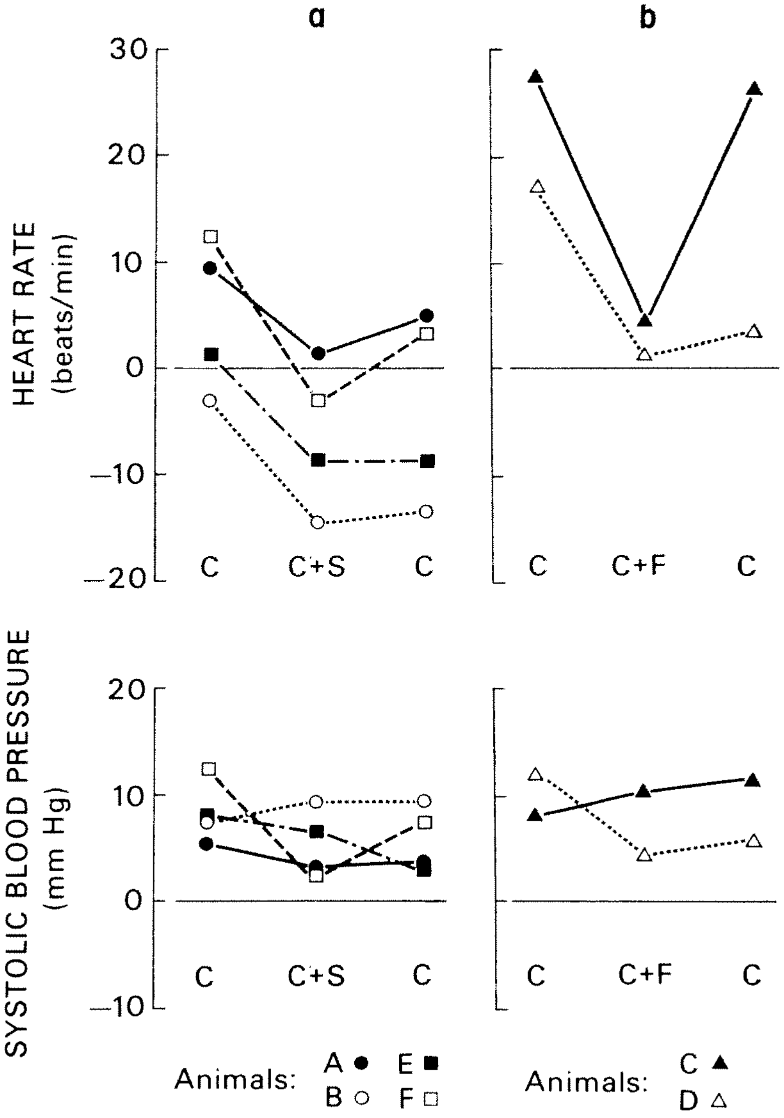

FIGURE 2. (a) Heart rate and SBP responses (warning cue − neutral cue) during classical conditioning sessions of animals operantly trained to slow HR. (b) Heart rate and SBP responses during classical conditioning sessions of animals operantly trained to speed HR. (Abbreviations: C = classical conditioning; C + S = combined classical conditioning and operant HR slowing; C + F = combined classical conditioning and operant HR speeding.)

animals which were trained to slow HR and which did not change their HR or BP responses after operant training. During sessions in which operant HR speeding and classical conditioning were combined (average number of sessions was 34), both animals continued to increase their HRs and SBP to the warning cue relative to the neutral cue. The HR responses during the combined sessions were attenuated significantly with respect to the previous postoperant, uncombined sessions. The BP responses were unchanged.

During the subsequent series of sessions of classical conditioning alone (average number was 29), both animals continued to increase HR and BP significantly to the warning cue. Animal C increased its HR to the precombined rate; however, Animal D maintained the same HR response which it had emitted during combined classical and operant conditioning.

Reversal of Operant Training

Animals initially trained to slow heart rate. Two animals, B and E, which were trained initially to slow HR operantly failed to speed HR after 90 and 106 training sessions, respectively. It should be noted here that we have trained about 20 animals to speed and to slow HR operantly, and these are the only 2 animals which failed to perform both responses reliably. The average responses for these animals were −20.5 beats/min (HR), 6.7 mm. Hg (SBP), and 4.3 mm. Hg (DBP).

Despite their failure to perform reliably during operant training, these animals were retested in the classical and combined paradigms. The results of these sessions are presented in Figure 3a. During classical conditioning sessions only (average number was 80) both animals persisted in slowing HR more to the warning cue than to the neutral cue. When classical and operant HR speeding conditioning was combined (average

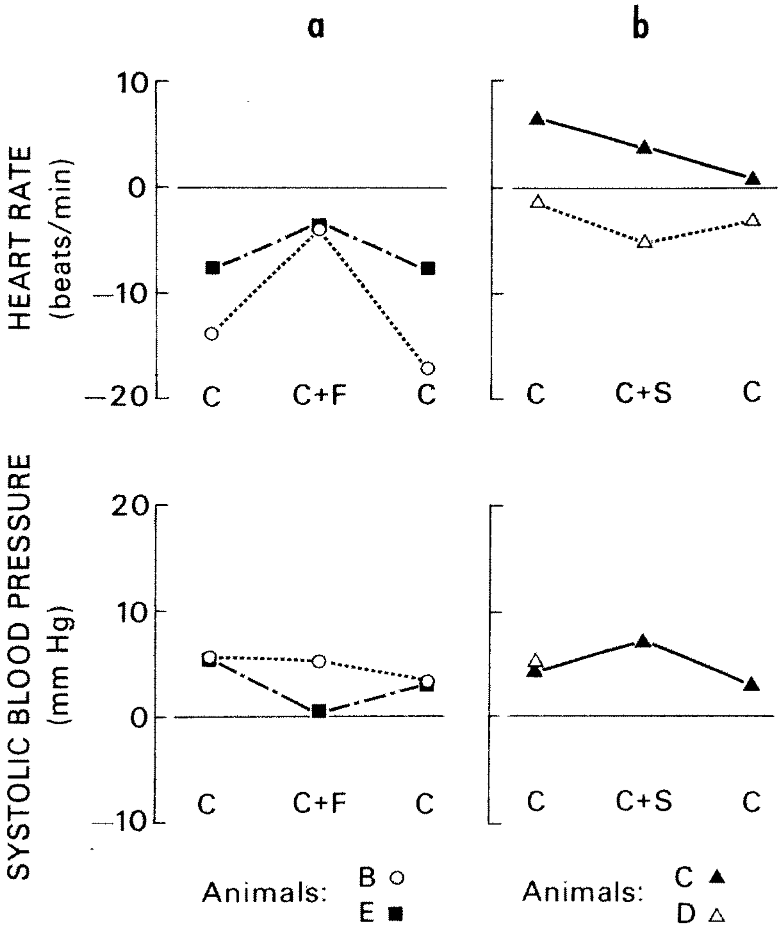

FIGURE 3. (a) Heart rate and SBP responses (warning cue − neutral cue) during classical conditioning sessions of animals which were operantly trained to speed HR following initial operant training to slow HR. (b) Heart rate and SBP responses during classical conditioning sessions of animals which were operantly trained to slow HR following initial operant training to speed HR. (C = classical conditioning; C + F = combined classical conditioning and operant HR speeding; C + S = combined classical conditioning and operant HR slowing.)

number o sessions was 42), the animals still responded with bradycardias to the warning cue; however, the difference between the warning cue and the neutral cue was attenuated significantly relative to the classical conditioning sessions which preceded the combined classical and operant sessions. During the postcombined, classical-conditioning-only sessions (average number was 42) HR responsivity returned to the precombined levels. Throughout all of these sessions BP responses remained relatively unchanged.

Animals initially trained to speed heart rate. The 2 animals which were trained initially to speed HR readily learned to slow HR operantly. During these sessions BP responses were inconsistent. The average responses for the animals (baseline − training) were −20.3 beats/min (HR), −.9 mm. Hg (SBP), and 1.6 mm. Hg (DBP).

During postoperant, classical conditioning sessions (average number of sessions was 36), Animal D showed the HR response reversal which the animals that were trained to slow HR first emitted (Figure 3b). During combined operant and classical conditioning (average number of sessions was 40), Animal D continued to emit the cardiac reversal response. Finally, during postcombined sessions (average number was 38), Animal D continued to emit the cardiac reversal response. During this sequence of sessions the difference between Animal C's response to the warning cue and neutral cue progressively decreased. Blood pressure responses throughout these 3 sets of sessions remained consistent for Animal C. Animal D's blood pressures were measured only during the postoperant, classical conditioning sessions, because attenuation of the pulse pressure wave (presumably attributable to a thrombus in the catheter) precluded valid pressure measurements.

DISCUSSION

It is possible to establish a reliable, differentiated, classically conditioned HR response in the monkey using clicks as the CS and electric shock to the tail as the US. It is also possible operantly to condition HR differentially using electric shock to the tail as the negative reinforcement. When these 2 sets of contingencies are paired: (*a*) The HR response to the warning cue is changed both in terms of its magnitude and in terms of its direction; (*b*) the operant cardiac contingencies produce changes in the HR response which persist indefinitely after the operant contingencies are withdrawn; and (*c*) the change that follows operant training in HR speeding is qualitatively different from the change that follows operant training in HR slowing. The discussion will consider some of the possible mechanisms which could mediate these effects.

Since the operant reinforcer was the same as the original US, it can be said that the change in HR response to the warning cue did not represent a CR to a new US. Nor did the operant task simply direct attention from the classical stimuli, since the subjects continued to respond differentially to these stimuli. Furthermore, the fact that the animals responded differentially in HR and BP, and the fact that the animals responded selectively to the CS depending upon the operant contingencies, rule out habituation as a mechanism mediating the cardiac responses. The cardiac CR apparently was changed by the operant task.

The change in some subjects' responses to the CS went beyond the point required by the operant schedule and became a positive response in the other direction. This change at least partially persisted long after the operant schedule was withdrawn and in 2 subjects even after an opposite schedule was introduced. Why might such an overreaction have occurred? Two hypotheses present themselves:

1. The subjects' behavior may not have been controlled by the true contingencies of reinforcement. It has been reported that monkeys which have learned an ordinary skeletal muscle operant to avoid shock emit this operant more when given additional unavoidable shock. They continue to emit the operant even when it produces shock (Kelleher, Riddle, & Cook, 1963; McKearney, 1969; Pomerleau, 1970). The subjects seem to react to the unavoidable shock as information that they have not responded enough, rather than that the avoidance re-

sponse is ineffective (see Dreyer & Renner, 1971). This experiment may have produced an analogous phenomenon using a visceral avoidance task.

2. Voluntary cardiac slowing may mitigate the aversiveness of electric shock. DiCara and Weiss (1969) have found that rats react to shock with much less emotionality after operant training in HR slowing than after training in HR speeding, and Sirota, Schwartz, and Shapiro (1973) have claimed recently that human subjects report a given stimulus as less painful if it occurs while they are voluntarily slowing their HRs. Increased vagal activity, which may be a mechanism of voluntary HR slowing, reduces a rat's tendency to learn a shock avoidance task (Slaughter, 1971). The subjects of the present experiment may have discovered a protective response that they can emit whenever they are in danger of shock.

If Hypothesis 1 were the sole mechanism operating in this experiment, one would expect the CR to change in the direction of the last HR operant learned, and subjects should have difficulty in learning a new operant regardless of the direction of the first operant. If Hypothesis 2 were the sole mechanism, there should be an asymmetry toward slowing: Operant speeding should not enhance conditioned speeding responses, and difficulty in learning the opposite HR operant should follow training in slowing more than training in speeding. The data show some evidence for both interpretations. Initial HR training in either direction changed the HR response to the warning cue in that direction, as Hypothesis 1 predicts. However, the effects of operant speeding and slowing were not symmetrical. In Subjects C and D, a combination of conditioning and operant speeding reduced rather than increased their tendency to speed, even though these subjects had received no training in HR slowing. Also, Subjects B and E were unable to learn operant speeding after having learned operant slowing, although C and D learned slowing after speeding. Thus the nature of the operant process engendered by the avoidable shocks in this experiment is not clear. Both hypotheses could be correct, or a third mechanism may be operating. It might be possible to minimize the occurrence of the mechanism in Hypothesis 1 by giving subjects HR training with cues and reinforcers as different as possible from the stimuli with which they are classically conditioned.

It could be argued that the procedure described here did not alter the subjects' CRs, but replaced them with operants. This would be to say that an operant response came under control of the previously conditioned stimulus and inhibited or overshadowed the CR. This argument is definitional, since there is no way of testing whether or not a new response to a CS is still a CR. But if one chooses to adopt this definition, he still must account for the situation where operant and classical responses directly compete with each other. Given a situation where there is operant incentive to slow and classical conditioning to speed HR, something has to determine whether the subject will speed or slow its HR. The process that causes conditioning of responses must have a common dimension with the process that causes operant learning. They must each have a number that represents ability to compete for the final common path of behavior. If CRs cannot be "modified" by reward, they can at least be obliged to bargain for expression with a coin that has some corresponding value in the currency of reward. In terms of such a theory (2 separate, competing processes), the significance of this experiment concerns the relative strength of the 2 opponents: The reinforcing effect of a contingent stimulus on cardiac behavior can be greater than the effect of the same stimulus presented in a classical conditioning paradigm, a phenomenon which previously had not been found. Application of this 2-process theory becomes awkward when the BP data also are taken into account. It would have to be said that a CS which was evoking conditioned HR and BP responses suddenly became a cue for operant HR responses, while remaining a CS for conditioned BP responses. Certainly it would be simplest to say that the subjects' CRs were altered by the operant reinforcement.

The concept of classical conditioning as a process distinct from operant learning has been necessary because there seemed to be learned connections that were beyond the reach of the law of effect (Schlosberg, 1937). While this still could be true with respect to learned connections between stimuli, there is now reason to doubt it in the case of connections between a stimulus and a response. If CRs must compete with operants, is there any reason to hypothesize that the property of the US which gives the CR its competitive ability is different from operant reinforcement? This experiment provides more reason to adopt Schlosberg's view (private communication reported in Kimble, 1961, p. 99) that all responses require operant reinforcement.

REFERENCES

DiCara, L. V., & Weiss, J. Effect of heart-rate learning under curare on subsequent noncurarized avoidance learning. *Journal of Comparative and Physiological Psychology,* 1969, **69,** 368–374.

Dreyer, P., & Renner, E. Self-punitive behavior—Masochism or confusion? *Psychological Review,* 1971, **78,** 333–337.

Engel, B. T. Operant conditioning of cardiac function: A status report. *Psychophysiology,* 1972, **9,** 161–177.

Engel, B. T., & Gottlieb, S. H. Differential operant conditioning of heart rate in the restrained monkey. *Journal of Comparative and Physiological Psychology,* 1970, **73,** 217–225.

Ferreira, S., Gollub, L., & Vane, J. The release of catecholamines by shocks and stimuli paired with shocks. *Journal of the Experimental Analysis of Behavior,* 1969, **12,** 623–631.

Forsyth, R. P., & Rosenblum, M. A. A restraining device and procedure for continuous blood pressure recordings in monkeys. *Journal of the Experimental Analysis of Behavior,* 1964, **7,** 367–368.

Gormezano, I., & Moore, J. Effects of instructional set and UCS intensity of the latency, percentage, and form of the eyelid response. *Journal of Experimental Psychology,* 1962, **63,** 487–494.

Hilgard, E., & Humphreys, L. The effect of supporting and antagonistic voluntary instruction on conditioned discrimination. *Journal of Experimental Psychology,* 1938, **22,** 291–304.

Kelleher, R., Riddle, W., & Cook, L. Persistent behavior maintained by unavoidable shocks. *Journal of the Experimental Analysis of Behavior,* 1963, **6,** 507–517.

Kimble, G. *Hilgard and Marquis' conditioning and learning.* New York: Appleton-Century-Crofts, 1961.

McKearney, J. Fixed-interval schedules of electric shock presentation: Extinction and recovery of performance under different shock intensities and fixed-interval duration. *Jour- of the Experimental Analysis of Behavior,* 1969, **12,** 301–313.

Miller, N. Learning of visceral and glandular responses. *Science,* 1969, **163,** 434–445.

Miller, R., & Caul, W. Influence of uncertainty on conditioned heart rates of monkeys. *Physiology and Behavior,* 1969, **4,** 975–980.

Nicholls, M., & Kimble, G. Effect of instructions upon eyelid conditioning. *Journal of Experimental Psychology,* 1964, **67,** 400–402.

Norris, E., & Grant, D. Eyelid conditioning as affected by verbally induced inhibitors, set and counter reinforcement. *American Journal of Psychology,* 1948, **61,** 37–49.

Ominsky, M. Instructions and interstimulus interval in eyelid conditioning. *Psychonomic Science,* 1968, **12,** 49–50.

Perkins, C. The stimulus conditions which follow learned responses. *Psychological Review,* 1955, **62,** 341–348.

Pomerleau, O. F. The effects of stimuli followed by response-independent shock on shock-avoidance behavior. *Journal of Experimental Analysis of Behavior,* 1970, **14,** 11–21.

Rescorla, R., & Solomon, R. Two-process learning theory: Relationships between Pavlovian conditioning and operant learning. *Psychological Review,* 1967, **73,** 151–182.

Schlosberg, H. The relationship between success and the laws of conditioning. *Psychological Review,* 1937, **44,** 379–394.

Sirota, A., Schwartz, G., & Shapiro, D. Effects of feedback control of heart rate on judgments of electric shock intensity. *Psychophysiology,* 1973, **10,** 212–213. (Abstract)

Slaughter, J. Effects of avoidance performance of vagal stimulation during previous fear conditioning. Unpublished doctoral dissertation, University of Denver, 1971.

Smith, O., & Stebbins, W. Conditioned blood flow and heart rate in monkeys. *Journal of Comparative and Physiological Psychology,* 1965, **59,** 432–436.

Smith, R. Discriminative heart rate conditioning with sustained inspiration as a respiratory control. *Journal of Comparative and Physiological Psychology,* 1966, **61,** 221–226.

Snapper, A., Pomerleau, O., & Schoenfeld, W. Similarity of cardiac CR forms in the Rhesus monkey during several experimental procedures. *Conditional Reflex,* 1969, **4,** 212–220.

Spence, K., & Ross, L. A methodological study of the form and latency of eyelid response in conditioning. *Journal of Experimental Psychology*, 1959, **58,** 376–385.

Stebbins, W., & Smith, O. Cardiovascular concomitants of the conditioned emotional response in the monkey. *Science*, 1964, **144,** 881–883.

Tighe, T., Graves, D., & Riley, C. Successive reversals of a classically conditioned heart-rate discrimination. *Journal of Experimental Analysis of Behavior*, 1968, **11,** 199–206.

Zener, K. The significance of behavior accompanying conditioned salivary secretion for theories of the conditioned response. *American Journal of Psychology*, 1937, **50,** 384–403.

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