

PARTIAL REINFORCEMENT OF A
CONDITIONED EMOTIONAL RESPONSE

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SCOPE AND CONTENTS:

Experiments were conducted, with rats, to ascertain the effects of partial reinforcement in aversive classical conditioning. Conditioned suppression of bar-pressing was more resistant to extinction following intermittent reinforcement of a conditioned stimulus than following consistent reinforcement. This effect was obtained whether or not bar-pressing was permitted during conditioning as well as during extinction. The effect was amplified by interpolating a large block of nonreinforced trials early in the partial schedule; it was eliminated by adding more reinforced trials prior to the partial schedule. The effect was not obtained by interpolating a large block of nonreinforcements in a continuous schedule. The data were related to current theoretical conceptions of partial reinforcement.

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CHAPTER ONE
INTRODUCTION

The present thesis is concerned with "partial reinforcement" within a classical conditioning procedure. Classical, or Pavlovian, conditioning customarily involves the presentation of two stimuli to an animal in close temporal sequence. One of these, the Unconditioned Stimulus (US)¹, is a relatively important event (e.g., food, shock) to the animal, while the other, the Conditioned Stimulus (CS), is relatively unimportant prior to conditioning. If the CS is presented just before the US a sufficient number of times, an "association" may be formed between the two so that the CS acquires some degree of control over the animal's behavior (which may or may not be similar to the effect that the US has on behavior). In this case the US is said to have "reinforced" the CS or "reinforced" the association. "Partial reinforcement" (PRF) is a procedure in which the CS is sometimes followed by a US, but sometimes is simply presented alone; with "continuous reinforcement" (CRF), the CS is always followed by a US.

There is at least a formal similarity between the above procedure and the partial reinforcement conditioning procedure for instrumental responses. With instrumental PRF, however, it is a specified response which sometimes is and sometimes is not followed by the "reinforcing" event.

¹While each symbol or abbreviation will be defined when first used, a list of all of them will be found in Appendix A.

Given the formal similarity of procedures used in classical and instrumental PRF, the question arises as to whether their behavioral effects are, in any way, similar; and if so, whether the effects arise from the same mechanisms. In the literature on PRF, the tendency over the years has been to lump classical and instrumental studies together (e.g., Lewis, 1960). But, as will be made clear in our historical review, some evidence has been accruing to suggest that the two classes of PRF procedures might best be kept distinct.

There has been one PRF phenomenon which has received by far the greatest amount of experimental attention, and which has therefore acquired the status of the "partial reinforcement effect" (PRE), even though there are other effects of PRF. Paradoxically, or so it has seemed to many, PRF has typically led to greater resistance to extinction² than has CRF (Jenkins and Stanley, 1950; Lewis, 1960). The apparent paradox stems from the traditional notion that reinforcements increase response strength while nonreinforcements simply decrease it. Resistance to extinction is assumed, of course, to vary directly with response strength. The PRE, however, was a case in which nonreinforcements, interspersed among reinforcements, actually increased resistance to extinction over that which would have been generated by reinforcements alone.

The PRE has been a very frequent and easily replicable result. But it has happened that most of the empirical and theoretical work on PRF has come from instrumental studies with animals. This has been the case

²In extinction reinforcement is omitted permanently with the result that the response in question eventually decreases in strength or probability.

even though an historically important stimulus was provided by the classical conditioning studies of Humphreys (1939, 1940) with humans. Those relatively few studies which have employed classical conditioning PRF with animals have encountered extreme difficulty in demonstrating a substantial or reliable PRE (cf. Spence, 1966; Wagner, Siegel & Fein, 1967). Since several human eyelid or galvanic skin response (GSR) conditioning procedures (presumably "classical") have provided PREs, a current hypothesis (e.g., Spence, 1966) is that the species of the subjects is very important: that a Pavlovian PRE requires that the animal be capable of "higher psychological processes" or have a well-developed cerebral cortex, etc. This view, however, leaves unexplained why lower animals should so readily display a PRE within instrumental training procedures, unless different mechanisms underlie the two types of PRE. Another possibility, however, is that a more sensitive classical conditioning procedure is required in order to detect a reliable Pavlovian PRE in infrahuman Ss.³

We have been discussing PRF in terms of classical and instrumental procedures. It is true that we are primarily concerned with processes. But at present there is no way of being certain that a given Pavlovian procedure involves only Pavlovian associations⁴, nor that any instrumental procedure produces only instrumental associations. For that matter, it is

³Kimble (1961) was one of the first to emphasize a difference between classical and instrumental PRF, but he was concerned with what seemed to be important differences in acquisition effects. If anything, the evidence on acquisition PRF would now indicate a strong similarity in that regard.

⁴Not even eliminating peripheral responses with curare would solve the problem since central neural processes could occur and be reinforced (and partially reinforced if the reinforcer were intermittent).

not at all certain that the processes which we refer to as "associations" will ultimately fall neatly into a classical-instrumental dichotomy.

Nevertheless, the distinction between classical and instrumental procedures lies at the root of much contemporary theorizing about learning. The distinction with respect to the PRE would be of major significance, of course, if it were indeed the case that a PRE could not be obtained within Pavlovian procedures, or if any PRE so obtained responded to parametric variation in ways radically different from those already known in the case of the instrumental PRE.

Since a recurrent question throughout this thesis will be whether or not a given experiment has demonstrated a PRE, it is important that the criteria for such an effect be considered in some detail. In general, a PRE will be said to have been produced if the PRF treatment leads to significantly greater resistance to extinction than does the CRF treatment with which it is compared, although additional control groups will sometimes be required. The greater resistance to extinction of the PRF group may be indexed by the number of trials to an extinction criterion, by the performance on a given trial or block of trials following acquisition, by the slope of the extinction curve, etc. Some of these indices might provide more striking evidence of a PRE than others, of course. Furthermore, in attempting to demonstrate a PRE, there are some experimental outcomes which are relatively unambiguous and others which are not at all clear. The degree of ambiguity depends on the performance of the PRF group relative to the CRF group in acquisition, as well as in extinction. Consider, for example the following possible outcomes:

Case 1. The most dramatic type of PRE would be obtained if the PRF group showed the poorer performance in acquisition (the lesser response

strength) but the better performance in extinction; in other words, if the PRF group, in extinction, reversed its relationship to the CRF group, A PRE would have been obtained in spite of an initial advantage for the CRF group.

Case 2. If the two groups show identical terminal acquisition performance, while the PRF group then shows greater resistance to extinction, a PRE would again be demonstrated.

Case 3. An ambiguous situation arises if the PRF group shows the better performance in both acquisition and extinction. Even if the separation of the two groups is greater in extinction than in acquisition, it cannot be ascertained with confidence whether any of the PRF's superiority in extinction is attributable to the PRF schedule per se. The behavior of the PRF group might, e.g., have been duplicated by a second CRF group having the same terminal acquisition level.

Case 4. It is not infrequent that a PRF group shows the poorer acquisition performance but then does not seem to differ from the CRF group in resistance to extinction. Or both groups may reach an extinction criterion at the same time, with the result that the CRF group has shown a faster rate of extinction (in this case the CRF group would have shown the greater response strength on many extinction trials). It could be argued that this type of outcome qualifies as a PRE on the grounds that the PRF group, with its weaker response strength at the start of extinction, should otherwise have reached the extinction criterion more rapidly than did the CRF group. However, it is conceivable that the rate at which extinction performance changes is a function of the distance over which the performance can change, and that the schedule of reinforcement is

irrelevant. Again, a CRF control group displaying the same terminal acquisition performance as that of the PRF group would be desirable.

From a practical standpoint, a detailed analysis of the PRE would presumably require the establishment of an unambiguous PRE as a standard against which other treatments could be compared. The experiments to be reported were designed with this consideration in mind.

The experiments begin, therefore, with an effort to demonstrate a clear-cut PRE with a classical conditioning procedure. The procedure employed was a modification of the Estes-Skinner (1941) conditioned emotional response (CER) technique, according to which the degree of association between a CS and an aversive US is indicated by the degree to which the CS interrupts (suppresses) food-rewarded instrumental activity. Not only do the experimental operations of the CER conform to the Pavlovian paradigm, but the procedures have been shown to be appropriately sensitive to the variation of a number of parameters important in Pavlovian salivary conditioning -- e.g., temporal relations between CS and US, CS intensity, US intensity (cf. Kamin, 1965).

Once a Pavlovian PRE was obtained, using the CER, the objective was to investigate variables which might be expected, on the basis of data and theories derived from the study of instrumental PRF, to affect the size of the PRE. Whether these variables affect a Pavlovian PRE in the same ways in which they affect an instrumental PRE will be, of course, one focus of interest.

CHAPTER TWO

HISTORY

Since our first experimental objective was to demonstrate a PRE with a classical conditioning procedure, the literature relevant to this matter will be reviewed first. Although the studies employing classical conditioning procedures have been placed by their authors in one or another theoretical context, they have been primarily concerned with the presence or absence of a PRE, rather than with a general theory of the effect. However, the final experiments of the thesis involved the study of variables assumed to affect the magnitude of a Pavlovian PRE. The second section of the History will therefore review several attempts to account theoretically for the major empirical findings associated with PRF procedures. The empirical work supporting such theories has been primarily instrumental, of course, and it will be of interest later to note whether there are discrepancies between the more important and reliable instrumental findings and those of the CER PRE. An absence of any such discrepancies would suggest that the same basic processes were involved in the two types of PRE.

Classical Conditioning Studies of PRF

The review of the Pavlovian experiments is organized, roughly, by species, since one of the current hypotheses is that "lower" animals are incapable of manifesting a Pavlovian PRE. There is little doubt that a PRE can be obtained using certain Pavlovian procedures (GSR, eyeblink)

with human subjects (e.g., Grant & Schipper, 1952; Bridger & Mandel, 1965; cf. Lewis, 1960). Partly for this reason, the human studies will not be reviewed. But, in addition, the procedures and results of human conditioning experiments seem very far removed from those of animal experiments. The use of instructions, the linguistic capabilities of humans, and certain methodological problems (e.g., the classification of "voluntary responses" in eyelid conditioning) certainly suggest that human and animal data should be treated separately for the time being.

Dogs

As might be expected, Pavlov (1927) had occasion to observe some effects of PRF, although neither he nor his students appear to have conducted detailed studies on the topic. One instance (Chapter 22, p. 384) involved a single animal which was trained with a series of different CSs, each with a different fixed ratio of reinforcement (food). Reinforcement of every other presentation of the first CS produced conditioned salivation with accustomed speed, according to Pavlov. Acquisition was even faster with the next CS, which was reinforced every third time. However, when the third CS was reinforced on a four to one ratio, the conditioned reflex failed to develop even after a large number of trials. Extinction was not studied in this dog.

In another section (Chapter 4, pp. 53-4), however, Pavlov (1927) has described what was, apparently (the passage is not absolutely clear), the opposite of a PRE when dogs were given repeated acquisitions and extinctions; that is, successive extinctions were found to lead to increasingly less resistance to

extinction. But the result is in contrast to a more recent one-dog experiment by Konorski and Szwejkowska (1950) in which increasing resistance to extinction was reported with successive acquisitions and extinctions. It is clear that neither of these studies provides conclusive information on extinction PRF effects.

A more detailed study of the acquisition effects of PRF was made by Brogden (1939), with leg flexion as the conditioned response and shock as the US. While the flexion response did not vary significantly with the different percentages of reinforcement, ranging from 100% to 20%, Brogden did observe a decrease in such responses as vocalization with a decrease in percentage of reinforcement. Again, no extinction data were reported.

PRF of the salivary response in dogs has been studied by Fitzgerald (1963) who used acid as a US for salivation. Three groups were run, comparing CRF with 50% and 25% PRF. A significantly higher level of response strength in acquisition was achieved by the CRF group than by either PRF group. An extinction PRE was said to have been demonstrated, but the results seem ambiguous in this regard. As the measure of resistance to extinction, a S's response strength in extinction was expressed as a percentage of its terminal acquisition level. With this measure, the CRF group showed a significantly greater percentage decrease in response strength than did either PRF groups. However, the PRF group never were superior in absolute performance at any time in extinction. This is an example of one of the "ambiguous" PREs discussed in Chapter 1 (Case # 4). The CRF group, which showed the fastest rate of extinction,

had also achieved a higher acquisition level, and had received many more (two to four times as many) reinforcements than had the PRF groups. It is quite possible that extinction performance equivalent to that of the PRF groups could have been obtained with CRF groups which were given the same number of reinforcements as the PRF groups, or which had been trained to the same terminal acquisition level.

Wagner, Siegel, Thomas, and Ellison (1964), as part of a larger study, obtained quite similar results with a comparison of CRF and 50% food reinforcement of a tone CS. The CRF group showed greater conditioned salivation both at the end of acquisition and, characteristically, on the first trial of each extinction day; this, of course, suggests the opposite of a PRE. But within extinction days the CRF group tended to drop to the level of the PRF group. The CRF group thus displayed a greater decrement in response strength than did the PRF group. The overall superiority of the CRF group from day to day was interpreted as evidence against the existence within Pavlovian conditioning of the dramatic PRE typically obtained with instrumental PRF. The within-session effect, however, was considered to be a rudimentary (and in our terminology, ambiguous) form of PRE, which is critically dependent on the use of short intertrial intervals (ITIs). The so-called "aftereffects" theory (cf. p. 35, below) of PRF was suggested as a possible explanation of this and any other PRE which might be demonstrable with classical reward situations. This view suggests that, with short ITIs, the aftereffects of nonreinforcement persist until the next trial. For the CRF Ss these aftereffects

are never part of the conditioned stimulus complex prior to extinction, and a drop in responding occurs early in extinction as a result of generalization decrement. The PRF Ss, for whom acquisition included many reinforced trials with these aftereffects present, would show less decrement during extinction.

However, as with the Fitzgerald (1963) experiment, these results are difficult to interpret in the absence of controls for differences in terminal acquisition performance. Whatever the explanation is for these results, it is clear that the ambiguous PRE in this study is relatively unimpressive compared to the clear, large effects which are commonplace with instrumental PRF.

Further evidence of an extinction PRE has been reported by Fitzgerald and his associates in a series of experiments on conditioned heart-rate acceleration with shock reinforcement. Fitzgerald (1966) reported two experiments. In the first, a 100%, a 50%, and a backward control group were equated for trials (12), but no PRE was obtained. In the second, similar groups were equated for reinforcements, such that the PRF group had 12 reinforcements (Rs) and 12 nonreinforcements (Ns) instead of 6Rs and 6Ns.

The CRF and PRF groups responded similarly throughout most of the acquisition period, although late in acquisition there was some tendency for the PRF animals to show a greater heart-rate increase to the CS than did the CRF Ss. This, however, was reported to be the result of struggling by PRF animals, which evidently occurred only on those acquisition trials on which PRF Ss made extra-large heart rate responses. There are, of

course, unconditioned reflex relations between skeletal activity and heart rate acceleration. The authors report that struggling during extinction was too infrequent to affect the results. The PRF animals showed greater heart-rate acceleration throughout extinction, during which the separation between groups increased, resulting in a significant Treatments by Trials interaction. Thus, on the assumption that the apparent difference between groups at the end of acquisition was an artifact related to struggling, the PRE would seem to be unambiguous -- unless the struggling during acquisition by PRF Ss had effects which persisted into extinction.

Fitzgerald, Vardaris, and Teyler (1966) obtained a similar PRE using the same type of procedure but with an even longer acquisition. They also found, in agreement with instrumental PRF studies, that the additional resistance to extinction produced by a PRF schedule would survive an interpolated period of CRF with very little diminution, although the PRE under these conditions fell barely short of statistical significance. As in the Fitzgerald (1966) study, the PRF Ss showed somewhat higher response strength in the latter portion of acquisition as a result of struggling; but again, it was reported that struggling was not an important factor with respect to the extinction PRE. Additional information concerning the relation between heart-rate changes and skeletal responses would certainly be helpful in interpreting these data. Perhaps the use of curarized Ss (whose skeletal responses are prevented) would eliminate the problem.

In discussing their results, Fitzgerald et al. make reference to a "competing response" theory of PRF which is taken up in a later

section of this chapter (cf. p. 29).

Rabbits

Thomas and Wagner (1964) conducted an eyelid conditioning study with rabbits, using an extremely long acquisition period, with CRF and 50% PRF groups equated for reinforcements (220). In acquisition, the PRF Ss required twice as many trials as the CRF Ss to make the first conditioned response, and to reach an 80% anticipatory response criterion. They also received only half as many reinforcements within a given number of trials, so that there was almost no observable effect during acquisition of the non-reinforcements. The groups did not differ in terminal acquisition performance. Finally, the extinction curves were practically identical. The authors suggested, on the basis of the evidence available at that time, that a classically conditioned PRE was probably unattainable with most infrahuman organisms.

Pigeons

Longo, Milstein, and Bitterman (1962) reported two experiments which were unsuccessful in producing an extinction PRE. Both measured pigeons' activity in response to a light CS which was paired with shock. In the first, a partial group reached, in acquisition, a somewhat higher level of responding than did two CRF groups with which the PRF group was matched for trials and for reinforcements, respectively. This relationship persisted in extinction, but apparently was not significant: however, within the number of trials given, no group showed a significant loss of response strength. In the second experiment, the CRF group reached

and maintained a higher performance than did a PRF group, equated for reinforcements, but there was no significant extinction difference.

Slivka and Bitterman (1966) also employed the activity measure, but with appetitive (food), rather than aversive, conditioning. A CRF group and PRF group were matched on the basis of their performance at the end of an initial CRF period. During differential training, the groups did not differ in magnitude of conditioned response (CR). And in extinction there was no significant main effect of schedule of reinforcement. But within each extinction day, there was a significant tendency for the PRF group to show the greater CR magnitude on the later trials of the session. The activity displayed by the two groups during the CSs indicates, therefore, a small PRE. However, one of the figures of the report presents group curves for baseline activity (that is, activity during periods other than the CSs). From these curves, it appears that during acquisition and the first part of extinction the PRF group had a higher baseline activity level. The possibility exists, then, that the relatively small "PRE" was simply the result of generally high activity on the part of the PRF Ss. The baseline differences (assuming that they were large enough to be important) could, of course, have been the result of any of a number of factors, including the reinforcement schedules themselves. The Slivka and Bitterman study would therefore seem to be a suggestive, but not entirely conclusive, demonstration of a PRE.

Fish

Most of the studies on fish are also from Bitterman's laboratory and have provided, for the most part, negative results. Three papers

(Gonzalez, Longo, & Bitterman, 1961; Gonzalez, Milstein, & Bitterman, 1962; and Berger, Yarczower, & Bitterman, 1965) reported a total of 13 goldfish aversive conditioning experiments with an activity CR and a large number of procedural variations: different ITIs, patterns of PRF, CS-US intervals, N-lengths (runs of nonreinforcements); some PRF and CRF groups were equated for trials and others were equated for Rs. The only result in all these goldfish studies which might offer even ambiguous evidence of a PRE was a significant Treatments by Days by Trials interaction in Experiment 4 of the Berger et al. report: within some extinction days, a CRF group showed greater CR strength on the first trials of a session and less CR strength on the final trials than did one of two PRF groups. The authors did not consider this to be a convincing demonstration of a PRE.

In contrast to the goldfish studies, an experiment by Gonzalez, Eskin, and Bitterman (1963), using a different species of fish (mouth-breeders), did provide a PRE. Again, the CR was an aversively conditioned increase in general activity; the CRF and two PRF groups were equated for Rs; and both PRF groups, which differed in amount of initial CRF prior to PRF, showed greater resistance to extinction than did the CRF group. The PRE was particularly impressive since the CRF group had had a significantly higher terminal acquisition performance. These investigators thus emphasize the importance of species differences in reference to the discrepancy between goldfish and mouthbreeder results. However, if species differences are important, they certainly do not, in the light of available data, fit any obvious phylogenetic pattern.

One final experiment with goldfish (Geller, 1964) employed a conditioned-suppression technique in which pairings of a light CS and a shock US were superimposed on lever-pressing for food reinforcement. CRF and 50% PRF fish were trained to an acquisition criterion and then extinguished. The 50% group took significantly fewer trials to extinguish -- i.e., the opposite of a PRE was obtained.

Earthworms and Planaria

A convincing demonstration of a PRE with either of these organisms would be an outstanding achievement, particularly in view of the lack of agreement as to the existence of true conditioning in such creatures. Experiments by Wyers, Peeke, and Herz (1964) with earthworms and by Kimmel and Yaremko (1966) with planaria attempt to make such a demonstration. Of the two, the study by Wyers et al. appears to be the better controlled. The conditioning procedure involved the pairing of a vibration from a doorbell (the CS) with the onset of a "photo-flood" light (US). CRF and PRF groups were equated for trials, with the CRF group thereby receiving more CS-US pairings than the PRF group (which, except for initial CRF trials, received 50% reinforcement). One control group received only CS presentations and a second group received both stimuli in blocks of 10 USs followed by 5 CSs. These somewhat unorthodox controls were included as a replication of groups run by Ratner and Miller (1959). The CR was a rearing and withdrawal movement by the worm, very much like the unconditioned response to the light. A check was made on the reliability of scoring CRs, and all subjects were run with their assignments to treatments hidden from the scorer. The CRF and PRF groups

showed equivalent acquisition performance, much superior to the two controls. The PRF Ss made significantly more CRs during extinction than did the CRF Ss, suggesting a clear PRE. There are, however, at least two major objections to the study. According to the first (cf. Underwood, 1966), backward conditioning control groups should have been included for both CRF and PRF groups; this might have produced more responding to the CS than did the presentation of CSs and USs in long blocks. Secondly, Wyers, et al. (1964), themselves, have pointed out that previous data with earthworms showed that massed trials lead to less responsiveness following a rest interval than do spaced trials. This in itself would seem to provide a satisfactory account for the alleged "PRE", since the CRF group's conditioning trials were more massed than those of the PRF group.

Kimmel and Yaremko (1966) studied three groups of planaria, equated for total numbers of CSs and USs. For a 100% group (CRF), all CSs and USs were paired; a 50% PRF group received half of each in CS-US pairings, the rest "unpaired"; and a 0% group received all CSs and USs unpaired. The degree of "unpairing" is not at all clear, since the CS-US interval was two seconds and the inter-trial interval was approximately 15 seconds. Inter-observer reliability was indicated as being "almost perfect"; there was no indication that in extinction the E was unaware of each S's acquisition treatment. In acquisition, both 100% and 50% groups were credited with more CRs than the 0% group; in extinction the 100% did not differ from the 0%, while the 50% group gave more CRs than either of the others. This outcome is suggestive of a PRE, though it is disconcerting that the 100% group should have displayed no more resistance

to extinction than the 0% group. However, the earlier experiment by Wyers et al. (1964) suggests a further serious problem, even granting the existence of any conditioning: namely, the lack of control for the different intervals between CS-US pairings of the 100% and 50% groups. This is not the place to go into the larger question of demonstrating any conditioning in planaria, but presumably a convincing replication would have to include more information concerning procedures, as well as additional control groups.

Rats

Rat experiments have been held until the last, since they relate most directly to the experiments to be reported in the body of the thesis.

The first two studies to be reviewed do not involve classical conditioning procedures exclusively, but rather show "transfer" effects from classical pairings to instrumental behavior. Crum, Brown, and Bitterman (1951) administered classical, buzzer (CS)-shock (US) pairings to rats on PRF and CRF schedules; they then gave escape training to shock alone; and finally they tested for resistance to extinction of the same escape response to the buzzer alone. Under these conditions the PRF rats took longer to reach an extinction criterion for the instrumental response.

Knott and Clayton (1966) found that rats preferred to press a lever which produced a tone, rather than a lever which did not, when the tone had been paired with rewarding brain stimulation either continuously or intermittently. However, this conditioned reinforcement effect was stronger following intermittent pairings than following continuous pairings.

Again, a PRE based upon classical pairings was manifested through instrumental responding.

Turning to procedures that are more directly "Pavlovian", we may first mention a heart-rate conditioning experiment on rats, by Fitzgerald, Vedaris, and Brown (1966). A tone was the CS and shock the US. There was no significant difference during acquisition between CRF and 50% PRF. In extinction, no loss of response strength took place within the number of trials allotted, so that extinction effects of PRF could not be determined.

Geller, Kailan, Stein, and Brady (1957) performed what is apparently the first study of PRF using the Estes-Skinner conditioned emotional response (CER) technique. The study has never been published and, except for graphs and brief notes provided by Geller and Stein in personal communications, a detailed account has not been available. However, it appears that PREs were obtained in two separate CER experiments. The CER procedure involved, first, training rats to bar-press for food on a variable interval schedule, and then, while the animal was engaged in that activity, presenting either CS-shock pairings or CSs alone. Conditioning is indicated by the degree to which the CS comes to suppress the rat's bar-pressing relative to the pre-CS rate of pressing. Both experiments equated CRF and PRF groups for trials, so that the CRF Ss received many more reinforcements. When 100% and 50% schedules were compared in a spaced trials (6 - 7 hours apart) study, the two curves for mean amount of suppression were similar during acquisition, but diverged in extinction, with the PRF group showing more resistance to extinction. In the second study, a 25% PRF group showed

slower acquisition and a lower asymptote of suppression⁵ than did a 100% group, under massed trials (15 minutes apart) conditions. However, the two curves crossed each other in extinction, with the CRF Ss extinguishing more rapidly. These results certainly suggest a PRE; but, unfortunately, data on baseline rates of bar-pressing were not available. The different frequencies of shock reinforcement, particularly under massed trial conditions, might well have influenced the baseline rates and, indirectly, the rates of extinction as indexed by the suppression ratios.

A report of much the same type of result has been published by Brimer and Dockrill (1966). In two separate experiments, significant PREs were obtained in comparisons of a PRF and each of two CRF groups. The two CRF groups in each case were equated with the PRF group for trials and for reinforcements, respectively, within a daily session of four trials. In one experiment the percentage of PRF was 50%, in the other, 25%. The relative efficacy of the two schedules could not be ascertained because of procedural differences between experiments; but both were very effective.

There is one procedural aspect of the Brimer and Dockrill experiments which complicates interpretation of the PREs which they observed. Each S was maintained on its schedule of reinforcement until an acquisition criterion was met, at which point the S was placed on extinction. A

⁵Willis and Lundin (1966) have provided data on the maintenance of the CER under PRF. They rotated three rats through different percentages of reinforcement, and found that suppression was maximum with the highest percentages. Extinction was not studied in this experiment.

consequence was that there were significant differences among groups in the number of trials to criterion, with the PRF groups requiring the most trials. The rationale for employing an acquisition criterion was presumably to equate groups for level of performance at the outset of extinction, so that any extinction PRE would be unambiguous. However, the use of an acquisition criterion raises problems of its own. Individual Ss will certainly differ with respect to such variables as amount of reinforcement, and, more critically, the groups, too, may differ in the same respects. Baseline response rates (which might have been affected by differences in number of reinforcements) were not reported.

Nevertheless, the Brimer and Dockrill results, together with those of Geller et al., suggest that the CER procedure might be especially sensitive for a detailed study of the effects of PRF.

The final study to be reported, by Wagner, Siegel, and Fein (1967), has had considerable influence on the present thesis (a prepublication report was made available by the authors), since it strongly suggested that the CER was not, in fact, suitable for the study of a Pavlovian PRE. Two quite different experiments were conducted, the second of which was most directly relevant. The first experiment involved the measurement of a startle response in conjunction with PRF and CRF fear conditioning. With an equated-trials procedure, the CRF Ss received twice as many CS-US pairings as the 50% PRF Ss, and the CRF Ss showed the greater startle response throughout acquisition and extinction. I.e., there was no evidence for a PRE.

The second experiment was addressed to the discrepancy between the PRE obtained by Geller et al. (1957) and the otherwise almost universal absence of PREs with classical conditioning procedures. Wagner et al. speculated that the basis for the discrepancy might be the fact that the customary CER procedure, as used by Geller et al., involves the "...intermingling of classical and instrumental acquisition experiences..." (1967, p. 162); i.e., fear conditioning (the pairing of CS and US) is superimposed on the appetitive behavior (bar-pressing). The authors therefore employed two pairs of PRF and CRF groups in a CER experiment. One pair received the usual CER procedure, but the other pair was given all but the last four trials of its CER training with the foodcup and bar covered over. During the preliminary bar-press training, the last four CER trials, and the subsequent CER extinction, bar-pressing was permitted to all Ss. (Bar-pressing was necessarily permitted in CER extinction, of course, in order to assess the Pavlovian conditioning.) The "bar-out" procedure (as we will call it) precluded the possibility of "fortuitous" associations between instrumental bar-pressing and shock during CER training.

The four groups in this study displayed equal suppression on the last day of acquisition. In extinction, a significant PRE was obtained with the CRF and PRF groups which had been allowed to bar-press; but there was no evidence for a PRE with the bar-out groups. The fact that the PRE was observed only when the CS-US pairings were superimposed on on-going instrumental behavior was interpreted by Wagner et al. as indicating that the observed PRE was in some way dependent upon instrumental acquisition experiences.

These data indicate, therefore, that the one conditioning technique which had appeared sensitive enough to warrant extensive use in Pavlovian PRF studies was, in fact, not appropriate for this purpose.

Summary of Classical Conditioning Studies of PRF

The number of reasonably convincing demonstrations of a Pavlovian PRE has obviously been quite small compared to the quantity of negative results. The conditioned heart rate experiments on dogs, by Fitzgerald and his associates, provided quite reliable PREs, but a more convincing outcome might have been achieved had the contribution of skeletal (instrumental) movements been investigated more thoroughly.

The substantial PRE reported by Gonzalez, Eskin, and Bitterman (1963), using mouthbreeder fish, contrasts sharply with a large number of unsuccessful goldfish studies which employed the same procedures.

The two remaining demonstrations of a PRE which have not, as yet, been challenged by other research were the two rat experiments in which instrumental PREs were observed following differential training (PRF vs. CRF) in classical conditioning situations (Crum, Brown, & Bitterman, 1951; and Knott & Clayton, 1966).

The one Pavlovian procedure which, at first, appeared well suited for the study of the PRE was the CER technique⁶. This procedure, as we

⁶In a sense, the CER procedure is similar to the procedures employed by Crum et al. (1951) and by Knott and Clayton (1966): both involve instrumental behavior in the assessment of Pavlovian associations. But a critical aspect of the CER is that the instrumental behavior (bar-pressing) is maintained by a reinforcer (food) which is quite unrelated to the CS or US of the Pavlovian conditioning in question.

have seen, has been sharply challenged by the results of the Wagner, Siegel, and Fein (1967) experiment which indicated that the CER PRE was dependent on some fortuitous relationship between bar-pressing and either the CS or the US of the Pavlovian conditioning. Whether some such dependency is necessary to produce a CER PRE was the subject of the first formal experiment of the present thesis.

Acquisition effects of PRF are not a major concern of the experiments to be reported, but we may note that there is substantial agreement in this regard among the Pavlovian studies reviewed: if PRF does have an influence on acquisition performance, it is to produce a lower terminal level of performance and/or slower initial acquisition.

Theories of Partial Reinforcement

The theoretical interpretations of PRF have arisen, almost entirely, from studies of appetitive instrumental reinforcement, which constitute the major portion of the empirical work on PRF. While some of the theories do make reference to aversive and/or classical conditioning, most are quite specific to the situation in which rats are trained to traverse a runway for food reinforcement on a discrete trial basis⁷.

Nevertheless, since we shall be making comparisons between the empirical

⁷The extensive research with "free-operant" procedures has been carried on more or less independently of the major PRF theories. The lack of control over inter-response times and the problems of response classification consequent upon unrestricted responding have made the free-operant situation less satisfactory than discrete trial procedures for the investigation of a number of theoretically relevant PRF issues.

effects of various experimental manipulations on Pavlovian and instrumental PREs, it will be useful to have some theoretical guidance in organizing the data. Particularly, as we shall soon indicate, some of the major theories designed to account for instrumental PRF data can be quite easily extended to apply to the classical aversive case.

For detailed discussion of the empirical work on instrumental PRF, reviews by Jenkins and Stanley (1950), Lewis (1960), Amsel (1962), and Capaldi (1966) should be consulted. However, we should mention here a number of characteristics of instrumental PRF which have been both reliable and of theoretical concern. An extinction PRE has been obtainable under very widely spaced trials (Weinstock, 1954, 1958); a PRE can be reduced, but only to a very minor degree, by the interpolation of CRF between a PRF schedule and extinction (Jenkins, 1962; Theios, 1962); and it can be considerably reduced or eliminated by preceding the PRF by a large amount of CRF. (Sutherland, Mackintosh, & Wolfe, 1965; Hothersall, 1966) In spite of the sizable literature on PRF, the effects of many other theoretically relevant variables on the extinction PRE are either unknown or have not been consistent across experiments. As Lewis (1960) has noted, adequate parametric studies have been infrequent.

With regard to initial training of an instrumental response, there is considerable agreement that acquisition is more rapid under CRF than under PRF. Thereafter, however, the CRF superiority is sometimes maintained for the duration of acquisition, but sometimes PRF leads to a higher level of response strength, particularly if acquisition is extremely long (Wagner, 1961).

There have been two general types of PRF theories, "cognitive" and "conditioning". The former type has been the less productive of research, and can be quickly reviewed.

One of the earliest of the cognitive hypotheses was that of Humphreys (1939). It was said that a S on continuous reinforcement would build up a strong "expectancy" of regular reinforcement; that the first nonreinforcement in extinction would sharply contradict the expectancy, and would soon lead to the expectancy of no reinforcement. But for partially reinforced Ss, nonreinforcements would not be unexpected, and it would require many more of them in extinction to build up an expectancy of consistent nonreinforcement. The main criticism of this view has been that the nature of an animal's expectancy in a particular situation would have to be inferred either from what the animal in fact does (ex post facto interpretation) or from what the human observer feels, intuitively, ought to be "expected" (anthropomorphism). The theory lacks explanatory and predictive power, therefore.

Lawrence and Festinger (1962) have attempted to revive a cognitive interpretation of PRF, using a concept of "cognitive dissonance" to integrate evidence from studies of PRF, delay of reinforcement, and effortfulness of response. One of the hypothesized processes is that the nonreinforcements within a PRF schedule induce S to find "added attractions" in an empty goal box. The added attractions are assumed to reduce "dissonance", which is aversive, and which would otherwise be consequent upon making responses for "insufficient reward". The theory is an imaginative one. But it is also complex, even with respect to humans to whom it might seem

more appropriate to assign such intricate mental processes. However, in addition to its anthropomorphic flavor, the theory suffers from a lack of convincing evidence for critical mechanisms, especially for the assumed effects of nonreinforcement.

Of the many "conditioning" theories of PRF (sometimes referred to as "stimulus-response", "mediational-conditioning", or "multi-stage conditioning" theories), particular attention will be devoted to the Amsel (1962) "frustration" interpretation and the Capaldi (1966) "conditioned-aftereffects" account. As we shall see, neither of these theories, by itself, provides an entirely satisfactory account of the instrumental PRF data, but they have suggested profitable ways of studying the PRE, and have substantially influenced the experiments of the present thesis. We shall discuss these two theories in some detail, following a more general account of other forms of "conditioning" theories.

In a sense, all conditioning theories of PRF have incorporated either the specific or the general version of the "discrimination" hypothesis. According to the specific version, the PRE is the result of there being a more abrupt, localized stimulus change from acquisition to extinction for a continuously reinforced S than for a partially reinforced S. However, this notion has been discredited by experiments of Theios (1962), Jenkins (1962), and Sutherland, Mackintosh, and Wolfe (1965), all of which show that introducing long periods of continuous reinforcement between partial reinforcement and extinction does not eliminate the PRE. Theios did find a slight diminution in size of PRE with the largest of three amounts of interpolated continuous reinforcement.

This suggested that there might be a very small role played by a local-discrimination factor. But clearly the major portion of any PRE was contributed by an alternative process. More general types of discrimination hypotheses have avoided a dependence on events which occur at the transition from acquisition to extinction. They have, instead, assumed that for PRF (vs. CRF) Ss there must be a greater similarity between extinction and some (any) portion or aspect of acquisition; e.g., both contain nonreinforced trials.

However, in its loss of specificity about what was discriminated from what, the discrimination hypothesis lost most of its usefulness; it became an ex post facto explanation, postulating the differences in similarity when a PRE could be demonstrated and no differences when there was no PRE.

There have been a number of conceptions of PRF effects (in both acquisition and extinction) involving the notion of "competing responses". They are not entirely consistent with one another and vary in degree of precision and complexity. One such interpretation was developed by Weinstock (1954) for instrumental PRF in the runway. According to this notion, competing responses occur within a nonreinforced trial as a reaction to the empty goal box. The competing responses then gradually "habituate" or "drop out" through the action of subsequent nonreinforced trials. Although the exact nature of the "habituation" is not made clear, an extinction process would seem to be involved. In any case, by the time extinction has begun, PRF Ss would not be likely to make the competing responses, and the primary instrumental response could then occur

without that source of interference. But for CRF Ss, the competing responses would occur for the first time in extinction, and would thereby produce a decrement in the strength of the instrumental response of CRF Ss relative to the performance of PRF Ss. A major difficulty for Weinstock's account is that it is the pre-goal-box running behavior which provides the dependent measure for the PRE, and why this behavior should be affected by "competing responses" in the goal box is not specified. Secondly, even if a second set of competing responses is postulated for the runway part of the apparatus, it must be assumed that the competing responses are more susceptible to "habituation" on nonreinforced trials than is the running response itself. Finally, it is not obvious that a PRE, rather than the opposite of a PRE, would be predicted from the theory. In extinction, CRF Ss are assumed to have greater strength of competing responses than PRF Ss have, and therefore display less strength of the running response. But the occurrence and nonreinforcement of competing responses on the first few extinction trials should not affect the potential strength of the running response. Thus, the CRF Ss should eventually resume running at full strength at a time when the running response would already be partly extinguished for PRF Ss. The opposite of a PRE would then have been obtained. The last two criticisms would seem to be relevant to any extension of the theory to Pavlovian PRF situations.

As mentioned earlier, in the review of classical PRF studies, (p. 12), Fitzgerald (1966) and Fitzgerald et al. (1966) have used a competing response theory to account for their results with aversively

conditioned heart rate acceleration. According to this hypothesis, which is somewhat different from Weinstock's, competing responses occur during PRF on trials which follow nonreinforcements, and they are then punished when they occur on reinforced trials. They would therefore not be likely to occur in extinction for PRF Ss. But for CRF Ss, the competing responses would occur for the first time in extinction and would go unpunished, thereby producing an apparent decrement in the conditioned response strength as compared to that of PRF Ss. The nature of the competing responses again is not clearly specified, but one might assume the "response" of keeping the heart rate constant would be the primary one. A necessary (and somewhat dubious) assumption for such a competing response hypothesis would seem to be that the "response" of keeping the heart rate constant can come under instrumental (punishment) control while the response of heart rate acceleration, which should be punished for both CRF and PRF Ss, cannot. Moreover, if the hypothesis were applied to Pavlovian situations with a US which could function as a reward, it would seem to predict the opposite of a PRE. In any case, the theory is clearly at a relatively imprecise stage of development.

We may conclude, therefore, that neither the Weinstock (1954)⁸ nor the Fitzgerald et al. (1966) competing response theories provide a

⁸Weinstock (1958) has employed a different interpretation of PRF to account for an instance in which PRF led to a higher acquisition level of performance. Briefly, he proposed that, in addition to the running response, "non-functional" responses occur in a runway and are rewarded in the case of CRF; but that with PRF, nonreinforcements cause them to drop out. Thus PRF Ss may reach a goal faster than CRF Ss. However, this hypothesis was not applied to an extinction PRE.

convincing account of either instrumental or Pavlovian PREs.

Estes (1959) has presented still another competing response hypothesis. In this case, competing responses are assumed to occur as a result of non-reinforcement, but they do not habituate out (disappear); further, they are not so competitive as to necessarily eliminate the running response. Rather, on a reinforced trial, competing responses occur and are followed by the running response which culminates in reward: ... "therefore, response-produced stimuli associated with them will become conditioned cues for the reinforced response and will tend to maintain the latter during a subsequent extinction series" (Estes, 1959; p. 437). A CRF animal will have no opportunity for the competing-response-produced-stimuli to occur and to become conditioned to the running response prior to extinction. When the competing responses finally do occur, in extinction, they will simply replace the running response. The effects of PRF are therefore developed through two stages of conditioning, in which the conditioning, itself, is assumed to take place according to Guthrie-contiguity principles.

We will now consider a theory which is structurally very similar to Estes', but which is more specific about the nature of the competing responses, and which grew out of Hull-Spence theory instead of Guthrie's. This interpretation, developed by Amsel (1958, 1962), emphasizes the role of "frustration". An outline of his view as applied to the food-rewarded, runway response is as follows (abstracted from the 1962 paper)⁹.

⁹In a later chapter, we will attempt to apply the Amsel theory to an aversive classical conditioning situation, and we will then need to devise concepts analogous to "frustration", etc.

There are four general stages in PRF which are thought to be the necessary and sufficient conditions for producing an extinction PRF:

1. First, an animal must be rewarded for running down the alley. Initially, non-rewards are presumed to have no important effect (unless so many are given that the response is not maintained). Following a number of rewarded trials, an anticipatory response begins to occur as the animal is running down toward the goal box: it is known as "fractional anticipatory reward" or " $r_R - s_R$ ", meaning that it is a conditioned response (r) with stimulus (s) consequences.
2. After the development of $r_R - s_R$, non-rewards will have motivational consequences, namely, "frustration". To demonstrate "frustration", Amsel employs two runways in series. There is a startbox, a first runway, a first goalbox (G1), a second runway, and a second goalbox (G2). Reward and non-reward are varied in G1, and the rat is always rewarded in G2; speed of running in runway 2 is taken to be a direct reflection of how frustrated the rat is following, e.g., non-reward in G1. Within the Hullian framework, any specific increase in motivation is assumed to be funneled into a general drive source which energizes whatever response is ongoing. Thus, if running speed in runway 2 is increased over some baseline speed immediately after the rat has been non-rewarded in G1, it suggests that the non-reward has had motivational consequences. In the case of the usual single-runway, the "frustration effect" (FE) would not be observable; but Amsel would still assume that the S was "frustrated".
3. After some number of non-reinforcements, the cues of the runway come, again through classical conditioning, to evoke "fractional anticipatory

frustration", or $r_F - s_F$, in addition to $r_R - s_R$. These two response tendencies are now assumed to be in competition.

4. At this stage, if the percentage of reward were low, the rat might refuse to run at all; i.e., the $r_F - s_F$ might completely overshadow the $r_R - s_R$. However if the percentage of reward is high (or if there were a second goalbox which would maintain the overall sequence of responses), the conflict would be resolved in favor of running. This may happen on trials in which there is a reward with the result that s_F (stimulus component of anticipatory frustrative responses) becomes conditioned to the instrumental response. Thus, by the time genuine extinction is begun, the PRF rats have been conditioned to run down the alley in the presence of stimuli (s_F) which are the (conditioned) consequence of previous non-reinforcement.

Amsel's experimental work seems to have been largely concerned with showing that there is an immediate, observable, and manipulable effect of nonreinforcement which can take the form of the FE. There are relatively few instances in which he has tried to show, experimentally, a relationship between frustration and an extinction PRE. The degree of emphasis is indicated by Amsel (1962, pp. 307 - 308). Examples of the experimental findings would be:

1. Non-reward following consistent reward does invigorate responses which follow it (i.e., in runway 2 of the double runway) (Amsel & Roussel, 1952).
2. The FE will develop during partial reinforcement but does so gradually (Amsel & Hancock, 1957). Whereas, after enough CRF trials, the FE is manifested immediately after the first nonreinforcement. That is, the most

efficient procedure for getting a FE would appear to be to provide CRF initially.

3. A number of studies provide evidence that factors which, by definition (within the Hullian system), are favorable to the development of r_R (anticipatory reward responses) also are favorable to the development of the FE.

A difficulty with Amsel's treatment of partial reinforcement is that it is not perfectly clear that "frustration" is a necessary condition for producing a PRE in extinction. He and his co-workers have done experiments in which large PREs are manifested (e.g., SurrIDGE & Amsel, 1966). The results are always interpreted in terms of frustration. But the possibility remains that the special motivational factor is simply, so far as the PRE is concerned, a non-functional by-product and that the PRE can be interpreted in terms which do not involve the frustration concept. E.g., Amsel (1958) finds that a PRE can be demonstrated more easily with a long acquisition than with a short one; it is said, therefore, that a critical number of trials must be necessary to condition " s_F " to the running response. But most other theories of PRF (not involving frustration) would make the identical prediction. A second prediction is that partial animals should show greater variability of performance in the early-middle portions of acquisition when r_R and r_F are supposed to be in conflict, and data support this (Amsel, 1958). However, there are other theories (e.g., Weinstock, 1954; Estes, 1959) which consider similar acquisition phenomena but which do not employ frustration as an explanatory concept for the extinction PRE.

Of particular import for the Amseian view is an experiment by Jones (1966), in which rats were partially or consistently exposed to food in a goalbox, but in which this differential training occurred while the animals were satiated for food: both the initial training to run down the runway for food, as well as extinction, were carried out under food deprivation. A significant PRE was obtained during extinction. Assuming that these results are reliable (and a more recent experiment by Jones and Bridges, 1967, is somewhat supportive), it is difficult to see how "frustration" could have been involved in producing the PRE. There have been other reports (cf. McCain, 1967) of PREs with very short acquisition, but these might be interpreted as demonstrating rapid development of frustration effects rather than their absence.

There have been other multi-stage conditioning hypotheses which postulate different or more neutral mediating variables. One of the earliest of all the PRF interpretations was Sheffield's (1949) "aftereffects" hypothesis. She proposed that the aftereffects of nonreinforcement or reinforcement might persist until the next trial. Many, if not all, of these aftereffects were apparently peripheral, e.g., presence or absence of food-residues in the mouth. The extinction PRE would be the result of the aftereffects of nonreinforcement being paired with reinforcement. However, when it was later found by Weinstock (1954, 1958) that a PRE could be obtained with 24 - hour inter-trial intervals, it was generally assumed that the aftereffects hypothesis was untenable. This was because, intuitively and a priori, it seemed that no "aftereffect" could last that long. Presumably, a parametric study of inter-trial intervals would indicate

the importance of this kind of aftereffect for a particular PRE.

More recently, Capaldi (cf. his 1966 review) has developed a particularly detailed treatment of PRF which involves "aftereffects", but not aftereffects which are shortlived. The aftereffects are stimulus consequences of a response, or "mediating reaction", which has been conditioned to the external stimuli of the apparatus. The aftereffects occur, therefore, when the S is placed in the apparatus, no matter how much time has elapsed since the preceding trial. It is these stimulus consequences, " S^N " and " S^R ", of reward (R) and nonreward (N), respectively, with which Capaldi is primarily concerned. The pairing of S^N with R is considered to be a necessary condition for the development of a PRE. Further, it is assumed that S^N takes on different values for different numbers of N-trials in a row (different N-lengths); e.g., three consecutive Ns would produce an " S^{N3} ", etc. Resistance to extinction for a partially rewarded animal is considered to be a direct function of N-length, number of different N-lengths, and the number of occurrences of each N-length. These relationships are based primarily on the assumption that resistance to extinction is a function of the degree to which habit strength generalizes from S^N that have been reinforced in acquisition to S^N that are encountered in extinction. There are additional assumptions concerning the way in which this generalization can be maximized. E.g., the longer the acquisition N-lengths are, the greater will be the generalization of habit strength to N-lengths which are experienced in extinction at the time the S would, ordinarily, be about to cease responding. The use of multiple, rather than single, values of acquisition N-length produces generalization

to a larger number or extinction S^N ; summation of the different generalization tendencies is also assumed to occur. The more times a given N-length occurs (and is followed by a R) the more habit strength there will be for the S^N of that N-length.

An R-trial performs two functions, according to the Capaldi account. First, it conditions stimuli, including S^N , to the instrumental response. Secondly, it "replaces" all S^N with S^R . However, for the second function, only a single R is considered necessary, so that there would be no difference in the consequences of a single R and several Rs in a row.

The Capaldi scheme is obviously not a simple one, and he has discussed at some length the implications of, and support for, these many assumptions (Capaldi, 1966). Much of Capaldi's own experimentation to test his theory has involved an unusual procedure called "inter-trial reinforcement" (ITR). According to Capaldi, placing the S directly in the goalbox (with food) between trials permits S^R to replace S^N without at the same time reinforcing the instrumental response. The procedure has, therefore, been used by him to investigate such variables as N-length without having to worry about many normally confounded variables (Capaldi, 1964). While Capaldi has presented some evidence to support his interpretation of ITR (e.g., Capaldi and Spivey, 1963) there is at least one instance of a failure to replicate the effects of ITR (Black and Spence, 1965). However, irrespective of the status of the ITR procedure, Capaldi's theory at least is relatively specific and testable, as PRF theories go.

There is one major PRF finding for which Capaldi's theory, like others, does not seem to provide an adequate account, namely, the diminution

of an extinction PRE if the PRF schedule is preceded by a large amount of CRF. This "initial-CRF" effect is a result which Capaldi, himself, was one of the first to obtain (Capaldi & Capaldi, 1963), and it has also been found by Sutherland, Mackintosh, and Wolfe (1965) and by Hothersall (1966). The latter was a parametric study showing that the size of a PRE was inversely related to the amount of initial CRF. This same factor would seem to have been the basis for Jenkins' (1962) finding, in one experiment, of less resistance to extinction when CRF preceded PRF than when it followed PRF, although the effects of amount of initial and terminal CRF were confounded in that experiment.

Apparently, the only attempt to explain the initial-CRF effect has been Sutherland's (1964), which was derived from his two-stage model of discrimination learning. The model was originally intended to account for the fact that an animal might "attend" to less than the total available number of stimulus "dimensions" during a discrimination task. According to Sutherland, a stimulus input is fed into a system of "analysers", each of which is sensitive to one dimension of the overall stimulus complex. An output of an analyser (e.g., the output corresponding to "black" on the brightness analyser) can become conditioned to a response. Discrimination learning involves learning which analyser to switch in and which response to attach to which output of the analyser. There are a number of "rules" which Sutherland has drawn up to cover the operation of these mechanisms; e.g., "...the more strongly one analyser is switched in, the less strongly are others switched in". (Sutherland, 1964, p. 150). "Inconsistent" reinforcement is considered to result in the switching in and out of a large

number of analysers, with the further result that a larger number of them (i.e., their outputs) get conditioned to the response than is the case for consistent reinforcement. Thus when extinction begins, there are more response-analyser attachments to extinguish for PRF Ss than for CRF Ss. However, if a large amount of initial-CRF is given prior to PRF, certain analysers (one or a few) get switched in very strongly, making it more difficult for any other analysers to get switched in and become conditioned.

The main difficulty with the Sutherland conception is that it is imprecise, particularly in regard to different schedules of PRF (e.g., the term "consistency" is not well defined). With respect to the theory's application to PRF, it is not clear what the "dimensions" are. They might have to do with environmental stimuli to which the animal might become more attentive, or they could refer to internal states of the organism, which suggests a wealth of possibilities. Since the concept of "dimension" is unclear, it is even less certain how one would expect the consequences of reinforcement and nonreinforcement to be allotted or distributed between "switching" operations and conditioning processes.

We may note with some emphasis that Sutherland's idea that a PRF animal develops a larger number of associations than does a CRF animal is also an essential aspect of the conditioning theories of either Amsel or Capaldi. Both CRF and PRF Ss are conditioned to apparatus cues or to a CS, but, in addition, the PRF Ss acquire associations between the reinforcement and either "anticipatory frustration" (for Amsel) or the "aftereffects" of nonreinforcement (for Capaldi). The location of the "cues" is internal, of course, for the Amsel and Capaldi hypotheses, whereas

Sutherland's theory might emphasize or include external cues, as well. It is an interesting question whether only one or both of these sources of cues is important. If the external source is important, the size of a PRE might be varied by manipulating the external experimental environment in some way. Perhaps an enriched environment would produce a large PRE by providing a large number of cues to which the Ss could become conditioned.

To conclude, in one of the more recent reviews of PRF, Lewis (1960) presented a rather bleak picture of the theoretical state of affairs, concluding that there was no adequate reason for preferring any one PRF theory over another. We would suggest that this view is overly pessimistic and that there has been a turn-over of hypotheses. Certainly, the cognitive and local-discrimination notions, as well as some competing response theories, would seem to be less fruitful than the multi-stage conditioning views of Amsel or Capaldi which are much more explicit and therefore more subject to experimental test and modification. These two theories, and to a lesser extent that of Sutherland, will prove useful in the organization of our own experimental data.

CHAPTER THREE

GENERAL METHOD

To avoid unnecessary repetition, a description of methodological details common to all experiments of the thesis will be given first. At one time or another all of the CER procedures involved training rats to bar-press for food reward, presentation of a white noise CS in conjunction with an electric-shock US, and the measurement of the degree of suppression of bar-pressing during the CS.

Subjects

The Ss throughout were hooded rats, differing from each other in weight within an experiment by not more than approximately 50 grams (usually much less), and having an overall range of weights of from 250 to 350 grams when on ad libitum diet. Rats obtained from a supplier were placed on an ad lib diet for approximately five days and were then reduced gradually to 75% of their final ad lib weight. Thereafter they were weighed daily and fed enough to keep them at the 75% level; during an experiment they were weighed and/or fed within half an hour following their experimental session.

Apparatus

The equipment consisted of standard Grason-Bradley operant conditioning boxes and associated programming and recording equipment. The

boxes were housed in sound-proof containers which were located in a separate room from the programming apparatus.

The conditioned stimulus (CS) was white noise from a Grason-Stadler Model 901B Noise Generator, the output of which was passed through a unit which prevented interactions in noise intensity between boxes. Noise intensity (which was not the same in all experiments) was checked with a General Radio Co. Type 1551-C Sound Level Meter; measurements were made with the ventilation fans off, and with the meter microphone in a standard position within the experimental unit.

The unconditioned stimulus (US) was a 0.5 second shock delivered to the rat's feet from a Grason-Stadler Model E1064GS Shock Generator (one per box). Periodic checks were made to insure that the shock circuits retained an approximate electrical equivalence from box to box. Grids were cleaned after every rat's session, and room dehumidifiers were sometimes used to prevent condensation and grid shorting. Stated shock intensities refer only to the nominal settings on the shock generators, and not to the actual amperage. Within an experiment, treatments were counterbalanced in order to allow for possible effects of differences between experimental chambers or differences in the time of day at which a rat was run.

Procedure

Magazine training -- All animals were first taught to press a lever ("bar") to receive food (45 mg. Noyes Pellets) on a continuous reward schedule. Their first 40 food pellets were delivered automatically on a

variable-interval (VI-1 minute) schedule and/or whenever the rat lever-pressed; thereafter lever presses alone brought food. On all sessions after the first, bar-pressing was rewarded on a 2.5 - minute variable interval food reinforcement schedule. Preliminary training included at least five (at most, seven) days of lever-pressing, for either one two-hour session, or for two forty-minute sessions, per day.

Since one of the experimental variables in all the studies was the intermittent pairing of CS and US, the CS was never "pretested" or "habituated". The relatively minor unconditioned effects of the white noise CS in the CER situation have been documented in previous research from this laboratory (e.g., Hilton, 1964) and are not of critical importance to the experiments to be described.

CSs were three minutes in duration, except in one experiment which varied CS duration. On each reinforced trial, the CS was terminated with the onset of shock.

The index of conditioning was the amount of suppression of bar-pressing during the CS. This was measured by comparing the number of responses during the CS to the number made in the 3 - minute period immediately preceding the CS (the pre - CS period). The suppression ratio employed was that adopted by Kamin (1961), and was of the form, $B/(A+B)$, where B is the number of responses during the CS and A is the number in the Pre-CS period. A ratio of .50 indicates no suppression (or facilitation); .00 means complete suppression; and 1.00 represents the theoretically possible case in which there is at least one response during

the CS and none in the Pre-CS period. In one experiment, a 30 - second CS was employed; in this case the number of responses in the CS was multiplied by six before insertion into the formula.

Note: the formal experiments of the thesis employed primarily a "Bar-Out" procedure, according to which the bar (lever) was made inaccessible to the rat from the beginning of the first CER training day through the last CER day. Under these circumstances suppression scores are available only for test days during which the CER is progressively extinguished. With the "Bar-In" procedure, the CS-US pairings (or CSs alone) were simply superimposed on the rats' on-going lever-pressing activity. Under this procedure, suppression data is available during CER acquisition, as well as during CER extinction.

CHAPTER FOUR
PRELIMINARY EXPERIMENTS

The present work on PRF within a CER procedure was begun before the recent studies on this problem (Brimer & Dockrill, 1966; Wagner, Siegel, & Fein, 1967) had been published. The attempt to develop a workable procedure involved a considerable amount of trial and error in the form of pilot experiments. While the preliminary experiments do not provide a very systematic body of data, they do contain some information of value, and the present chapter will discuss very briefly the main findings of four of these experiments. An expanded description of this work can be found in Appendix B. The basic procedures of the preliminary experiments involved slight modifications of the standard CER training technique described by Kamin (1965). Within this procedure, a number of CS-US pairings (or nonreinforced CS presentations) are superimposed on on-going bar-pressing behavior.

Experiment P-1

This experiment was part of a larger study, primarily concerned with "habituation". The extinction effects of PRF were not observed. However, the experimental design was such that comparisons of CRF and 50% PRF were available for CER training under two intensities of shock (US). With the weaker shock intensity, the nonreinforcements quite clearly impeded or prevented acquisition of the CER: the trial-to-trial behavior of the

PRF group suggested an alternating pattern of acquisition and extinction. CRF groups which were equated with the PRF group for reinforcements and trials, respectively, both acquired the CER.

When stronger shock was employed, a CRF and 50% PRF group acquired complete suppression at approximately the same rate. However, the PRF group showed a subsequent loss of suppression during continued PRF training. Why the CER should have been attenuated after it had been acquired, rather than during its initial development, is not obvious. Conceivably, this effect might depend on the fact that the CS was never pretested or "habituated" prior to CER training, with the result that unconditioned suppression was especially strong during the first few trials.

Experiment P-2

This experiment was specifically designed to examine both acquisition and extinction effects of PRF. There were three groups, two PRF and one CRF. All groups received eight consecutive reinforced trials at the outset of training¹⁰. One PRF group was then given a single alternating pattern of reinforced and nonreinforced trials. The other PRF group received an irregular 50% schedule of PRF. The CRF group was equated with each PRF

¹⁰One reason for this initial CRF was that, according to Amsel (1958), "frustration" can be developed most efficiently by giving CRF rather than PRF at the beginning of training. With the CER, it was thought that a process analogous to frustration (e.g., "relief"); might be operating in which case initial-CRF might be a good tactic (cf. outline of Amsel's theory in Chapter 2).

group for number and spacing of reinforcements. Relative to later experiments, short inter-trial intervals were used: within a two-hour session the CRF Ss had four trials and the PRF Ss had eight. Following eight days of CER training, extinction of the CER was begun, with four non-reinforced CSs presented in each daily session to each group.

The only noteworthy acquisition result was an almost immediate attenuation of the CER for both of the PRF groups, following introduction of PRF. The two PRF groups behaved similarly. In extinction, there was only slight evidence of a PRE. The result was of the ambiguous variety described in Chapter 1 under "Case 4". While the PRF groups showed the least suppression at the end of acquisition, they did not differ from the CRF group in extinction. The inference of a PRE in such a case rests on the assumption that the PRF groups would have extinguished faster than the CRF group had there been no "PRE". In any case, the experiment did not produce a PRE of sufficient magnitude or reliability to permit detailed further study of the effect.

Experiment P-3

This study was a further attempt to achieve a large PRE. Both US intensity and CS intensity were increased, and the ITI was lengthened so that one trial, at most, was given per session. There were two sessions per day, spaced approximately five hours apart. After an initial block of four continuously reinforced trials, which produced complete suppression, subjects were divided into two groups which then received CRF and 50% PRF, respectively, over 13 CER days. Both groups remained completely suppressed

during the CER training, and in extinction the CRF group showed a more rapid loss of suppression. This was a clear PRE, but the effect barely achieved statistical significance, in spite of a relatively large number of Ss per group (16). The prospect of a series of studies on PRF, employing such a time-consuming procedure which produced so slender a result, was not inviting.

Experiment P-4

This experiment was conducted solely to see if a PRE at least of the size obtained in Experiment P-3, above, could be obtained with a shortened acquisition period. The attempt was totally unsuccessful, however; PRF and CRF treatments resulted in virtually identical extinction curves.

The next experiment provided a clear-cut PRE, in conjunction with a procedure which was judged to be sufficiently sensitive to allow continued systematic investigation of the effect. This study, which in itself investigated some procedural variables, is described in the next chapter as the first formal experiment of the thesis proper.

CHAPTER FIVE

While the experiments in this and subsequent chapters have been set apart from those of Chapter 4, largely on the basis of their fruitfulness, the general method described in Chapter 3 still applies. Further, many of the more specific procedures to be described below were retained from Experiments P-3 and P-4. Experiment 1 provides significant information about PRF within a classical conditioning procedure, as well as giving a practical method for further study of the PRE. The subsequent experiments are concerned with answering a number of specific questions about the PRE.

EXPERIMENT 1.

Personal communication with A. R. Wagner had acquainted us with the provocative results of some PRF experiments which he and his associates had conducted, and which have since been published (Wagner, Siegel, & Fein; 1967). An outline of their findings was presented earlier (Chapter 2). The most important result for present purposes was that a PRE was obtained with a standard "Bar-In" CER procedure, but not with a "Bar-Out" procedure. That is, the PRE was obtained only if the animals were allowed to bar-press during CER training sessions. Wagner et al. concluded that any PRE observed with a CER procedure was probably an artifact of some fortuitous instrumental contingency between bar-pressing and shock, and not attributable to a bona fide classical conditioning procedure. The Wagner

et al. results seemed surprising to us. Particularly, a significant PRE had been observed in Experiment P-3, and most rats in that study had become completely suppressed by the time PRF training was instituted for the PRF group. It appeared unlikely, therefore, that bar-pressing could have been differentially affected by CRF and PRF. Because the theoretical implications derived by Wagner et al. from their results were of critical importance both for our demonstration of a PRE and for any subsequent investigations, an attempt to replicate their findings with respect to the Bar-In vs. Bar-Out factor seemed highly desirable. We also decided to examine the effects of CS duration, since Wagner et al. had used a 30-second CS (relatively short), and our preliminary experiments had employed a 3-minute CS. The first formal experiment, then, was a 2 x 2 x 2 factorial, comparing Bar-In vs. Bar-Out; 30-second vs. 3-minute CS duration; and CRF vs. an irregular PRF schedule.

Method

There were eight groups of rats (one group assigned to each of the eight combinations of the three factors), with four rats in each group. The supplier was Canadian Research Animal Farms.

The CS was an 80 decibel white noise of either 30-seconds or 3-minutes duration. The US was a 2.0 ma. shock of .5 second duration, the onset of which was simultaneous with CS offset.

All Ss were given two 45-minute sessions each day, with the first session in the morning and the second session about five hours later. Ss were fed a small portion of their daily food ration after the first session, so that baseline response rates would remain roughly the same for the

two sessions. They were weighed and fed an additional amount (enough to keep them at 75% of ad lib weight) after the second session.

Within a session there was only one trial-period, located so that the CS onset (if there was a CS) occurred 20 minutes after the start of the session. This location had been found in Experiment P-3 to maintain reasonably stable baseline bar-pressing rates throughout the experimental session.

Preliminary training consisted of the usual magazine training, plus six days of bar-press training on the usual 2.5 minute VI schedule. CER training was then begun.

Half the animals received the Bar-Out procedure, in which the lever was removed from the Skinner box during all CER training sessions. The hole in the wallpanel created by removal of the bar was covered with a metal plate. The remaining Ss received the standard Bar-In procedure, i.e., they were allowed to lever-press during all phases of the experiment. Within each of these divisions, half the Ss received a 3-minute CS and half received a 30-second CS.

During CER Days 1 and 2, there were four reinforced trials (i.e., initial CRF) for all Ss. The Ss were then divided for a third time, with half the animals in each sub-division receiving PRF, for CER Days 3 through 16, and the other half receiving CRF. The schedule of PRF, following the initial CRF, was an irregular pattern of 14 reinforced trials (R) and 14 nonreinforced trials (N). The patterning of CER trials, for both PRF and CRF Ss, is indicated below.

CER Day: 3 4 5 6 7 8 9 10 11 12 13 14 15 16

PRF: NN RN RR NN NR RR NR NN RN RR NN NR RR NR

CRF: R RR R RR R R RR R RR R

The CRF and PRF groups, as can be seen, were equated for number and spacing of reinforced trials. When PRF Ss were receiving an N, the CRF Ss received neither CS nor US (a "blank trial").

Extinction of the CER (presentation of CS without US) was begun on the day after the last CER training day. In the first session, the CS was omitted for all Ss to insure that the Bar-Out groups would be at a high level of instrumental responding prior to the start of CER extinction. The first extinction trial (ET-1) was on the second session of that day. Each S received one such extinction trial per session, thereafter, until the S achieved a criterion of two consecutive suppression ratios greater than or equal to .40. This criterion was based on the results of Experiment P-3, and it ensured that all PRF-CRF pairs of groups would have an opportunity to display a PRE. Further, all Ss were, in fact, run for a minimum of 45 extinction trials.

Results

There was virtually complete suppression in all groups on the first extinction trial. All but one S had a ratio of .00; the exception was a 30-second CS rat (CRF, Bar-In) with a ratio of .18. (This S subsequently provided .00 ratios through trial 42.)

The differences between groups which appeared in the course of extinction were examined in two ways. First, a comparison was made

of the amount of suppression which had been displayed over the course of the first 45 extinction trials. Figure 1 presents the mean suppression ratio of each group for the first extinction trial, and for blocks of four extinction trials, with the curves for each PRF-CRF pair of groups plotted separately. Differences in treatment effects already seem considerable within this number of trials. While only the 3-minute CS groups have yet displayed a substantial loss of suppression, a greater resistance to extinction on the part of PRF groups is evident, particularly with the 3-minute CS, Bar-Out procedure.

For statistical analysis, the amount of overall suppression for each S was indexed by calculating the mean of that S's suppression ratios for trials 2 through 45. Table 1 presents the group mean overall suppression ratios (i.e., the means of the subject means for each group). Table 2 summarizes an analysis of variance performed on the same overall ratios. The main effect of each of the three factors was highly significant. As indicated by the mean ratios, the 30-second CS duration produced more resistance to extinction (i.e., greater suppression) than did the 3-minute CS duration. The Bar-In procedure produced more resistance to extinction than did the Bar-Out procedure. Of more immediate interest, the PRF procedure produced more resistance to extinction than did the CRF procedure. Two interactions achieved significance. A greater PRE was observed with the 3-minute CS than with the 30-second CS. Further, a greater PRE was observed with the Bar-Out procedure than with the Bar-In. The interactions are somewhat misleading, however: since the 30-second CS groups had shown very little loss of suppression by the 45th trial, they had no adequate opportunity to display a PRE; to a lesser degree, the same can be said for the Bar-In treatments.

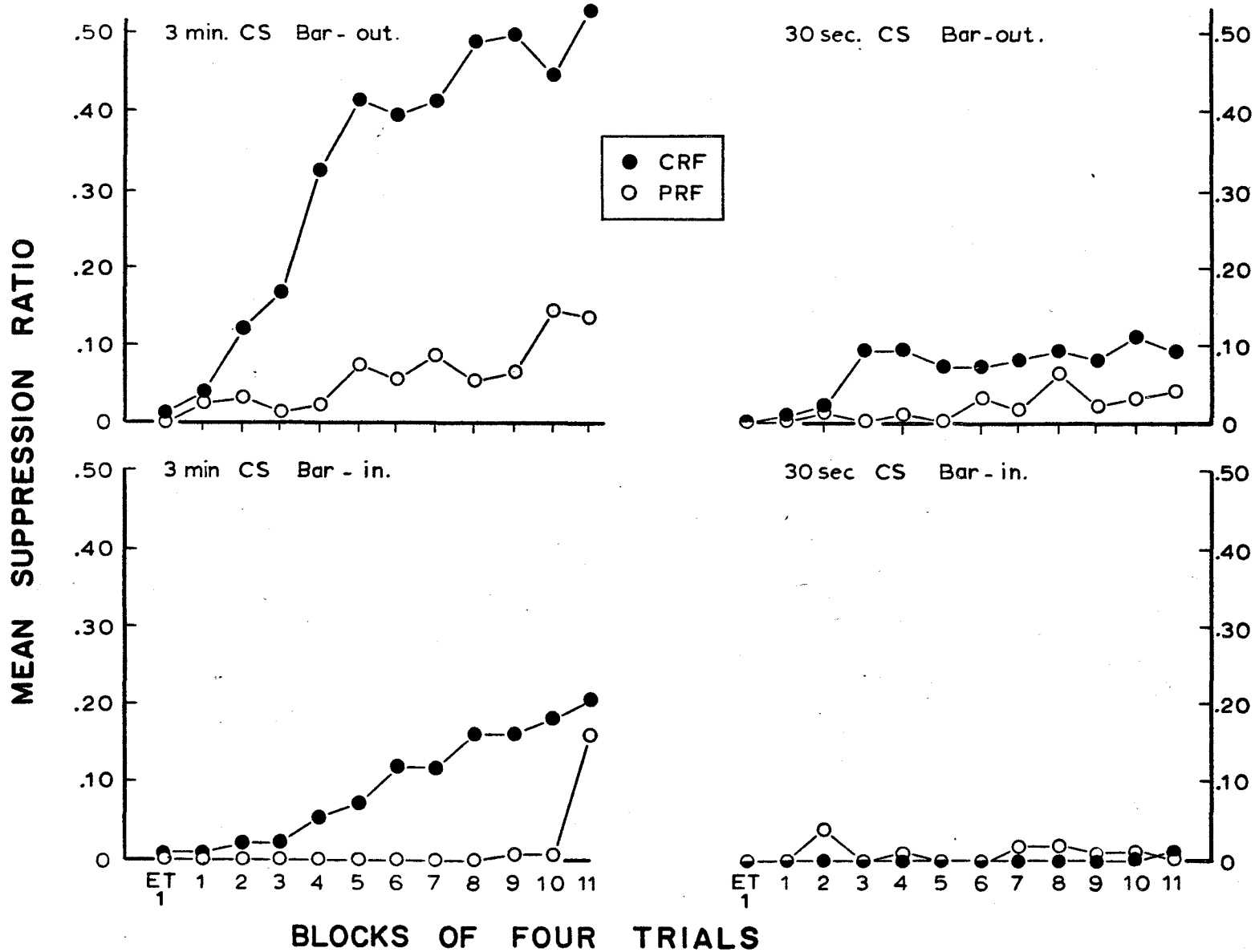


FIGURE 1 EXTINCTION OF CER AS A FUNCTION OF SCHEDULE OF REINFORCEMENT, CS DURATION, AND PRESENCE OR ABSENCE OF BAR DURING ACQUISITION (EXPERIMENT 1).

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Table 1

Mean overall suppression ratios for
each group, Experiment 1. Ranges are in parentheses.

	CRF	PRF
3-minute CS, Bar-Out	.35 (.20 - .43)	.06 (.00 - .12)
3-minute CS, Bar-In	.10 (.00 - .24)	.02 (.00 - .05)
30-second CS, Bar-Out	.07 (.00 - .28)	.02 (.00 - .04)
30-second CS, Bar-In	.00 (.00 - .00)	.01 (.00 - .02)

Table 2

Summary of analysis of variance of overall suppression ratios, Experiment 1.

SOURCE	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A (Schedule of reinforcement)	1	1 631 269.5	14.08	p < .001
B (Bar)	1	1 298 063.3	11.20	p < .005
C (CS duration)	1	1 744 245.0	15.05	p < .001
AB	1	687 671.3	5.93	p < .025
AC	1	990 241.6	8.54	p < .01
BC	1	400 736.3	3.46	n.s.
ABC	1	197 597.9	1.70	n.s.
w Cells	24	115 892.7		

A more equitable measure of resistance to extinction would therefore be the number of trials each S required to reach an extinction criterion (two consecutive ratios of .40 or greater). Table 3 presents the mean number of trials to criterion for each group, and Table 4 provides a summary of an analysis of variance of these data. With this measure, the tendency for the Bar-Out procedure to produce faster extinction than the Bar-In was not quite significant. However, the other two main effects, schedule of reinforcement and CS duration, were again significant. There were no significant interactions. As indicated by the mean scores in Table 3, all four comparisons of PRF with CRF are suggestive of the usual PRE. However, the largest mean difference occurred with the 3-minute CS, Bar-Out procedure, which was the only case with no overlap between CRF and PRF Ss. This finding by itself constitutes a statistically significant PRE ($p = .014$).

We should note, finally, that there was no evidence at any point of differences between groups in baseline rate of bar-pressing. Two analyses of variance were performed: the first was on the number of bar-presses each S made in the pre-CS period of the first extinction trial; the second was on the sum, for each S, of the responses for the pre-CS periods of trials 1 through 45 (cf. Table 5). In neither case did group differences approach significance. In the remaining experiments of this thesis, baseline responding was monitored regularly, but since systematic differences between groups were never observed, no further reference to baseline rates will be made.

Discussion

The most striking finding in this experiment is the fact that

Table 3

Mean number of trials to extinction criterion, Experiment 1.
 Ranges are in parentheses.

	<u>CRF</u>	<u>PRF</u>
3-minute CS, Bar-Out	21 (9-41)	66 (46-80)
3-minute CS, Bar-In	54 (34-61)	59 (46-72)
30-second CS, Bar-Out	68 (12-98)	98 (56-134)
30-second CS, Bar-In	87 (69-103)	117 (99-138)

Table 4

Experiment 1. Summary of analysis of variance
of trials to extinction criterion.

SOURCE	<u>df</u>	<u>MS</u>	<u>F</u>	
A (Schedule of Reinforcement)	1	6 160.5	12.81	p<.005
B (Bar-In Bar-Out)	1	1891.1	3.93	(p<.10)
C (CS duration)	1	14 535.1	30.23	p<.001
AB	1	780.1	1.62	n.s.
AC	1	45.1	--	n.s.
BC	1	72.0	--	n.s.
ABC	1	800.0	1.66	n.s.
w CELLS	24	480.8		

Table 5

Summary of analysis of variance of the sums
of bar-presses made during the Pre-CS periods, trials 1-45 of Experiment 1.

SOURCE	<u>df</u>	<u>MS</u>	<u>F</u>	
A (Reinforce- ment schedule)	1	1023.8	--	
B (Bar)	1	26.3	--	
C (CS duration)	1	2227.8	1.17	n.s.
AB	1	3300.8	1.73	n.s.
AC	1	457.5	--	
BC	1	639.0	--	
ABC	1	3061.6	1.60	n.s.
w CELLS	24	1911.9		

the use of a Bar-Out procedure did not prevent the occurrence of a PRE. In fact, the largest PRE obtained, and the only one which was significant by itself (there were only four Ss per group), was with the Bar-Out procedure with a 3-minute CS.

The reason for the discrepancy between this result and the findings of Wagner, Siegel, and Fein (1967) is not clear. (They, it should be recalled, observed a PRE only with a Bar-In procedure.) There are a number of procedural differences which could have been important. Some of the more obvious are: (1) We have equated CRF and PRF groups for reinforcements, whereas Wagner et al. equated for trials, with the result that their CRF Ss received twice as many reinforcements as their PRF Ss. (2) Inter-trial interval: our ITI was at least five hours, with, at most, one trial per session. Wagner et al. used a 15-minute ITI, four trials per hour. It may be noted that a short ITI was used in our Experiment P-2 (Chapter 4) which gave very little evidence of a PRE. (3) Shock Intensity: 2.0 ma in this experiment, 1.0 ma in Wagner's. Our Experiment P-2 also employed 1.0 ma shock. (4) CS: we have used an 80 db white noise; Wagner et al. used a 2000 cps tone accompanied by an intermittent change in illumination.

At present there does not seem to be any reason for singling out any of these factors over another as a basis for the discrepancy. While it might not be surprising to find that any of the above mentioned procedural variations could affect the size of a PRE, it is difficult to conceive of such a variation reversing the relative efficacies of Bar-In and Bar-Out procedures. Perhaps sheer sampling error is as plausible

an explanation of the discrepancy as any other. In this connection we can anticipate our subsequent experiments and note that we have repeatedly replicated the PRE with the Bar-Out procedure.

The fact that the Bar-Out groups of this experiment extinguished significantly faster than did the Bar-In groups seems attributable simply to a form of discrimination. The Bar-Out groups had received their CS-US pairings in a situation very different from that in which resistance to extinction of the CER was measured. For these groups, the CS had been followed by shock only when the bar was not present, and thus when no bar-press behavior was occurring. The CS when the bar was present, and when bar-press behavior was occurring, was never followed by shock. This obvious basis for a discrimination was not, of course, available to the Bar-In groups.

The significant effect of CS duration is consistent with previous demonstrations (Kamin, 1965) of stronger CER conditioning with short CS-US intervals than with long intervals.

This experiment, finally, appears to provide a procedure which is effective in producing a PRE and, at the same time, is not overly time-consuming: namely, the 3-minute CS, Bar-Out procedure. This procedure was adopted for use in subsequent experiments.

Summary

The present experiment has provided evidence that a clear-cut PRE can be obtained with a classical conditioning procedure, and with substantial intertrial intervals. At the least, it has indicated, contrary to the Wagner et al. findings, that the PRE obtained with a CER procedure is not a consequence of accidental contingencies between instrumental bar-

pressing and shock. Finally, the experiment has provided a reasonably efficient procedure for a more extensive analysis of the PRE.

CHAPTER SIX
EXPERIMENT TWO

The 3-minute CS, Bar-Out PRF and CRF treatments, successfully employed in Experiment 1, seem to provide a basic "baseline" or reference procedure for further experimental study of a Pavlovian PRE.

The experimental manipulations employed in the remaining experiments of this thesis had a dual purpose. First, it will be of interest to observe whether a Pavlovian PRE would respond in a similar fashion to the same variables known to influence an instrumental PRE; second, parametric investigation of the Pavlovian PRE should help in the development of a theoretical account of the phenomenon. Within Experiment 2, we now turn to an experimental procedure for which, in the instrumental case, no exactly analogous data are available. However, the procedure is relevant to theoretical interpretations of the PRE.

The number of variables which could conceivably affect the size of a PRE and which might therefore be chosen for study, is practically limitless; and, as Capaldi (1966) has pointed out, it is extremely difficult, if not impossible, to manipulate certain factors without at the same time involving other factors which are confounded. The choice is best determined by theoretical considerations rather than by an attempt to cover, systematically, all logically possible manipulations.

The next experiment varied the length of a single block of non-reinforced (N) trials, which was interpolated between initial CRF and the standard PRF schedule of Experiment 1. The rationale for this experimental

manipulation was derived largely from the Amsel (1958, 1962) hypothesis, converted to aversive conditioning terms. However, the Capaldi (1966) account of PRF is equally pertinent.

Amsel (1958) has indicated that his scheme ought to apply to aversive conditioning, but he has not discussed such an application in any detail. We can, however, in the spirit of Amsel's theory, hazard the following account for the case of CER conditioning: 1. With initial Rs (i.e., CS + shock), the CS gradually acquires the capacity to elicit fear or "fractional-anticipatory-pain" responses (" $r_P - s_P$ "). 2. Once fear has been conditioned to the CS, an N (i.e., nonreinforced CS) should produce "relief" (the counterpart of "frustration" for the appetitive case). The degree of relief would be a direct function of the amount of fear elicited during the N trial. 3. After some number of Ns, and as a consequence of a second stage of conditioning, the CS should come to evoke "fractional-anticipatory-relief" or "hope" (" $r_H - s_H$ "). At first the CS would elicit fear and hope in competition; but with enough Ns, hope would become the dominant emotional reaction to the CS. 4. The final requirement would be that the hope ($r_H - s_H$) elicited by the CS be paired with shock on R trials a sufficient number of times so that s_h becomes a conditioned stimulus for fear.

According to Amsel's account of the instrumental PRE in a runway, a PRF S is reinforced during acquisition for responding in the presence of anticipatory-frustrative responses ($r_F - s_F$); our extrapolation to the aversive case suggests that a PRF animal might continue to experience fear during the CS after many extinction trials because hope in the presence of

the CS has, in the past, been followed by shock. There is a kind of paradox in each case. In the first, anticipatory-frustrative responses have become a cue that reward will follow; in the second, hope has become a cue that shock will follow.

The above outline suggests that perhaps a PRE could be amplified considerably if, prior to extinction, care were taken to ensure that an especially large amount of hope occurred on a trial which ended in shock. This could presumably be accomplished by interpolating a long block of N trials at some point in the PRF schedule.

Capaldi's (1966) theory of PRF (cf. Chapter 2) makes essentially the same prediction concerning an interpolated block of Ns. In fact, his account may be said to begin with this stage of PRF conditioning, leaving largely undiscussed the question of initial conditioning processes (other than to assume that some initial Rs are necessary to get the S running down the runway). As indicated earlier, N-length is one of the three factors considered to be important in determining resistance to extinction following PRF; the others are number of different N-lengths and number of occurrences of each N-length. (Capaldi has reviewed a number of empirical studies which support his interpretations). In extinction, a relatively long N-length will have occurred prior to the cessation of responding by a continuously reinforced subject. For a PRF subject, the longer the N-length experienced in acquisition, the greater the generalization, during extinction, to what would ordinarily have been an N-length sufficient to produce the cessation of responding. The more the generalization (of fear in the present case), the less likely that responding will cease (that suppression will

cease) within the normal number of trials. Thus as with our extrapolation of Amsel's theory, Capaldi's account also suggests that interpolation of long N-length into the PRF schedule should increase the magnitude of the PRE.

Method

The overall experiment included two sub-experiments, 2A and 2B, containing five and three groups of rats, respectively. The rats were obtained from the Quebec Breeding Farms, Inc., St. Eustache, P. Q.

Most of the procedures of Experiment 1 were retained. The CS was 80 db white noise of three minutes duration, and the US was 2.0 ma shock. The Bar-Out procedure of the previous experiment was employed; i.e., the bar was removed during the CER conditioning. Again, there were two 40-minute sessions per day and no more than one trial per session. Preliminary training consisted of magazine training plus seven days of VI-training. After CER conditioning, the bar was replaced and all Ss given a session of bar-pressing. All Ss were then given 25 CER extinction trials while bar-pressing.

Experiment 2A. The five groups, of varying ns (due to deaths and experimental oversights/errors), consisted of four PRF groups and one CRF "control" group. The CRF group and one of the PRF groups were treated identically to the 3-minute, Bar-Out groups of Experiment 1. That is, they both received an initial 4R over CER days 1 and 2; this was followed by a 50% schedule of 1⁴R and 1⁴N for the PRF group, and by the 1⁴R alone for the CRF Ss. As in Experiment 1, the schedule of CER trials for PRF and CRF Ss was as follows (after the initial 4R):

CER day: 3 4 5 6 7 8 9 10 11 12 13 14 15 16
 PRF : NN RN RR NN NR RR NR NN RN RR NN NR RR NR
 CRF : R RR R RR R R RR R RR R

The remaining three PRF groups differed from the 4R PRF group in that a block of Ns was interpolated between the initial 4R and the standard PRF schedule. These added blocks (N-lengths) were either 4N, 10N, or 20N. All groups were equated for reinforcements, not trials. Since the N-lengths differed, the beginning of CER training was staggered for the groups so that all five groups would begin extinction simultaneously. The five treatments are listed below in an arrangement which indicates the order in which each group received each portion of its treatment. The number of Ss per group is listed at the right.

Groups

4R 14R	(Extinction)	n = 8
4R PRF	"	n = 13
4R 4N PRF	"	n = 12
4R 10N PRF	"	n = 12
4R 20N PRF	"	n = 9

Experiment 2B. Three groups were run in a separate set of equipment (but at the same time as 2A) to determine whether extinction differences which might occur among the PRF groups of 2A could be the result of differences in the time elapsing between initial CRF and introduction of the standard PRF schedule. Differences in this time interval among groups in Experiment 2A were, of course, confounded with the differences in N-length. The control groups of 2B were, essentially, three replications of group 4R

PRF, but with varying numbers of sessions with blank trials (BT), having neither CS or US, interpolated between the 4R and the PRF. The three treatments are listed below:

<u>Groups</u>					
4	R	PRF	(Extinction)		n = 6
4R	4BT	PRF	"		n = 7
4R	20BT	PRF	"		n = 7

Results

Experiment 2A. The performances of the five groups in 2A are presented in Figure 2, which plots mean suppression ratios, by group, over the 25 extinction trials. Again, there was virtually complete suppression by all groups on the first extinction trial (ET - 1). On subsequent trials, not only does the previously observed PRE appear to be replicated, but the interpolation of the largest N-length seems to have increased the PRE substantially. The interpolation of the smaller N-lengths had no apparent effect. (See also Table 6, which presents the group mean overall suppression ratios for trials 2 through 25.)

Statistical analysis confirmed the above picture. An analysis of variance of the mean overall suppression ratios showed a significant treatments effect ($F = 3.76$, $df = 4.49$; $p < .025$). The Newman-Keuls multiple comparison procedure (cf. Winer, 1962) showed only that the 4R 20N PRF group was significantly different from the 4R 14R group ($p < .01$). This, of course, indicates that the interpolation of 20N produced a large PRE. However, multiple t-tests further clarify the group differences. The CRF group,

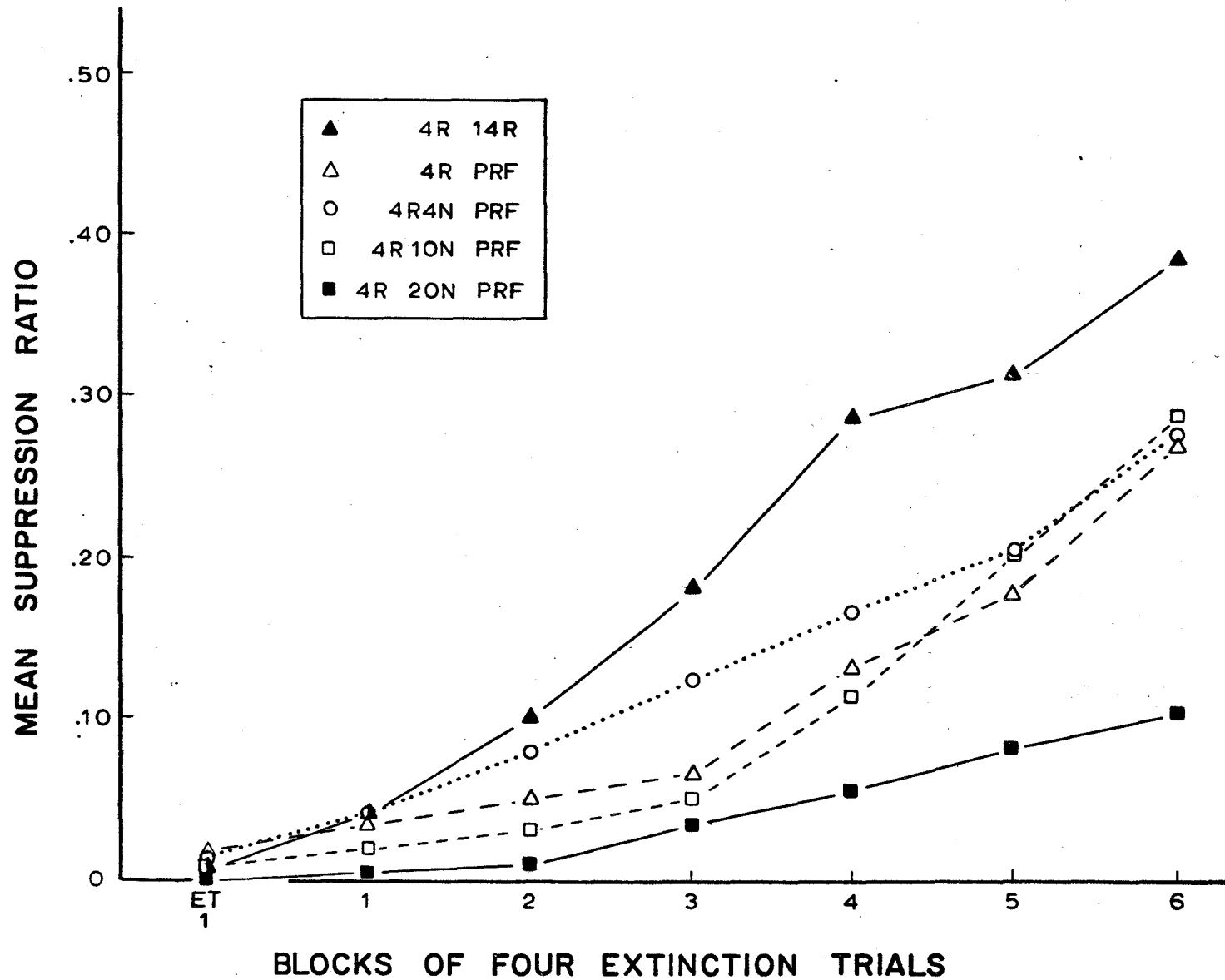


FIGURE 2 EXTINCTION OF CER AS A FUNCTION OF SCHEDULE OF REINFORCEMENT AND LENGTH OF A SINGLE INTERPOLATED BLOCK OF N-TRIALS (EXPERIMENT 2A).

Table 6

Mean overall suppression ratios for each group of Experiment 2A (trials 2-25).

<u>Treatment</u>	<u>Group Mean Ratio</u>	<u>Range of Mean Ratios</u>
4R 14R	.22	(.10 - .38)
4R PRF	.12	(.00 - .26)
4R 4N PRF	.15	(.02 - .34)
4R 10N PRF	.12	(.00 - .20)
4R 20N PRF	.05	(.00 - .26)

4R 14R, was significantly different, at at least the .025 level, from all PRF groups except 4R 4N PRF; in this later case, the difference approached significance ($.05 < p < .10$). The 4R 20N PRF group showed significantly greater suppression (resistance to extinction) at at least the .05 level, than did all the other groups. Differences within the cluster of groups having intermediate resistance to extinction (groups 4R PRF, 4R 4N PRF, and 4R 10N PRF) did not approach significance.

Experiment 2B. Figure 3 shows the mean extinction suppression ratios, over blocks of trials, for the three groups of the control experiment. Table 7 provides the group mean overall suppression ratios (for trials 2 - 25). There are no apparent differences among the three groups, and an analysis of variance of the overall mean suppression ratios (for trials 2 - 25) showed no significant difference. The numbers of blank trials interpolated between the initial 4R and the subsequent PRF matched the numbers of interpolated N trials of groups which differed significantly in Experiment 2A; it seems clear, therefore, that the differences observed in Experiment 2A cannot be attributed to differences in time elapsed between 4R and PRF, to differences in amount of handling, or to other confounded variables.

Discussion

The results of Experiment 2A provide, first of all, a clear replication of the PRE obtained in Experiment 1. That is, group 4R PRF demonstrated significantly greater resistance to extinction than did group 4R 14R.

The additional result of Experiment 2A was that interpolation of a substantial number of nonreinforced trials (20N in this case) between the initial 4R and the subsequent PRF greatly increased the magnitude of the PRE.

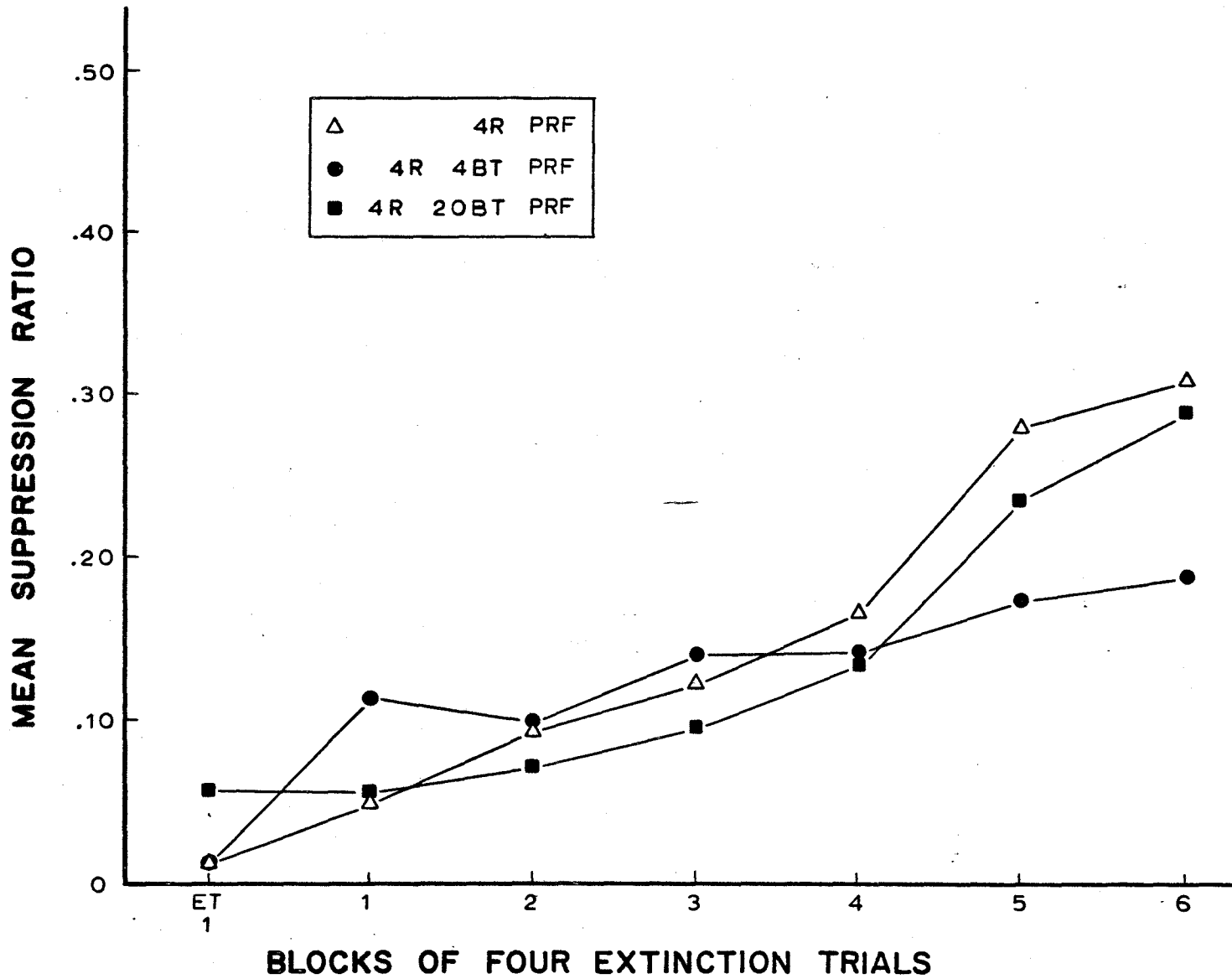


FIGURE 3 EXTINCTION OF CER FOLLOWING PRF AS A FUNCTION OF NUMBER OF INTERPOLATED SESSIONS WITH BLANK TRIALS (EXPERIMENT 2B).

Table 7

Mean overall suppression ratios for each group of Experiment 2B (trials 2-25).

<u>Treatment</u>	<u>Group Mean Ratio</u>	<u>Range of Mean Ratios</u>
4R PRF	.17	(.07 - .34)
4R 4BT PRF	.14	(.00 - .55)
4R 20BT PRF	.14	(.01 - .38)

But when smaller N-lengths were employed, i.e., 4N or 10N, the results did not differ from the outcome obtained with the standard 4R PRF group, which had had no interpolated N trials. It is also clear from Experiment 2B that the increase in PRE magnitude is not attributable to variables such as inter-reinforcement interval, etc., which were confounded with the differences in N-length.

It should be noted that the importance of the specific location of the 20N within the overall schedule cannot be ascertained from the present experiment. It is quite conceivable that interpolation of the same 20N at other points in the PRF schedule would accomplish the same end; for that matter it is logically conceivable that any distribution of the 20N trials within the PRF schedule could produce the same result!

However, as indicated in the introduction to this chapter, the empirical effect obtained with the interpolated N-length seems quite consistent with the theoretical accounts of the PRE of both Amsel and Capaldi. That is, according to an Amselian interpretation of our results, the 20N presumably produced an exceptionally high level of "hope" which was then paired with shock; thus, during extinction, an even more exceptional amount of hope would have to be developed before the S could display a loss of suppression, because the amount of hope which would ordinarily (with CRF Ss) lead to a loss of suppression has been made a cue for fear (in the case of PRF Ss).

For Capaldi's theory, the aversively conditioned aftereffects of 20N would provide greater generalization of fear than would the aftereffects of shorter N-lengths, to those aftereffects encountered in extinction which would ordinarily produce a loss of response strength.

There does not appear to be any study of an instrumental PRE which, in a precisely similar way, investigates the effect of interpolation of a single long N-length into a PRF schedule. However, theories of the instrumental PRE predict the type of effect which we have observed within our Pavlovian procedure, so that the results of this experiment in no way suggest a difference in the mechanisms which underlie classical and instrumental PREs.

CHAPTER SEVEN

EXPERIMENT 3

The final experiment to be reported was designed to provide further information concerning the results obtained in Experiment 2, and to investigate an additional aspect of the PRE.

The results of Experiment 2 indicated clearly that the PRE produced by the standard 4R PRF treatment could be greatly enhanced by the interpolation of a long N-length between the initial 4R and the PRF schedule. This result, as indicated in the previous chapter, seemed quite consistent with the theoretical views of Amsel and of Capaldi. That is, either the large amount of "hope" or the special "aftereffects" produced by the 2ON presumably became, as a result of their association with shock, fear-eliciting conditioned stimuli. These conditioned stimuli, in contrast to those produced by short N-lengths, would tend to occur in extinction at about the stage in which the \underline{S} would normally have lost its fear of the regular CS; fear would thus be reinstated or its loss delayed. Theoretically, the critical aspect of the 4R 2ON PRF treatment, as far as the contribution of the 2ON is concerned, would be the occurrence of one or more shock trials following the 2ON.

To extend this argument a little further, it ought to be possible to produce a PRE by following the N-length simply with GRF rather than with PRF. In fact, it is conceivable that a single R-trial, following the N-length, might be sufficient for a PRE, provided that the single R

was also sufficient for producing a recovery from the extinctive effects of the N-length. However, if a single R-trial were not enough, certainly 14R should be, since that was the number of Rs within the PRF portion of the 4R 20N PRF treatment. Thus, the first portion of Experiment 3 included two PRF treatments containing a long N-length; the N-length was followed, in one case, by 1R and in the other by 14R. In order to assess a possible PRE, each PRF group was, of course, paired with a CRF group which received the same total number of Rs.

The second portion of Experiment 3 was concerned with whether or not the size of the CER PRE might be affected by the amount of CRF presented prior to PRF. As indicated in Chapter 2, with instrumental PRF a very reliable inverse relationship between magnitude of the PRE and number of initial CRF trials has been obtained (e.g., Hothersall, 1966). It is this initial-CRF effect which is central in Sutherland's theory of PRF (cf. Sutherland, Mackintosh, & Wolfe, 1965).

Method

There were seven groups of rats, with from 8 to 10 Ss per group. The Ss were obtained from Quebec Breeding Farms. The groups were run simultaneously as one experiment.

Most procedural details were identical to those of Experiment 2, with the exception that, during CER training, instead of removing the bar and covering the hole with a metal plate, a black plastic cover was employed which prevented access to the lever and food cup. Following CER training, all Ss were given 33 extinction trials.

The seven treatments are listed below, schematically. As in Experiment 2, all groups began extinction at the same time and the start

of CER training for the PRF groups (A, C, E, and F) was staggered in the manner indicated. The R-trials for each CRF group were thus arranged so that they occurred simultaneously with the R-trials of the PRF group with which that CRF group was matched.

<u>Group</u>	<u>Treatment</u>			<u>Number of Subjects</u>
A	4R	20N	1R (Extinction)	8
B	4R		1R "	8
C	4R	20N	14R "	9
D	4R		14R "	9
E	4R		PRF "	10
F	20R		PRF "	9
G	20R		14R "	9

Within this set of seven treatments, we shall be primarily concerned with four comparisons of PRF and CRF. Groups A and B will indicate whether a single long N-length followed by a single R-trial is enough to produce a PRE. A similar comparison may be made between Groups C and D. For Group C, however, the N-length is followed by 14R rather than by only 1R. A comparison between Groups E and D provides still another replication of the basic PRE already demonstrated in Experiments 1 and 2. Whether prolonged CRF (20R vs. 4R) prior to PRF attenuates the PRE might be determined by comparing Groups F and E. However, it should be noted that, within Group F, a weakening of the effect of PRF (relative to Group E) could conceivably be compensated for by an increased resistance to extinction produced by the extra R trials received by Group F. Thus a

more straightforward test of an initial-CRF effect would be a comparison of Group F with its own CRF control group (Group G), which has the same total number of Rs. This comparison would be appropriate no matter what the effect of prolonged CRF, per se, might be.

Some evidence concerning resistance to extinction following different amounts of CRF is, in fact, available within the experiment, since Groups B, D, and G received 5R, 18R, and 34R, respectively.

Results

Figure 4 presents the extinction performance of the groups which had received a single large N-length followed only by R-trials rather than by PRF. As indicated in the left-hand panel (Groups A and B), when the N-length was followed by a single R-trial, the result was the opposite of a PRE, even though both PRF and CRF groups were (essentially) completely suppressed on the first extinction trial. A Mann-Whitney U-test (Siegel, 1956) on the overall suppression ratios (for extinction trials 2 through 33) indicated that the difference was significant at the .005 level.

When the number of R-trials following the large N-length was increased to 14R (cf. Groups C and D in right-hand panel of Figure 4) there was no evidence of a systematic difference between PRF and CRF. It is clear, therefore, that the use of a single N-length followed by R-trials alone was not sufficient to produce a PRE.

Figure 5 presents the results concerning the addition of a prolonged period of CRF prior to PRF. The left-hand panel shows the outcome for the standard CRF and PRF treatments (Groups D and E), and the usual

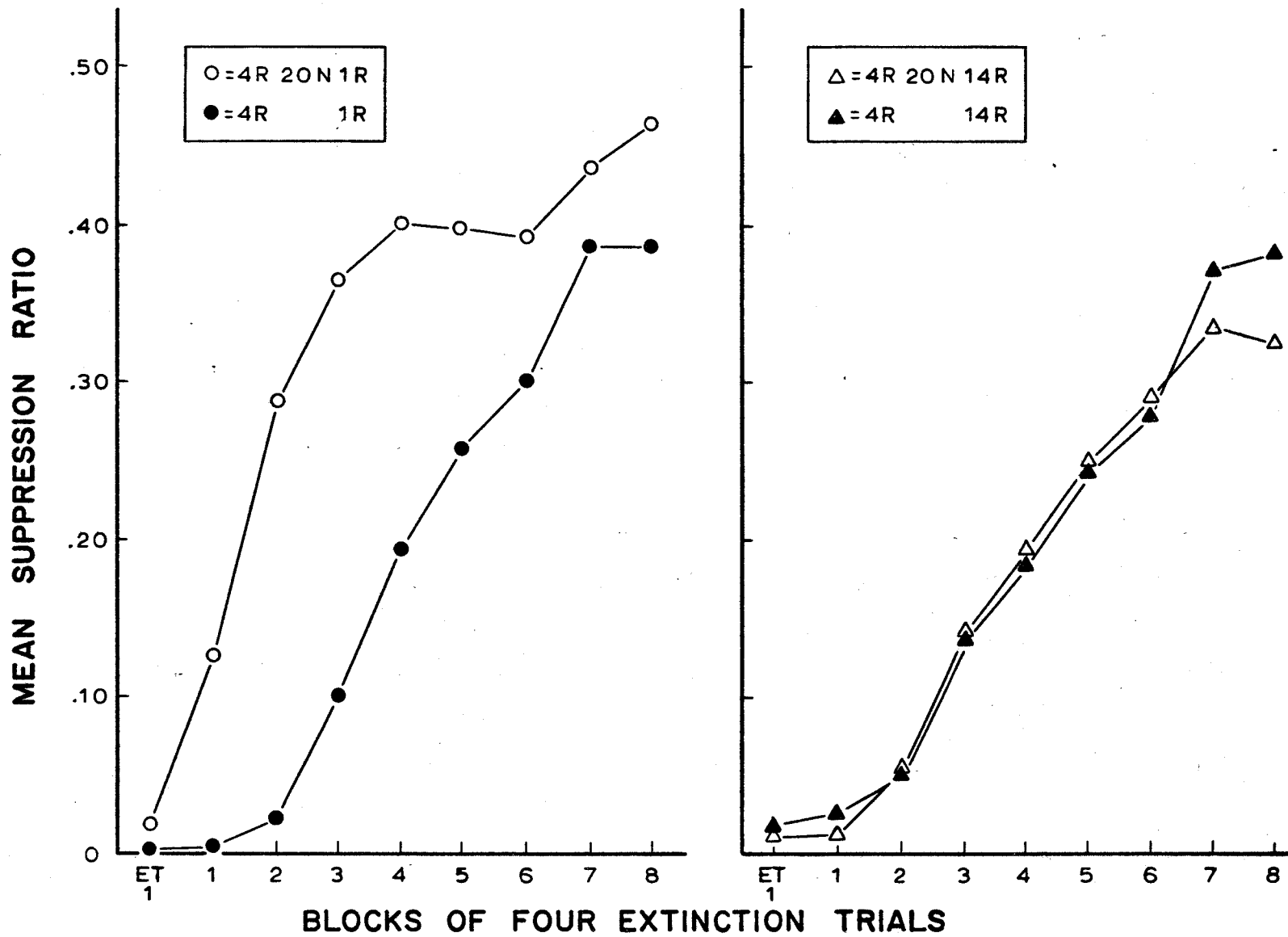


FIGURE 4 EXTINCTION OF CER AS A FUNCTION OF SCHEDULE OF REINFORCEMENT AND OF NUMBER OF R-TRIALS FOLLOWING A SINGLE N-LENGTH (EXPERIMENT 3).

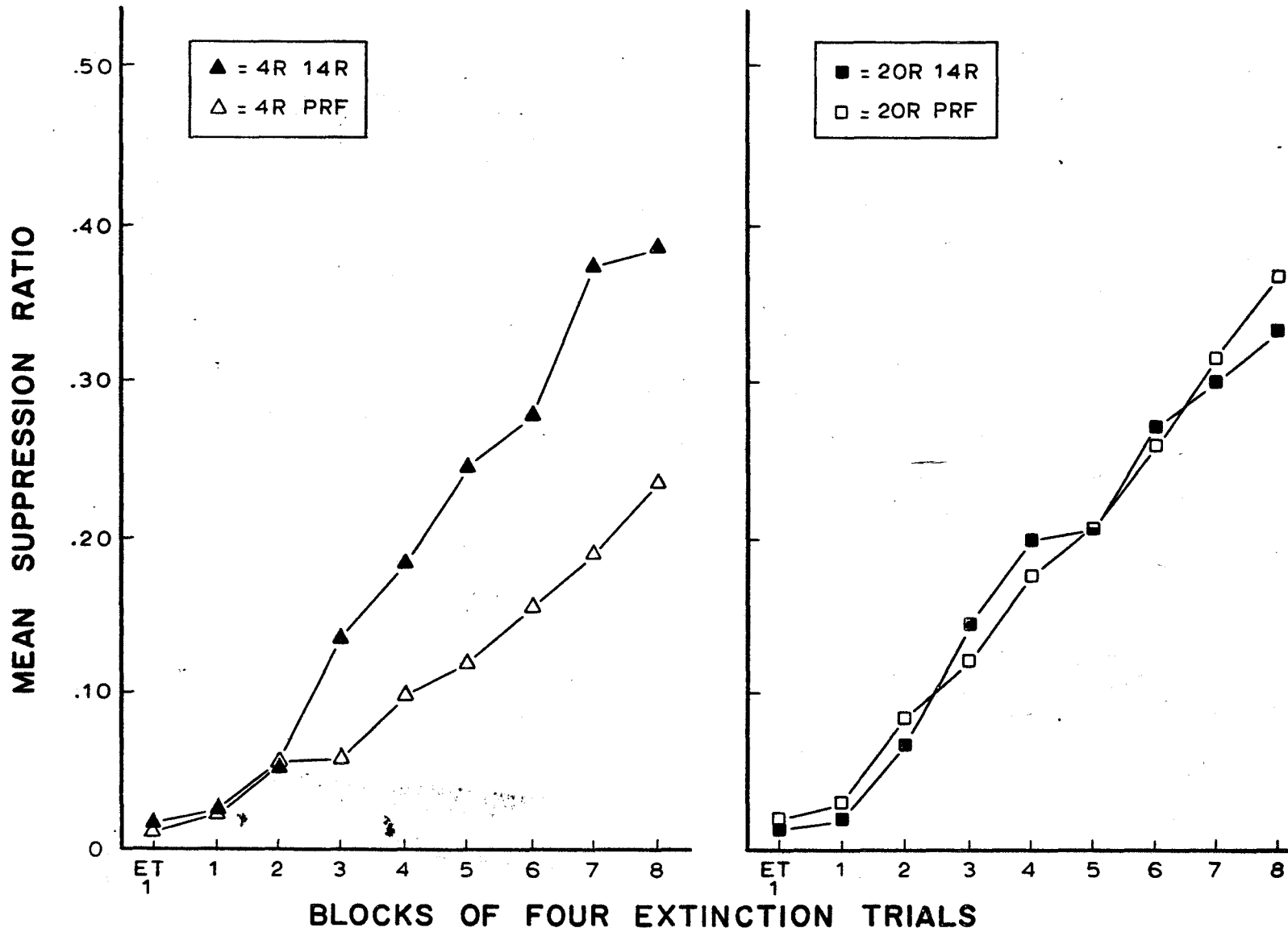


FIGURE 5 EXTINCTION OF CER AS A FUNCTION OF SCHEDULE OF REINFORCEMENT AND AMOUNT OF INITIAL CRF (EXPERIMENT 3).

PRE is evident. While the difference between the two groups was barely significant at the .05 level (U-test on the overall suppression ratios), it constitutes still another replication of the basic PRE. However, when additional 16 R-trials were given prior to the standard CRF and PRF treatments (Groups G and F in the right-hand panel of Figure 5) all evidence of a PRE disappeared. The remaining comparison, between the two PRF groups (E and F), showed a decrement in resistance to extinction with the additional initial CRF, but the difference between groups fell short of significance.

The general pattern of results for the seven treatments of Experiment 3 is indicated in Table 8, which contains the overall group mean suppression ratios (over trials 2 through 33). Only two groups stand out: Group E, which is the PRF group of the basic PRE; and Group A, which received the 4R 20N 1R treatment and displayed the opposite of a PRE. The remaining five groups had nearly identical mean suppression ratios, ranging from .19 to .21. Moreover, it follows that the three CRF groups, receiving 5R, 18R, and 34R, respectively, did not differ in resistance to extinction.

Discussion

There are three findings of particular interest within Experiment 3. The first is that the basic PRE demonstrated by the 4R PRF and 4R 14R treatments was replicated still another time, and it was roughly the same magnitude as the basic PRE of Experiment 2 (compare Figure 2 and the left-hand panel of Figure 5).

Second, a single large N-length followed by CRF was totally ineffective in producing a PRE. The long N-length led either to a "reverse

Table 8

Overall group mean suppression ratios (for trials 2-33), Experiment 3.
 Ranges of \bar{S} means are in parentheses.

<u>Group</u>	<u>Treatment</u>	<u>Mean Ratio</u>	<u>Ranges</u>
A	4R 2ON 1R	.36] opposite of PRE (.01 - .54)
B	4R 1R	.21	
C	4R 2ON 14R	.20	(.00 - .36)
D	4R 14R	.21] PRE (.04 - .35)
E	4R PRF	.12	
F	2OR PRF	.19	(.00 - .36)
G	2OR 14R	.19	(.05 - .36)

PRE" (when followed by 1R), or it had no detectable effect (when followed by 14R). The reverse PRE displayed by the 4R 20N 1R group does not seem especially surprising, for it is presumably attributable to extensive extinction within the N-length. The single R following the N-length might, in theory, have counteracted this extinction in either or both of two ways: the R might have re-conditioned the CER to the white noise CS, and it might have made "hope" (or the "aftereffect" of 20N) a conditioned cue for fear. The data strongly indicate that some reconditioning of the CER was accomplished by the single R, for the .00 suppression ratio displayed by the animals on the first extinction trial was considerably lower than the ratio which would presumably have been observed had the Ss been tested immediately after the 20N. The rapid extinction of these Ss, however, makes clear that the effects of the long N-length were not completely counteracted. Obviously the observation of a .00 suppression ratio on a single trial is no guarantee that reconditioning has been asymptotic.¹¹ Thus, with these animals, any tendency for the single R trial to have produced a PRE via the mechanisms proposed by Amsel or Capaldi seems simply to have been overwhelmed by the extinctive effects of the 20N.

We also, however, failed to obtain a PRE when the 20N was followed by 14R. In this case, reconditioning of the CER following the 20N seems to have been asymptotic; at least, there was no difference in resistance to extinction between the 4R 20N 14R group and the 4R 14R group. The data

¹¹ A similar account may apply to the reverse PRE obtained by Geller (1964). The complete suppression displayed by PRF Ss on early trials of extinction is in no way inconsistent with the fact that N-trials during the PRF schedule have in fact facilitated extinction. (Cf. earlier discussion of this experiment, p. 16.)

thus make clear that the facilitations of the PRE by interpolation of an N-length of 20N, observed in Experiment 2, depends upon an interaction between the N-length and the subsequent PRF; if the N-length is followed by the same number of R-trials employed in our PRF schedule, but if the R-trials are administered consecutively, no contribution of the N-length to resistance to extinction can be detected.¹²

Unfortunately, neither the Amsel nor the Capaldi theories seem sufficiently detailed and specific either to have predicted this outcome unambiguously, or to be gravely embarrassed by it. From the Amselian view, it might of course be argued that a single pairing of hope and shock (the first R-trial after the N-length) is not sufficient to produce a PRE. The ambiguity arises, however, in considering whether, for the 4R 20N 14R group, hope is present to any substantial degree during the remaining 13R. If the answer were negative, one might then argue that, in the 4R 20N PRF treatment, N-trials during PRF "re-activate" hope, which is then repeatedly paired with shock on the R-trials.

The Capaldi account is more specific with respect to the question of whether the aftereffects of the N-length persist through a subsequent series of R-trials. According to Capaldi, the aftereffects of any given N-length are completely eliminated by the delivery of a single reinforcement. Thus, in a sense, the contribution of an interpolated 20N to the PRE is "fixed" once the first subsequent R-trial is administered. However, Capaldi's theory is not very specific in describing the ways in

¹²Strictly, the N-length in our 4R 20N 14R treatment should have been 22N, since our standard PRF schedule, employed with the 4R 20N PRF group of Experiment 2, begins with 2N. This minor discrepancy, however, does not seem likely to have affected the data.

which various numbers of different N-lengths might generalize, summate, and interact, in ultimately determining the resistance to extinction generated by a given PRF schedule. Thus, the present outcome would be embarrassing to Capaldi's theory only under the extreme assumption that a PRF schedule could be fractionated into a number of component N-lengths, each of which, in a strictly additive fashion, contributes to resistance to extinction.

In any event, however these data are interpreted, a practical consequence of these results is that the strategy of assessing the effects of single N-lengths, independent of the precise nature of the reinforcement schedules which follow them, appears infeasible.

The third major finding of Experiment 3 is the disappearance of the basic PRE when 20R, rather than 4R, were given prior to the standard PRF schedule. The evidence from this experiment alone does not, of course, show that the same result could not have been obtained had the additional 16R been interpolated at some other point within the 4R PRF treatment. But the finding is the same as that obtained with instrumental PRF, and with instrumental PRF the location of the added CRF is important. The equivalence between the present result and that observed in instrumental PRE studies, offers no ground to support an assumption that the mechanisms which underlie the two types of PRE are different.

The initial-CRF effect obtained here with the CER was not as dramatic as that found in instrumental studies by e.g., Hothersall (1966): while there was no trace of a CER PRE when 20R PRF was compared to its CRF control group, 20R 14R, the difference between the two PRF groups themselves (i.e., 4R PRF and 20R PRF), though in the appropriate direction,

did not reach significance (two-tailed) in terms of either overall suppression ($.20 > p > .10$) or suppression on the trial of least overlap between groups ($.10 > p > .05$). However, it seems likely that the initial-CRF effect might be maximized by employing a "reference" PRF procedure which would lead to a larger PRE. For example, a difference between 1R PRF and 2OR PRF might be greater than the difference obtained between 4R PRF and 2OR PRF. This prospect is heightened by a comparison of our second preliminary experiment (P-2 of Chapter 4) and a somewhat similar CER study by Brimer and Dockrill (1966) described in Chapter 2. The PRE observed in their experiment was much larger than that of our Experiment P-2. The explanation may be that their PRF schedule contained a fewer number of R-trials at the start (at most two, compared with the eight of Experiment P-2).

As was indicated in Chapter 2, Sutherland's theory of PRF is explicitly concerned with the initial-CRF effect, while Capaldi's does not deal at all with the problem. Amsel's theory is not specific enough in this regard to be easily categorized. An account by Amsel and Surridge (1964, pp. 321-322) would seem to predict the opposite of the initial-CRF effect, however: a PRE is assumed to be a function of anticipatory frustration at the time of reward; anticipatory frustration is a function of amount of frustration; frustration is a function of amount of anticipatory reward; and anticipatory reward is presumably related to number of initial R-trials (as well as amount of reward, etc.). The PRE might therefore be a direct function of number of initial R-trials.

One final aspect of Experiment 3 which should be mentioned is the absence of any differences in resistance to extinction among the three

CRF groups (which received 5R, 18R, and 34R respectively). In contrast to this result, a comparison of the CRF groups of the Preliminary Experiments P-3 and P-4 suggests that with a Bar-In procedure extinction differences can be obtained between CRF groups having numbers of acquisition trials within the range of 7R to 17R (cf. Appendix B).

The different functions relating resistance to extinction to number of CRF trials observed with Bar-In and Bar-Out procedures might be interpreted by reference to the results of Experiment 1. With the CRF groups of that experiment, the Bar-Out procedure tended to produce much more rapid extinction than did the Bar-In procedure. We have previously indicated that this effect could be attributed to the formation of a discrimination by Bar-Out CRF Ss: for them, the CS presented while the bar was absent was always reinforced by the US, while the CS with the bar present (during extinction) was never reinforced. This discrimination, which greatly facilitated extinction, might have "washed out" differences in resistance to extinction as a function of number of CRF trials, differences which can be detected with a Bar-In procedure. The data of Experiment 1 indicate, however, that the Bar-Out procedure did not facilitate extinction to the same degree for PRF Ss; for such Ss, of course, the basis for a discrimination between Bar-Out and Bar-In conditions was not so substantial, since the CS had sometimes been nonreinforced during the Bar-Out condition.

An implication of this interpretation is that other methods of producing a large change in the environmental stimulus complex at the onset of extinction should produce a similar increase in the size of the PRE -- not because the stimulus change would make the PRF Ss more

resistant to extinction, but because it would make the CRF Ss less resistant to extinction. This account of the difference in effects of the Bar-In and Bar-Out procedures might be considered a special case of the "local-discrimination" theory of the PRE mentioned in Chapter 2.

There remains the possibility, of course, that conditioning of an emotional response might simply occur more rapidly in the Bar-Out condition than in the Bar-In condition. To hold to this position we would have to assume that the CER, in the Bar-Out condition, reaches an asymptote within five CRF trials. This is not logically inconsistent with the fact that re-introduction of the bar facilitates extinction.

CHAPTER EIGHT
GENERAL DISCUSSION

The first objective of this thesis was to demonstrate a reliable PRE with a classical conditioning procedure. This objective would certainly seem to have been achieved, in as much as the basic PRE obtained in Experiment 1 was replicated in both Experiments 2 and 3. Contrary to the implication of the findings of Wagner, Siegel, and Fein (1967), the CER PRE was not dependent on any fortuitous relationship between bar-pressing and either the CS or the US of the Pavlovian pairings. In other words, we have eliminated at least one obvious possible source of an instrumental contingency from the CER PRF procedure. However, as indicated in Chapter 1, we cannot logically rule out all possibility of "instrumental" associations being responsible for the PRE observed with a CER procedure. As with any Pavlovian procedure, the S could conceivably make instrumental responses which were unobservable by E. We can at least ask, however, whether our "Pavlovian" PRE can be shown to differ in any important way from the "instrumental" PRE.

The data of Experiments 2 and 3 provide no evidence for such a difference. The CER PRE was obtained using widely spaced trials (at least five hours apart), as is also the case with instrumental PREs (Weinstock, 1954, 1958). In Experiment 2, the interpolation of a single long N-length in the basic PRF schedule increased the size of the PRE. While there is no exactly comparable body of instrumental data, this effect was at least consistent with a number of theoretical interpretations of the instrumental PRE. The failure to obtain a PRE when a single N-length was followed by

R-trials alone might seem puzzling, but there is no reason to suppose that an analogous procedure with instrumental conditioning would be any more successful. Finally, the effect on the CER PRE of prolonged initial CRF was similar to its effect on the instrumental PRE: with both types of procedure, the initial CRF reduces, if it does not entirely eliminate, the PRE.

Thus, the most parsimonious assumption which we could adopt, for the present, would be that the PRE either has very similar mechanisms for both types of conditioning procedures or that both PREs are at bottom attributable to one form of conditioning. The one form of conditioning might be either a "Pavlovian" or an "instrumental" process, irrespective of the type of experimental procedure. The total evidence available, in any event, does not justify an attempt to distinguish classical from instrumental conditioning on the ground that PRF affects the two forms of conditioning in different ways.

We may next consider whether a satisfactory theoretical account can be made of the results of Experiments 2 and 3. Experiment 2 showed that the interpolation of a large N-length between an initial 4R and a standard PRF schedule produced a substantial increase in the size of the PRE. This result, by itself, seemed quite consistent with the views of either Amsel or Capaldi. According to our extension of Amsel's concepts to the CER situation, the long N-length developed an exceptionally high level of "hope" (analogous to Amsel's "anticipatory frustration") which, after having been paired with shock, then became a conditioned stimulus for fear. Ordinarily, this much hope occurring in extinction would produce a loss of suppression; but it would not do so under the present circumstances, since it elicits fear.

The Capaldi interpretation of the interpolated N-length effect would be essentially similar, but the more neutral concept of "aftereffects" is employed rather than that of "hope".

What is not clear is how either the Amsel or Capaldi theories would account for the fact that the long N-length contributed to resistance to extinction when followed by a PRF schedule (Experiment 2) but not when followed by R-trials alone (Experiment 3). A simple additivity view would suggest that the contribution of the N-length could be separated from that of the remainder of the PRF schedule. The empirical results, however, make it clear that the N-length interacts with the properties of the subsequent PRF schedule. There appears to be no need to saddle either the Amsel or Capaldi theory with the simple additivity view, but the unfortunate fact remains that no extant theory of PRF deals in any very explicit fashion with the interactions between N-lengths, R-lengths, numbers of N-R and R-N transitions, etc., involved in PRF schedules. Indeed, empirical information about such interactions is still scanty, and, as Capaldi (1966) has pointed out, the confounding of these variables makes their unraveling a formidable experimental task.

The initial-CRF effect of Experiment 3 is, as we have seen, analogous to the results obtained with instrumental PRF. The effect is also an important basis for Sutherland's theory (described in Chapter 2), according to which certain analyser-response connections formed during initial CRF somehow prevent the subsequent formation of the multiple analyser-response connections assumed to underlie the PRE. However, it appears doubtful that the Amsel and Capaldi theories can account for the initial-CRF effect without being considerably expanded. As Capaldi's theory stands now, primary emphasis

is on N-length variables, with Rs serving the three functions of response reinforcement, replacement of the aftereffects of nonreinforcement with those of reinforcement, and the conditioning of the response to the aftereffects of nonreinforcement. Given the quite unambiguous initial-CRF results of both instrumental and CER experiments, the effects of R-length variables would presumably have to be included -- in an already complex theory -- if it were to try to encompass all the available data.

For Amsel's theory, too, Rs maintain the conditioned response, but particular importance is attached to the necessity of Rs occurring prior to Ns so that the absence of reinforcement can function as "nonreinforcement". That is, to take the appetitive situation, Rs must occur before "frustration" can be produced; and the frustration is a precursor to anticipatory-frustration, which must be then paired with reinforcement in order to produce a PRE. However, while the PRE may indeed be directly related to frustrative effects, and while these in turn may be directly related to initial CRF, the empirical evidence indicates that initial CRF involves another factor. One possible factor is suggested by Amsel's emphasis on the competition between anticipatory-reward and anticipatory-frustration at intermediate stages of PRF. This competition is assumed to affect S's running behavior, but it might also affect the likelihood of anticipatory-frustration being associated with reinforcement.

The latter result might be accomplished if the anticipatory-reward responses were to "overshadow", or "dominate", the anticipatory-frustration responses. In that case, prolonged initial-CRF might be the means by which anticipatory-reward could achieve a position of domination. However, since

initial R-trials also increase the potential for frustration, presumably there would be some critical (for a PRE) amount of initial CRF which would produce some frustration but not too much anticipatory-reward. Direct experimental support for such a scheme is obviously lacking, at present. However, the "competition" notion might well prove to be helpful in a further development of either Amsel's theory or Capaldi's. (With a modified Capaldi theory, the aftereffects, S^R and S^N , would be in the competitive relationship.)

Finally, the initial-CRF effect suggests another way of making at least some sense out of the large N-length results of Experiments 2 and 3, i.e., the finding that the contribution of the N-length to resistance to extinction depended on its being followed by PRF, rather than by R-trials alone. The notion is that the main function of the long N-length in the 4R 20N PRF treatment of Experiment 2 was to undo the effects (whatever they were) of the initial 4R. That is, the 20N did not make a contribution to resistance to extinction in its own right which was any greater than that of any other N-length. The assumption is that as few as 4R are still too many initial R-trials for maximizing a PRE. If such an interpretation were correct, it should be possible to obtain an amount of resistance to extinction at least equivalent to that produced by the 4R 20N PRF treatment simply by deleting the 4R 20N from the overall schedule, or by substituting smaller numbers of Rs and Ns for the 4R 20N part of the treatment.

Perhaps the major conclusion to be drawn from our discussion is that to arrive at a satisfactory account of the PRE, the effects of N-

lengths and R-lengths cannot be considered in isolation. The failure of existing theories to deal explicitly with their interaction is a major shortcoming, and it seems clear that the further development of experimental procedures designed to assess the effects of interpolation of individual trials and blocks of trials into PRF schedules will be useful in establishing the more detailed empirical information which might in turn make possible a more satisfactory theoretical account of PRF.

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APPENDICES

APPENDIX A
LIST OF SYMBOLS AND ABBREVIATIONS

List of Symbols and Abbreviations

CS	=	Conditioned Stimulus
US	=	Unconditioned Stimulus, or reinforcing stimulus
CER	=	conditioned-emotional-response or conditioned-suppression technique
R	=	a reinforced trial or reinforcement
N	=	a nonreinforced trial or nonreinforcement
R-length	=	a block of consecutive Rs
N-length	=	a block of consecutive Ns
CRF	=	continuous, consistent, or 100% reinforcement
PRF	=	partial or intermittent reinforcement
PRE	=	partial reinforcement effect: specifically, the observation of greater resistance to extinction following PRF than following CRF. Note: acquisition effects of PRF have not been given special abbreviations.
GSR	=	Galvanic Skin Response
ITI	=	Intertrial Interval

APPENDIX B
PRELIMINARY EXPERIMENTS
P-1 THROUGH P-4

PRELIMINARY EXPERIMENTS

EXPERIMENT P-1

Method

The Ss were five groups of McMaster Colony rats, eight rats per group. The CS was a 3-minute, 70 db white noise. The US was a .5-second shock of either .5 ma or 1.0 ma.

Each S received one two-hour session per day. Within a session there were eight trial-periods, equally spaced, with the first CS occurring 11 minutes after the start of the session.

After a preliminary five days of bar-press training, the five groups were given the following treatments, which compared PRF and CRF at the two different US intensities.

Groups

- PL PRF with .5 ma shock; CS + US on the odd (1,3,5,& 7) trials, CS alone on even trials.
- 4CL CRF with .5 ma shock; CS + US on the odd trials; remaining trial periods were blank (neither CS nor US). Groups 4CL and PL were therefore equated for reinforced CSs, but not for trials.
- 8CL CRF with .5 ma shock; CS + US on all 8 trials. Groups 8CL and PL were equated for trials (CSs) but not for reinforcements.
- PH PRF with 1.0 ma shock; CS + US on odd trials; CS alone on even trials.
- 4CH CRF with 1.0 ma shock; CS + US on odd trials; remaining trials blank. Groups 4CH and PH were equated for reinforcements.

All groups were given 10 days of CER training, with the exception of group PH which received only five. Extinction was not studied in this experiment.

Results

Group PL displayed almost no suppression. While some individual Ss suppressed gradually, the group median ratio remained close to .50, crossing that value at least once during each CER day. There was a tendency for a nonreinforcement to be followed by a loss of suppression on the next trial; in only three out of 30 "opportunities" did a nonreinforcement lead to a lower median suppression ratio on the next trial.

The two CRF groups receiving .5 ma shock (4CL and 8CL) showed a moderate and gradual increase in suppression up to the fourth CER day, after which there was a gradual loss of suppression.

With the 1.0 ma shock, groups PH and 4CH both displayed very little suppression on the first CER day but became completely suppressed during CER day 2. Thereafter, the 4CH group remained well suppressed through CER day 10. But the PH group (PRF) showed a loss of suppression by the end of CER day 5 (this group did not receive additional training).

Median suppression ratios for each group are listed below.

Median Suppression Ratios Experiment P-1

<u>Group</u>	Trial Period							
	1	2	3	4	5	6	7	8
PL	.52	.50	.54	.50	.51	.50	.46	.44

CER Day 1, cont'd

4CL	.50		.48		.50		.50	
8CL	.47	.46	.48	.44	.42	.44	.42	.42
PH	.46	.48	.54	.50	.58	.44	.51	.29
4CH	.49		.44		.43		.37	

CER Day 2

<u>Group</u>	<u>Trial Periods</u>							
	1	2	3	4	5	6	7	8
PL	.44	.42	.50	.44	.52	.50	.50	.50
4CL	.42		.40		.41		.34	
8CL	.36	.32	.32	.25	.38	.35	.32	.30
PH	.06	.00	.00	.00	.00	.00	.00	.00
4CH	.06		.01		.00		.04	

CER Day 3

<u>Group</u>	<u>Trial Periods</u>							
	1	2	3	4	5	6	7	8
PL	.44	.39	.49	.47	.51	.42	.49	.42
4CL	.24		.30		.24		.22	
8CL	.22	.23	.29	.22	.29	.26	.26	.17
PH	.02	.00	.00	.00	.05	.09	.14	.11
4CH	.02		.02		.05		.01	

CER Day 4

<u>Group</u>	<u>Trial Periods</u>							
	1	2	3	4	5	6	7	8
PL	.38	.43	.47	.44	.52	.49	.53	.48
4CL	.08		.18		.29		.22	
8CL	.19	.16	.26	.24	.28	.28	.28	.38

CER Day 4, cont'd

PH	.00	.00	.00	.23	.22	.08	.16	.06
4CH	.01		.03		.06		.03	

CER Day 5

Trial Periods

<u>Group</u>	1	2	3	4	5	6	7	8
PL	.50	.40	.48	.46	.52	.54	.52	.47
4CL	.32		.27		.18		.20	
8CL	.35	.30	.30	.35	.34	.36	.36	.32
PH	.02	.16	.11	.28	.23	.25	.37	.22
4CH	.05		.08		.06		.04	

CER Day 6

Trial Periods

<u>Group</u>	1	2	3	4	5	6	7	8
PL	.50	.49	.50	.42	.52	.49	.60	.58
4CL	.37		.21		.22		.32	
8CL	.42	.34	.40	.38	.42	.36	.40	.35
PH								
4CH	.01		.00		.01		.02	

CER Day 7

Trial Periods

<u>Group</u>	1	2	3	4	5	6	7	8
PL	.48	.42	.48	.44	.44	.44	.48	.50
4CL	.33		.28		.30		.34	
8CL	.41	.39	.35	.38	.42	.40	.44	.42
PH								
4CH	.03		.00		.00		.04	

CER Day 8

<u>Group</u>	Trial Period							
	1	2	3	4	5	6	7	8
PL	.50	.44	.48	.47	.52	.48	.50	.52
4CL	.40		.29		.34		.32	
8CL	.42	.35	.32	.38	.37	.41	.34	.36
PH								
4CH	.03		.01		.00		.05	

CER Day 9

<u>Group</u>	Trial Period							
	1	2	3	4	5	6	7	8
PL	.48	.43	.54	.51	.56	.56	.53	.59
4CL	.46		.24		.32		.36	
8CL	.46	.40	.37	.38	.38	.38	.43	.40
PH								
4CH	.04		.02		.02		.03	

CER Day 10

<u>Group</u>	Trial Period							
	1	2	3	4	5	6	7	8
PL	.51	.51	.48	.44	.55	.53	.59	.59
4CL	.40		.20		.28		.39	
8CL	.44	.40	.40	.38	.41	.46	.40	.38
PH								
4CH	.01		.02		.01		.01	

Experiment P-2

Method

The Ss were three groups of McMaster Colony rats, eight rats per group. The CS was a 3-minute, 70 db white noise. The US was a 1.0 ma shock of .5 sec. duration.

As in the previous experiment, there was one two-hour session per day, with eight trial periods in a session. Lever pressing was permitted throughout (Bar-In procedure).

Following five days of preliminary bar-press training, all Ss received four reinforced trials on each of CER days 1 and 2, for a total of 8R (CRF). Thereafter there were three treatments:

- CRF 4R per day, on the odd trial-periods (1, 3, 5, & 7).
- PR 50% PRF with the Rs on odd trial-periods, and Ns on the even trial-periods. (I.e., single alternation).
- PI 50% PRF with an irregular schedule of Rs and Ns. The patterns from CER days 3 - 8 were: RNRNRNRN, RNNRRNRR, RRNNRRNR, RNRNNRRR, RRNNNNRR, and RNNNNRRR.

Extinction for all groups began on the day after CER day 8. In extinction there were four trials per day, located in the odd trial periods.

Results

All groups were well suppressed by the end of the initial CRF of acquisition. Introduction of Ns on CER day 3 produced a moderate loss of suppression for both PRF groups. The two PRF groups, which did not differ from one another, remained for the rest of acquisition, at a level of lesser suppression than the CRF group.

In extinction there was some evidence of a small PRE: the median curves for the PRF groups, which had stabilized during acquisition at a

moderate level of suppression above the CRF group, crossed over the CRF curve and displayed a slightly slower course of extinction. However, within extinction there were no significant differences between groups.

The mean suppression ratios of the three groups for the last CER acquisition day and the first five CER extinction days are given below.

<u>Group</u>	CER-8	Ext. 1	Ext. 2	Ext. 3	Ext. 4	Ext. 5
PR	.25	.21	.25	.37	.41	.43
PI	.21	.25	.31	.29	.38	.39
CRF	.09	.15	.30	.40	.46	.47

Experiment P-3

Method

The subjects were two groups of 16 rats each, obtained from Canadian Research Animal Farms, Bradford, Ontario.

The CS was a 3-minute white noise of 80 db (rather than 70 db). The US was a .5 second shock of 2.0 ma (rather than 1.0 ma).

Each rat was given two 45-minute sessions each day, the first session in the morning and the second approximately five hours later. Ss were fed a small portion of their daily food ration after the first session, and were weighed and then fed an additional amount after the second session. Within a session there was only one trial period, located so that a CS onset would occur 24 minutes after the start of the session.

Following six days of VI-training, CER training was begun, using the Bar-In procedure.

All Ss received a reinforced trial in each session of CER days 1

and 2, for a total of 4R (CRF). During the next 13 CER days, there were two treatments, PRF and CRF, having the following patterns of Rs and Ns:

CER day:	3	4	5	6	7	8	9	10	11	12	13	14	15
PRF :	NR	NR	RN	NR	RN	NR	RN	NR	RN	NN	RR	NN	RR
CRF :	R	R	R	R	R	R	R	R	R	RR	RR	RR	RR

The CRF and PRF groups were, therefore, equated for number and spacing of Rs. The CRF group received a blank trial when the PRF group received an N.

All Ss were given 34 trials of CER extinction.

Results

The Ss of both CRF and PRF groups were essentially completely suppressed before the differential treatments were initiated (i.e., by the end of CER day 2). Both groups remained completely suppressed throughout acquisition.

In extinction, the PRF group displayed greater resistance to extinction than the CRF group; however, the PRE was barely significant. Mann-Whitney U-tests indicated that the two groups were significantly different, at at least the .05 level, on extinction trials 25, 26, and 27 in terms of suppression. They also differed in the number of trials required to reach a criterion of two consecutive ratios of .40 or greater ($p < .05$).

The median suppression ratios for each group for the last two trials of CER acquisition and the 34 trials of extinction are given below.

Median Suppression Ratios
Experiment P-3

	<u>CER trials</u>		<u>Extinction trials</u>									
	29	30	1	2	3	4	5	6	7	8	9	10
PRF:	.00	.00	.00	.01	.02	.00	.02	.06	.02	.01	.04	.04
CRF:	.00	.02	.01	.00	.01	.00	.02	.02	.02	.04	.10	.06

	<u>Extinction trials</u>											
	11	12	13	14	15	16	17	18	19	20	21	22
PRF:	.10	.08	.06	.06	.12	.07	.12	.08	.14	.14	.16	.16
CRF:	.10	.08	.12	.12	.17	.18	.28	.18	.34	.31	.36	.38

	<u>Extinction trials</u>											
	23	24	25	26	27	28	29	30	31	32	33	34
PRF:	.18	.11	.16	.21	.22	.26	.26	.36	.36	.45	.38	.38
CRF	.45	.44	.39	.46	.40	.46	.45	.46	.43	.44	.48	.46

Experiment P-4

Method

There were two groups of 16 rats each (from Canadian Research Animal Farms) which were treated identically to the groups of Experiment P-3 except that there were only three days of PRF after CER days 1 and 2. The schedules of Rs and Ns were as follows:

CER day:	1	2	3	4	5
PRF :	RR	RR	NR	RN	NR
CRF :	RR	RR	R	R	R

Results

All Ss were given 16 extinction trials, by the end of which both groups had median suppression ratios greater than .40. There was no sign of a systematic difference between groups at any point in extinction.

APPENDIX C

EXTINCTION SUPPRESSION RATIOS, TRIALS TO CRITERION
AND BASELINE RESPONSES FOR EXPERIMENT 1.

EXPERIMENT 1

Sums of Suppression ratios over blocks of trials and group mean ratios.

Treatment: 3-Min. CS Bar-Out CRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.02	.27	1.25	1.08	1.79	2.03	1.73	2.00	2.16	2.03	1.48	2.67
2	.00	.00	.02	.01	.11	.69	.37	1.09	1.55	1.52	1.54	1.84
3	.00	.00	.00	.09	1.43	1.77	1.65	1.56	1.87	2.22	2.05	2.12
4	.02	.32	.63	1.43	1.85	2.01	2.44	1.96	2.24	2.24	1.98	1.80
Mean Ratio	.010	.037	.119	.163	.324	.406	.387	.413	.489	.501	.441	.527

Treatment: 3-Min. CS Bar-Out PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.25	.00
2	.00	.09	.11	.00	.20	.18	.00	.32	.42	.49	1.36	1.13
3	.00	.26	.38	.21	.07	.95	.71	.76	.19	.37	.35	.85
4	.00	.05	.02	.00	.03	.04	.13	.26	.16	.14	.20	.06
Mean Ratio	.000	.025	.032	.013	.019	.073	.052	.084	.048	.062	.135	.128

Treatment: 3-Min. CS Bar-In CRF

<u>Trial(s):</u>	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.00	.00	.00	.00	.00	.05	.00	.00	.00	.05	.08	.28
2	.00	.00	.00	.00	.00	.00	.00	.06	.00	.00	.04	.17
3	.03	.08	.32	.19	.38	.82	1.32	1.29	1.50	1.52	1.52	1.51
4	.00	.12	.06	.07	.36	.21	.52	.58	1.00	.95	1.31	1.45
Mean Ratio	.008	.012	.024	.016	.046	.068	.115	.121	.156	.158	.184	.213

Treatment: 3Min. CS Bar-In PRF

<u>Trial(s):</u>	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.00	.00	.00	.00	.00	.00	.03	.00	.00	.01	.00	.01
2	.00	.04	.00	.00	.00	.00	.00	.03	.00	.03	.59	1.50
3	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.18
4	.00	.02	.00	.00	.01	.00	.00	.02	.06	.06	.30	.80
Mean Ratio	.000	.004	.000	.000	.001	.000	.002	.003	.004	.006	.056	.156

Treatment: 30-Sec. CS Bar-Out CRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.00	.00	.00	.00	.00	.00	.00	.08	.00	.00	.00	.00
2	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
3	.00	.00	.34	1.48	1.32	1.18	1.03	1.22	1.34	1.36	1.80	1.43
4	.00	.11	.00	.00	.09	.00	.03	.00	.03	.00	.00	.06
Mean Ratio	.000	.007	.021	.092	.088	.074	.066	.081	.086	.085	.112	.093

Treatment: 30-Sec. CS Bar-Out PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
2	.00	.00	.00	.00	.19	.00	.00	.00	.07	.00	.00	.00
3	.00	.00	.15	.00	.00	.00	.12	.00	.33	.40	.52	.40
4	.00	.00	.00	.00	.00	.00	.27	.15	.57	.00	.00	.26
Mean Ratio	.000	.000	.009	.000	.012	.000	.027	.009	.061	.025	.032	.041

Treatment: 30-Sec. CS Bar-In CRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.00	.00	.00	.00	.00	.00	.00	.00	.00	.07	.00	.12
2	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.11
3	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
4	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
Mean Ratio	.000	.000	.000	.000	.000	.000	.000	.000	.000	.004	.000	.014

Treatment: 30-Sec. CS Bar-In PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>21-25</u>	<u>26-29</u>	<u>30-33</u>	<u>34-37</u>	<u>38-41</u>	<u>42-45</u>
<u>Subject</u>												
1	.00	.00	.46	.00	.00	.00	.00	.22	.13	.15	.00	.00
2	.00	.00	.22	.03	.09	.03	.08	.02	.02	.03	.01	.03
3	.00	.00	.00	.00	.00	.00	.00	.00	.11	.00	.00	.00
4	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.12	.00
Mean Ratio	.000	.000	.042	.002	.006	.002	.005	.015	.016	.011	.008	.002

Number of trials to a criterion of two consecutive suppression ratios $\geq .40$ for each subject in Experiment 1.

		Subject	Number of trials to criterion
3-Min. CS	Bar-Out	1	9
		2	41
		3	19
		4	16
	PRF	1	70
		2	46
		3	80
		4	70
	Bar-In	1	60
		2	59
		3	34
		4	61
30-Sec. CS	Bar-Out	1	72
		2	46
		3	59
		4	59
	PRF	1	82
		2	98
		3	12
		4	82
	Bar-In	1	112
		2	134
		3	92
		4	56
30-Sec. CS	Bar-Out	1	75
		2	69
		3	100
		4	103
	PRF	1	99
		2	123
		3	108
		4	138

Experiment 1

Baseline responses: number of bar-presses in Pre-CS
period of the first extinction trial.

3-Min. CS				30-Sec. CS			
Bar-Out		Bar-In		Bar-Out		Bar-In	
<u>C</u>	<u>P</u>	<u>C</u>	<u>P</u>	<u>C</u>	<u>P</u>	<u>C</u>	<u>P</u>
41	19	11	8	28	36	27	36
29	48	11	35	7	0	44	0
23	20	32	12	12	31	1	33
55	36	75	110	31	19	8	40

Experiment 1

Mean number of bar-presses in Pre-CS periods for
trials 1-45 of extinction.

3-Min. CS				30-Sec. CS			
Bar-Out		Bar-In		Bar-Out		Bar-In	
<u>C</u>	<u>P</u>	<u>C</u>	<u>P</u>	<u>C</u>	<u>P</u>	<u>C</u>	<u>P</u>
32	11	23	33	39	27	51	38
66	42	18	33	43	99	27	237
31	44	36	18	44	31	17	41
49	93	55	66	131	16	31	45

APPENDIX D
EXTINCTION SUPPRESSION RATIOS FOR
EXPERIMENT 2A & 2B

EXPERIMENT 2A

Sums of suppression ratios over blocks of trials

Treatment: 4R 14R

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subject</u>								
1	.00	.04	.14	.26	.42	1.06	1.18	3.10
2	.03	.00	.00	.05	.42	.86	1.17	2.50
3	.00	.02	.47	1.54	1.69	1.08	1.37	6.17
4	.01	.00	.00	.12	1.69	1.77	2.01	5.59
5	.00	.02	.02	.43	.90	1.27	1.50	4.14
6	.00	.15	.07	.43	.57	.79	1.16	3.11
7	.00	.80	1.37	1.60	1.77	1.69	1.90	9.13
8	.03	.26	1.29	1.42	1.85	1.51	2.05	8.38
Mean Ratio:	.009	.040	.105	.183	.289	.314	.386	(.219)

Treatment: 4R PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subject</u>								
1	.00	.01	.01	.01	.53	.44	.77	1.77
2	.02	.29	.63	.38	.32	.43	1.24	3.29
3	.00	.00	.00	.00	.05	.85	1.75	2.65
4	.05	.36	.76	.69	.92	1.67	1.39	5.79
5	.05	.48	.69	.88	1.32	1.50	1.48	6.35
6	.00	.00	.00	.28	.93	.95	2.01	4.17
7	.00	.04	.08	.06	.36	1.24	1.56	3.34
8	.08	.33	.18	.53	.62	.73	1.55	3.94
9	.00	.00	.09	.41	1.44	.89	1.09	3.92
10	.00	.00	.00	.00	.00	.00	.00	.00
11	.00	.06	.02	.10	.32	.49	1.16	2.15
12	.00	.00	.00	.04	.00	.00	.00	.04
13	.00	.00	.02	.00	.00	.00	.00	.02
Mean Ratio	.015	.032	.048	.065	.131	.177	.269	(.120)

Treatment: 4R 4N PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subject</u>								
1	.00	.00	.01	.01	.08	.28	1.06	1.44
2	.00	.00	.00	.00	.00	.19	.59	.78
3	.03	.15	.60	.75	1.30	1.23	1.57	5.60
4	.03	.00	.00	.10	.12	.53	1.57	2.32
5	.00	.00	.13	.12	.20	.32	.25	1.02
6	.03	.06	.08	.07	.06	.02	.27	.56
7	.01	.05	.12	.19	.33	.29	.88	1.86
8	.04	.94	.98	1.48	1.68	1.57	1.43	8.08
9	.00	.00	.00	.26	1.06	1.33	.91	3.56
10	.00	.70	1.19	1.36	.99	1.46	1.55	7.25
11	.02	.07	.71	1.54	1.91	1.98	1.87	8.08
12	.00	.00	.00	.02	.15	.54	1.35	2.06
Mean Ratio	.013	.041	.080	.123	.164	.203	.277	(.148)

Treatment: 4R 10N PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subjects</u>								
1	.00	.15	.09	.50	.78	1.30	1.45	4.27
2	.02	.23	.54	.50	.79	1.35	1.15	4.56
3	.01	.15	.09	.17	.39	.61	1.40	2.81
4	.00	.00	.00	.02	.48	.55	1.81	2.86
5	.02	.09	.01	.00	.00	.06	.21	.37
6	.00	.02	.00	.02	.52	1.42	1.70	3.68
7	.05	.00	.04	.09	.11	.18	.05	.47
8	.00	.00	.03	.02	.40	.62	1.58	2.65
9	.00	.00	.02	.03	.00	.00	.02	.07
10	.01	.13	.29	.43	.51	1.15	.88	3.39
11	.00	.08	.25	.68	.86	1.23	1.69	4.79
12	.00	.00	.00	.00	.70	1.30	1.84	3.84
Mean Ratio	.009	.018	.028	.051	.115	.203	.287	(.117)

Treatment: 4R 20N PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subject</u>								
1	.00	.00	.09	.00	.00	.00	.00	.09
2	.00	.00	.00	.42	.16	.17	.57	1.32
3	.00	.00	.00	.11	.00	.19	.28	.58
4	.00	.00	.04	.00	.00	.17	.11	.32
5	.00	.07	.09	.03	.00	.00	.00	.19
6	.00	.00	.00	.02	.00	.19	.05	.26
7	.00	.05	.08	.11	.01	.31	.64	1.20
8	.00	.01	.02	.50	1.76	1.92	2.02	6.23
9	.00	.00	.00	.00	.00	.00	.03	.03
Mean Ratio	.000	.004	.009	.033	.054	.082	.103	(.047)

EXPERIMENT 2B.

Sums of suppression ratios over blocks of trials

Treatment: 4R PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subject</u>								
1	.00	.07	.71	1.06	.72	.59	.87	4.02
2	.00	.00	.00	.02	.61	1.66	1.86	4.15
3	.02	.10	.04	.25	.89	1.29	1.25	3.82
4	.00	.12	.08	.20	.38	.96	.68	2.42
5	.00	.80	1.28	.95	1.23	1.82	2.13	8.21
6	.04	.03	.11	.46	.16	.40	.58	1.74
Mean Ratio	.010	.047	.092	.122	.166	.280	.307	(.169)

Treatment: 4R 4BT PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subject</u>								
1	.00	.03	.00	.00	.06	.46	.11	.66
2	.00	2.45	1.92	2.06	2.38	2.00	2.40	13.21
3	.00	.00	.00	.00	.00	.37	.93	1.30
4	.02	.11	.02	.05	.02	.06	.00	.26
5	.00	.02	.00	.01	.00	.05	.03	.11
6	.00	.00	.00	.00	.00	.00	.06	.06
7	.05	.60	.81	1.79	1.43	1.88	1.75	8.26
Mean Ratio:	.010	.114	.098	.140	.140	.172	.189	(.142)

Treatment: 4R 20BT PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>2-25</u>
<u>Subject</u>								
1	.08	.11	.41	.12	1.09	1.64	1.61	4.98
2	.00	.00	.00	.00	.06	.12	.67	.85
3	.04	.05	.31	1.07	.94	.76	.77	3.90
4	.00	.00	.00	.00	.01	.64	.84	1.49
5	.27	1.34	1.21	1.41	1.33	1.67	2.20	9.16
6	.00	.00	.02	.00	.23	1.73	1.72	3.70
7	.00	.00	.00	.00	.00	.03	.27	.30
Mean Ratio:	.056	.054	.070	.093	.131	.235	.289	(.145)

APPENDIX E
EXTINCTION SUPPRESSION RATIOS FOR
EXPERIMENT 3

EXPERIMENT 3

Sums of suppression ratios over blocks of extinction trials.

Treatment: 4R 20N 1R

<u>Trial(s):</u>	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>2-33</u>
<u>Subject</u>										
1	.00	.00	.44	1.19	1.41	1.77	2.03	1.90	2.01	10.75
2	.00	.04	.00	.01	.02	.02	.02	.00	.33	.46
3	.02	.21	.53	1.37	1.35	1.42	1.33	1.92	2.40	10.53
4	.00	.72	1.91	1.94	2.74	2.45	2.31	2.58	2.47	17.12
5	.00	.77	1.40	1.74	1.91	1.57	1.80	1.71	1.57	12.47
6	.02	.38	1.66	1.80	1.86	2.03	2.01	1.78	1.95	13.47
7	.07	1.42	1.60	1.93	1.82	1.69	1.16	2.14	2.23	13.99
8	.03	.45	1.66	1.74	1.73	1.83	1.91	1.97	1.93	13.22
Mean Ratio:	.018	.125	.288	.366	.401	.399	.393	.438	.465	(.359)

Treatment: 4R 1R

<u>Trial(s):</u>	<u>ET-1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>2-33</u>
<u>Subject</u>										
1	.00	.00	.00	.63	1.70	1.63	2.00	1.82	1.55	9.33
2	.00	.00	.00	.02	.04	.02	.05	.28	.57	1.00
3	.00	.00	.04	.04	.10	.08	.41	.71	1.17	2.55
4	.01	.04	.28	.92	1.34	1.71	1.70	2.13	1.98	10.10
5	.00	.00	.00	.65	1.53	1.47	1.08	1.69	1.68	8.10
6	.00	.00	.06	.00	.11	.80	1.32	1.57	1.98	5.84
7	.00	.00	.00	.00	.15	1.39	2.25	2.62	1.65	8.06
8	.00	.02	.32	.94	1.19	1.20	.82	1.56	1.79	7.84
Mean Ratio:	.001	.002	.022	.100	.192	.259	.301	.387	.387	(.206)

Treatment: 4R 20N 14R

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>2-33</u>
<u>Subject</u>										
1	.04	.04	.10	.34	.92	1.22	1.78	1.44	1.64	7.48
2	.03	.05	.25	.53	.44	.50	.58	1.17	1.05	4.57
3	.00	.09	.59	.94	1.36	1.83	1.84	1.78	1.69	10.12
4	.00	.07	.23	.81	.24	1.01	.64	1.37	1.03	5.40
5	.00	.00	.02	.00	.78	.73	1.25	1.09	1.15	5.02
6	.00	.02	.32	.45	.54	.92	1.20	1.66	1.84	6.95
7	.00	.00	.00	.00	.00	.04	.00	.03	.00	.07
8	.00	.04	.23	1.49	1.85	1.75	2.00	2.19	2.03	11.58
Mean Ratios:	.009	.010	.054	.142	.192	.250	.290	.335	.326	(.200)

Treatment: 4R 14R

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>2-33</u>
<u>Subject</u>										
1	.00	.06	.02	.00	.01	.00	.06	.36	.74	1.25
2	.00	.00	.03	.18	.56	.20	.71	1.75	1.57	5.00
3	.03	.03	.11	.24	.77	1.01	1.29	1.49	1.76	6.70
4	.00	.03	.06	.26	.11	.77	.88	1.34	1.32	4.77
5	.05	.58	.66	1.20	1.55	1.61	1.76	1.76	1.89	11.31
6	.00	.00	.19	1.04	1.04	1.35	1.53	1.67	1.70	8.52
7	.03	.16	.65	.76	1.00	1.00	1.19	1.56	1.78	8.10
8	.03	.00	.00	.17	.55	1.39	.93	1.42	1.49	5.95
9	.00	.00	.10	1.06	1.02	1.49	1.67	2.01	1.54	8.89
Mean Ratios:	.015	.024	.051	.136	.184	.245	.278	.371	.383	(.210)

Treatment: 4R PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>2-33</u>
<u>Subject</u>										
1	.00	.11	.67	.31	.32	.33	.53	.40	1.07	3.74
2	.00	.00	.01	.00	.02	.02	.11	.56	.77	1.49
3	.00	.00	.03	.05	.42	.93	1.46	1.67	2.09	6.65
4	.05	.05	.00	.00	.01	.00	.04	.34	.49	.88
5	.00	.00	.03	.03	.03	.06	.06	.09	.05	.35
6	.03	.17	.23	.61	1.15	1.26	1.67	1.89	1.86	8.84
7	.02	.39	.84	.97	1.44	1.56	1.59	1.65	1.81	10.27
8	.00	.00	.00	.00	.05	.08	.02	.08	.30	.53
9	.00	.02	.10	.06	.07	.05	.02	.11	.02	.45
Mean Ratios:	.010	.021	.053	.056	.098	.119	.153	.189	.235	(.115)

Treatment: 20R PRF

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>2-33</u>
<u>Subject</u>										
1	.01	.39	.97	1.20	1.14	1.44	1.74	1.77	1.97	10.62
2	.09	.34	1.10	1.64	1.71	1.78	1.59	1.67	1.69	11.52
3	.00	.00	.03	.65	1.13	1.46	1.45	1.44	1.52	7.78
4	.00	.00	.04	.02	.00	.00	.00	.00	.02	.08
5	.00	.00	.04	.06	.74	.24	.36	1.53	2.37	5.34
6	.03	.03	.11	.00	.13	.36	1.16	1.37	1.66	4.82
7	.00	.05	.04	.17	.46	1.27	1.47	2.20	2.50	8.16
8	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
9	.03	.23	.70	.56	.99	.83	1.50	1.26	1.40	7.47
Mean Ratios:	.018	.029	.084	.119	.175	.205	.258	.312	.365	(.194)

Treatment: 20R 14R

Trial(s):	<u>1</u>	<u>2-5</u>	<u>6-9</u>	<u>10-13</u>	<u>14-17</u>	<u>18-21</u>	<u>22-25</u>	<u>26-29</u>	<u>30-33</u>	<u>2-33</u>
<u>Subject</u>										
1	.00	.00	.00	.00	.00	.37	.72	.77	1.40	3.26
2	.00	.00	.06	.51	1.07	1.43	1.47	1.40	1.71	7.65
3	.04	.10	.06	.87	1.58	1.60	1.84	2.14	1.97	10.16
4	.02	.03	.89	1.50	1.67	1.74	1.85	1.94	1.82	11.44
5	.03	.03	.00	.05	.18	.08	.12	.07	.02	.55
6	.00	.00	.00	.04	.04	.00	.01	.00	.07	.16
7	.00	.02	.67	1.18	1.40	1.16	1.76	1.55	1.72	9.46
8	.00	.00	.00	.00	.00	.02	.20	.78	1.24	2.24
9	.02	.49	.70	1.04	1.16	.96	1.76	2.05	1.94	10.10
Mean										
Ratios:	.012	.019	.066	.144	.197	.204	.270	.297	.330	(.191)