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THE EFFECTS OF CUED, RESPONSE-INDEPENDENT FOOD DELIVERY ON CARDIAC RATE

City University of New York PH.D. 1984

The effects of cued, response-independent food delivery on cardiac rate.

by

Malcolm R. McCullough

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, the City University of New York.

1984

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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Abstract

The effects of cued, response-independent food delivery on cardiac rate.

by

Malcolm R. McCullough

Advisor: Professor Brett K. Cole

When comparing the data of studies using "Pavlovian" procedures to condition cardiac rate responses with those of studies involving "operant" conditioning of lever presses, it appears that: 1) the response variability in the "Pavlovian" studies is relatively greater; and, 2) with only a few exceptions, "Pavlovian" studies have not found increases in CR strength with a lessening of p(US/CS) from 1.0; however, "operant" studies often report enhanced response strength accompanying a reduction in probability of reinforcement.

The present study drew on findings concerning both response variability and the effects of response-independent reinforcement and on an account of proposed effects of extinction on the distribution of response subclasses, in order to analyze the available "Pavlovian" findings. That analysis led to predictions regarding variation in the characteristics of the cardiac rate CR between subjects and

the effects of within-subjects manipulation of probability of reinforcement with cued, response-independent conditioning procedures ("Pavlovian").

Those predictions were tested by examining changes in heart rates of ten hooded rats exposed to a cued, response-independent conditioning procedure. During the initial conditioning sessions, .08cc milk (US) followed every presentation of the 8-sec cue (CS). After that initial condition (p(US/CS)=1.0), p(US/CS) was reduced to 0.50 and then to 0.25, 0.12, and 0.06, after which a recovery point (p(US/CS)=1.0) was presented.

The major results were: 1) rats developed heart rate responses during the cue that differed in direction, despite being exposed to procedurally identical conditioning operations; and, 2) more than half of the rats displayed larger heart rate responses at p(US/CS)=0.50 than at p(US/CS)=1.0. Both these findings were consistent with the present predictions. It was suggested that the temporal relation between the reinforcer and the heart rate response was critical in determining which response (acceleratory or deceleratory) was eventually conditioned. Since this relation was largely uncontrolled in the present response-independent procedure, such between-subjects variability would not be unexpected. It was also suggested that the introduction of unreinforced trials, with the move from p(US/CS)=1.0 to p(US/CS)=0.50, increased the variability of the conditioned heart rate response which

allowed for a change in the prevailing response-reinforcer relations, which could result in either increased or decreased CR magnitudes at p(US/CS)=0.50.

To anyone who finds this work either
of interest or of some value.

Acknowledgements

The present work was made possible by the direct assistance, and published works, of Drs. Brett K. Cole and William N. Schoenfeld. I wish to thank Brett for his continuing interest in this work, for freely sharing his thoughts, and for his sympathetic attitude. I also wish to acknowledge my debt to Dr. Schoenfeld for the training he provided me during my graduate student years and to thank him for his contributions to this work. I offer my sincere thanks to all the members of my supervisory committee for their contributions.

I would like to express my gratitude to several other people who contributed in some way to the completion of this dissertation.

My deepest thanks to Patricia Meyers. I cannot really thank Patty enough for her help with countless hours of inspecting data, entering data at the terminal, and proof-reading. Nor can I adequately express my appreciation for the willingness with which she offered her help during those years of running and writing.

Special thanks to my parents, Doris and Bill, for their unconditional emotional support over those years and for their fiscal support when it was most needed.

Thanks to Dr. Ron Kadden for his advice and help over the complete course of the dissertation. Thanks also to the many individuals who assisted me in various ways during the many stages of this work: my friends and colleagues at Queens College, C.U.N.Y. -- Julian Albert, Lupita Coll, Gene Fisch, Ed Korber, Elizabeth Ramirez, and Dave Coleman; Paul Tremont and Bill Steele of the Montrose laboratory; and, John Leong, of the Queens College Computer Center.

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Conditioning paradigms, which are among the known procedures to establish and maintain behavior, may involve either response-dependent or response-independent presentation of stimuli. With response- dependent delivery of the stimulus, a member of a particular response class is a necessary condition for presentation of the stimulus. Response-independent presentation of a stimulus uses a criterion other than presence of a particular response (e.g., passage of time since the last stimulus) to determine the moment of stimulus presentation. With respect to the traditional "types" of conditioning procedures, "operant" procedures usually involve response-dependent stimulus deliveries, and "Pavlovian" procedures usually are based on response-independent stimulus delivery.

Skinner's early writings (see, for example, Skinner, 1938) contain a distinction between "types" of behavior in addition to the typing of the conditioning procedures. One category included responses of smooth muscles and glands, responses that had traditionally been labelled "involuntary". These were called "respondents" by Skinner since they seemed to occur as specific responses to specific stimulus presentations. Pavlov (1927) demonstrated that such responses could be conditioned with response-independent procedures. However, subsequent work has shown that respondents may be conditioned by response-dependent procedures also (e.g., Black, 1971; DiCara and Miller, 1968; and Miller, 1969). In the latter,

a stimulus, such as shock or food, is presented if and only if a change in the response measure occurs (e.g., an increase in blood pressure), and large changes have been achieved by continually raising the criterion for stimulus delivery.

Skinner's second category of behavior included responses of striated (skeletal) muscles. These responses had traditionally been labelled "voluntary" but were called "operants" by Skinner. Unlike respondents, operants did not occur in response to an identifiable, prior stimulus presentation[1]. Response-dependent procedures were the first to be used extensively to condition operant responses in the laboratory (Skinner, 1938). Perhaps the initial use of response-dependent as against response-independent procedures strengthened Skinner's view that operant responses operated on the environment instrumentally to secure the reinforcer. However, subsequent work by Skinner (1948) has shown that operants may be conditioned when the reinforcers are delivered according to a clock rather than the animal's behavior. Skinner's later study was taken by some workers as demonstrating that an operant response did not have to operate on the environment instrumentally in order to be conditioned (Schoenfeld, Cole, Lang, and Mankoff, 1973). From the literature, it seems safe to conclude that either response-dependent procedures or response-independent procedures may be used to condition responses of either the "respondent" or "operant" type.

The finding that a respondent could be conditioned with either an "operant" or a "Pavlovian" procedure seemed to force the abandonment of the notion, advanced by Skinner (1938), that the "types" of conditioning procedures (Pavlovian and operant) could be distinguished by the "type" of behavior they affected (respondent and operant, respectively). From those same findings it also follows that stimuli presented response-dependently with respect to an operant might at the same time be acting, response-independently, on a respondent. Such a case was reported by Shapiro (1961), who measured salivation while the subject responded on a fixed interval schedule of food (reinforcer) presentation[2]. The usual pattern of lever pressing behavior was observed but in addition a salivary response had been conditioned, presumably by the periodic food deliveries. It might appear then that reinforcers, dependent on the occurrence of a particular operant response for their delivery, will inevitably affect either respondents, or other operants (these operants may be either responses that qualify for reinforcement or they may be entirely different responses). These multiple effects of reinforcer deliveries, which were predictable from works like Skinner's "superstition" experiment (1948), may also occur in cases not involving response-dependent reinforcement of an operant. For instance, Pavlov's time reflex (periodic presentation of food led to development of a conditional salivary response) and Skinner's

"superstition" study (periodic presentation of food led to the conditioning of identifiable muscular movements) were isolated experiments; however, since the procedures were identical, either investigator could have included the measurements of the other and presumably would have seen simultaneous, response-independent conditioning of an operant and a respondent. The two preceding examples (Shapiro; and, Pavlov and Skinner) suggest that when performing one "type" of conditioning the experimenter may inadvertently be performing the other "type". The inevitability of such occurrences has been stated by Schoenfeld (1971), who pointed not only to the fact that the organism is continually behaving (the "behavior stream" notion), but also to the fact that many response systems will be affected by delivery of a single reinforcer (the notion of "conditioning the whole organism").

The above paragraphs point to the possibility of conditioning any response with either of the conventionally defined conditioning procedures[3]. Furthermore, they suggest that reinforcers delivered to a behaving organism will inevitably affect behaviors beyond the single operant or respondent that the experimenter may be measuring. That is, such reinforcers may simultaneously act as "operant SRs" and "Pavlovian USs". Additionally, the above paragraphs brought out facts that appear to make it impossible to sustain distinctions between "operant" and "Pavlovian" "types" of conditioning based on either "type" of behavior affected or

the presence or absence of a response-reinforcer dependence. Now that some general considerations regarding conditioning procedures and the effects of such procedures on "categories" of behavior have been discussed, attention will be given to some more specific behavioral phenomena.

In the initial work with conditioning procedures, certain events, either response occurrences or stimulus presentations, were both the necessary and sufficient conditions for performance of the reinforcement operation (delivery of a particular class of stimulus). Specifically, in most of Pavlov's (1927) reports, an unconditional stimulus (US) was presented for each occurrence of the conditional stimulus (CS), while in the early work with the lever press response by Skinner (1938), a reinforcing stimulus (SR) was presented each time that a lever press occurred. Designation of either the occurrence of a response or the presentation of CS as both necessary and sufficient conditions for presentation of either SR or US, respectively, came to be called continuous, regular, or 100% reinforcement by various workers. The findings were an increase toward asymptote in either conditional response (CR) magnitude (Pavlov, 1927) or response rate (Skinner, 1938) with successive reinforcer deliveries. It appeared that presentations of reinforcing stimuli were responsible for the increases in either response magnitude (Pavlovian) or response frequency (operant) that were labelled "conditioning".

Once the conditioned behavior reached asymptote the reinforcer presentations could be continued, which would maintain the new behavior, or they could be discontinued. Procedurally, extinction was defined as the discontinuance of reinforcer presentations. With Pavlovian procedures, CS was no longer accompanied by US, while in operant procedures, responses no longer "produced" the reinforcer. With either procedure extinction produced a gradual loss of strength in the conditioned behavior, a result that further established the critical role of reinforcers in the acquisition and maintenance of behavior. Although Pavlovians focused upon CR magnitude while operant workers measured response rate, there was a basic similarity across these two areas with regard to the procedures and behavioral effects of extinction.

Soon after the initial work with 100% reinforcement, workers began to study the effects on behavior of removing the sufficiency condition, that is, not presenting a stimulus for every CS or for every response response (e.g., Humphreys, 1939; and, Skinner, 1938). The "omission" of stimuli, either USs or SRs, became known as partial or intermittent reinforcement. Before the effects of intermittent reinforcement were actually determined the known effects of regular reinforcement and extinction seemed to permit prediction of the likely effect of such schedules. If those two operations (reinforcement and nonreinforcement) were alternated in some fashion, a simple summing of their

effects in isolation might be predicted. Given that reinforcement increased response strength and nonreinforcement decreased response strength, a mixture of these operations might yield a response strength somewhere between the levels seen with regular reinforcement and extinction. This prediction was not uniformly supported in the laboratory.

In the case of the rat's lever press response, omission of some SRs, after acquisition with response-dependent reinforcement, was shown to have unexpected effects. For instance, greater resistance to extinction (a slower decline of the response measure in the absence of further reinforcement) was usually observed for animals with prior exposure to intermittent reinforcement than for animals with a history of 100% reinforcement (e.g., Hearst, 1961; and, Skinner, 1938). This outcome, labelled the partial reinforcement extinction effect (PREE), appeared paradoxical because the interpolation of the extinction operation, which had already been shown to decrease one measure of response strength (response rate), actually increased a different measure of response strength (the persistence of responding in a later extinction period)[4]. Another finding that was not predicted from the above mentioned view that reinforcement strengthened behavior and that nonreinforcement weakened behavior and that mixtures of these two operations would produce effects intermediate to those seen at the endpoints, was that omission of some SRs

from a response-dependent schedule of regular reinforcement could increase the rat's rate of response. That is, behavior occurred more frequently with less frequent presentations of the very same stimulus that was responsible for the establishment of that behavior (e.g., Boren, 1953 and Brandauer, 1958, with ratio schedules; Skinner, 1938, with a change to an extinction schedule; Reynolds, 1961 (a, b), with a decrease in reinforcer frequency in one component of a multiple schedule; and, Schoenfeld, Cole, Lang, and Mankoff, 1973, with a lessening of response-independent p(SR)). Intermittent reinforcement, as against regular reinforcement, was shown to produce either enhanced response rates or increased resistance to extinction for "operants" reinforced response-dependently.

Turning from the typical "operant" study to the "Pavlovian" case, omission of some USs in the case of response-independent reinforcement of respondents (e.g., heart rate, salivation, galvanic skin response) yielded effects mostly consistent with the above mentioned prediction. That is, presenting US on only 50% instead of 100% of the trials usually resulted in decreases in the measure of CR strength. Specifically, Pavlovian studies examining resistance to extinction of a CR using a variety of infrahuman subjects and a number of response measures, more often than not, have failed to report increased resistance to extinction after intermittent, as against continuous, reinforcement (no PREE)[5]. Comparing these

Pavlovian findings with the earlier-mentioned "operant" findings, there appears to be a difference between the "operant" and "Pavlovian" literatures regarding the effects of intermittent versus regular reinforcement on later extinction performance. For measures of CR strength taken during reinforced trials (CR magnitude or CR probability), intermittent reinforcement only rarely produced increases in the measure of CR relative to that seen with p(US/CS)=1.0 [6]. (Note: the probability of US, given a CS, is an expression of intermittency for Pavlovian procedures and is abbreviated p(US/CS)). Most studies of the effects of manipulating p(US/CS) on CR strength involved comparisons of CRs at values of 1.0 and 0.50, and reported either diminished measures of CR at values of p(US/CS) less than 1.0 or equality of CR measures at these values [7]. From these findings, it appears that the "operant" and "Pavlovian" literatures differ with respect to the response strength that can be maintained with intermittent, as against 100%, reinforcement.

The effects of intermittency in "Pavlovian" procedures and "operant" procedures may be capsulized as follows: 1) with operant procedures intermittent reinforcement usually produces increased resistance to extinction, but with Pavlovian procedures a PREE has not usually been observed; and, 2) with operant procedures some amounts of intermittency of reinforcement have reliably yielded higher response rates, but with Pavlovian procedures there has not

been a parallel result. These comparisons raise the question of why intermittent reinforcement more frequently enhances behavior for cases of response-dependent reinforcement of "operants" than for cases of response-independent reinforcement of "respondents".

To answer this question it will be necessary to examine effects of extinction other than the weakening effect just mentioned. Antonitis (1951) provided some information concerning the effects of extinction on response variability. He observed that response variability increased with a change from regular reinforcement to extinction (Millenson and Hurwitz, 1961, also report such a finding). Schoenfeld (1950, 1968) took the Antonitis paper, in which the response variability was defined in terms of spatial location, and constructed a similar argument for the lever press response. The lever press, a discrete event involving the closure of a switch, was designated the "response class". Schoenfeld used the notion that members of such a response class were also members of response subclasses. That is, one lever press might be a member of a low force or a short duration subclass while another lever press might be a member of a high force or a long duration subclass. Extinction of a lever press would increase the number of subclasses of leverpress emitted (i.e., increase variability).

Given the Antonitis finding and the notion of response subclasses, Schoenfeld attempted to explain the PREE for

operant responses[8]. He suggested that animals exposed to intermittent reinforcement schedules would have had many more subclasses of the response reinforced than would animals with a history of only regular reinforcement. These differences would arise from the interspersing of extinction periods, which produce increases in response variability, between the reinforcers of the intermittent schedules. With regular reinforcement, a response subclass that is increased in strength by reinforcer delivery will surely be reinforced if emitted again and will eventually become the predominant subclass. However, with intermittent reinforcement, a response subclass strengthened by reinforcer delivery is immediately exposed to some extinction (unreinforced emissions). The effects of this extinction period are: 1)to weaken this recently strengthened subclass, if a member of that subclass is emitted and not followed by SR; and, 2)to increase the variability of response subclasses emitted These two consequences of extinction presumably increase the likelihood that the next reinforcer will follow, and strengthen, a response subclass different from the one the previous reinforcer followed. The result would be that the intermittently reinforced organism acquires a history that includes more reinforced subclasses than the organism conditioned with regular reinforcement. Given such a history, the intermittently reinforced animals would have a greater variety of response subclasses to extinguish. Assuming that all these response subclasses had approached

their asymptotic strength, the intermittently reinforced animals should emit more responses in extinction, indicating a PREE. As stated by Schoenfeld, the above argument reduces finally to the statement, consistent with general reinforcement theory, that response subclasses reinforced during conditioning will appear during extinction.

The above case of exposing an animal to extinction after a history of either regular or intermittent reinforcement is in some way similar to the case of an animal that is placed on an intermittent schedule after a history of regular reinforcement. Both of these cases involve increases in the frequency of extinction trials during the experimental session. Stated differently, both cases are instances of an animal being changed from a higher to a lower frequency of reinforcement. It would seem, therefore, that the analysis of the events occurring during a test for the PREE might also apply to an animal changed from a schedule of regular reinforcement to a schedule of intermittent reinforcement.

In the case of an animal moved to intermittent reinforcement after a history of regular reinforcement, Antonitis's finding would suggest that response variability would increase during the first extinction period that was encountered after switching from regular reinforcement. This increased response variability, according to Schoenfeld's argument, would result from the reappearance of response subclasses that had been reinforced at one point in

the animal's history but were not necessarily still a part of its repertoire at the point of the schedule change. Since reinforcers are still being delivered on the response-dependent intermittent schedule, it is highly likely that some of these "new" response subclasses will be followed by reinforcers. Given such occurrences, particular subclasses would have an increased chance of recurring and being followed again by a reinforcer. In this way the animal's behavior might be changed as a result of a change in the response subclasses that are reinforced. The prevailing subclass under regular reinforcement may no longer be the prevailing subclass if the intermittently delivered reinforcers strengthen a new subclass from the broadened array of subclasses produced by the interpolated extinction. Clearly, it would not be possible to predict exactly which subclasses would be "selected" for strengthening from those "brought out" by extinction. Therefore, the overall effect of such a decrease in the frequency of reinforcement on the response involved could not be predicted. A shift in subclasses might lead to responses with shorter durations that could facilitate higher response rates. On the other hand, the change in schedule might result in the strengthening of a less "efficient" topography of the lever press resulting in lowered response rates. Both these cases do, however, involve response-dependent reinforcement of a response class (lever press) and, at the same time, response- independent

reinforcement of a particular response subclass. It is this "superstitious" reinforcement of response subclasses, with the change in schedule, that may produce a change in the properties of the response class (e.g., lower or higher response rates). Such response-independent reinforcement of response subclasses would be expected to produce different effects in different animals, with the effects being determined by idiosyncratic instances of response subclass-reinforcer contiguity[9].

The preceding paragraphs have reviewed Schoenfeld's (1950,1968) account of the PREE, an account that utilized certain facts about the effects of extinction on response variability and certain proposed consequences of the response definition: the inevitable response- independent reinforcement of different response subclasses. These same findings and ideas were then applied to the case of an animal switched from regular to intermittent reinforcement, a case that was seen as basically similar to the test for the PREE. Both of these cases involved response-dependent reinforcement with respect to the response class. In the paragraphs that follow, a similar analysis will be undertaken for the same two cases when response-independent reinforcement procedures are employed.

Heart rate will be the response measure in the discussion that follows. However, before discussing the effects of response-independent reinforcement on that response, it seems appropriate to relate the the measure of

heart rate to the lever press, the response used in the prior analysis. Although obviously involving different muscles, the individual heart beat has variations in its occurrences (as reflected, perhaps, in varying electrocardiograms for different beats), just as the lever press shows variations from one occurrence to another (as reflected by electromyograms of the muscles involved). It would therefore be reasonable to use the notion of response subclasses with respect to the heart beat, since any beat may be further classified into a subclass, depending on the specific properties of that particular beat[10].

Allowing that both lever presses and heart beats are response classes and that they may be further divided into subclasses, it may now be asked what happens to the distribution of those subclasses with a switch from reinforcement to extinction. Millenson and Hurwitz (1961) reported that lever press variability increased with a change from regular reinforcement to extinction, suggesting a broadening of the subclass distribution, but for heart rate the parallel finding has not been clearly established. However, it has been shown that both lever pressing and heart rate are conditionable by means of appropriate presentations of reinforcers, and furthermore, it has been shown that by presenting the reinforcer only after members of specific response subclasses (e.g., a hard lever press or a short interbeat interval), the distribution of those response subclasses can be altered (e.g., Notterman and

Mintz, 1965; and Miller and DiCara, 1967, respectively). Additionally, the discontinuance of reinforcer presentations has a similar effect on both conditioned lever pressing and conditioned heart rate: an eventual decrease in the strength of the observed response. Given the similarities between lever presses and heart rate with respect to the effects of conditioning and extinction operations, and the existence of evidence for response subclasses with both these responses, one might predict that heart rate would also display an increase in variability with a change in schedule from 100% reinforcement to extinction[11].

Turning to the analysis of the effects of response-independent reinforcement on heart rate, it would appear that Schoenfeld's explanation of the PREE would apply directly to such a case. That is, "heart rate response" (defined as CS rate minus pre-CS rate) could be substituted for "lever press" in the Schoenfeld paper. While such application of Schoenfeld's argument would predict a PREE with heart rate or any respondent behavior, the literature usually reports no PREE with Pavlovian conditioning of respondents[5]. The discrepancy between the prediction and the experimental data may arise from a feature inherent in response-independent procedures: no restriction regarding the behavior that US may follow. Unlike response-dependent procedures where the reinforcer occurs only after a member of a specified response class, with response-independent (e.g., "Pavlovian") procedures US may directly follow a

member of any response class. Therefore, with response-dependent reinforcement the reinforcer consistently affects members of the measured response class ("R") but with response-independent reinforcement the reinforcer may affect many response classes other than the measured response class ("not-R")[10]. That reinforcement of "other behavior" does occur in response- independent procedures is suggested by the commonly observed decreases in response rates when an animal is moved from a response-dependent schedule to a response-independent schedule with an equal frequency of reinforcers (e.g., Herrnstein, 1966; Lachter, 1971; Lachter, Cole, and Schoenfeld, 1971; Schoenfeld, Cole, Lang, and Mankoff, 1973; and, Zeiler, 1968). The reinforcers delivered on the response-independent schedule are presumably affecting behavior but they are acting on "other behaviors" in addition to the measured response. The degree of intermittency for the measured response is thereby increased while behaviors exclusive of the measured response are strengthened.

In the paragraphs that follow, the details of the Schoenfeld proposition will be altered to accomodate the features of response- independent reinforcement procedures.

Briefly, an animal placed on a schedule where every CS is followed by US (p(US/CS)=1.0) would, according to Antonitis's data, approach a lower asymptote of variability. However, in approaching that asymptote the animal passes through a period of greater response variability which

presumably involves both reinforcement of various response subclasses and reinforcement of different response classes. That is, given a response class of heart rate acceleration, the reinforcer might have followed either various subclasses of the acceleratory response or the reinforcer might have followed instances of "not-CR" (no change in heart rate or a heart rate deceleration, in this particular example). Continuing with Schoenfeld's argument, these various subclasses of response (CR), along with the instances of "not-CR", will reappear during an extinction session. This will be true for both the animals with a history of regular reinforcement and for those with a history of intermittent reinforcement. However, the animals exposed to p(US/CS)<1.0 would have had more CR subclasses reinforced and more other response classes ("not-CR") reinforced than would the animals exposed to p(US/CS)=1.0. This is predictable based on the extinction inherent in intermittent reinforcement schedules, which acts to increase response variability. For the response-dependent case, reinforcement of a greater number of response subclasses yielded a PREE; however, for the response-independent case, reinforcement of "not-CR" must be considered along with reinforcement of CR subclasses. When comparing groups with p(US/CS)=1.0 and groups with p(US/CS)<1.0, a crucial factor might be the relative frequencies of reinforcement of response subclasses as against reinforcement of other response classes. The greater the frequency of the latter, the faster the

extinction of the CR in a test for the PREE, since there would be relatively more trials on which the animal would not produce the CR (that is, its response would be "not-CR"). If the animals with p(US/CS)<1.0 have a higher proportion of reinforcement for "not-CR" than for subclasses of CR, then the results in the literature[5,7] would be predicted. While the Schoenfeld account can be applied to the response-independent case, as done above, it is not clear whether on not it agrees with the experimental findings unless the data on relative frequency of reinforcement of subclasses of CR, as against reinforcement of "not-CR", are available.

Turning from the case of the PREE to the case of an animal moved from p(US/CS)=1.0 to p(US/CS)<1.0, there again appear to be similarities and differences between the response-dependent (usual "operant" case) and the response-independent (usual "Pavlovian" case) cases with respect to the application of Schoenfeld's analysis. Assuming CR approached a lower asymptote of variability on p(US/CS)=1.0 and then increased in variability with introduction of extinction trials (that is, the response subclass distribution broadened), the situation would be similar to the "operant" case (Antonitis, 1951). But unlike the "operant" case, "Pavlovian" extinction, as already mentioned, should bring out responses in the "not-CR" category. It would appear that the larger the number of cases of "not-CR" brought out by extinction, the greater the

probability of one of those cases of "not-CR" being followed by US. Reinforcement of "not-CR" would lessen the animal's tendency to respond with CR, thereby producing a diminished strength of CR at $p(US/CS)<1.0$. That is, in fact, a frequent result in the Pavlovian literature: diminished CRs at $p(US/CS)<1.0$ compared to those at $p(US/CS)=1.0$[7]. On the other hand, the effect on the CR of the increased number of response subclasses resulting from the extinction trials cannot be predicted so definitely. As in the "operant" example, it cannot be specified in advance which subclass will be affected by reinforcer delivery. For the response-dependent case, SR was presented after a selected response class member and "superstitiously" reinforced the subclass represented by that particular response. However, for the "Pavlovian" case there is no response-dependency, so variation in CR subclasses interacts with reinforcer delivery differently than in the "operant" case. For example, a short-latency CR subclass brought out by extinction might serve to increase the temporal separation of response and reinforcer. Such an increase in the time between CR and US delivery might allow "not-CR" to occur in the period immediately before US. The probable result of such an occurrence would be a lessening of either CR magnitude or CR probability with the switch to $p(US/CS)<1.0$. On the other hand, if the extinction trials brought out a long-latency CR subclass, the temporal distance between CR and US might be decreased, relative to that at $p(US/CS)=1.0$.

Such an eventuality would very likely result in either enhanced response magnitude or increased response probability, as a result of the reduced opportunity for instances of "not-CR" to occur in the period between CR and US. These two hypothetical examples illustrate the possibility of either CR enhancement or CR diminution resulting from the rearrangement of temporal relations between the conditioned response and the reinforcer occasioned by the change in p(US/CS) from 1.0 to a value less than 1.0.

In summary, for the "Pavlovian" case of a change from 100% reinforcement to intermittent reinforcement there are two factors affecting the strength of CR on the intermittent schedule. The first, reinforcement of other response classes ("not-CR") brought out by the extinction trials, would tend to weaken the CR. The second, reinforcement of the various "new" subclasses of CR, brought out by the extinction trials, could have either an enhancing or a weakening effect on CR. In cases where the first factor is weak and the second factor operates to enhance CR it should be possible to see greater CR magnitudes at $p(US/CS)<1.0$ than at $p(US/CS)=1.0$. Such a result is almost unknown in the Pavlovian literature[6,7]. A factor that may be largely responsible for the absence of such results is the particular training procedures used in Pavlovian studies that compare CR magnitudes or CR probability at $p(US/CS)=1.0$ and at values less than 1.0.

Almost exclusively, Pavlovian studies of the effects of probability of reinforcement employ group designs with each group getting a single value of p(US/CS). With such a design, animals exposed to values of p(US/CS)<1.0 immediately encounter extinction trials. Exposure to even a few extinction trials when either CR magnitude or CR probability is still low will probably have a greater weakening effect than would exposure to the same number of extinction trials with CR at asymptotic strength. By weakening the tendency towards CR, the organism will be more likely to respond with "not-CR". Increasing the frequency of occurrence of "not-CR" will also increase the chances for US to follow and strengthen "not-CR". It appears, then, that animals acquiring a CR under conditions of intermittent reinforcement would have a greater tendency to respond with "not-CR" than would animals conditioned from the outset with p(US/CS)=1.0, and the conclusion is that the typically used group design almost assures a finding of weaker CRs on the intermittent schedule .

While a within-subjects manipulation of p(US/CS), starting with a value of 1.0, would avoid exposing the response immediately to extinction, there is another reason for performing such a manipulation. Only with a switch from a higher p(US/CS) to some lower value of p(US/CS) can the events proposed in this paper actually occur. Briefly, those proposed events were: 1) during exposure to p(US/CS)=1.0, response (CR) variability approaches a lower

asymptote; 2) with the introduction of extinction trials into the session, response variability increases (the source of the variability increase being the reappearance of previously reinforced behavior); 3) the increased response variability for the response-independent case is comprised of both CR subclasses and responses in the "not-CR" category as against the response-dependent case where the variability increase results only from changes in the response subclasses. This difference arises from the possibility of reinforcement of instances of "not-CR" with response-independent procedures whereas cases of "not-R" are not usually reinforced in response-dependent procedures; 4) if the decrease in p(US/CS) causes a reappearance primarily of cases of "not-CR", then a diminution of CR should result. However, if the decrease in p(US/CS) causes a reappearance primarily of subclasses of CR, then either increases or decreases in response strength may occur (this was the outcome predicted for the "operant" case earlier in this paper).

The present study will manipulate p(US/CS) within-subjects, starting at a value of 1.0 and decreasing to 0.06, with heart rate being the measured response. Use of a response-independent procedure might allow observation of effects consistent with the above-mentioned predictions. Specifically, it was argued that: 1) between-subjects variability in the direction of the heart rate CR would not be unexpected when using a response-independent

reinforcement procedure; and, 2) a decrease in p(US/CS) from 1.0 to 0.50 could result in either increases or decreases in response magnitudes for different animals.

1 Skinner (1931) proposed that operant responses were in fact preceded by stimuli, thereby allowing the operant to remain in the reflex tradition. Since the antecedent stimulus was usually unspecifiable, it was initially referred to as "little S". The matter of identification of these antecedent stimuli seems to have been ignored in Skinner's later works.

2 The present work will use the term reinforcer to refer to stimuli delivered in both "operant" and "Pavlovian" procedures. Both Skinner (1938) and Pavlov (1927) used the term "reinforcer", although for Skinner the stimulus reinforced the response while for Pavlov the stimulus reinforced a stimulus-response connection (reflex). Consistent with the current usage of "reinforcer", both "SR" and "US" will be used to symbolize the reinforcer.

3 There is an asymmetry in the use of "response-dependent" and "response-independent", with respect to the traditional "types" of conditioning and "types" of responses, that should be mentioned to avoid confusion. When a response-dependent procedure is used, the label "operant conditioning" is applied regardless of the "type" of behavior affected (i.e., operant or respondent). However, when a response-independent procedure is used, the label "operant conditioning" is applied only when an operant response is affected. If the response-independent procedure affects respondent behavior, then the label "Pavlovian conditioning" is applied. Restated, all response-dependent procedures are called "operant conditioning", while response-independent procedures may be called either "operant" or "Pavlovian", depending on the behavior affected. 4 The "paradoxical" status of the PREE finding stems from the view that both response rate during conditioning and number of responses to an extinction criterion are measures of a single aspect: response strength. However, certain approaches to the PREE (e.g., Sheffield's discrimination hypothesis (1949) and Capaldi's sequential hypothesis (1966)) have taken these two dependent measures as not reflecting a unitary property and therefore do not regard the PREE as paradoxical.

5 Gibbon, Farrell, Locurto, Duncan, and Terrace (1980) surveyed the classical conditioning literature with infrahuman subjects, examining those studies for evidence of a PREE. Of the twenty six studies with such data, nine showed more trials to extinction in animals with histories of intermittent reinforcement, as against histories of 100% reinforcement. While two thirds of the studies showed no PREE on a per trial basis, 24 of 26 studies showed no PREE when responses per omitted reinforcer was the criterion. Their review is in agreement with Gormezano and Coleman (1975) who stated:"... a number of infrahuman classical conditioning studies have revealed: little differences in

acquisition asymptotes under partial and continuous reinforcement; and failures to obtain the PREE" (emphasis supplied).

It should be noted that the findings from the human literature, with respect to the PREE, are different from those with infrahuman subjects. Gormezano and Coleman (1975) summarized these findings as follows: "In studies of human classical defense conditioning, two findings have consistently appeared: a relatively permanent detrimental effect of partial reinforcement (PR) on acquisition performance; and the partial reinforcement extinction effect (PREE) of greater resistance to extinction under partial than continuous reinforcement". Further examples of such findings are as follows. Humphreys (1939, 1940) observed greater resistance to extinction after intermittent reinforcement than after 100% reinforcement for both the eyeblink CR and the psychogalvanic CR in humans. Notterman, Schoenfeld, and Bersh (1952), measuring heart rate CR magnitude in humans, saw a definite PREE. Grant and Schipper (1952) observed greater resistance to extinction of the eyeblink CR in the 50% and 75% reinforcement groups than in the 100% group (using the same response measure, essentially the same result had already been found by Grant and Hake, 1951, and was later reported by Grant, Schipper, and Ross, 1952, and Hartman and Grant, 1960).

6 Razran (1955), measuring magnitude of salivary CR with humans, reported larger CRs with intermittent as against 100% reinforcement. Powell and Milligan (1975) conditioned two groups of rabbits with either 50% or 100% of CSs followed by shock to the eyelid. They measured both conditional eyelid responses and conditional heart rate responses. The initial deceleratory portion of the heart rate CR was seen to be larger in the group with p(US/CS)=0.50 than in the group with 100% reinforcement.

7 In the classical conditioning literature with infrahumans, reports of equal magnitude or probability of CR with 50% and 100% reinforcement predominate. While Pavlov (1927) reported roughly comparable rates of acquisition with 100% and intermittent reinforcement, Brogden (1939), measuring salivary CRs in dogs, may have been the first to report equal asymptotic levels with 100% and 50% reinforcement. Many subsequent works confirmed this latter finding. Two groups of such studies are available from Bitterman and his colleagues (Berger, Yarczower, and Bitterman, 1965; Gonzalez, Eskin, and Bitterman, 1963; Gonzalez, Longo, and Bitterman, 1961; Gonzalez, Milstein, and Bitterman, 1962; and Slivka and Bitterman, 1966) and from Fitzgerald and Vardaris (Fitzgerald, 1966; Fitzgerald, Vardaris, and Brown, 1966; and Vardaris, 1971). While most of the studies in the two preceding groups measured magnitude of the conditional response, studies by Thomas and

Wagner (1964) and Gormezano and Coleman (1975) employed the measure typically used in the human studies, percentage of trials with a CR, and contrary to the findings with humans, saw no differences between the 50% and 100% reinforcement groups. While certainly a minority finding, lower asymptotic levels with intermittent reinforcement have been reported with infrahuman subjects. For example, Holmes and Gormezano (1970), measuring percentage of trials with jaw movement, and Wagner, Siegel, Thomas, and Ellison (1964), working with magnitude of the salivary CR, both reported decreased reflex strength with 50% reinforcement in a classical conditioning paradigm using appetitive US.

Turning to the studies using human subjects, most of the reports are of weaker conditioning at 50% reinforcement than at 100%. Although Humphreys (1939, 1940) observed equal probability of eyelid CR and equal magnitude of psychogalvanic CR at both 50% and 100% reinforcement, a collection of subsequent reports from the Iowa and Wisconsin laboratories during the 1950s and the early 1960s revealed weaker conditioning with intermittent reinforcement (e.g., Grant and Hake, 1951; Grant, Schipper, and Ross, 1952; Hartman and Grant, 1960; Ross, 1959; Ross and Spence, 1960; and Spence and Trapold, 1961). In most of the above studies each group received a single value of p(US/CS). However, Ross (1959) employed within-subjects manipulation of p(US/CS). In that study subjects switched from 100% to 50% reinforcement fell to the same average level as subjects maintained at 50% reinforcement while subjects changed from 50% to 100% reinforcement increased response probability to the level of the subjects maintained on 100% reinforcement.

In the human literature there is at least one report of equal CR strengh with 100% and 50% reinforcement, from Notterman, Schoenfeld, and Bersh (1952), with heart rate as the measure.

8 Schoenfeld's (1968) account of events responsible for the PREE is not the only one available in the literature. Amsel's frustration account (1962), Capaldi's sequential hypothesis (1966), and Sheffield's discrimination explanation (1949) all attempt to explicate the PREE. The present paper is not comparing the relative merits of these explanations. Schoenfeld's account is being used because it is based on findings and assumptions that relate directly to the manipulations of the present study.

9 Although the present paper predicts a mixture of behavioral effects of lowering reinforcement frequency, there are cases for which uniform effects of such changes might be seen. As an example, consider an animal changed from a very low fixed ratio (e.g., 1:1) to a higher ratio (e.g., 10:1). Increased response rates are commonly observed with such a change (Ferster and Skinner, 1957). The prevalence of rate increases, rather than a mixture of rate increases and decreases, may result from a different

behavioral pattern now being followed by the reinforcer. Rather than the "eat, pause, press, eat ... " sequence of FR 1:1, the sequence is now "eat, pause, press ten times, eat ...". The reinforcer now follows and presumably strengthens a "run" of responses, a run which could not occur when the animal was interrupted by food after every response. Perhaps both increases and decreases in response rates would occur with a change to FR 10:1 if the bar was withdrawn after each unreinforced response for the length of time reinforcement would have taken.

10 Schoenfeld and Farmer (1970) used the notation "R" to indicate the measured response class. Behavior exclusive of that class was designated "not-R", and could be thought of as a class of behavior itself. The "not-R" class was defined as a specific period of time without the occurrence of "R". Just as there are subclasses of "R" (e.g., different forces of lever press), it could be argued that there should also be subclasses of "not-R" (e.g., all the different sets of behavior that could possibly satisfy a particular "not-R" requirement). There does not appear to be any reason to restrict the use of "R" and "not-R" to the case of response-dependent reinforcement. Reinforcers delivered response-independently may affect either a particular response class, such as lever press, or behavior other than lever pressing. Furthermore, there also does not appear to be any reason to restrict the use of "R" and "not-R" to responses typically called "operants" (e.g., lever presses or key pecks). The present study will use the notions of "R" and "not-R" when dealing with response independent reinforcement of heart rate. As a link with the Pavlovian tradition, such categories will be referred to as "CR" and "not-CR". In addition, the notion of subclasses will be used with respect to both "CR" and "not-CR". Although there may not be a precedent for this in the literature, it seems a legitimate extension of the Schoenfeld and Farmer idea.

11 Given the behavioral unit of either a lever press or a heart beat, investigators often use a change in the rate of occurrence of such events as an indication of conditioning (Pavlov, 1927, took increased rate of salivation during the cue as a measure of conditioning, while Estes and Skinner, 1941, took a change in lever press rate during the cue as evidence of conditioning). Consistent with such precedents, the present study will define the heart rate response as the difference in heart rate between the pre-CS period and the CS period (Schoenfeld, 1976, offers a comprehensive discussion of issues concerning definition of the response). Although it was argued that the heart beat could be further classified into subclasses, it remains a question whether or not the heart rate <u>response</u> can be so treated. To the extent that not every change in heart rate is the same (the latency from onset of a cue or the size of the change may

differ from one occasion to the next), it may be said that "heart rate responses" may be taken as a response class capable of division into subclasses. However, when successively larger changes in heart rate are "shaped" by means of requiring larger and larger heart rate responses for delivery of the reinforcer, it is not clear exactly what the reinforcer is affecting. Are the individual beats being affected? Is the entire pattern of beats that comprise the heart rate response being affected? Is the activity between the heart beats being affected (as the rat's activity between lever presses can be affected by reinforcer deliveries)? These questions will not be addressed in the present study but the notion of subclasses of the heart rate response will be employed.

12 In contrast to Pavlovians, operant workers almost routinely perform response "shaping", provide a history of regular reinforcement, and gradually increase intermittency of reinforcement. Through some or all of those manipulations, operant workers avoid having the response rate fall to near zero. If the response rate were to fall to near zero, the combination of a long pause followed by a response would probably be followed by reinforcer delivery. That combination of "long pause followed by a response" would be strengthened by such a delivery, perhaps leading to an increase in the strengh of such behavior and eventually to the establishment of stable low rates of responding. Rather, the above manipulations of the operant workers usually result in relatively small increases in the length of extinction (compared to what the animal has experienced up to that point), which may actually raise the prevailing response rates. Those increased rates may then be followed by a reinforcer, and the elevated rate may then become the predominant rate. In such fashion it is possible for decreased reinforcement rates to maintain higher rates of response (Morse, 1966). For that to happen, the response must have sufficient strength at the beginning of the extinction period and the extinction must not be so long as to cause response rates to fall. The utility of this approach is supported by the "success" of operant workers in sustaining behavior with intermittent reinforcement. Some Pavlovian workers have suggested and used prior exposure to 100% reinforcement as a means of sustaining the CR at intermittent values (Jenkins and Stanley, 1950; Notterman, Schoenfeld, and Bersh, 1952, respectively).

Method

Subjects

Eighteen experimentally naive, male, hooded rats (Long-Evans strain) served as subjects. The rats were approximately 100 days old upon arrival into the laboratory and they were first exposed to the experimental procedure approximately 60 days after their arrival. The rats were housed in individual cages for the duration of the study.

Apparatus

The experimental sessions were conducted in a standard, small-animal, operant conditioning chamber (Lehigh Valley Electronics). The chamber was 30.5 cm across the front face, 25-cm deep, and 27-cm high. It was enclosed in a large container designed to minimize extraneous acoustic and visual stimuli. The chamber was equipped with a solenoid operated dipper which was made available to the animal 1.5 cm up from the floor bars in the center of the side wall. A few centimeters to the right of the dipper was a standard operant conditioning lever which could be activated by a force of 20 gm. Three other exteroceptive stimuli could also be presented on this side wall. One of these stimuli, the dipper light, was a #327 (28 v) miniature bulb

positioned at the top of the 3-cm circular opening which provided access to the dipper. The house light, a 5-w, 28-v bulb (#CM1820) positioned in the middle of the side wall 2 cm from the ceiling, was used as CS for some rats. A small acoustic speaker, mounted midway between the dipper opening and the house light, presented CS for those rats not receiving the light CS. The electrocardiogram (ECG) was obtained using recording electrodes of the external belt type described by Ferraro, Silver, and Snapper (1965). This recording apparatus utilizes a belt wrapped around the rat's body behind the forepaws. A velcro strip was used as the actual belt with copper rivets serving both as the electrodes and as fasteners for the wires connected to the electrodes. The electrodes were approximately 4 cm apart and were equidistant from the thoracic midline when the belt was positioned. A spring, wound from .028-in diameter piano wire, was riveted to the belt and ran up to a swivel connection at the roof of the chamber from its attachment point over the rat's back. The wires from the belt electrodes were enclosed in metal shielding and were routed through the coil spring and out of the chamber and its outer casing to a mercury commutator mounted on the top of the outer chamber. This arrangement allowed the rats freedom of movement without twisting of the electrode wires.

Shielded wires connected the stationary side of the mercury commutator to a voltage coupler of a Beckman R611 polygraph. After passing through the coupler, preamplifier,

and amplifier, the voltage was inputted to an ink writer and to a digitizer fabricated from BRS solid state logic packages. The first two packages were an emitter follower, EF101 (an impedance matching device), and a Schmitt trigger, ST102, which was adjusted to fire only on large voltage changes, corresponding to the R wave of the ECG waveform. The Schmitt trigger output went to a one-shot (a pulse former) and then to a relay driver before inputting to a State Systems Inc. interface from which the signal finally reached a Digital Equipment Corp. PDP-8/e computer. The computer control of events in the experimental chambers and the on-line recording of data was accomplished by programs written in SKED (Snapper, Kadden, and Inglis, 1982).

Procedure

The adaptation of the rat's heart rate to the conditioning situation and to the electrode belt has been shown to be a slow process that can continue for 15 to 25 sessions (Snapper, Ferraro, Schoenfeld, and Locke, 1965). In order to avoid having this adaptation occur during the collection of experimental data, all subjects were given adaptation sessions prior to the experimental manipulations. A total of 44 adaptation sessions were conducted with the first 13 sessions lasting 30 min each and the remaining sessions lasting 50 min each. For the first few adaptation sessions, only the velcro strip was placed around the rat. Subsequent sessions involved shaving of the subject prior to

the session (see Snapper, et al., 1965, for details), fitting the electrode harness, and fastening the protective spring to the swivel at the top of the chamber. During the adaptation sessions no stimuli were presented to the animals. After these 39 sessions all rats received the final five adaptation sessions with the only change being the application of electrode paste to the recording electrodes before fastening the belt.

Before the adaptation was completed, rats 5, 13, and 18 became so agitated and difficult to handle that they were dropped from the study. This agitation, which resulted from the animals becoming entangled with the spring leading from the belt to the roof of the chamber, made removal of the belt exceedingly difficult. This difficulty was also encountered by Schoenfeld, Matos, and Snapper (1967) and appears to be a major drawback of the belt/spring arrangement.

Of the 15 rats remaining, 9 were randomly selected to be advanced to the experimental conditions (rats 1, 2, 3, 7, 8, 9, 14, 15 and 16). For these animals, CS was a 3000-cps tone with an intensity of 80 db (re .0002dynes/sq cm) and US was presentation of 0.01-cc water and illumination of the dipper light. These animals received an initial session consisting of 30, 8-sec CS presentations. On the following day, these rats received a session of 30, 4-sec presentations of the 0.01-cc water US. The rats then received training to insure they would actually consume US

(dipper training). In those sessions the dipper was repeatedly presented until the animal reliably moved towards it and consumed US. After those sessions CS/US pairings commenced.

The first experimental condition, like all subsequent conditions, consisted of trials composed of 12 sec of CS with access to US, if scheduled for delivery, during the final 4 sec of CS. Data recording commenced 8 sec prior to CS onset and terminated 8 sec after US offset. The data taken were the number of heart beats occurring in each 2-sec segment (bin) of the 28-sec recording period and whether or not the animal consumed US. The drinking response data were manually recorded by the experimenter based on visual observation of S's behavior in the chamber.

For the first three experimental conditions, 36 trials were presented each session. Intertrial intervals were randomly selected from seven durations ranging from 86 sec to 146 sec. In the first experimental condition, all nine rats received 10 sessions of CS/US pairings, with both CS and US presented on every trial (p(US/CS)=1.0), and US being 0.01cc water. The second condition consisted of 5 sessions with the US magnitude increased to 0.08cc. For both the first and second conditions the rats had not had access to water for 22 hr prior to the experimental sessions. The third experimental condition consisted of five sessions with the rats at 48-hr water deprivation and US magnitude of 0.08 cc. The changes in US magnitude and deprivation level were

intended to increase the magnitude of the observed cardiac rate conditional response.

Based on a preliminary analysis of the data from the above conditions, it was decided to substitute milk for water as the US in a further attempt to increase the magnitude of the cardiac rate CR. The rats were given free access to water but had restricted food intake and were thereby gradually reduced to 90% of their free feeding body weights and maintained at that level by measured daily feedings right after the experimental sessions. The milk was a mixture of 2-parts water to 1-part Borden's Eagle Brand sweetened condensed milk.

The six animals that had been previously adapted to the experimental procedure but not exposed to the CS-water pairings (rats 4, 6, 10, 11, 12, and 17) were given 4 more adaptation sessions. They were then given a CS-only session, a US-only session, and then shaped to drink from the dipper. These procedures were carried out in a manner identical to those for the first group of rats with the exception that CS was illumination of both the houselight and the dipper light, instead of presentation of a tone.

The entire group of 15 rats was then exposed to 10 sessions of conditioning with p(US/CS)=1.0 and a 0.08-cc milk US at 90% body weight. After these sessions, in another effort to increase the cardiac rate CR magnitude, the rats were further reduced to 80% of their free feeding weights and were maintained at that level for the remainder of the study.

During the conditioning sessions with the milk US, several additional rats (# 2, 4, 9, 14, and 17) became too difficult to harness and unharness and were dropped from the study. Thus, 10 rats remained for exposure to the values of p(US/CS). All of these 10 animals successfully completed the remaining experimental conditions. Of those 10 rats, six had a prior conditioning history with water US and received tone CS, while the four others had no prior history with CS/water pairings and received light CS.

All subsequent experimental conditions were conducted with 0.08-cc milk US and at 80% body weight. Sessions consisted of 32 trials with p(US/CS) meeting the programmed value during both the first 16 and the second 16 trials of each session (e.g. if p(US/CS) was 0.50 then eight USs would occur in the first 16 trials of the session and eight would occur in the second 16 trials of the session). The selection of a given trial as either US or non-US was made by randomly sampling a finite population of elements without replacement. As a general rule, data were taken only from the second half of the session in order to allow the within-session adaptation, observed by Snapper, et al. (1965), to occur. Only data from the last two sessions of an experimental condition were used for analysis.

The sequence of experimental conditions with the animals at 80% of their free-feeding weights was: 5 sessions with p(US/CS)=1.0 at 22-hr deprivation; 8 sessions with p(US/CS)=0.50 at 22-hr deprivation followed by 4 sessions

with p=0.50 at 0-hr deprivation; 6 sessions with p(US/CS)=0.25 at 22-hr deprivation followed by 4 sessions with p=0.25 at 0-hr deprivation; 6 sessions with p(US/CS)=0.125 at 22-hr deprivation followed by 4 sessions with p=0.125 at 0-hr deprivation; 6 sessions with p(US/CS)=0.06 at 22-hr deprivation followed by 4 sessions with p=0.06 at 0-hr deprivation; and finally, 6 sessions with p(US/CS)=1.0 at 22-hr deprivation followed by 4 sessions with p=1.0 at 0-hr deprivation. For those sessions with 0-hr deprivation, the rats were given their daily ration of food 1 hr prior to their experimental sessions.

After the probability manipulations, 24 discrimination training sessions were carried out. For all rats, sessions consisted of 8 presentations of a 4-kHz tone followed by US and 16 presentations of a 5-kHz tone not followed by US. Timing of CS and US and data recording were the same as in the previous conditions.

Results and Discussion

Figures 1-22, which comprise the first part of this section, contain basically two kinds of data presentation. One variety involves displaying cardiac rate during periods immediately before, during and subsequent to a CS/US pairing. These data depict the "shape" of CR (the time course of cardiac rate changes) and, when individual subjects are displayed, they give the reader a feel for response variability between subjects. The other type of data presentation shows one of a number of dependent variables as a function of the independent variable, p(US/CS). These latter functions are group averages and, while more removed from the raw data, reveal the effects of the independent variable more clearly than the bin-by-bin presentation.

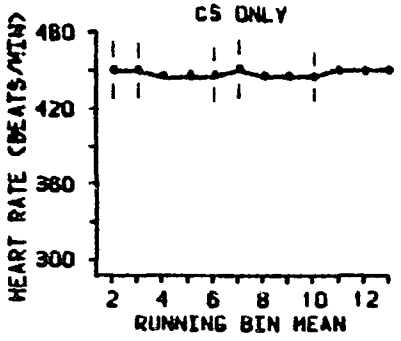
Pre-CS, CS, and post-US heart rate at 22-hr deprivation.

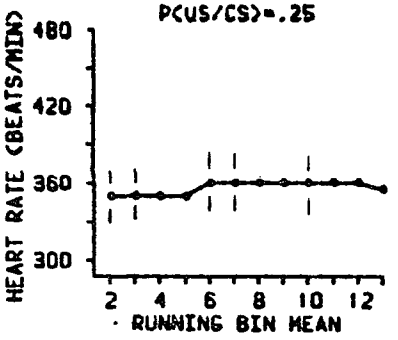
Cardiac rates during each 2-sec bin of the 28-sec recording period are presented for individual subjects in Figures 1-9. The data in Figures 1-9 were collected as the number of heart beats occurring in each of the 14, 2-sec bins that made up a recording period. Data in these figures were obtained with the rats at 22-hr food deprivation.

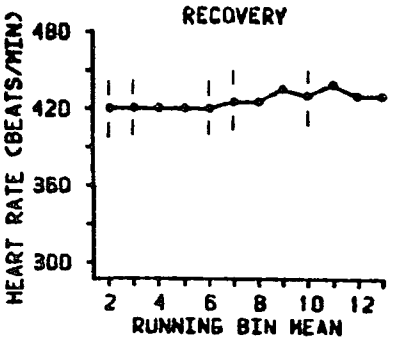
Rather than present heart rate in every 2-sec bin, running means of heart rates in three successive bins are plotted. Three bin running means were used in order to reduce the variability in heart rate between bins, thereby making the differences between pre-CS heart rate and CS heart rate more visible. However, the reading of Figures 1-9 needs explanation in light of this use of running means. Running bin means 2 and 3, the means of bins 1, 2, and 3, and bins 2, 3, and 4, respectively, best represent pre-CS heart rate (the 8 sec before CS onset). Likewise, running bin means 6 and 7, the means of bins 5, 6, and 7, and bins 6, 7, and 8, respectively, most accurately represent heart rate during CS (only CS was presented during bins 5-8). Finally, running bin mean 10, the mean of bins 9, 10, and 11, most closely reflects heart rate during presentation of US which occurred during bins 9 and 10. These data have been plotted as heart-beats/min (bpm) to make them more comparable with other cardiac conditioning studies.

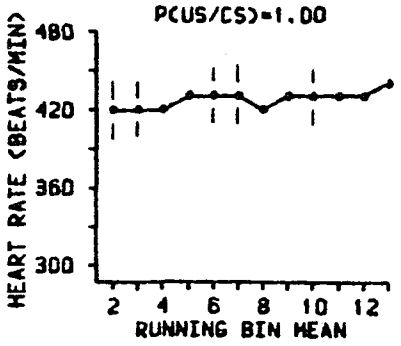
In Figures 1-9, panel 1 is intended to show the unconditional response to CS and thereby provide a basis for evaluating whether or not a conditional cardiac rate response developed when CS/US pairings commenced. For rats 6, 10, 11, and 12 the first panel indicates the cardiac rate changes in sessions composed only of CS presentations. However, for rats 1, 3, 7, 8, and 15, the first panel shows the cardiac rate during the first ten CS/US pairings. The data from the "CS-only" trials for the latter animals were

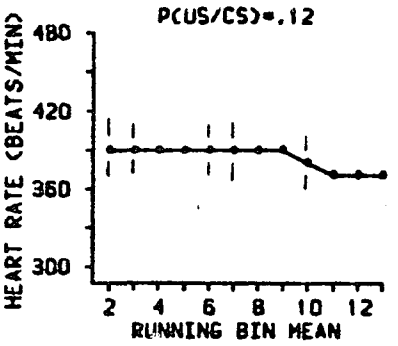
Figure 1-9. Individual rats' heart rates in 2-sec recording periods before, during, and after a CS/US pairing at 22-hr food deprivation. Three successive 2-sec bins have been averaged together to give three-bin running means. For example, the running mean for bin n is the mean of heart rate in bins n-1, n, and n+1. The running means that best represent heart rate during pre-CS, CS, and US are indicated by vertical lines. The individual bin heart rates were obtained by taking a median of all heart rates for a particular bin during the second half of the session for the last two days at an experimental point. Each panel in Figures 1-9 represents a different experimental condition.

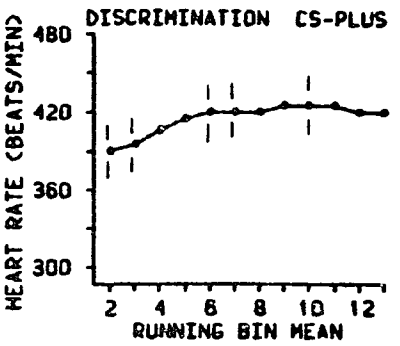

CS ONLY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

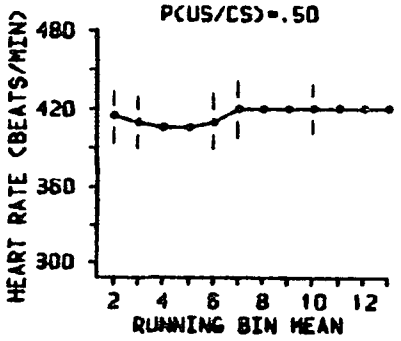

P(US/CS)=.25
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

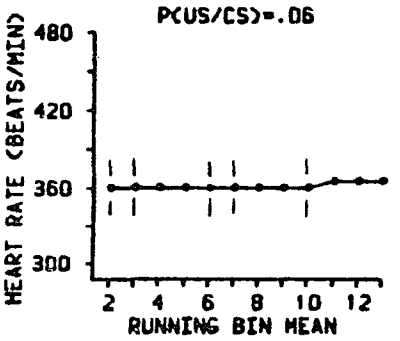

RECOVERY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=1.00
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.12
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.50
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.06
HEART RATE (BEATS/MIN)
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420
360
300
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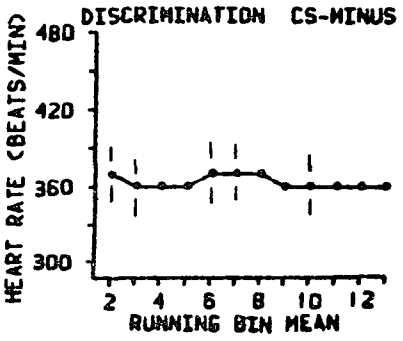
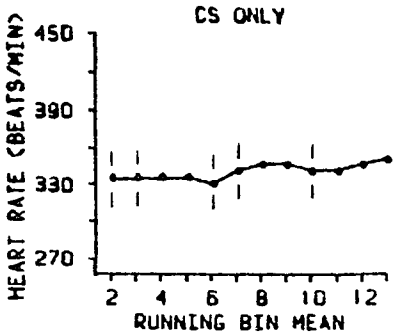
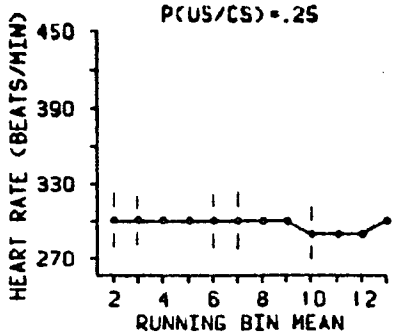
RUNNING BIN MEAN


DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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390
330
270
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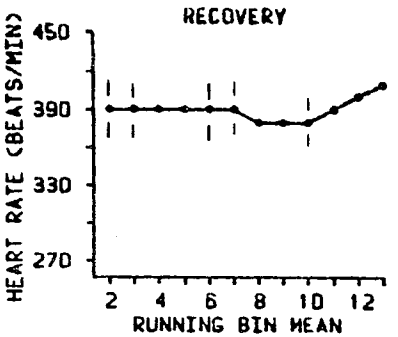
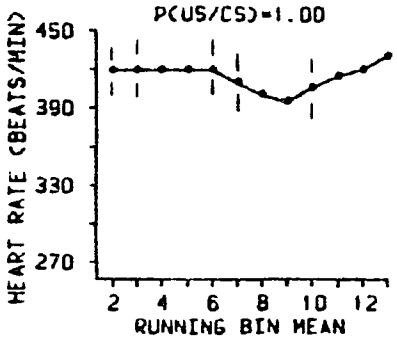
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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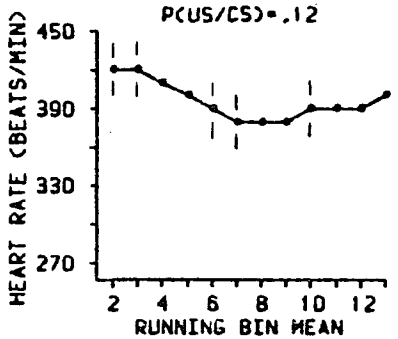
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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390
330
270
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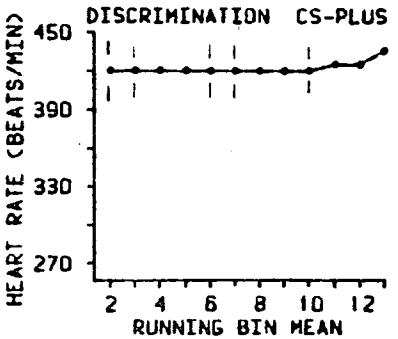
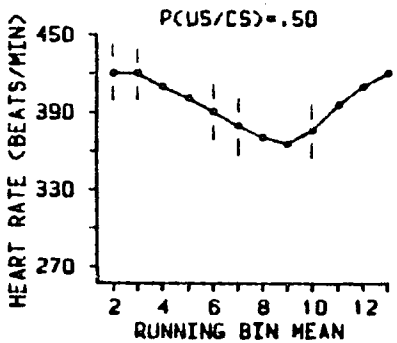
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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HEART RATE (BEATS/MIN)
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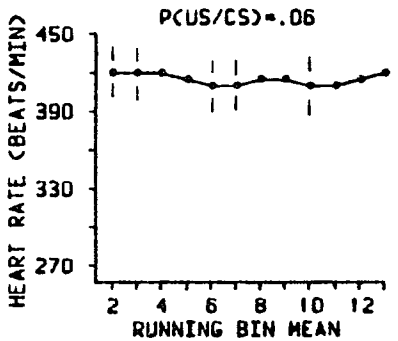
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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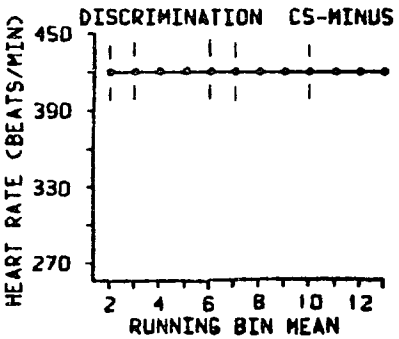
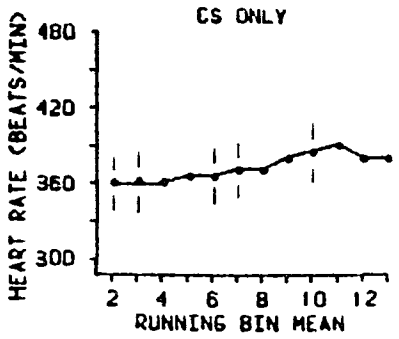
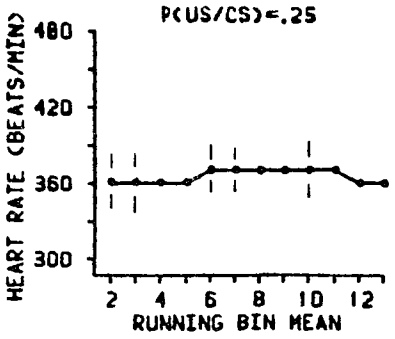
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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360
300
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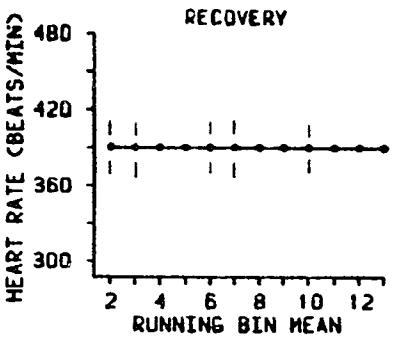
RECOVERY
HEART RATE (BEATS/MIN)
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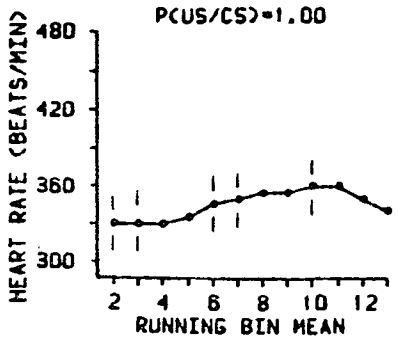
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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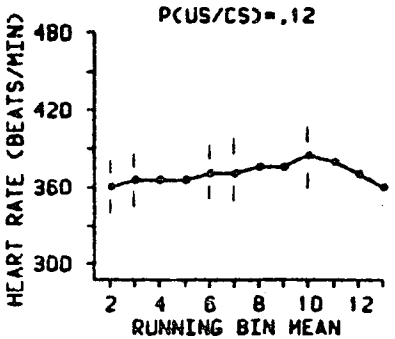
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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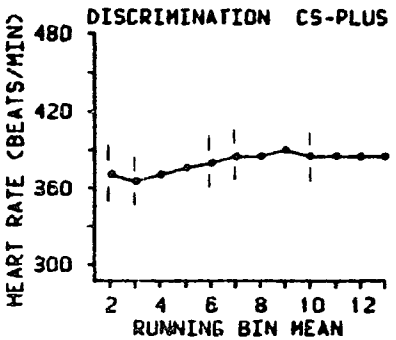
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
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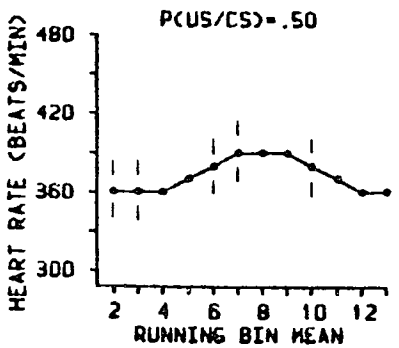
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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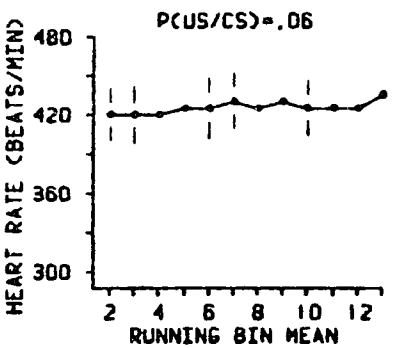
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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300
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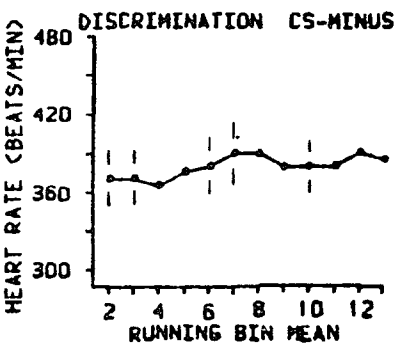
DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
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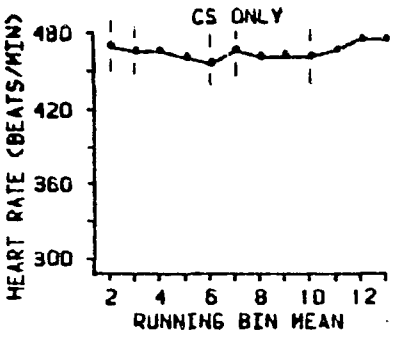
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
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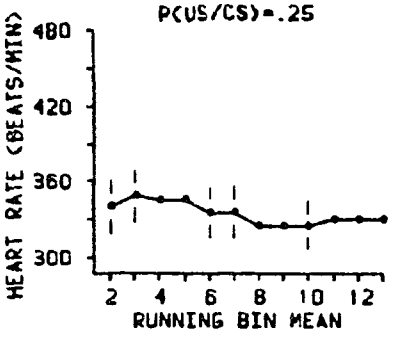
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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360
300
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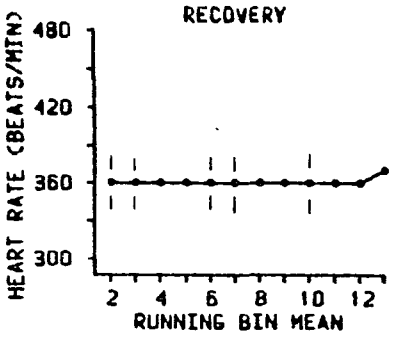
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
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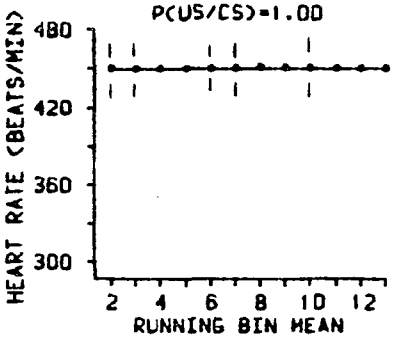
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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HEART RATE (BEATS/MIN)
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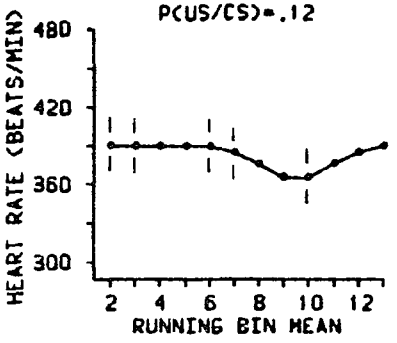
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.12
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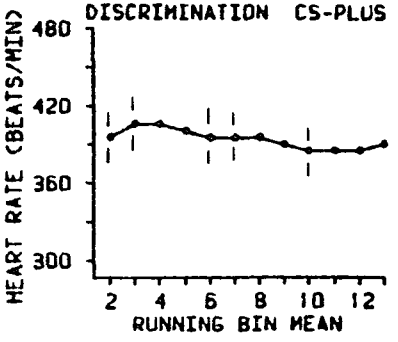
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
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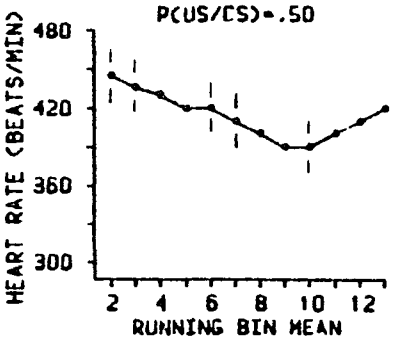
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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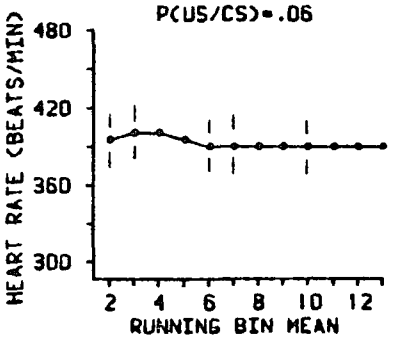
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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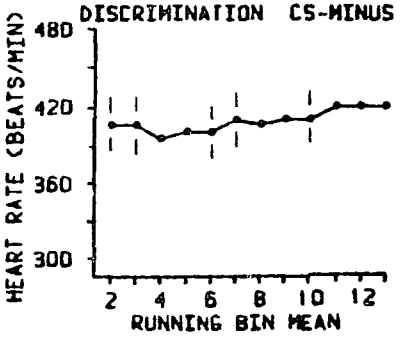
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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HEART RATE (BEATS/MIN)
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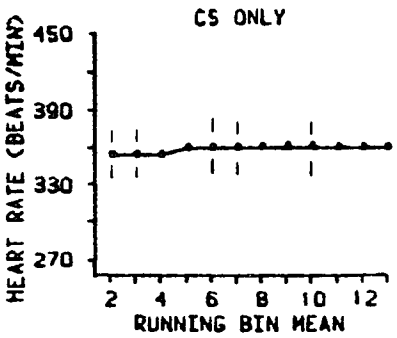
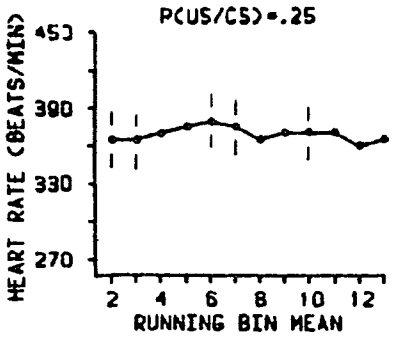
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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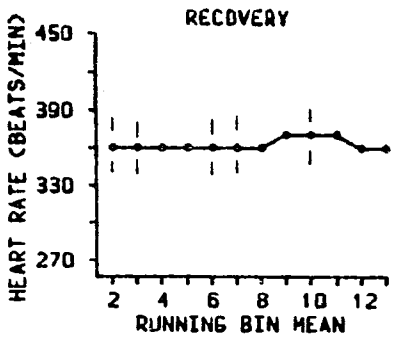
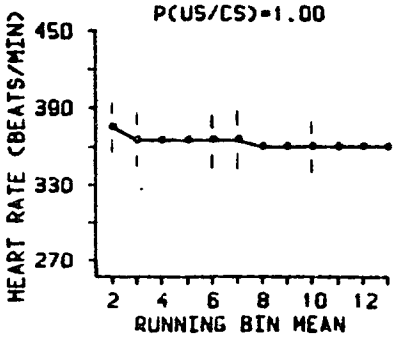
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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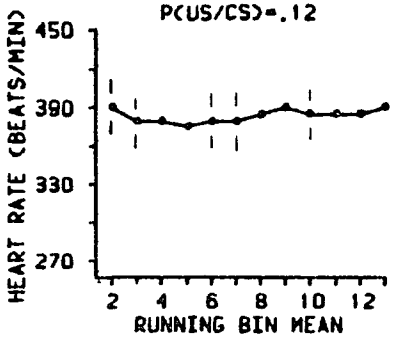
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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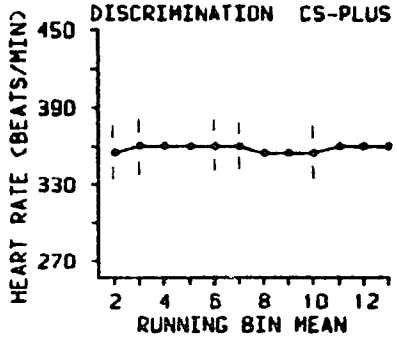
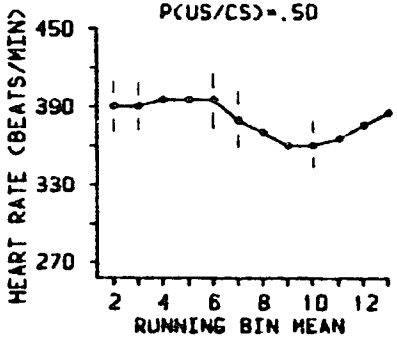
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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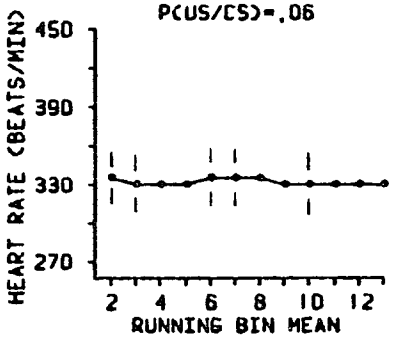
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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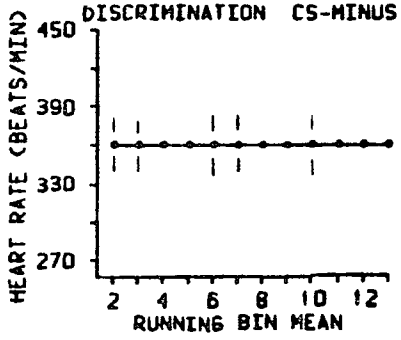
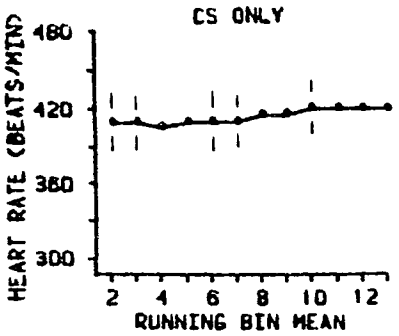
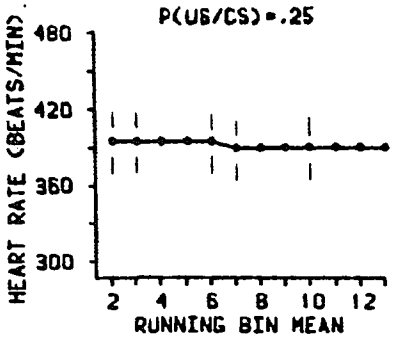
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.25
HEART RATE (BEATS/MIN)
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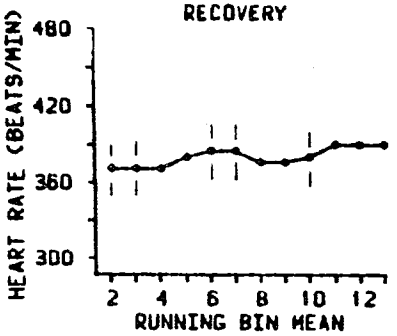
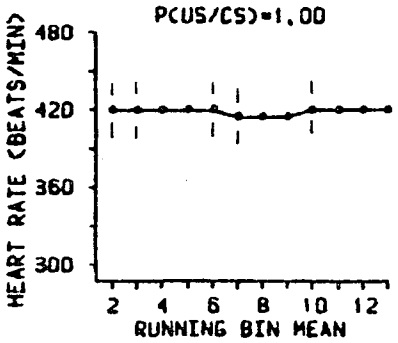
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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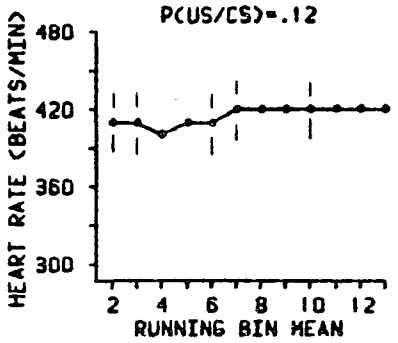
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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300
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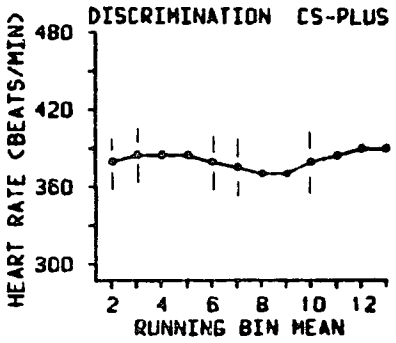
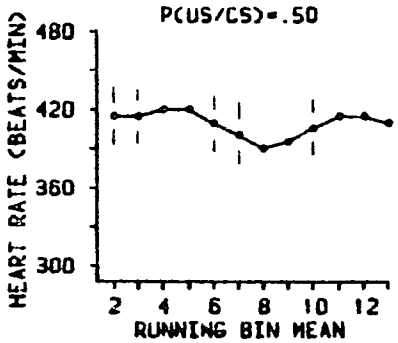

DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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HEART RATE (BEATS/MIN)
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300
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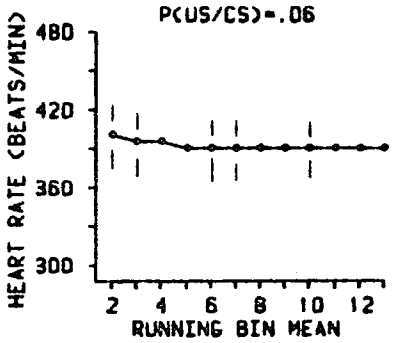
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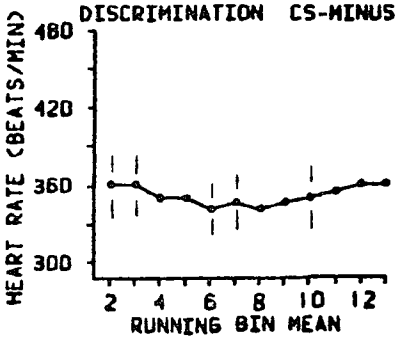
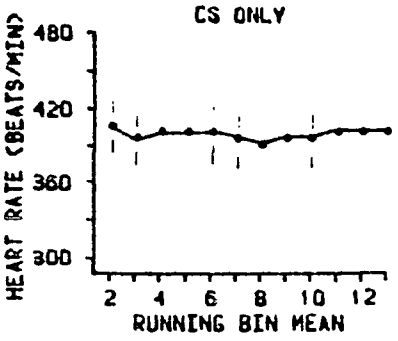
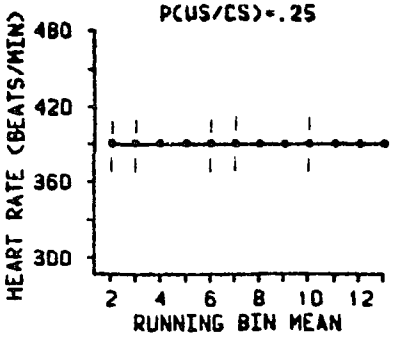

DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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300
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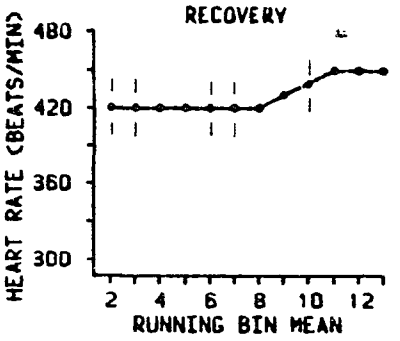
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
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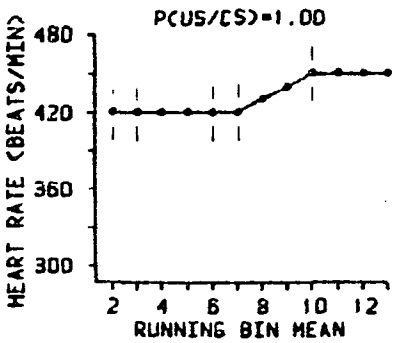
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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360
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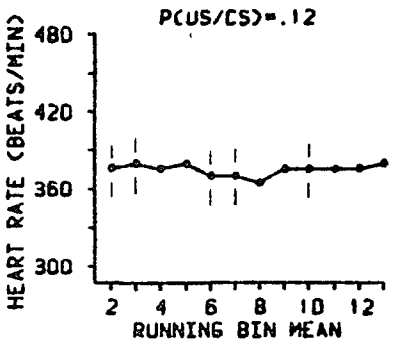
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420
360
300
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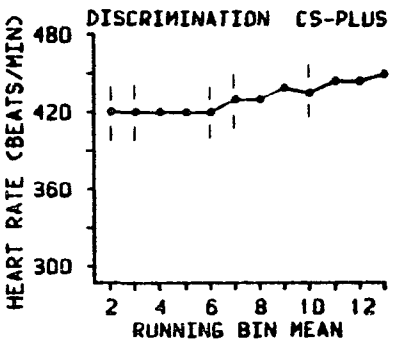
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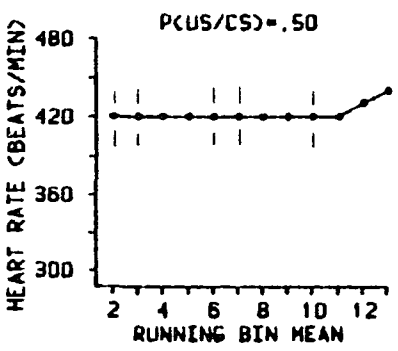
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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420
360
300
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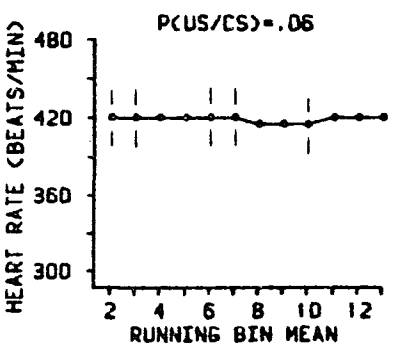
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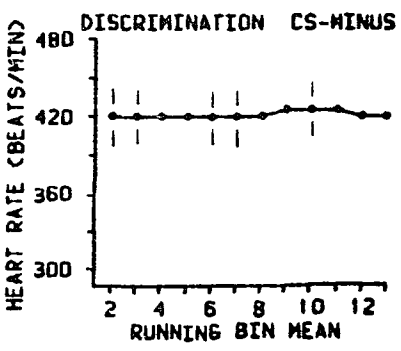
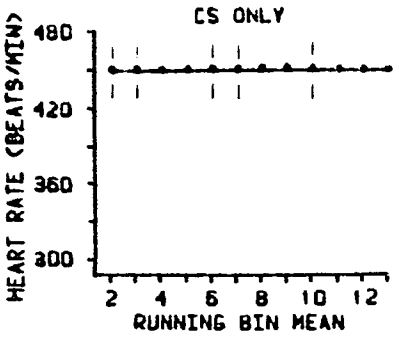
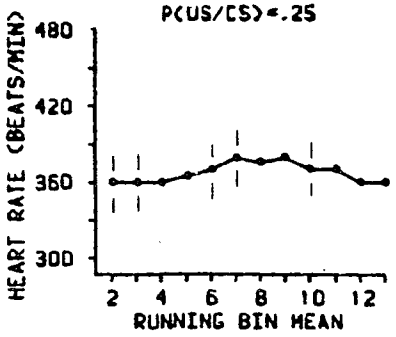
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.25
HEART RATE (BEATS/MIN)
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420
360
300
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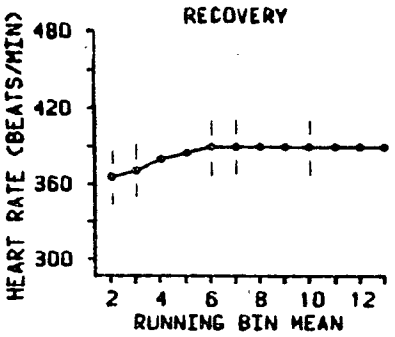
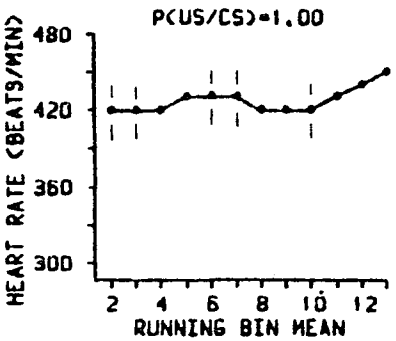
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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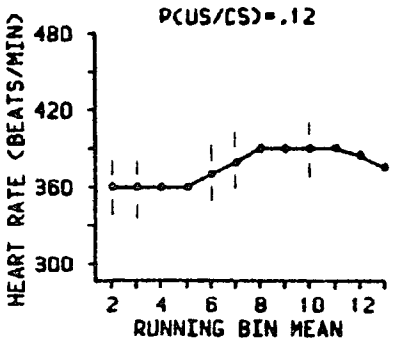
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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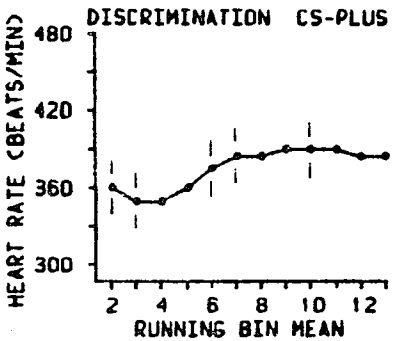
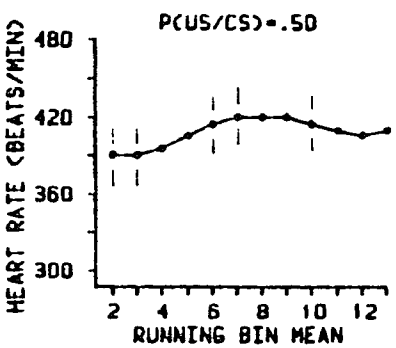
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN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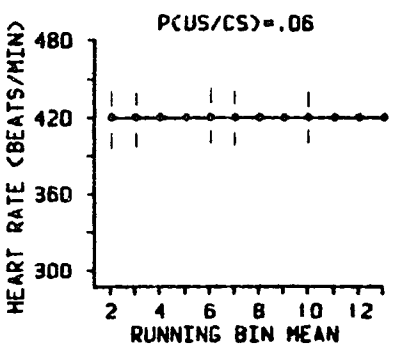
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.06
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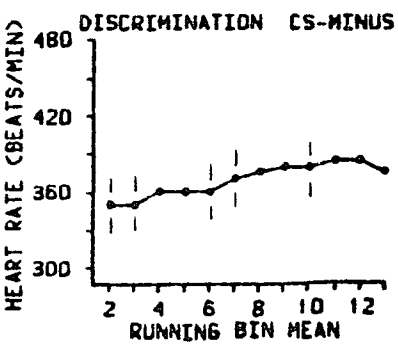
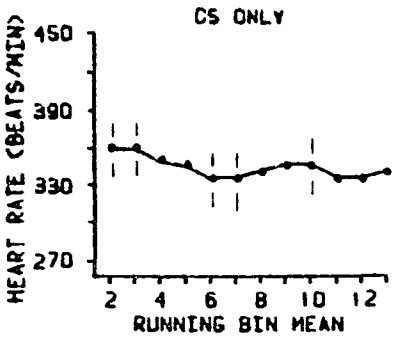
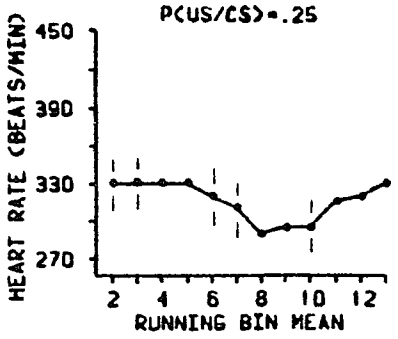
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


CS ONLY
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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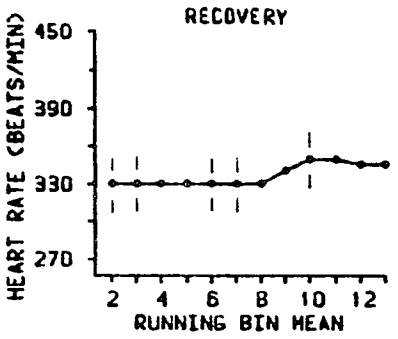
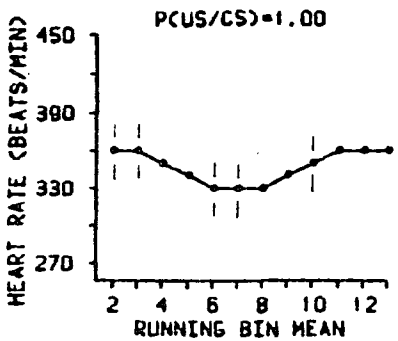
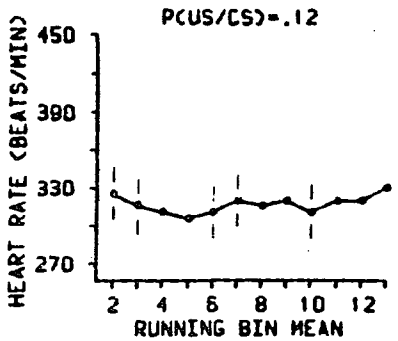
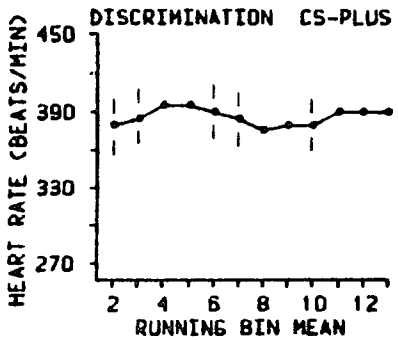
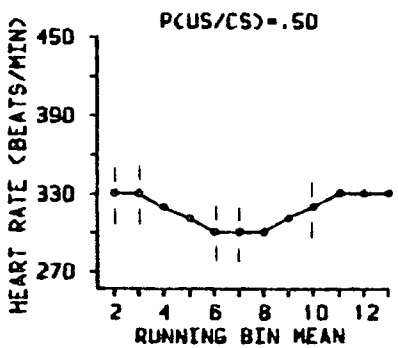
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=1.00
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.12
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN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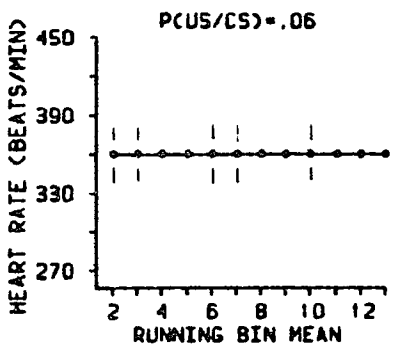
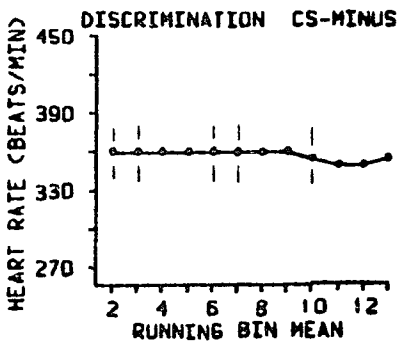
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.06
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
450
390
330
270
2 4 6 8 10 12
RUNNING BIN MEAN

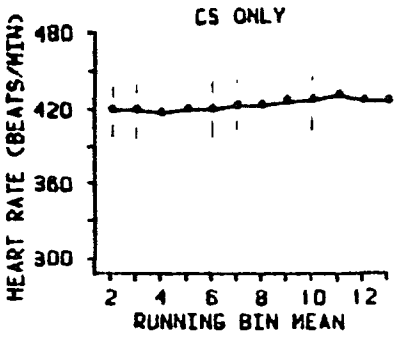
lost as a result of a problem with the data recording system. Therefore, data from the first few conditioning trials were the next best source to evaluate the unconditional response to CS. Available evidence indicates very little cardiac conditioning in rats in only ten trials when the parameters have values similar to those of the present study (Caul and Miller, 1968; Schoenfeld, Matos, and Snapper, 1967). The first panels of Figures 1-9 show the unconditional response to CS to be of very small magnitude for almost all rats. Of the nine rats, only rat 15 displays a sizeable response to CS during either CS-alone or the early conditioning trials. Inspection of individual trial data revealed that this animal's responses to CS were of large magnitude for conditioning trials 1-5 of Day 1 and then averaged zero magnitude for trials 6-36 of Day 1. During rat 15's subsequent conditioning sessions a conditional heart rate response slowly developed from the zero level seen on both Day 1 (after trial 5) and on Day 2 (as a session average). Therefore it seems reasonable to include rat 15 in the general statement that the unconditional heart rate response to CS was of small magnitude.

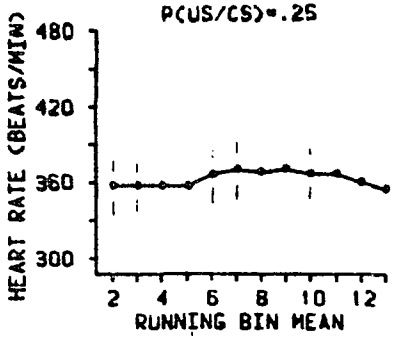
The effects of repeated pairings of CS with milk (US) on the cardiac rate response to CS may be seen by comparing panels 2 and 3 with panel 1 in each Figure. Approximately half the rats show an increased heart rate response to CS in panel 2, p(US/CS)=1.0, compared to that seen in panel 1.

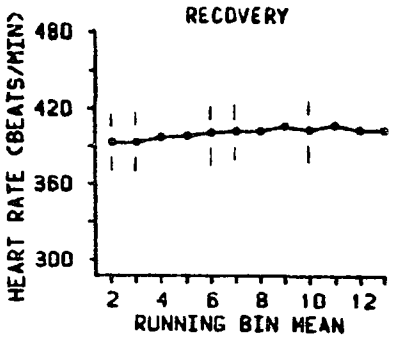
The other half of the subjects show larger heart rate changes to CS in panel 3, p(US/CS)=0.50, than in panel 1. Comparing panel 1 with either panels 2 or 3 in Figures 1-9, it appears that almost all rats developed conditional cardiac rate responses after exposure to either the 1.0 or 0.50 value of p(US/CS). Two exceptions to this general pattern deserve mention. Rat 15 had as large a response to CS during the first ten CS/US pairings (panel 1) as it did after several weeks of CS/US pairings (panel 2). However, given the transient nature of the initial large magnitude response to CS (only 5 trials) and the subsequent gradual acquisition of the large magnitude response seen in panel 2, it seems justifiable to conclude that rat 15 also developed a conditional cardiac rate response as a result of repeated CS/US pairings. Rat 11, the other exception, appears to have not developed a conditional cardiac response at any value of p(US/CS). Since this rat's CR magnitude was consistently close to zero, it was not included in most of the subsequent analyses.

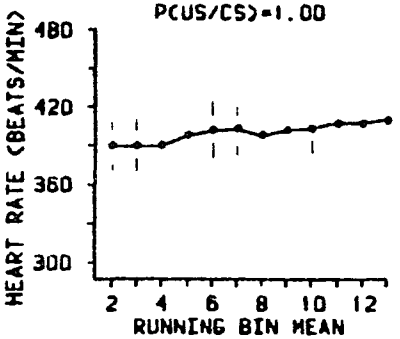
Since the subjects were in a sense naturally divided by the direction of their conditional heart rate responses, average responses were computed for the rats with acceleratory CRs (rats 1, 6, and 12) and for the rats with deceleratory CRs (rats 3, 7, 8, 10, and 15) and are shown in Figures 10 and 11. These averaged data, as would be expected from the individual functions, confirm the existence of two distinct topographies in the experimental subjects.

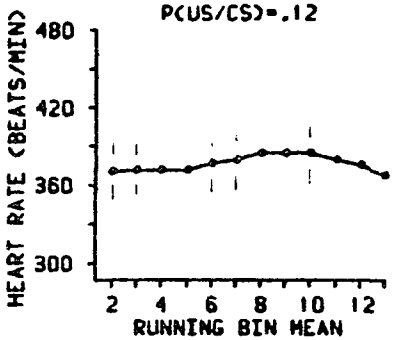
Figure 10 and 11. Group heart rate in 2-sec recording periods before, during, and after a CS/US pairing. Running bin means are used in these Figures exactly as in Figures 1-9. The average for the acceleratory CR group (rats 1, 6, and 12) is shown in Figure 10 while the average for the deceleratory CR group (rats 3, 7, 8, 10, and 15) is shown in Figure 11. Each panel in these figures was obtained by averaging across bins of the appropriate panels from Figures 1-9.

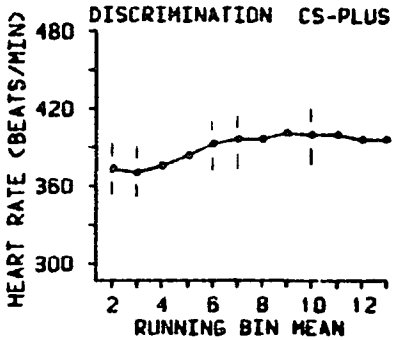

CS ONLY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

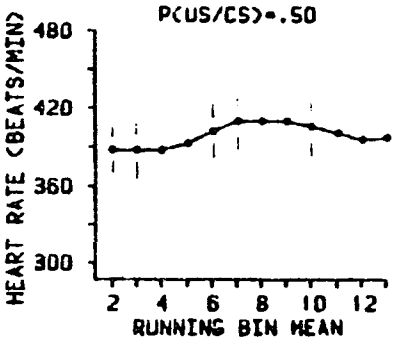

P(US/CS)=.25
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


RECOVERY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=1.00
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.12
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.50
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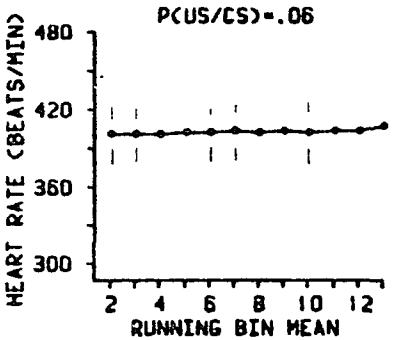
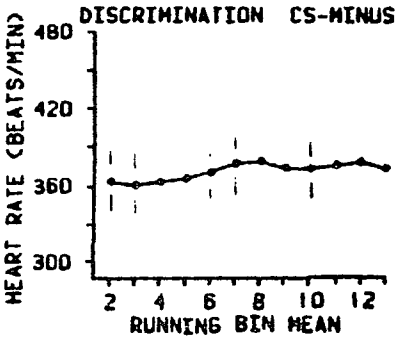
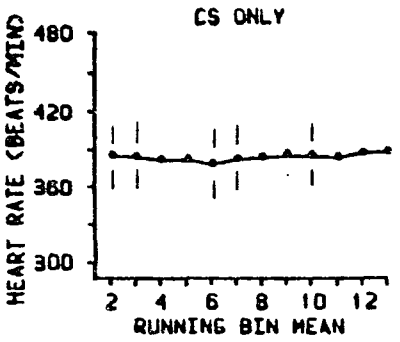
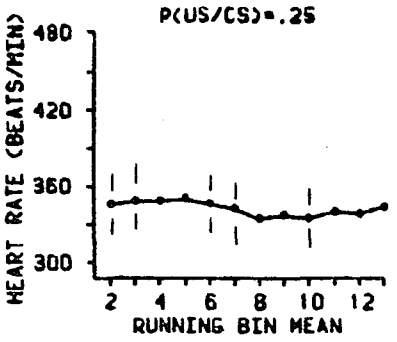
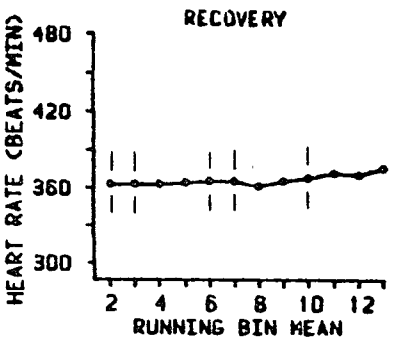
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

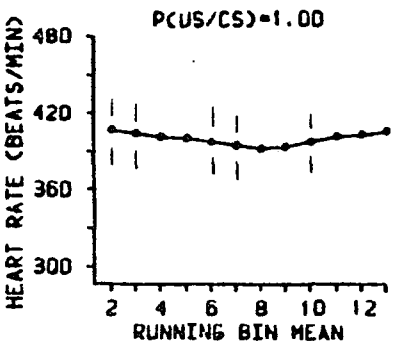

P(US/CS)=.06
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

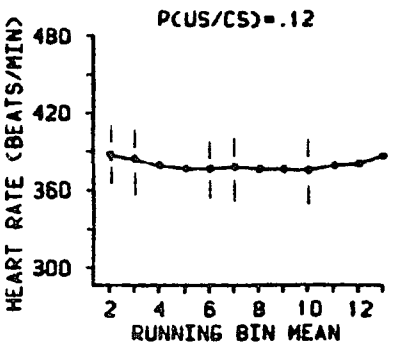

DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

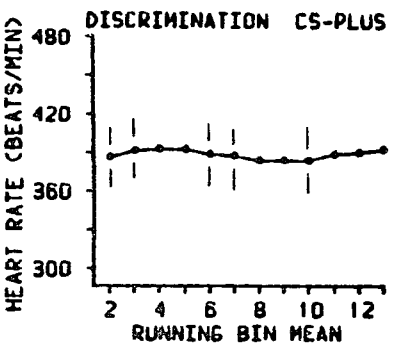

CS ONLY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

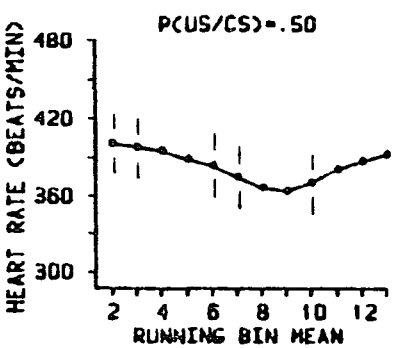

P(US/CS)=.25
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

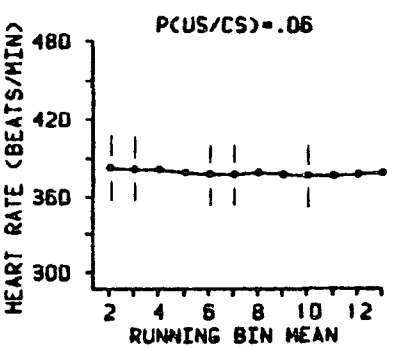

RECOVERY
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

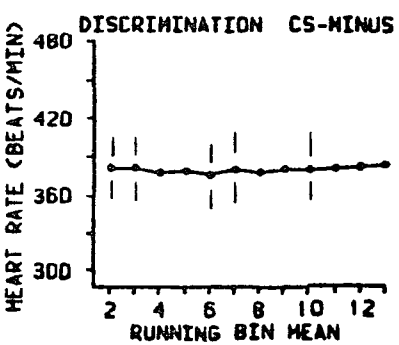

P(US/CS)=1.00
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.12
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-PLUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.50
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


P(US/CS)=.06
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN


DISCRIMINATION CS-MINUS
HEART RATE (BEATS/MIN)
480
420
360
300
2 4 6 8 10 12
RUNNING BIN MEAN

Up to this point only the data in the leftmost column of Figures 1-11 have been mentioned. The center column indicates the bin-by-bin heart rate at the 0.25, 0.12, and 0.06 values of p(US/CS). The upper panel of the rightmost column contains the data from a second exposure to p(US/CS)=1.0 at 23-hr deprivation. From Figures 10 and 11 it can be seen that the accelerators display CRs at the original and second exposure to p(US/CS)=1.0 that are more similar in magnitude than those of the decelerators at those same conditions. The next two panels of the rightmost column display the responses to CS-plus and CS-minus at the end of discrimination training. Turning again to Figures 10 and 11, it is clear that the rats with acceleratory CRs displayed differential responding while the rats with deceleratory CRs failed to develop a discrimination.

The present report is an addition to the small group of studies that have conditioned cardiac rate using appetitive unconditional stimuli instead of electric shock US (Randall, Brady, and Martin, 1975; and, Schoenfeld, Matos, and Snapper, 1967). The fact that all but one subject developed a cardiac rate CR with this US indicates that cardiac rate conditioning can be reliably accomplished with a "non-stressful" US. Additionally, the data from individual rats in the present study puts to rest completely the notion of the existence of a "true" direction of the cardiac rate CR. With both species and procedure remaining constant (either of which may influence the direction of the heart

rate CR), some rats in the present study developed acceleratory CRs (as a session average) while others developed deceleratory CRs.

Note: It was stated in the Method section that 10 animals successfully completed the adaptation procedure and moved through the experimental points. However, rat 16, whose data were not included in the individual functions (Figures 1-9), experienced an inappropriate experimental condition and therefore was considered too compromised to be a valid subject after that point. The error was receiving an entire session with the light CS, on Day 4 of p(US/CS)=0.50 at 22hr deprivation, instead of the tone CS which this animal had been receiving before that session.

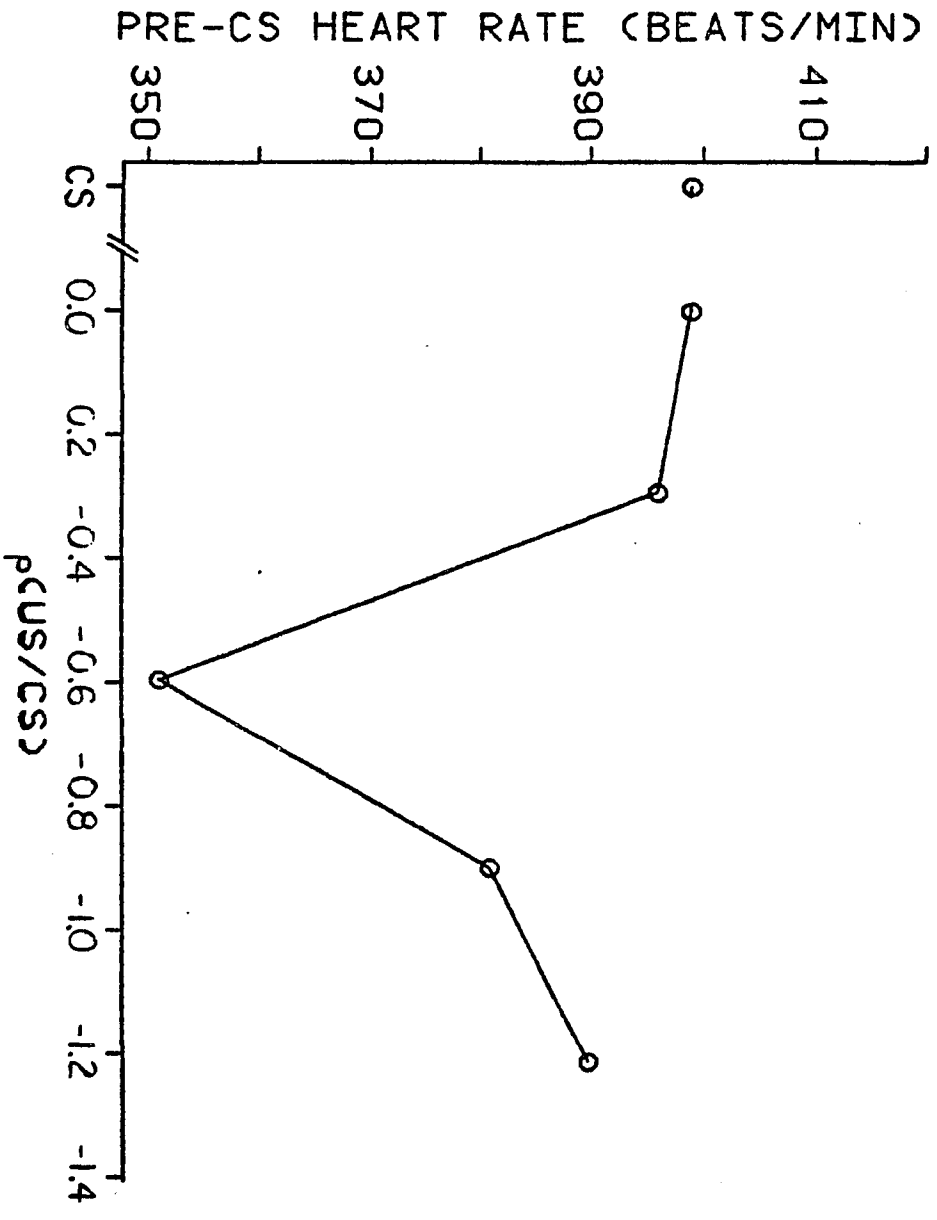
Effects of p(US/CS) on pre-CS heart rate (high deprivation)

Turning from the temporal pattern of heart rate changes during conditioning trials presented in Figures 1-11, Figure 12 presents changes in the pre-CS heart rate as a function of p(US/CS). Pre-CS heart rate is defined as the mean heart rate in bins 1-4 and is presented as a group average for both the rats with acceleratory CRs and the rats with deceleratory CRs. Both of these functions are clearly nonmonotonic with minima at p(US/CS)=0.25. The similarity in the shapes of the two functions in Figure 12 shows that changes in p(US/CS) have similar effects on the pre-CS heart rates of both the rats with acceleratory CRs and those with deceleratory CRs. Given the similarity of the functions for

Figure 12. Pre-CS heart rate, the mean heart rate in bins 1-4, as a function of p(US/CS). Each function is based on an aver- age of either the rats with acceleratory CRs (circles) or the rats with deceleratory CRs (triangles). These data were obtained at 22-hr food deprivation. Note the logarithmic scale on the abscissa. The first point of the function on the left end is for p(US/CS)=1.0. Moving to the right, the other points are for 0.50, 0.25, 0.12, and finally 0.06 on the extreme right.

PRE-CS HEART RATE (BEATS/MIN)
330
370
410
CS
0.0
-0.2
-0.4
-0.6
-0.8
-1.0
-1.2
-1.4
p(US/CS)

Figure 13. Pre-CS heart rate as a function of p(US/CS) for all eight rats at 22-hr food deprivation. This function is an aver- age of the two functions in Figure 12. Note the logarithmic scale on the abscissa. The first point of the function on the left end is for p(US/CS)=1.0. Moving to the right, the other points are for 0.50, 0.25, 0.12, and finally 0.06 on the extreme right.


PRE-CS HEART RATE (BEATS/MIN)
350
370
390
410
CS
0.0
-0.2
-0.4
-0.6
-0.8
-1.0
-1.2
-1.4
pCUS/CS)

these two subgroups, it seemed appropriate to combine all the rats to obtain a grand mean function. Figure 13 presents pre-CS heart rate as a function of p(US/CS) averaged across all eight rats. As expected, there is agreement between the shapes of the functions for the two subgroups in Figure 12 and the grand mean function in Figure 13.

The finding of changes in pre-CS heart rate with changes in p(US/CS) extends to single organisms the effects seen by Miller, Banks, and Caul (1967) and by Caul and Miller (1968) in their group designs. After exposing groups of animals to values of p(US/CS) of 1.0, 0.50, and 0.0, Caul and Miller observed a minimum in pre-CS heart rate at p(US/CS)=0.50 and suggested that "US uncertainty" was responsible for the decrease in pre-CS heart rates. The present study observed a definite minimum in the function relating pre-CS heart rate to p(US/CS) at $\underline{p}$=0.25. This location of the minimum does not support the suggestion of Caul and Miller since the $\underline{p}$-value producing minimum heart rate was not the $\underline{p}$-value corresponding to maximum unpredictability of US following CS.

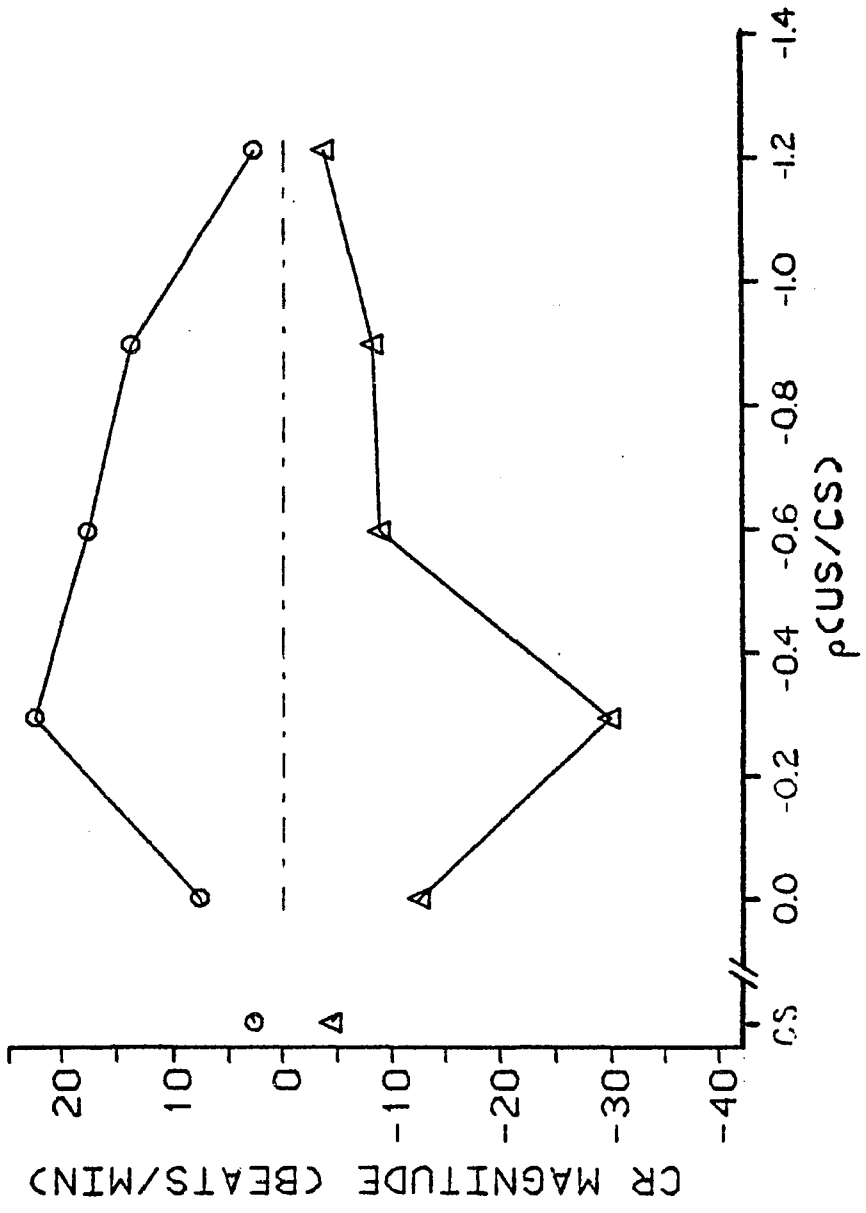
The above reports of changes in pre-CS levels are interesting since changes in this measure are often called adaptation and are known to occur during periods when no CS/US pairings are being presented (adaptation sessions). However, the changes noted by Miller, et al. (1967), Caul, et al. (1968), and the present study, occurred concurrent

with manipulations of stimulus events in the sessions. The present study's use of within-subject manipulation of p(US/CS) seems to permit concluding that these changes in pre-CS heart rate were somehow dependent upon the stimulus events of the sessions. Specifically, the change in pre-CS heart rate seen when p(US/CS) was moved from 1.0 to 0.25 reversed itself with further decreases in p(US/CS). This return of pre-CS heart rate to levels present at the beginning of conditioning is not typical of adaptation effects which usually involve changes from some high, initial value to a then stable, lower value. Whether or not these changes in cardiac rate during the intertrial interval (ITI) should be considered "conditioned" changes or "conditioning-facilitated adaptation" is not clear.

CR magnitude as a function of p(US/CS) at 22-hr deprivation.

Figure 14 presents the magnitude of the conditional cardiac rate response as a function of p(US/CS) for both the rats with acceleratory CRs and for the rats with deceleratory CRs. CR magnitude was defined as the difference between the mean heart rate in the last two CS bins (bins 7 and 8) and the mean pre-CS heart rate (mean of bins 1-4). Since the heart rate CR had a certain latency, bins 7 and 8 were taken as the measure of CS heart rate. CR magnitude is clearly a nonmonotonic function of p(US/CS) for both groups of animals, with a maximum response to CS occurring at p(US/CS)=0.50. Acknowledging that CR magnitude

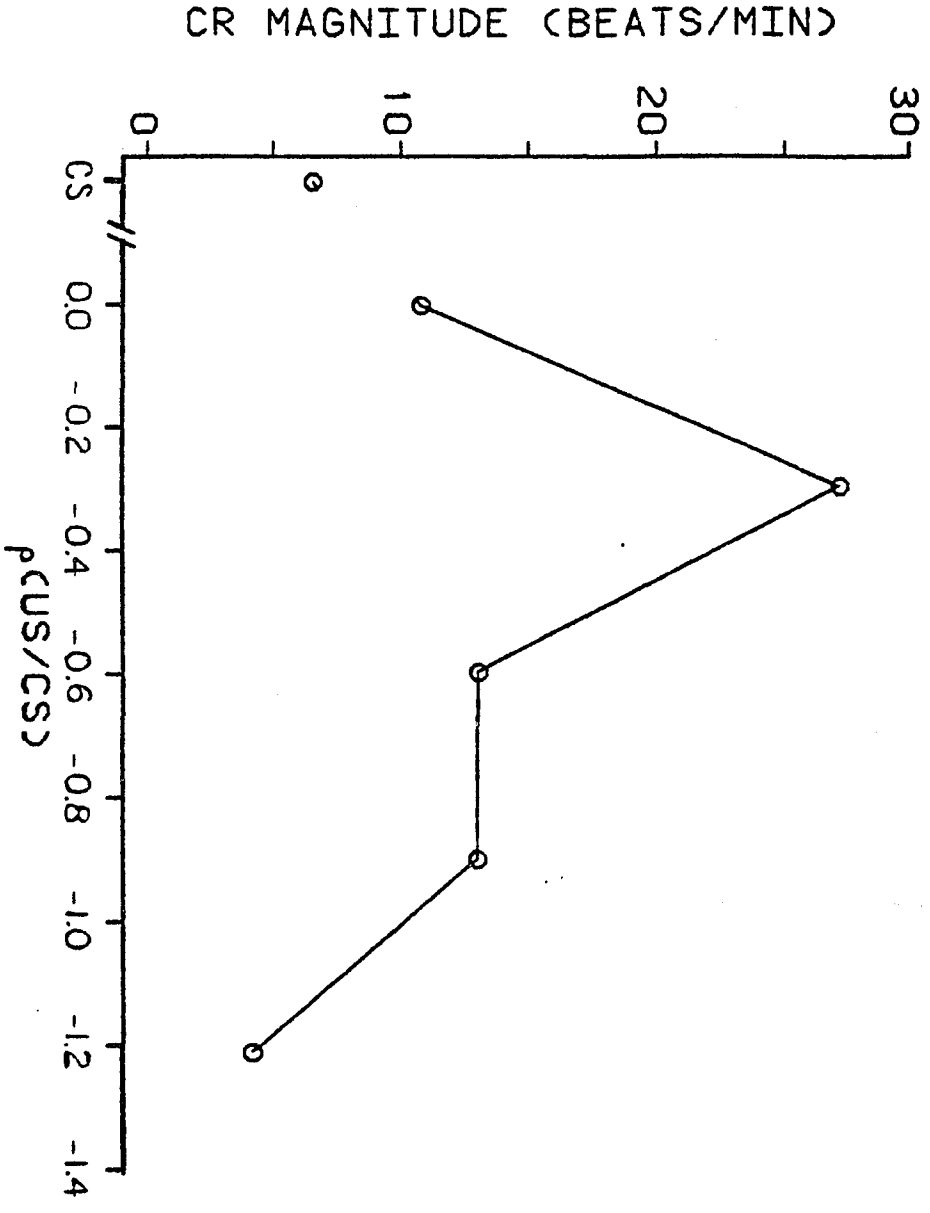
Figure 14. CR magnitude, the mean heart rate in bins 7 and 8 minus the mean heart rate in bins 1-4, as a function of p(US/CS). Each function is based on an average of either the rats with acceler- atory CRs (the circles) or the rats with deceleratory CRs (the triangles). These data were obtained from rats at 22-hr food deprivation. Note the logarithmic scale on the abscissa. The first point of the function on the left end is for p(US/CS)=1.0. Moving to the right, the other points are for 0.50, 0.25, 0.12, and finally 0.06 on the extreme right.


CR MAGNITUDE (BEATS/MIN)
20
10
0
-10
-20
-30
-40
CS
0.0
-0.2
-0.4
-0.6
-0.8
-1.0
-1.2
-1.4
p(US/CS)

has opposite signs for the acceleratory and deceleratory CR rats, Figure 14 indicates that the effects of p(US/CS) on CR magnitude were similar for both the acceleratory and the deceleratory CR rats. It therefore seemed justifiable to average the individuals, as was done in Figure 15, to present the grand mean function relating CR magnitude to p(US/CS). For this figure, the absolute values of the difference scores shown in Fig. 14 were used to calculate CR magnitude. The absolute differences were chosen so that the acceleratory and deceleratory CR magnitudes would not sum to zero. This grand mean function displays the same general characteristics as the functions for the subgroups.

Comparisons of CR magnitudes at two values of p(US/CS) are easily made if the pre-CS heart rates are equal at these two p-values. The similarity of pre-CS heart rates obviates the need for regression of CR magnitude on pre-CS level (such an analysis would be necessary to calculate what the CR magnitude would be at one of the p-values if the response was starting from the pre-CS level at the other p-value). Since such an equivalence of pre-CS heart rate was observed for the p(US/CS)=1.0 and 0.50 conditions, direct comparisons of the average CR magnitudes at these two p-values were made and revealed significantly larger CRs at 0.50 than at 1.0 (A one-way analysis of variance for repeated measures yielded an F of 5.07 which exceeds F.99 (4,28) of 4.07, indicating a treatment effect. Application of the Newman-Keuls method indicated that CRs at 0.50 were different from CRs at all

Figure 15. The absolute value of CR magnitude as a function of p(US/CS) for all eight rats at 22-hr food deprivation. The use of absolute values of CR magnitude allowed the two functions in Figure 14 to be averaged without summing to a flat, zero valued function while still reflecting the average size of the heart rate response to CS. Note the logarithmic scale on the abscissa. The first point of the function on the left end is for p(US/CS)=1.0. Moving to the right, the other points are for 0.50, 0.25, 0.12, and finally 0.06 on the extreme right.


CR MAGNITUDE (BEATS/MIN)
0
10
20
30
CS
0.0
-0.2
-0.4
-0.6
-0.8
-1.0
-1.2
-1.4
ρ(US/CS)

other p-values). Examining individual subjects, more subjects showed statistically significant differences between pre-CS and CS heart rates ($p<0.05$ for Wilcoxon test) at $p(US/CS)=0.50$ than at any other $p(US/CS)$. Four subjects had statistically significant CRs at $p(US/CS)=1.0$, six subjects at $p(US/CS)=0.50$, four at 0.25, three at 0.12, and one at 0.06. Additionally, inspection of Figures 1-9 indicates that 5 of 9 rats had greater heart rate CR magnitudes at $p(US/CS)=0.50$ than at 1.0 (rats 3, 6, 7, 10, and 12). The present study joins those of Razran (1955) and of Powell and Milligan (1975) in finding larger CRs at p-values less than 1.0 using a classical conditioning procedure with an autonomically controlled response.

As mentioned in the introduction, higher response rates with intermittent reinforcement, as against continuous reinforcement, have been reported for some cases of response-dependent conditioning of operants; however, a parallel result with classically conditioned respondents is all but unknown. The present finding of larger CRs at $p(US/CS)=0.50$ than at 1.0, serves to narrow the perceived gap between the "Pavlovian" and "operant" areas with respect to the effects of intermittency (vide, Terrace, 1973).

In the literature dealing with the conditioning of keypecks with noncontingent presentations of a cue paired with a reinforcing stimulus (autoshaping), a few studies have observed increases in steady-state response rate (automaintenance) with probabilities of food delivery less

than 1.0 (Gibbon, Farrell, Locurto, Duncan, and Terrace, 1980; Gonzalez, 1974; and Schwartz and Williams, 1972). The findings from the operant and autoshaping literatures, along with some from the Pavlovian literature (Razran, Powell and Milligan, and the present study), argue for the generality of the finding that intermittent reinforcement may increase certain measures of a response relative to the level seen at 100% reinforcement. Additionally, the similarity of the effects of intermittent reinforcement in the three above mentioned conditioning situations argues against distinguishing one from the other on the basis of the behavioral effects of intermittent reinforcement.

Pre-CS, CS, and post-US heart rate at 0-hr deprivation.

Figures 16-22 present heart rate data obtained from sessions during which the rats were 0-hr food deprived. That is, all data in Figures 16-22 were obtained from rats that had free access to food and water for one hour directly prior to the experimental session. In Figures 16, 17, and 18, heart rate is shown in successive running bin means exactly as in Figures 1-9. Several features of the heart rate data presented in Figures 16-18 deserve mention. Generally, the effect of feeding before the session was to lower the pre-CS heart rate. This lowered pre-CS rate appears to have altered the heart rate CRs of the rats with acceleratory heart rate CRs less than those of the rats with deceleratory heart rate CRs. This can be seen by comparing

Figure 16-18. Individual rats' heart rates in 2-sec recording periods before, during and after a CS/US pairing at 0-hr food deprivation. Each row presents data for a single rat. Within each row a panel represents a different p(US/CS). Three suc- cessive 2-sec bins have been averaged together to give three- bin running means. For example, the running mean for bin n is the mean heart rate in bins n-1, n, and n+1. The running bin means that best represent heart rate during pre-CS, CS, and US are indicated by vertical lines. The individual bin heart rates were obtained by taking a median of all heart rates for a particular bin during the second half of the session for the last two days at an experimental point.

RAT 1
RAT 3
RAT 6
HEART RATE (BEATS/MIN)
300
360
420
480
RUNNING BIN MEAN
2 4 6 8 10 12
P(US/CS)=.50
P(US/CS)=.25
P(US/CS)=.12
P(US/CS)=.06
P(US/CS)=1.0

RAT 7
RAT 8
RAT 10
HEART RATE (BEATS/MIN)
300
360
420
480
2 4 6 8 10 12
RUNNING BIN MEAN
P(US/CS)=.50
P(US/CS)=.25
P(US/CS)=.12
P(US/CS)=.06
P(US/CS)=1.0

RAT 11
RAT 12
RAT 15
HEART RATE (BEATS/MIN)
300
360
420
480
RUNNING BIN MEAN
2 4 6 8 10 12
P(US/CS)=.50
P(US/CS)=.25
P(US/CS)=.12
P(US/CS)=.06
P(US/CS)=1.0

the CRs of rats 6 and 12 (accelerators) at 0-hr deprivation with their CRs at 22-hr deprivation. The heart rate CRs of these rats at 0-hr deprivation were generally slightly reduced compared to those at 22-hr deprivation. However, in some cases (cf. rat 6 at low p(US/CS)) the CRs are larger at 0-hr than at 22-hr deprivation. By comparison, the rats with the most prominent deceleratory heart rate CRs at 22-hr deprivation, rats 3, 7, and 15, show substantially decreased CRs at 0-hr. Aside from the differences in magnitude of the heart rate CR at 0-hr deprivation, as against 22-hr deprivation, there was also a difference in the consistency of the direction of CR at these two deprivations for the deceleratory and the acceleratory rats. At 0-hr deprivation there was more variability in the direction of the heart rate change to CS in the group of rats that showed deceleratory CRs at 22-hr deprivation than in the rats with acceleratory CRs at the higher deprivation. Rats 3, 7, and 15, strongly deceleratory at 22-hr deprivation, all showed some acceleratory CRs at 0-hr while rats 1, 6, and 12, the accelerators at 22-hr deprivation, retained their acceleratory CRs at 0-hr deprivation (except for one value of p(US/CS) for rat 6). As another example of this, Rats 8 and 10 had marginally deceleratory heart rate CRs at 22-hr deprivation, but had no heart rate CRs at 0-hr deprivation except at p(US/CS)=.50. At this probability value both of these animals showed accelerations greater in magnitude than any of their heart rate changes to CS during the sessions at 22-hr deprivation.

It should be noted that the lowered pre-CS heart rate at 0-hr deprivation makes it difficult to interpret some of the above comparisons of CR magnitudes at 0-hr and 22-hr deprivation. However, for the decreased acceleratory CRs there is no problem. For acceleratory CRs, decreases in pre-CS heart rates alone would yield larger CRs according to the Law of Initial Value (LIV) as stated by Wilder (1950, 1957). Therefore, the diminished acceleratory CR magnitudes at 0-hr must be a result of the lowered deprivation and not merely the decreased pre-CS heart rate level. However, for the deceleratory CRs a decrease in pre-CS heart rate should produce smaller decelerations in CS and might even result in a change in direction to accelerations with a large enough change in pre-CS level. It remains a question, therefore, what role deprivation, as against changes in pre-CS heart rate, played in diminishing the magnitude, and increasing the variability in direction, of the deceleratory heart rate CRs at 0-hr deprivation.

Figures 16-18 reveal another difference in the findings for the rats with acceleratory CRs and for those with deceleratory CRs (at 22-hr deprivation). With respect to the last conditioning point, p(US/CS)=1.0 at 0-hr deprivation, the rats with acceleratory CRs at 22-hr deprivation all show an acceleratory heart rate CR at that experimental point. However, the rats with deceleratory heart rate CRs do not have any heart rate CRs at the final point. The only exception to this latter generalization is

rat 15 who displayed a slight acceleratory CR at p(US/CS)=1.0 at 0-hr deprivation.

In summary, the individual functions in Figures 16-18 suggest that decreases in the number of hours of food deprivation from 22 hr to 0 hr may have the following effects on heart rate CR: 1) for rats with acceleratory CRs, decreased deprivation seemed responsible for generally reduced CR magnitudes, although at lower probabilities of US the CR magnitudes were sometimes enhanced; 2) greater variability in the direction of heart rate change to CS for the group of rats with deceleratory CRs at 22-hr deprivation (a possible result of decreased pre-CS level); and, 3) better recovery of heart rate CRs upon reexposure to p(US/CS)=1.0 for the accelerators than for the decelerators.

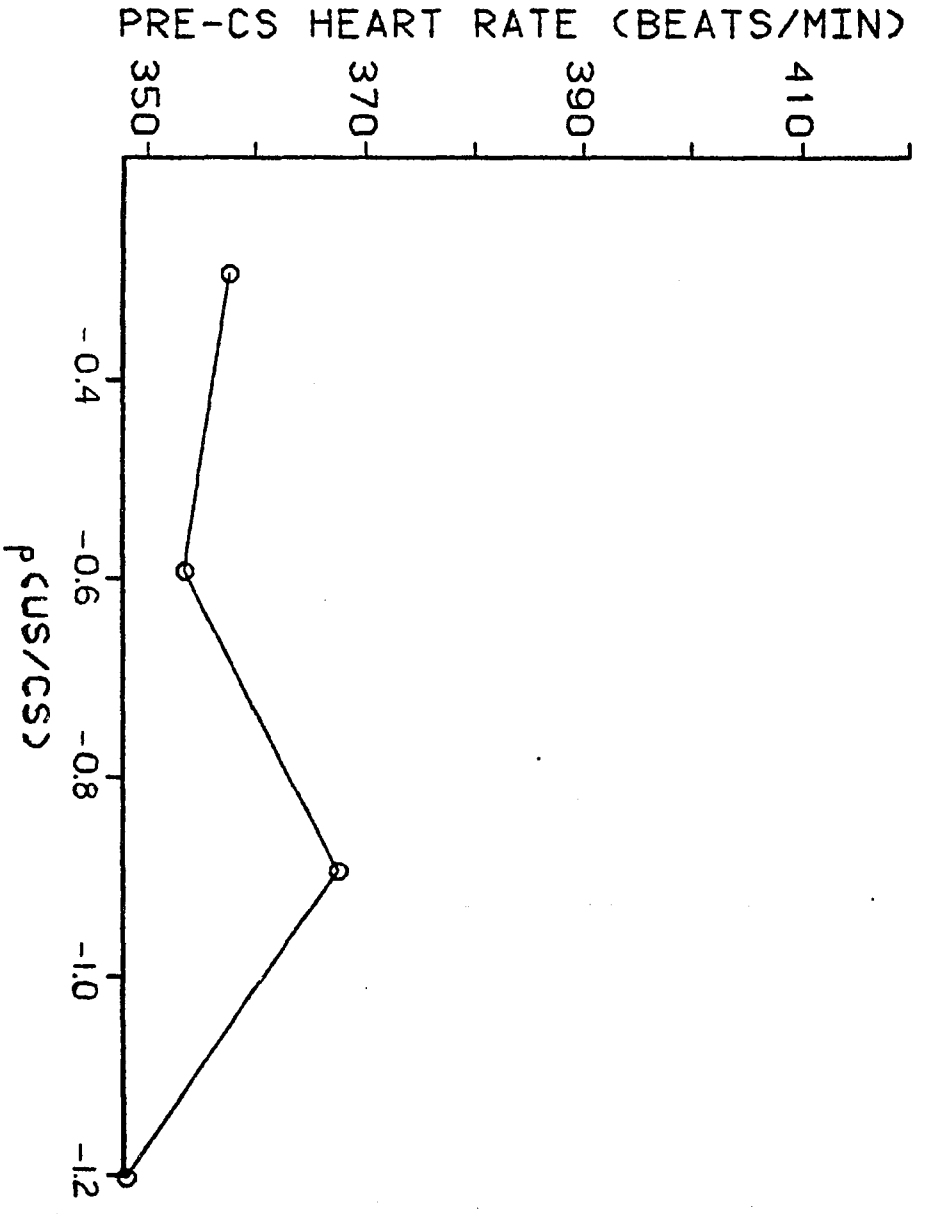
Effects of p(US/CS) on pre-CS heart rate (low deprivation)

Figure 19 presents averaged pre-CS heart rate levels for the rats with acceleratory CRs and for those with deceleratory CRs at 0-hr deprivation, just as in Figure 12 for the 22-hr deprivation condition. The accelerators display similar effects of p(US/CS) on pre-CS heart rate at the two deprivations (compare Figs 12 and 19) while the decelerators have very different functions over the range of p(US/CS) at the two deprivations. Thus, Figure 19 suggests an interaction between deprivation and the effects of p(US/CS) on pre-CS heart rate for the rats with deceleratory heart rate CRs at 22-hr deprivation.

Figure 19. Pre-CS heart rate, the mean heart rate in bins 1-4, as a function of p(US/CS) at 0-hr food deprivation. The rats that showed acceleratory CRs at 22-hr deprivation (rats 1, 6, and 12) are represented by the circles while the rats that showed deceleratory CRs at 22-hr deprivation (rats 3, 7, 8, 10, and 15) are represented by triangles. Note the logarithmic scale on the abscissa. The leftmost point on the function is for p(US/CS)=0.50. Moving to the right the other points are for 0.25, 0.12, and 0.06.

PRE-CS HEART RATE (BEATS/MIN)
330
370
410
-0.4
-0.6
-0.8
-1.0
-1.2
p(US/CS)

Figure 20. Pre-CS heart rate as a function of p(US/CS) for all eight rats at 0-hr food deprivation. This function is an averge of the two functions in Figure 19. Note the logarithmic scale on the abscissa. The leftmost point on the function is for p(US/CS)=0.50. Moving to the right, the other points are for 0.25, 0.12, and 0.06.


PRE-CS HEART RATE (BEATS/MIN)
350
370
390
410
-0.4
-0.6
-0.8
-1.0
-1.2
p(US/CS)

Given the possible differences between these two subgroups, a grand mean function (Figure 20) would not best represent the shape of the function for all the individuals; however, such a function allows rough comparison of the effects of p(US/CS) on pre-CS levels at the two deprivations. The effect of prefeeding, as seen in Figure 20, is to dramatically lower resting heart rate. The average pre-CS level at 0-hr deprivation was as low as the lowest level seen as a result of changing p(US/CS) at 22-hr deprivation. Comparing the heart rates in Figure 20 with other reports of resting heart rate levels in rats (Caul and Miller, 1968; Miller, Banks, and Caul, 1967), it appears that the pre-CS heart rates in the present study were about as low as can be seen in intact, undrugged rats.

CR magnitude as a function of p(US/CS) at 0-hr deprivation.

Figure 21, which is like Figure 14 except at the decreased deprivation level, shows CR magnitude as a function of p(US/CS) for both the accelerators and the decelerators. Figure 21 confirms some of the observations made from the individuals in Figures 16-18. Decreased CR magnitudes can be seen in Figure 21 for both the accelerators and decelerators, in comparison to those in Figure 14. However, this reduction in CR magnitude is unquestionably greater for the deceleratory CR rats (they move closer to the zero line) than for the acceleratory CR rats. Additionally, the reversals in direction of heart

Figure 21. CR magnitude, the mean heart rate in bins 7 and 8 minus the mean heart rate in bins 1-4, as a function of p(US/CS) at 0-hr food deprivation. The rats that showed acceleratory CRs at 22-hr deprivation (rats 1, 6, and 12) are represented by the circles while the rats that showed deceleratory CRs at 22-hr deprivation (rats 3, 7, 8, 10, 15) are represented by triangles. Note the logarithmic scale on the abscissa. The leftmost point on the function is for p(US/CS)=0.50, Moving to the right, the other points are for 0.25, 0.12, and 0.06.

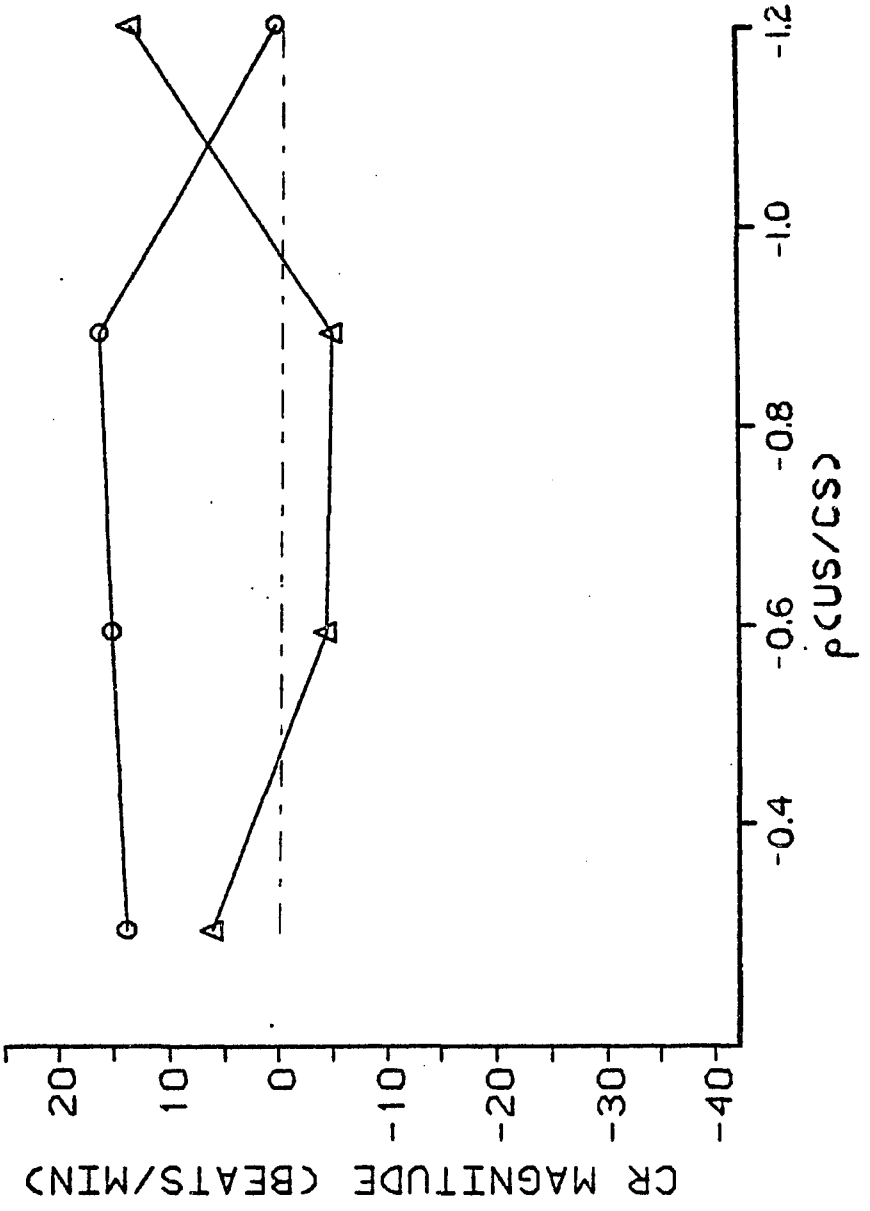
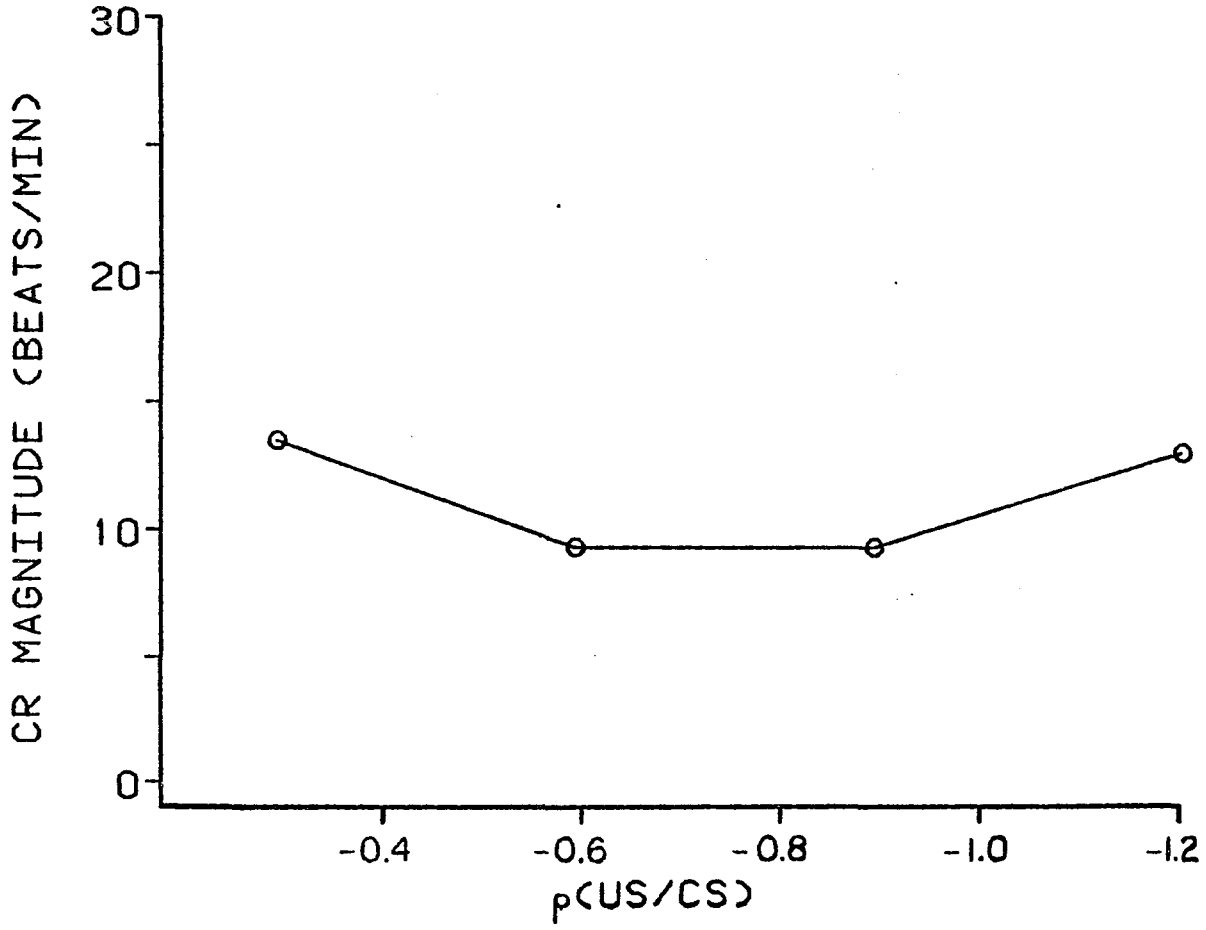

CR MAGNITUDE (BEATS/MIN)
20
10
0
-10
-20
-30
-40
-0.4
-0.6
-0.8
-1.0
-1.2
P(US/CS)

Figure 22. CR magnitudes as a function of p(US/CS) for all eight rats at 0-hr food deprivation. The use of absolute values of CR magnitude allowed the two functions in Figure 21 to be averaged without summing to a flat, zero valued function while still reflecting the average size of the heart rate response to CS. Note the logarithmic scale on the abscissa. The leftmost point on the function is for p(US/CS)=0.50. Moving to the right, the other points are for 0.25, 0.12, and 0.06.


30
20
10
0
CR MAGNITUDE (BEATS/MIN)
-0.4
-0.6
-0.8
-1.0
-1.2
p(US/CS)

rate CR by the decelerators can be observed in Figure 21 as heart rate changes in the positive range while all heart rate changes for these rats are negative in sign in Figure 13. These two observations point to a possible interaction between deprivation level and the direction of CR (such an interaction was already suggested by Figure 19). If such an interaction were present, averaging data across all the rats would yield a function that would not be representative of either subgroup. However, such a function facilitates comparison of the effects of p(US/CS) on CR magnitude at the two deprivations. Figure 22, a group function based on all the rats shows that the strong effects of p(US/CS) on CR magnitude (see Figure 15) have largely disappeared at the lowered deprivation, although the heart rate CR still remains at some appreciable magnitude at the lowered deprivation.

The second portion of this section consists of a more detailed examination of certain aspects of the data that are of interest for the present study.

CR magnitudes after reinforced versus unreinforced trials.

In the cardiac conditioning literature some attention has been paid to possible "local" effects of reinforcement and nonreinforcement. Some investigators (Holmes and Gormezano, 1970; and, Spence and Trapold, 1964) have seen a

difference in CR probabilities on trials following reinforced trials, as against trials following unreinforced trials. In those studies percentage of trials with a CR was lower at p(US/CS)=0.50 than at 1.0 (as a session average), and within sessions the CR probability was lower after unreinforced than after reinforced trials at p(US/CS)=0.50. The overall decremental effect of lowering p(US/CS) was therefore attributed to the "local" depressing effect of extinction trials. Other investigators, however, have failed to report "local" effects of reinforced, as against unreinforced, trials (Fitzgerald, 1966; and, Gormezano and Coleman, 1975). Gormezano and Coleman noted that only the studies seeing an overall depressant effect of decreasing p(US/CS) from 1.0 to 0.50 have seen local effects of extinction trials.

Turning to the data of the present study, Table 1 presents CR magnitude on trials following a single reinforced trial and on trials following a single unreinforced trial for six rats at p(US/CS)=0.50 with 22-hr food deprivation. A t-test for related samples revealed a nonsignificant difference in CR magnitudes. Table 2 presents exactly the same type of data as Table 1 except that now CRs are examined on trials preceded by either two consecutive reinforced trials or two consecutive unreinforced trials. Again there is no significant effect of these two different antecedents on CR magnitude. Only rats that had at least three instances of such trial runs

Table 1

Conditioned response magnitudes on trials following one reinforced trial versus CR magnitudes on trials following one unreinforced trial at p(US/CS)=.50.

	After US (beats/min)	After no US (beats/min)
Rat 1	5.6	5.8
Rat 3	-45.0	-38.7
Rat 7	-30.0	-30.0
Rat 8	-11.7	-13.8
Rat 12	30.0	12.3
Rat 15	-24.3	-22.5

Related samples *t*-test yielded t_{obs}=1.30 (*p* greater than .05).

Table 2

Pre-CS heart rates on trials following one reinforced trial versus pre-CS heart rates on trials following one unreinforced trial at p(US/CS)=.50.

	After US (beats/min)	After no US (beats/min)
Rat 1	414.6	414.3
Rat 3	423.0	417.9
Rat 7	440.1	442.5
Rat 8	393.3	396.3
Rat 12	382.5	405.9
Rat 15	337.5	340.5

Related samples *t*-test yielded t_{obs}=.93 (*p* greater than .05)

for both reinforced and unreinforced trials were included in this analysis.

Since basal heart rate levels have been shown repeatedly to interact with both the size and direction of the cardiac rate response (Ramsay, 1970; Snapper, Kadden, and Schoenfeld, 1971; and, Washton, 1978), the possibility exists that the observed equivalence of CR magnitude following the two types of trials (reinforced and unreinforced) is an artifact of unequal pre-CS heart rate levels. This possibility may be illustrated by the hypothetical case of pre-CS heart rate being differentially elevated after unreinforced trials. For rats with deceleratory heart rate CRs, larger CRs would be expected on those trials with higher pre-CS heart rate levels (the trials following unreinforced trials), based on the Law of Initial Value. If no difference were observed in actual magnitude of CR, given the difference in pre-CS levels, it would appear that non-reinforcement on the preceding trial served to decrease CR magnitude relative to that seen on trials following a trial with reinforcement. Because the above hypothetical case is a real possibility, Table 3 is included to determine whether or not pre-CS heart rates are different on trials following either one reinforced or one unreinforced trial, and Table 4 displays pre-CS heart rates on trials following either two reinforced or two unreinforced trials. Both Tables indicate no effect of type of preceding trial on pre-CS heart rate levels. The finding

Table 3

Conditioned response magnitudes on trials following two reinforced trials versus CR magnitudes on trials following two unreinforced trials at p(US/CS)=.50.

	After two US (beats/min)	After two no US (beats/min)
Rat 1	15.0	12.6
Rat 3	-46.8	-24.3
Rat 7	-27.6	-30.0
Rat 8	- 3.9	- 3.9
Rat 12	15.0	0.0
Rat 15	- 7.5	- 7.5

Related samples t-test yielded t_{obs}=1.52 (p greater than .05)

Table 4

Pre-CS heart rates on trials following two reinforced trials versus pre-CS heart rates on trials following two unreinforced trials at p(US/CS)=.50.

	After two US (beats/min)	After two no US (beats/min)
Rat 1	399.9	402.0
Rat 3	418.2	403.2
Rat 7	432.6	457.5
Rat 8	386.4	416.4
Rat 12	397.5	390.0
Rat 15	330.0	382.5

Related samples t-test yielded t_{obs}=1.77 (p greater than .05)

of equality of pre-CS heart rates removes the possibility that the observed equivalence of CR magnitude on trials following either reinforced or unreinforced CS presentations was an artifact of differing pre-CS levels after those two types of trials.

Tables 5 and 6 also examine CR magnitude and pre-CS heart rate, respectively, on trials following either a reinforced or an unreinforced trial. These Tables, however, present data obtained at p(US/CS)=0.25 with 22-hr food deprivation. Tables 5 and 6 reveal no differences in either measure as a function of the type of preceding trial, agreeing with the finding at 0.50. It was not possible to examine the effects of having either two reinforced or two unreinforced trials preceding a given trial at p(US/CS)=0.25 since there were very rarely occasions when two USs were presented on consecutive trials at that p-value.

It is possible, however, that differential effects of the type of preceding trial did exist in the sessions immediately following the change from p=1.0 to p=0.50 or in those following the change from 0.50 to 0.25. Such a finding would be similar to the cases of behavioral contrast where the changes in response rate are transitory. Such a possibility could perhaps account for the discrepancies among the studies examining such data. Since data were not taken from the first few days at an experimental condition, this possibility cannot be explored for the present study.

Table 5

Conditioned response magnitudes on trials following one reinforced trial versus CR magnitudes on trials following one unreinforced trial at p(US/CS)=.25.

	After US (beats/min)	After no US (beats/min)
Rat 1	12.6	19.5
Rat 3	- 7.5	2.4
Rat 6	11.4	12.6
Rat 7	-30.0	-15.0
Rat 8	15.0	0.0
Rat 10	- 3.9	3.9
Rat 12	12.0	21.0
Rat 15	-50.1	0.0

Related samples t-test yielded t_{obs}=1.04 (p greater than .05)

Table 6

Pre-CS heart rates on trials following one reinforced trial versus pre-CS heart rates on trials following one unreinforced trial at p(US/CS)=.25.

	After US (beats/min)	After no US (beats/min)
Rat 1	347.4	346.5
Rat 3	300.0	292.5
Rat 6	363.9	362.4
Rat 7	345.0	345.0
Rat 8	375.0	375.0
Rat 10	393.9	386.4
Rat 12	384.0	360.0
Rat 15	339.9	335.1

Related samples t-test yielded t_{obs}=2.05 (p greater than .05)

Heart rate CR as a predictor of the consummatory response.

Although primarily interested in the course of cardiac conditioning when food US, instead of shock US, was employed, Schoenfeld, Matos, and Snapper (1967) observed that for trials on which the rat consumed US there were mostly acceleratory heart rate CRs while for trials on which rat did not consume US the heart rate CRs were usually slightly deceleratory or absent. Thus, the presence of an acceleratory heart rate CR seemed to have promise as a predictor of the subsequent consummatory response. However, the observed correlation between heart rate CR and drinking, while interesting, was found in an experimental condition to which only two rats were exposed. Furthermore, the difference between the heart rate CRs on drink trials as against heart rate CRs on no-drink trials did not reach statistical significance (perhaps, as the authors suggested, this was because of the limited number of trials on which the rats failed to consume US).

The present study included a condition similar to the Schoenfeld et al. study (the initial value of p(US/CS)=1.0 at 22-hr deprivation), in an attempt to confirm their suggested finding in a larger sample of rats. Additionally, the present study hoped to extend that potential result by examining the relation between heart rate CRs and drinking in rats that had their heart rate CRs either diminished or eliminated as a result of decreases in either hours of food deprivation or probability of US. If the Schoenfeld et al.

finding is a real one, then affecting the heart rate CR in such fashion could have an effect on the consummatory response.

In the present study, the rats were observed throughout the course of the experiment to see whether or not they consumed the milk US. These data are easily summarized: US was consumed on 100% of the trials in all phases of the study, except during the initial acquisition sessions. Once an animal was consuming 100% of the presented USs, deprivation could be lowered to 0-hr or p(US/CS) decreased to 0.06 without decreasing the likelihood of US consumption. Six of the ten rats failed to consume every US during the early conditioning sessions (the others drank reliably from the first trial). Of those six, one rat (#3) had only a single missed US while the other five had varying numbers of missed USs before reaching 100% consumption.

Table 7 indicates the average pre-CS heart rates for trials on which the rat consumed US and for trials on which the rats failed to consume US. Most animals showed very small differences in their pre-CS levels on those two categories of trials. A t-test for related samples confirmed the absence of a difference between the pre-CS levels. Given this similarity of pre-CS levels, it becomes easy to evaluate the difference in CR magnitudes between drink as against no-drink trials. Using the same group of trials that comprised Table 7, CR magnitudes on drink and no-drink trials are given in Table 8. There was little

Table 7

Rat#	1	7	8	15	16	Mean
Pre-CS HR on Drink Trials	438.0	435.0	365.4	322.8	377.4	387.6
Pre-CS HR on No-Drink Trials	438.6	442.2	357.0	317.7	375.3	386.1

Related samples t-test yielded $t_{obs}=0.59$ (p greater than 0.05)

Table 8

Rat#	1	7	8	15	16	Mean
CR Magnitude on Drink Trials	-5.4	0.6	-6.9	-8.4	0.6	-4.2
CR Magnitude on No-Drink Trials	-4.5	-9.6	3.3	-30.0	4.2	-7.2

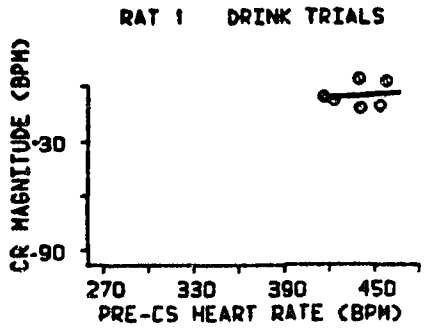
Related samples t-test yielded $t_{obs}=1.33$ (p greater than 0.05)

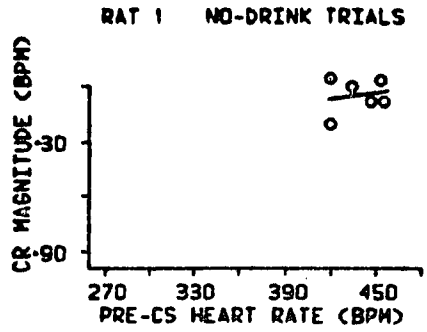
difference between CR magnitudes on trials for which the rat consumed US and on trials for which the rat failed to consume US. Using the absolute values of the CR magnitudes in Table 8, a t-test for related samples revealed no difference between CR magnitudes on drink as against no-drink trials. In both Tables 7 and 8 the drink trials selected were those that either directly preceded or directly followed the no-drink trials. Drink trials, the predominant case, were selected in this fashion to minimize differences in pre-CS levels between the drink and no-drink trials (pre-CS levels presumably varied more across a number of trials than between successive trials).

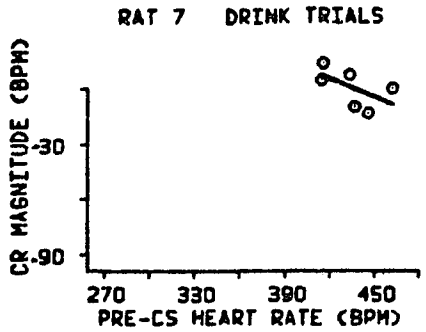
The data in Tables 7 and 8 are based on averages of drink or no-drink trials from as few as two to as many as six sessions, depending on the number of sessions containing no-drink trials. While an analysis based on data averaged across sessions could reveal a difference between CR magnitude on drink and no-drink trials, there is a possibility of not detecting such a difference even if one does exist. This possibility arises from the potential interaction of pre-CS heart rate level and CR magnitude. Consider the following hypothetical case: in session 1 a rat's pre-CS heart rate is relatively low and the CR on drink trials is an increase of 12 bpm, but in session 2 the rat's pre-CS heart rate is much higher and now the CR on drink trials is a decrease of 12 bpm (a result consistent with predictions based on LIV), yielding an average CR

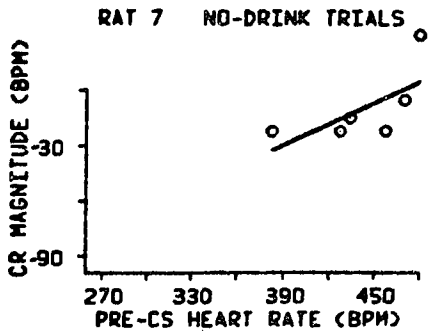
magnitude for the two sessions of 0-bpm; however, on the no-drink trials for both session 1 and session 2 the rat failed to exhibit any deviation from pre-CS levels during CS, that is, CR magnitude was 0 bpm. For such a case, averaging across sessions yields equal pre-CS levels and equal CR magnitudes for both the drink and no-drink trials, but in fact the rat exhibited cardiac rate changes to CS on drink trials but was unresponsive to CS on no-drink trials. Figure 23 is designed to reveal such a possibility. Each data point in this figure represents the average pre-CS heart rate level and the average CR magnitude from a single session. Data from drink and no-drink trials are shown in separate panels. In such fashion, this figure compares the relationship between pre-CS level and CR magnitude for each animal under conditions of either presence or absence of a consummatory response. For rats 1, 15, and 16 the relationship between these two measures seems independent of the presence or absence of the consummatory response. However, for rat 7 the relation between these measures does change as a function of the presence or absence of the consummatory response. Rat 7 displays the typical inverse relation between pre-CS level and CR magnitude on drink trials but has an unusual relation between pre-CS level and CR magnitude on no-drink trials. Rats 3 and 8 had fewer than three sessions with no-drink trials, so functions could not be obtained for them.

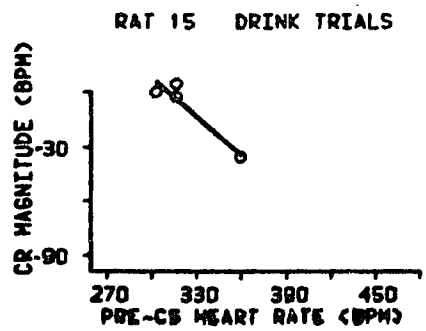
Figure 23. CR magnitudes plotted against pre-CS heart rate for both trials on which the consummatory response was observed and was not observed. Each point represents a session average. Only rats with no-drink trials occurring in at least three sessions are presented. The curves were fitted using the method of least squares. fitted lines are given.

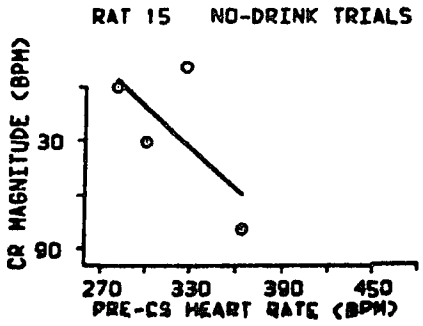

RAT 1 DRINK TRIALS
CR MAGNITUDE (BPM)
-30
-90
270
330
390
450
PRE-CS HEART RATE (BPM)

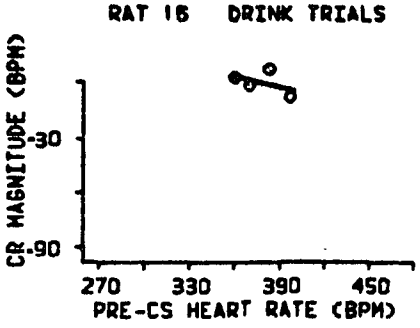

RAT 1 NO-DRINK TRIALS
CR MAGNITUDE (BPM)
-30
-90
270
330
390
450
PRE-CS HEART RATE (BPM)

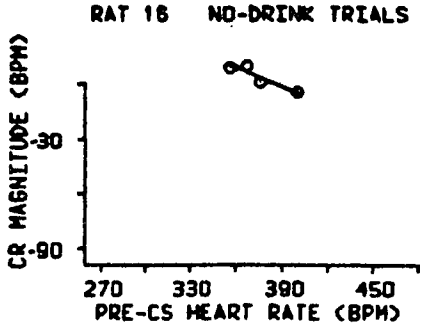

RAT 7 DRINK TRIALS
CR MAGNITUDE (BPM)
-30
-90
270
330
390
450
PRE-CS HEART RATE (BPM)


RAT 7 NO-DRINK TRIALS
CR MAGNITUDE (BPM)
-30
-90
270
330
390
450
PRE-CS HEART RATE (BPM)


RAT 15 DRINK TRIALS
CR MAGNITUDE (BPM)
-30
-90
270
330
390
450
PRE-CS HEART RATE (BPM)


RAT 15 NO-DRINK TRIALS
CR MAGNITUDE (BPM)
30
90
270
330
390
450
PRE-CS HEART RATE (BPM)


RAT 16 DRINK TRIALS
CR MAGNITUDE (BPM)
-30
-90
270
330
390
450
PRE-CS HEART RATE (BPM)


RAT 16 NO-DRINK TRIALS
CR MAGNITUDE (BPM)
-30
-90
270
330
390
450
PRE-CS HEART RATE (BPM)

The average heart rate CR magnitudes on drink as against no-drink trials were almost exactly equal in the present work. Therefore, it appears that differences in CR magnitudes are not a necessary condition for variability in the appearance of a subsequent consummatory response. Additionally, at the lowest value of p(US/CS) at 22-hr deprivation and also at p(US/CS)=0.12 at 0-hr deprivation, CR magnitudes were very small but the consummatory response still occurred to every US. In other words, the CR magnitude could vary over wide extremes while probability of the consummatory response remained at 1.00. The conclusion is that the heart rate CR is not necessary for the consummatory response to occur.

The present study reports no statistically significant difference in mean CR magnitudes on drink as against no-drink trials for the six rats examined. Although neither the present study nor the Schoenfeld et al. report observed a statistically significant difference in CR magnitudes on these two types of trials, the present study obtained a much smaller difference in CR magnitudes on drink as against no-drink trials (0.1 bpm) than the earlier work (6.0 bpm). It is not obvious why the present study found a smaller difference than Schoenfeld et al. between heart rate CRs on drink as against no-drink trials.

Neither the present study nor the Schoenfeld et al. report seem to have been ideally designed to establish a mediating role for the heart rate CR. For both studies the

strong cue provided by the raising of the dipper at US onset may have been a problem. By providing a strong discriminative stimulus for the consummatory response the potential for control over that response by internal responses of the organism might have been weakened. Perhaps this explains the reliable consumption of US at the lower values of p(US/CS) where CRs were virtually absent: "dipper up" had become a discriminative stimulus controlling dipper approach and US consumption. In contrast, during the initial sessions "dipper-up" might not have been serving as a controlling stimulus since the rat often failed to drink. During these initial sessions the acquisition of the cardiac rate CR presumably began to occur while the existing control by dipper-up continued to improve. However, if dipper-up came to control approach and drinking before the heart rate CR acquired much strength, there may never have been an opportunity for the heart rate CR to "mediate" the drinking response. Perhaps both the present study and Schoenfeld, et al. (1967) failed to see a stronger relation between these two responses because both the initial dipper training and the subsequent conditioning trials established dipper sound as a cue for approaching the dipper. (With respect to the preceding speculations, it may seem unexpected that the rats ever failed to consume US since they were given "dipper training" before conditioning began which established the sound of the dipper as a cue for approaching the dipper. However, since dipper training was only carried out until

the experimenter observed that the rats approached the dipper when it was raised, it is possible that either approach behavior was not 100% reliable or that it took more than 4 sec, which would be a no-drink trial once CS/US pairings began. In either case, there would have been imperfect control by the dipper sound at the start of CS/US pairings).

Admittedly the above-mentioned situation is problematic since in order to have cardiac conditioning the rat must be consuming US and the usual procedure is to start the rat drinking by dipper training. In order to increase the opportunity for control over dipper approach and drinking by the heart rate CR, the usual procedure must be modified. One possible change would be to omit dipper training entirely and to use a "silent" delivery of US. This would slow the rate of cardiac conditioning but might increase the chances of establishing control of drinking by the heart rate CR by reducing the control of the dipper sound. Another possible change would be to dipper train the rat, condition with p(US/CS)=1.0, and then fade out the sound of the dipper, thereby transferring control of the approach response to a stimulus other than the dipper sound.

Relationship between pre-CS heart rate and CR magnitude.

In the preceding section the relation between pre-CS heart rate and CR magnitude was examined to detect possible differences in cardiac activity on trials where the rat

consumed US as against those where US was not consumed. The relationship between pre-CS heart rate and the magnitude of the conditional heart rate response has, however, been examined as a datum by itself. The Law of Initial Value predicts an inverse relation between these two measures and, as already mentioned, has been demonstrated to be applicable to cardiac rate responses. Tables 9 and 10 give the product-moment correlations between pre-CS level and CR magnitude for each rat, at each p(US/CS), for 22-hr and 0-hr deprivations, respectively. Each entry in the tables represents the correlation computed from the same trials used in Figures 1-22. The averaged data from these Tables are plotted in Figure 24. In this figure the effect of p(US/CS) on the correlation can be seen to vary depending on deprivation.

Table 9

Correlations of pre-CS heart rate and CR magnitude obtained from rats at 22-hr deprivation

	Rat#	1	3	6	7	8	10	11	12	15	Mean
p(US/CS)	1.00	-.36	-.64	-.73	-.54	-.37	-.40	-.31	-.29	-.66	-.41
	0.50	-.73	-.59	-.70	-.26	-.31	-.45	-.57	-.51	-.22	-.48
	0.25	-.66	-.75	-.77	-.14	-.81	-.24	-.51	-.81	-.55	-.58
	0.12	-.65	-.36	-.41	-.39	-.62	.11	-.17	-.89	-.23	-.40
	0.06	-.73	-.35	-.47	-.50	-.61	-.22	-.45	-.41	-.18	-.44

Table 10

Correlations of pre-CS heart rate and CR magnitude obtained from rats at 0-hr deprivation

	Rat#	1	3	6	7	8	10	11	12	15	Mean
p(US/CS)	0.50	-.21	-.80	+.04	+.02	.44	-.67	-.04	.30	-.98	-.21
	0.25	-.46	-.55	-.68	.08	-.65	-.45	-.36	.37	-.58	-.36
	0.12	-.69	-.61	-.02	-.25	-.71	-.36	-.61	-.37	-.54	-.46
	0.06	.42	.45	.51	.53	.17	.82	.50	.86	.62	.54

Figure 24. The product-moment correlation between CR magnitude and pre-CS heart rate as a function of p(US/CS). The circles represent data taken at 22-hr deprivation while the triangles represent data taken at 0-hr deprivation. The initial value of p(US/CS) was 1.0 at 22-hr deprivation but was .50 at 0-hr deprivation. Note the logarithmic scale on the abscissa. For the function at 22-hr deprivation, the leftmost point is for p(US/CS)=1.0. Moving to the right the other points are for 0.50, 0.25, 0.12 and 0.06.

CORRELATION OF CR MAGNITUDE
WITH PRE-CS HEART RATE

General Discussion

A conspicuous feature of the cardiac rate conditional responses in the present study was the variation in the direction of those responses among animals. Some rats had predominantly acceleratory CRs while others had primarily deceleratory heart rate CRs. As stated in an earlier section, the existence of both acceleratory and deceleratory CRs in animals exposed to the same procedure argues against the notion of a "true" direction of the cardiac rate CR, even when such an idea is restricted to a single species (for an example of the use of the notion of a "true" direction for the cardiac rate CR; see, Fitzgerald, Martin, and O'Brien, 1973; Fitzgerald, Vardaris, and Brown, 1966; and, Powell and Milligan, 1975). While the variation in the direction of CRs is undeniable, the cause or explanation of such variation is not obvious. One possible explanation is the notion of "stimulus substitution" (Pavlov, 1927) which states that CS comes to control a response that is similar to the response controlled by US. In the present study, however, it appears that two of the rats that developed deceleratory heart rate CRs had acceleratory URs. In Figures 2 and 9 (Rats 3 and 15, respectively) there are heart rate decelerations during CS followed by accelerations

in heart rate after US delivery, at the higher values of p(US/CS). (The recovery condition (p(US/CS)=1.0) reveals the heart rate UR more clearly than the above- mentioned points since UR occurs from a "resting" level rather than from the "perturbed" value at the end of CS.) The present data therefore join several other reports that do not confirm the predictions of "stimulus substitution" theory (e.g., Fitzgerald, Vardaris, and Brown, 1966; and, Kadden, Schoenfeld, McCullough, Steele, and Tremont, 1980). At a strictly empirical level, it appears that the direction of heart rate UR to food may not be used as a predictor of the direction of the heart rate CR.

The lack of a satisfactory account of the variability in the cardiac rate CR, both between and within studies, and the inability to predict the outcome of "Pavlovian" studies with cardiac rate measures has been recognized in the literature (Harris and Brady, 1974). However, the intersubject variability in the direction of the heart rate CR may actually be the expected result if certain facts are taken into account. As mentioned earlier, it has been demonstrated that presentations of food or shock that are contingent on increases or decreases in either heart rate or blood pressure levels eventually affect the levels of the particular response system involved. In other words, respondent behaviors have been shown to be susceptible to influence by response-dependent procedures, the same procedures that are typically used to condition operant

responses. Carrying this parallel between operant and respondent behaviors one step further, the assumption is that "respondent" behavior could be affected by reinforcers delivered without any specified contingency, just as "operant" behavior was shown to be affected by reinforcers delivered without any specified contingency (Skinner, 1948, and Staddon and Simmelhag, 1971, provide examples of response-independent conditioning of "operants").

If the above assumption is correct, then "intruding" a stimulus into the organism's "behavior stream" would be expected to affect both "respondent" and "operant" behaviors, regardless of whether or not a contingency between the stimulus and the response was specified. However, with stimulus deliveries of this type it is not usually possible to specify in advance what the behavioral effect of the conditioning procedure will be. Such was the case in Skinner's "superstition" experiment, for which the behavioral effects of reinforcer presentations were only specifiable post hoc. For the case of heart rate, if the reinforcer happened to follow a deceleration in heart rate then such a change in heart rate would have an increased probability of occurring when the same conditions were encountered again (e.g., the same resting heart rate, or the same time since the last reinforcement or the presence of the same external stimulus). Given the occurrence of such response-independent stimulus deliveries, it would be possible for some animals to have increases in their heart

rates conditioned by US presentations, for other animals to have decreases in heart rate conditioned, and for some animals to have no change in heart rate conditioned (the latter case would not be absence of conditioning but rather conditioning of no change). Given the foregoing reasoning, the variability in the cardiac rate responses developed during response-independent stimulus deliveries could be seen as arising from the properties of the procedure and would not be considered to be either "noise" or anomalous. (Note: Under certain conditions there might be little variability in the direction of the heart rate CRs, even with response-independent delivery of reinforcers. If, for instance, an animal was given no adaptation to either the heart rate recording electrodes or to the experimental chamber, and displayed the predicted elevated resting heart rates, it might be expected that most conditioned responses that developed would be decelerations since those responses would have the highest likelihood of occurring, given the prevailing high heart rates. Another possible source of uniformity in the direction of the heart rate CR is the unconditional response to CS. If the subjects exhibit consistent, large magnitude URs to CS, it should be very likely that those URs will become the CRs if an appetitive US consistently follows such responses.)

In the introduction to the present study an extension of the analysis of the partial reinforcement effect by

Schoenfeld (1950, 1968) was made. The original work by Schoenfeld relied on both the data of Antonitis, showing an increase in response variability with the introduction of extinction, and on the notion of response subclasses. The present extension of that work presumed the existence of subclasses of heart rate CR and also took the Antonitis finding as applying to the cardiac rate response. The inevitable consequences of response-independent reinforcement, possible strengthening of responses in the "not-R" category, were then incorporated into the original argument.

In the present study, the schedule change from p(US/CS)=1.0 to 0.50 was essentially introducing extinction periods which, according to Schoenfeld's argument, would increase the number of different response subclasses emitted. With response-dependent deliveries of the reinforcer, some of these variations of the response would most likely be strengthened by reinforcer deliveries on the intermittent schedule. By contrast, with response-independent reinforcer deliveries the various response subclasses are not directly strengthened. The present paper argued that with cued, response-independent reinforcer deliveries (an operation typically called Pavlovian conditioning), increased response variability (as would result from a switch from regular to intermittent reinforcement) would change the temporal relations between the measured response (heart rate CR) and the stimuli (both

CS and US). The change in the temporal relations between CR and US result from both the increase in the number of CR subclasses and the independence of response and stimulus occurrences in response-independent procedures. Because of this independence of CR and US, the reinforcing stimulus can follow either "CR" or "not-CR". The changed CR-US relation might therefore increase the impact of the reinforcer either on the CR (if that response moved closer in time to US) or on behavior other than the CR (if the CR moved further away in time from US, thereby allowing instances of "not-CR" to precede US). Introducing extinction by switching the schedule from p(US/CS)=1.0 to 0.50 might therefore be expected to produce increases in CR magnitude for some animals and decreases for others. The particular behavioral effect would depend on which variations of the response were "brought out" by the extinction period and whether those subclasses served to enhance the US's effect on "CR" or on "not-CR". An additional possibility is that the extinction trials might bring out either relatively large or relatively small magnitude heart rate CRs and the subsequent response-independent reinforcement of either magnitude subclass would alter the magnitude of the conditioned heart rate response. The outcome would be the same as for the rearrangement of temporal relations between CR and US: the possibility of a variety of behavioral effects of a schedule change from a higher to a lower frequency of reinforcement.

The results of the present study are in agreement with the predictions of the above account. Slightly more than half of the subjects exhibited increased CR magnitudes with the change from p(US/CS)=1.0 to 0.50 while the remaining subjects showed either unchanged or diminished CRs with that change in p(US/CS). Increases in response strength with decreases in reinforcement frequency are commonplace in the "operant" literature but almost unprecedented in the "Pavlovian" literature. The present report adds to the small group of studies (Powell and Milligan, 1975; and, Razran, 1955) reporting such effects. As already mentioned, such findings argue against distinguishing "Pavlovian" and "operant" findings on the basis of the effects of intermittent reinforcement. Additionally, the present finding is not easily accounted for by either: 1) traditional explanations involving a summing of excitation from reinforcement operations and inhibition from extinction operations (Bush and Mosteller, 1955; Estes, 1950; and, Spence, 1960); or, 2) by the recent formulations involving "contingency" between stimuli, in this case between CS and US (Rescorla, 1967; and, Gibbon, Berryman, and Thompson, 1974). The latter accounts would predict that maximum response strength would result from maximum contingency between CS and US, which would occur at p(US/CS)=1.0. By contrast, the behavioral prediction of the present paper, based on: 1) the observed and the proposed effects of extinction; 2) the behavioral consequences of

response-independent reinforcement schedules; and, 3) the establishment of a CR before the introduction of extinction trials, was in agreement with the present finding. The last of the above conditions, the establishment of adequate CR strength before introduction of extinction trials, was accomplished by within-subject manipulation of p(US/CS). Use of such a design sets the present work apart from the body of literature exploring the effects of p(US/CS) on a "respondent" with infrahuman subjects.

The 0-hr deprivation condition was originally included with the expectation that lowered deprivation would increase the chances that "no-drink" trials would occur, by reducing the magnitude of, or eliminating entirely, the heart rate CR. If the lowered deprivation had such an effect, there would have been an opportunity to see if trials on which the animal failed to consume US were also trials on which the animal had no heart rate CR (an outcome suggested by the data of Schoenfeld, Matos, and Snapper, 1967). Although the lowered deprivation did not affect the drinking response, it did produce a diminution of the heart rate CRs, an effect that was not unexpected considering the usual effect of reducing hours of deprivation on appetitively maintained behavior. This is a case of a "respondent" (the heart rate CR) proving to be a more sensitive behavioral indicator of changed deprivation level than an "operant" (the consummatory response).

There is another point about the 0-hr deprivation condition that must be mentioned. Interpolating such a condition between exposures to values of p(US/CS) at 23-hr deprivation may have affected the data obtained at 23-hr deprivation. At 0-hr deprivation the rats with deceleratory heart rate CRs had substantially lowered pre-CS heart rates. The Law of Initial Value (Wilder, 1950, 1957) predicts that decreased resting heart rates would lower the magnitude of a deceleratory heart rate CR. Those rats displaying deceleratory heart rate CRs at 23-hr deprivation had CRs of approximately zero magnitude at 0-hr deprivation. When the rats with deceleratory CRs were exposed to p(US/CS)=0.50 at 0-hr deprivation, the sequence of events was as follows: pre-CS heart at a low level, CS onset occurs, heart rate remains at the pre-CS level, and US is presented following the 8-sec of steady heart rate in CS. Consistent with the previous reasoning in this paper, such a sequence of events should have served to strengthen "not-CR" (in this case, no change in heart rate) instead of CR (a deceleration in heart rate). After exposure to p(US/CS)=0.50 at 0-hr deprivation, the rats in the present study were then exposed to the next lower probability of reinforcement (p(US/CS)=0.25) at 23-hr deprivation. However, when comparing the performance at p(US/CS)=0.25, 23-hr deprivation with that at p(US/CS)=0.50, 23-hr deprivation, the effect of the reinforcement of "not-CR" at p(US/CS)=0.50 at 0-hr deprivation (the intervening condition) cannot be ignored. It seems

reasonable to expect that such a history of reinforcement would serve to lower CR magnitudes at p(US/CS)=0.25, 23-hr deprivation.

The alternation of deprivation conditions continued right through the lowest probability value (p(US/CS)=0.06) and for the final exposure to p(US/CS)=1.0. Exposure to p(US/CS)=1.0 at 23-hr deprivation, after the lowest probability value, was included as a "recovery" condition. The rats with acceleratory CRs displayed at least partial recovery of their heart rate CRs but the rats with deceleratory heart rate CRs displayed no recovery of their previous CRs at p(US/CS)=1.0. This difference may be accounted for by the history of reinforcement of "not-CR" for the rats with deceleratory heart rate CRs during the 0-hr deprivation conditions. That is, the deceleratory CR rats were not merely exposed to a series of conditions that decreased the magnitude of their heart rate CRs. Rather, they were exposed to a sequence of conditions that possibly involved reinforcement of responses other than the CR. If that were the case, then the change in p(US/CS) from 0.06 to 1.0 would serve to increase the opportunities for the reinforcer to follow the prevailing response to CS, no change in heart rate. Consistent reinforcement of "not-CR" would make recovery of the deceleratory CR seen on the initial exposure to p(US/CS)=1.0 all but impossible.

The discrimination data in Figures 1-9 may be taken as corroborating the above suggestion concerning the effects of

conditioning at 0-hr deprivation on the rats with deceleratory CRs at p(US/CS)=1.0 and 0.50. None of the rats with deceleratory CRs at the high p-values displayed such a response to CS-plus. Rather, those rats gave essentially no response to both CS-plus and CS-minus. Given their history of reinforcement of "not-CR" (mentioned above), this failure to develop a response to CS-plus is not surprising. Likewise, it is not surprising that two of the three rats with acceleratory heart rate responses (rats 1 and 12) displayed discriminated heart rate responses. These discrimination data may therefore be taken as being consistent with the explanations offered to account for other aspects of the data.

The present finding of equal heart rate CR magnitudes on trials following a reinforced trial and on trials following an unreinforced trial agrees with some reports in the literature (Fitzgerald, 1966; and Gormezano and Coleman, 1975), but is at odds with others (Holmes and Gormezano, 1970; and Thomas and Wagner, 1964). However, none of these other reports compared pre-CS heart rate levels on trials preceded by reinforced as against unreinforced trials. Without such a comparison there is the possibility that differences or equalities in CR magnitudes could arise as a by-product of different pre-CS levels on the two types of trials. Because of just such a possibility, the present study included a comparison of pre-CS heart rates on these

two types of trials and found no differences in this measure. The present study may therefore be considered the most legitimate report of equality of CR magnitudes after either one reinforced or one unreinforced trial and also after either two reinforced or two unreinforced trials.

The finding of an effect of varying p(US/CS) on pre-CS heart rate has already been discussed in the previous section. There is however, one additional comment to be made concerning this finding that relates to the position of the present paper that stimuli presented to an organism, either response-dependently or response-independently, may affect any behavior of that organism. Although CS was presented independently of the animal's behavior, it is still possible that CS could come to affect a particular response that preceded its onset. Such a possibility must be considered because stimuli presented response-independently do affect behavior (a fact that has already been brought out in this paper) and because a stimulus lacking the power to affect behavior may acquire such properties through "paired" presentations with a stimulus already able to influence behavior ("reinforcing" stimulus). Examples of the latter may be found in Pavlov's 1927 collection (CS acquired the ability to act as a new US after sufficient pairings with the original US) and also in Skinner's 1938 work (the sound of the food dispenser became a "reinforcing" stimulus, able to establish new behaviors,

by virtue of its pairing with food delivery). Since "pre-CS heart rate" was occurring immediately before the onset of CS it might be assumed that such behavior could be affected by CS presentations. For example, if heart rate was returning to baseline (from the high level after the last US presentation) when the CS was presented, decreases in rate or lowered heart rate levels might become more likely. Certainly the possibility of such an effect of CS presentations seems real, however there is the question of why so many subjects in this study and in Miller and Caul (1969) showed decreases in pre-CS heart rates with the change from p(US/CS)=1.0 to 0.50 or to 0.25 and then showed increased rates with a change to p(US/CS)=0.0 or 0.06. With response-independent presentation of the stimulus such between and within studies consistencies are surprising, considering the possible variation in the behavior followed by CS. However, since heart rate was elevated during US, or shortly after US, for most subjects in the present study, it may have been the case that after the intertrial interval, the heart rate was usually either decreasing towards the resting level or had already reached, or even "overshot", the resting level. Since CS consistently followed US at a delay equal to the intertrial interval, there was the possibility that on most trials CS was presented during periods when heart rate was either decreasing or at levels lower than the normal resting levels. This proposed situation would be analogous to a predicted effect of

response-independent reinforcement, a possibility already suggested by Schoenfeld (1972). The second half of the function, the increasing portion from p(US/CS)=0.25 to 0.06, may be accounted for either by the fact that as US presentations became less frequent so did the number of trials on which CS presentation had a chance to strengthen the decreasing or lowered heart rate, or by the fact that CS lost its secondary reinforcing powers as the frequency of US presentations approached 0.0.

The preceding paragraphs have reviewed the major findings of the present study and have attempted, where possible, to relate these findings to the propositions advanced in the introduction to this work. This paper has taken the suggestion that the observed increases in variability during extinction, seen with response-dependent delivery of the reinforcer, arose from the reappearance of previously reinforced response subclasses and extended it to the case of changes in p(US/CS) that involve increasing degrees of intermittency of response-independent reinforcement. It was argued that, within certain limits, the use of a "Pavlovian" as against an "operant" procedure, or the measurement of "respondent" as against "operant" behavior was of secondary importance in predicting the results of specific changes in the schedule of reinforcement. The factors that were put forth as being critical to the present results were: 1) a "substantial"

degree of response strength at the introduction of extinction trials; 2) the increase in response variability (broadening of the response subclass distribution) that occurs when extinction trials are introduced; 3) variations in the temporal relations between the measured response and the reinforcer, an inherent feature of response-independent conditioning procedures; and, 4) the possibility of reinforcement of responses other than the measured CR, also an inherent feature of response-independent conditioning procedures.

The differences in response-reinforcer interactions between response- dependent and response-independent procedures were recognized and utilized in the present paper. However, it was argued that those differences cannot be equated with the operant/Pavlovian distinction that underlies two- factor learning theory. Response-independent and response-dependent procedures have similarities and differences that cut across the boundaries of the traditional "types" of conditioning and "types" of behavior. The present findings of variability in the direction of the conditioned cardiac rate response and of increases in CR magnitude with a shift from p(US/CS)=1.0 to 0.50 in at least half of the subjects were taken as evidence that the operations of extinction and reinforcement have effects that are independent of the "type" of behavior or the "type" of conditioning procedure involved. That is, response-independent reinforcement appears to have similar

effects on both lever presses and cardiac rate CRs: a complement to the observed similarities of the effects of response-dependent reinforcement of lever pressing and heart rate. It may therefore be suggested that the independence or dependence of the reinforcer on the occurrence of a particular response class are more important determiners of the effects of the conditioning operation than the operant versus Pavlovian designation of procedures customarily indicate.

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