THE UNIVERSITY OF MANITOBA

SODIUM AMOBARBITAL AND THE PARTIAL REINFORCEMENT EFFECT: DIFFERENTIAL EFFECTS ON REINFORCED AND NONREINFORCED TRIALS

by

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A dissertation submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

MASTER OF ARTS

For my wife, Margitta.

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ABSTRACT

When subjects trained on a partial reinforcement schedule are compared with subjects trained on continuous reinforcement, it has usually been observed that the subjects trained on partial reinforcement exhibit greater resistance to extinction than the continuously reinforced group. This phenomenon has been termed the partial reinforcement extinction effect (PREE). Additionally, partial and continuous reinforcement schedules have been observed to affect acquisition performance differentially. The partial reinforcement acquisition effect consists of initial slower responding by the partially reinforced group with faster responding at asymptote relative to the continuously reinforced group.

The two major theories accounting for the partial reinforcement effects are Amsel's (1958) Frustration theory and Capaldi's (1967) Sequential theory, of instrumental learning. Both theories are concerned with internal stimulus control in explaining the PREE but they differ with regard to the mechanism. Amsel's theory suggests that the cues associated with anticipatory frustration become classically counter-conditioned to the instrumental response in partially reinforced subjects, thereby providing the mechanism or the PREE. Capaldi suggests that on a given trial partially reinforced subjects remember the reward condition of the previous trial and this memory is conditioned to the instrumental response or the followed trial via reinforcement. Thus Amsel's theory is an intratrial theory whereas Capaldi's theory involves intertrial conditions.

Controversy presently exists with regards to the emotional properties of nonreward-related internal stimuli for training under widely

spaced trials (e.g., 24 hr. intertrial-interval). In previous studies it has been demonstrated that the barbiturate sodium amobarbital attenuates the emotional components of nonreward related interval stimuli, but does not seem to affect the conditioning of emotional responses. By factorially manipulating sodium amobarbital injections on the reinforced and/or nonreinforced trials of acquisition in an alleyway under widely spaced training and testing conditions (24 hr. inter-trial-interval), the present study was designed to test both Amsel's theory and Capaldi's theory as well as investigate the emotional properties of nonreward related internal stimuli under widely spaced trials.

One group of rats received amobarbital injections prior to all reinforced trials and another group prior to all nonreinforced trials in a partially reinforced acquisition schedule. Two other groups received either the drug on all acquisition trials or saline on all acquisition trials. A continuously reinforced group served as a reference group for the PREE. Following acquisition (Phase 1) all groups received five continuously reinforced trials (Phase 2) followed by sixteen extinction trials (Phase 3). During phases 2 and 3 no injections were given to any of the groups.

The results indicated that the groups receiving the drug on either reinforced or nonreinforced trials only, exhibited patterned running in phase 1 of the experiment which was interpreted in terms of drug state dependent cues and emotional responses elicited by the runway. Amobarbital eliminated the reverse partial reinforcement acquisition effect (early in training) and the "goal box effect".

A robust partial reinforcement extinction effect (PREE) was found following partial reinforcement training under saline and when

nonrewarded trials only were preceded with saline injections. Sodium amobarbital administered on all trials of acquisition or only preceding nonreinforced trials, eliminated the PREE (except in the goal section). It was also shown that the drug exerted its effects on nonrewarded trials in acquisition but not on rewarded trials.

Interpretation of these results in terms of Amsel's Frustration theory and Capaldi's Sequential theory, indicated that the results of the present study did not support Amsel's theory but were not inconsistent with Capaldi's theory.

Chapter 1 Introduction

1.1 Partial Reinforcement and its effects

The term partial reinforcement refers to a procedure where some, but not all, of the responses an organism makes, are reinforced. In contrast, the terms continuous reinforcement and extinction refer to schedules where all the responses an organism emits are reinforced or not reinforced, respectively. When subjects trained on partial reinforcement (PRF) are compared with subjects trained on continuous reinforcement (CRF), several distinct differences in performance are observed.

The most notable difference between the PRF and CRF trained subjects is observed in their relative resistance to the decremental effects of continuous nonreward; i.e. resistance to extinction (Rn). Rn has most often been measured by the differential decreases in response speed during the extinction phase. It has become clear from a large body of literature that a pattern of PRF greatly increases Rn relative to CRF (cf. Robbins, 1971). Some of the earliest demonstrations of this phenomenon termed the partial reinforcement extinction effect (PREE) were reported by Skinner (1938) and Humphreys (1939), and many experiments have since repeated these results under a variety of conditions and procedures. Response decrements have been measured in a variety of ways and in a variety of situations to demonstrate extinction. Only the runway situation involving decrements in running speed as a measure of extinction will be dealt with here since the experiment reported involved a discrete trial runway situation.

In the discrete trial situation the reference experiment was performed by Weinstock (1958), who gave different groups of rats one trial

per day under a 17%, 33%, 50%, 67%, 88% or 100% reinforcement schedule. An inverse relation between resistance to extinction and percentage of reinforced trials during training, was found.

In addition to producing differential Rn, PRF and CRF schedules have also been observed to affect acquisition performance differentially. The characteristic effect of PRF on acquisition has been termed the partial reinforcement acquisition effect (PRAE). The PRAE actually consists of three acquisition findings (Robbins, 1971). Initially, PRF subjects run slower than CRF subjects, although at asymptote they run faster. This "cross over effect" has been reported by a number of investigators (Goodrich, 1959; Haggard, 1959; Ross, 1964; Wagner, 1961; Weinstock, 1958). The third finding is the "goal box effect". Reports by Goodrich (1959), Haggard (1959), McCoy and Marx (1965) and Wagner (1961), have demonstrated faster asymptotic running speeds by the CRF animals in the goal speed measure (reverse PRAE). The reference study for the PRAE is Weinstock (1958) who found an inverse relation between asymptotic start and alley speeds, and percentage of reinforced trials as well as an inverse relation between the trial block in which the cross over effect occurred and percentage of reinforced trials.

A graphic illustration of both the PREE and the PRAE is given in Figure 1. For a recent review dealing with the effects of partial reinforcement in alleyway studies with rats, see Robbins (1971). The major theoretical positions dealing with partial reinforcement effects are Amsel's "Frustration theory" (Amsel, 1958, 1967) and Capaldi's "Sequential theory" (Capaldi, 1966, 1967, 1971, 1974).

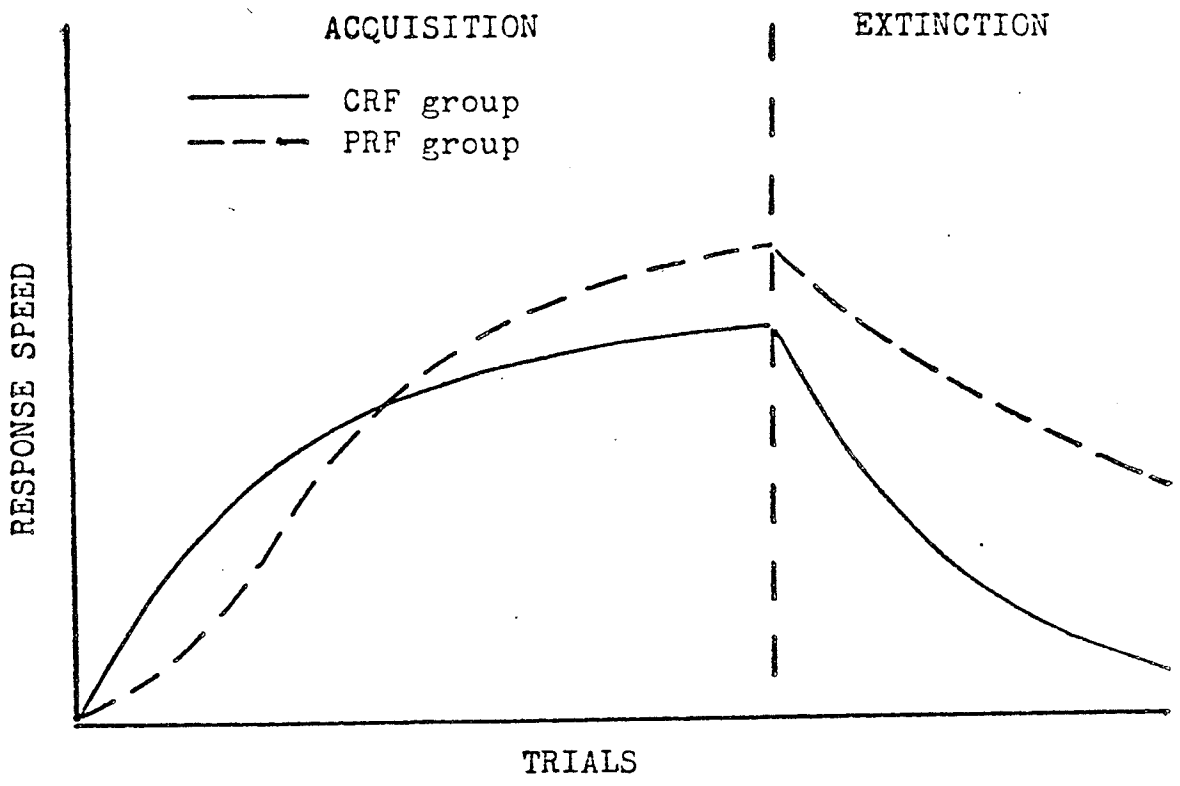

ACQUISITION
EXTINCTION
CRF group
PRF group
RESPONSE SPEED
TRIALS

Fig. 1a

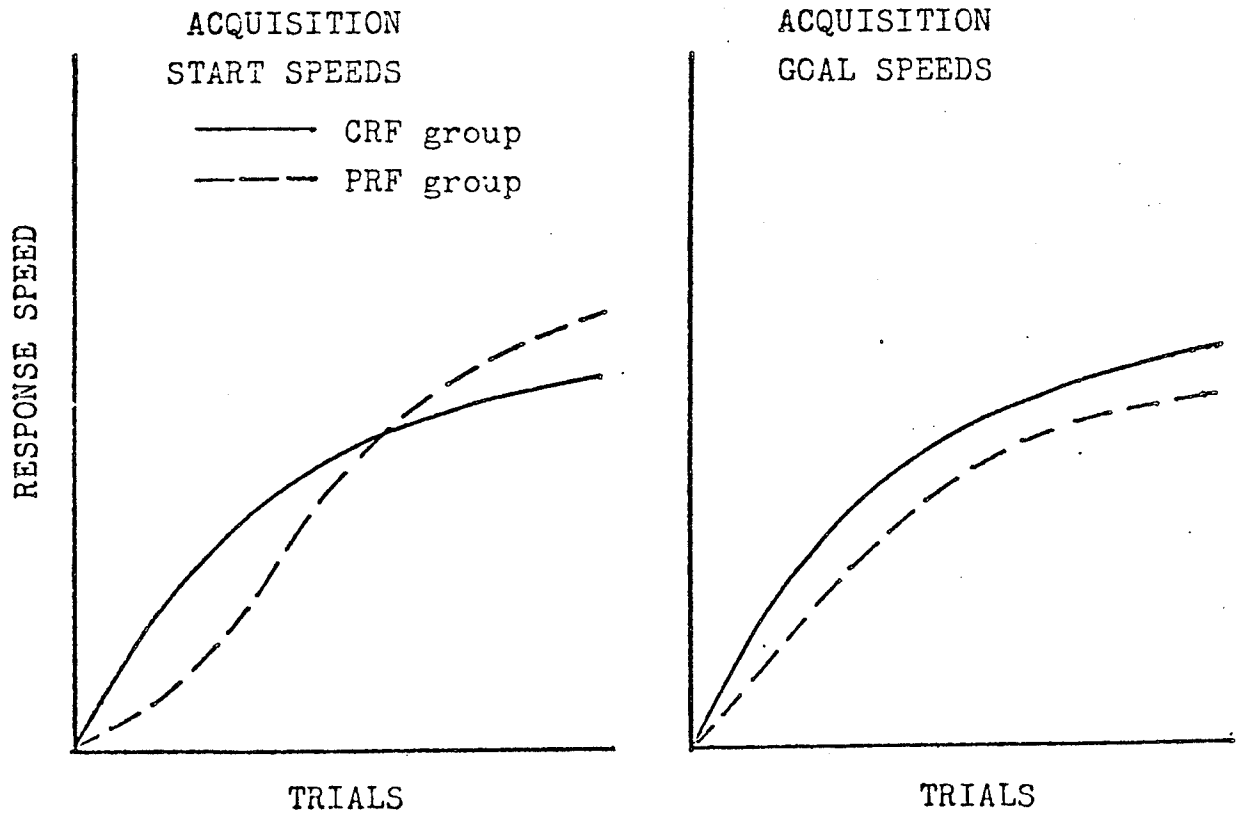

ACQUISITION
START SPEEDS
CRF group
PRF group
RESPONSE SPEED
TRIALS
ACQUISITION
GOAL SPEEDS
TRIALS

Fig. 1b

1.2 Amsel's Frustration Theory

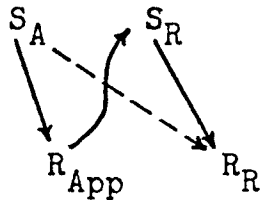
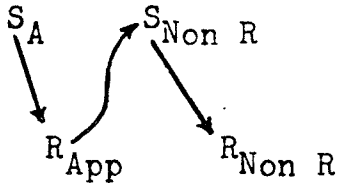
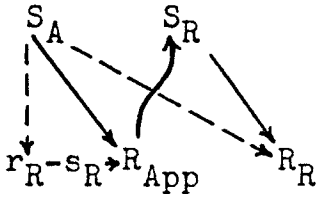
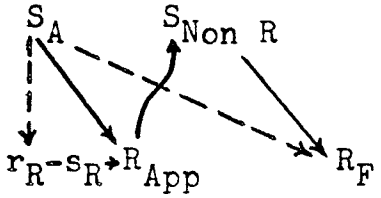
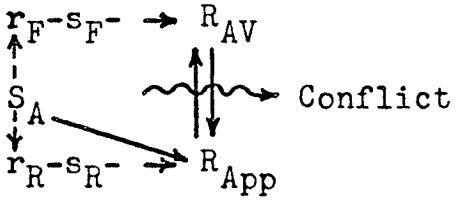
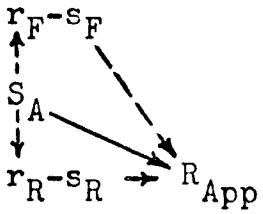
In an attempt to account for the effects of partial reinforcement, Amsel (1958, 1967) conceptually separated reward acquisition into four stages, each involving somewhat different processes. Figure 2 schematically represents the four stages which are conceptualized by Amsel (1958, pp. 108 - 109) as follows:

1. The development of $r_R - S_R$ ($r_g - S_g$) with early rewards: nonreward is ineffective at this stage.
2. With the development of $R_R - S_R$, nonrewards elicit frustration.
3. When nonrewards elicit frustration, the cues previously evoking r_R now also evoke r_F, and these antedating goal responses are temporarily in competition.
4. Since r_R and r_F cannot be elicited separately by differential cues in partial reinforcement, and since partial reinforcement training is such that running to the goal box is reinforced more than avoiding is, S_F becomes associated with the instrumental approach response in the latter stage of partial reinforcement training.

Briefly, Amsel assumes that during the initial stages of partial reinforcement training the subjects build an expectancy for reward or acquire an anticipatory goal response by means of a classical conditioning process (Stage 1). Once the animals have come to expect a reward in the goal box, an emotional response termed frustration (F) is elicited on nonrewarded trials. Initially, the frustration response occurs only in the goal box

PARTIAL REWARD

STAGES OF LEARNING

	REWARD TRIALS	NONREWARD TRIALS
1		
2		
3		
4		

(Stage 2) but eventually, by the same classical conditioning procedure which produced the anticipatory goal responses, the instrumental sequence cues previously evoking r_R now also evoke anticipatory frustration R_F (Stage 3). Since it is assumed that r_F inhibits responding, the antedating goal responses r_R and r_F are temporarily in competition. Both these antedating goal responses occur on both reinforced and nonreinforced trials and thus on reinforced trials the cues associated with r_F (S_F) become counterconditioned to the instrumental running response (R_I) (Stage 4). In extinction, the PRF trained subjects who have been trained to respond (approach) in the presence of the stimuli associated with anticipatory frustration (S_F), approach the goal box more readily, than do CRF trained subjects who are experiencing S_F for the first time. The anticipatory frustration cues in the CRF group, not previously conditioned to R_I, inhibit responding and produce rapid extinction. The major independent variables dealt with in Amsel's theory are "number" variables; such as percentage of reinforcement, number of reinforced trials, number of nonreinforced trials, and number of acquisition trials. The Frustration Theory of the PREE is consistent with much of the data, however, a recent series of studies using a small number of acquisition trials provide difficulty for the theory. Briefly stated, a PREE has been observed following limited training (Amsel, Hug and Surridge, 1968; Capaldi and Deutsch, 1967; Capaldi, Lanier and Godbout, 1968; Capaldi and Waters, 1970; McCain, 1966; McCain and Brown, 1967; Padilla, 1967; Shanab and Birnbaum, 1974; Ziff and Capaldi, 1971).

In an attempt to reconcile the small trial partial reinforcement effect with Frustration Theory, Amsel, Hug and Surridge (1968) modified

Frustration Theory so that one reinforced trial consisting of multiple pellets was assumed to develop anticipatory reward, because the animal is required to make multiple goal approaches in this situation, and thus provide frustration on the following nonreinforced trial. However, the Ziff and Capaldi (1971) study is damaging even to this notion since these authors demonstrated that the small trial PREE is not due to frustration. By eliminating the assumed frustrative emotional responses, and thus S_F, by means of sodium amobarbital, the PREE was still obtained under the drug conditions. In addition, McCain (1966) demonstrated that the PREE following limited acquisition occurred even when N trials were not preceded by R trials, thus preventing the occurrence of frustration as a result of reward expectancy.

Another finding, which has often been taken as support for the Frustration Theory is the "goal box" PRAE. Spence (1956, 1960) has argued that partially reinforced subjects run faster (at asymptote) in an alley because they are running under a "frustrative" drive, but that subjects receiving continuous reinforcement run faster in the goal box because they have developed higher levels of "incentive motivation". This effect, however, has been shown to disappear when discriminative stimulation due to the presence or absence of the reward is eliminated (Robbins, Chait and Weinstock, 1968; Dachowski and Dunlap, 1969). Thus, even the "goal box" PRAE cannot be taken as support for Frustration Theory. The mentioned inconsistencies provide a body of data that presents considerable difficulty for Frustration Theory.

1.3 Capaldi's Sequential Theory

The sequential hypothesis grew out of what is termed the

Hull-Sheffield hypothesis (Hull, 1952; Sheffield, 1949). Hull-Sheffield initially conceived of internal reward and nonreward-related stimuli (S^R and S^N respectively) as traces or after-effects which persisted from one trial to the next. The sequential hypothesis (Capaldi, 1966, 1967, 1971) in common with several other approaches to instrumental learning, places considerable emphasis on internal organismic stimuli which result from the occurrence of reward and nonreward. The unique assumption of Sequential Theory is that different goal box events occasion distinctive memory stimuli that may be modified by succeeding trials or conditioned to instrumental behaviors.

Thus, rather than postulating a trace mechanism, Capaldi suggests that the goal box events S^R and S^N (as well as others) are stored as memories (Capaldi, 1971). In a partial reinforcement situation, this capacity of stimuli related to nonreinforcements (S^N) to control the instrumental behavior (R_I), depends upon the sequence of reinforced and nonreinforced trials during acquisition training. If a nonreinforced trial, occasioning S^N is followed by a reinforced trial, then S^N, which is reinstated, is conditioned to the instrumental reaction (the running response). It can be seen from this brief description that in extinction the PRF group will show more resistance to extinction than a CRF group, due to the fact that S^N will occasion instrumental responding in the PRF group as a result of the conditioning of S^N to the instrumental approach response during acquisition (i.e. an $S^N - R_I$ association has been established). The CRF group, not experiencing S^N during acquisition, will exhibit less resistance to extinction since S^N during extinction does not occasion R_I. From this analysis it can be seen that the PREE will occur only if nonrewarded trials are followed by rewarded trials (N-R transitions) in a partial reinforcement schedule since it is only on

these sequences that S^N is conditioned to R_I.

Using this approach it would be predicted that a PRF group receiving the trial sequence N R R would show greater Rn than a PRF group with the sequence R R N since for the first group S^N would occur in the goal box on trial 1. On trial 2 S^N would be reinstated in the startbox and then be conditioned to R_I as a result of being rewarded on trial 2. For the second group, S^N would never be followed by the instrumental response R_I plus reward, such that S^N would never be conditioned to R_I. Even though both groups would receive the same number of reinforced trials, one group would show greater Rn. It is the N-R transitions which are the important variable. The above prediction is verified by the appropriate investigations (Grosslight, Hall and Murnin, 1953). In contrast, Amsel's Frustration Theory would predict equal Rn for the two groups since both have the same percentage of trials rewarded.

Capaldi (1966) further assumed the S^N is modified by successive N trials. The stimulus consequent of a single N trial or S^N_1, differs either quantitatively, qualitatively, or both from the stimulus consequent of two successive N trials S^N_2. Capaldi proposed that this modification process occurs according to a simple growth function so that $S^N \longrightarrow 0$.

> "Stimulus modification continues either until an R trial occurs, in which event S^N is conditioned to the instrumental response and is replaced by S^R, or until the limit of the modification process is reached, a possible event given greater number of consecutive N trials as in extinction." (Capaldi, 1966, p. 461).

The four major determiners of Rn, according to the present hypothesis, are: a) N length b) number of different N lengths c) number of occurrences of each N length and d) magnitude of reward following N trials. Indeed, Capaldi (1971) argues that his theory is an extinction theory. He

maintains that extinction provides a better measure of learning in acquisition than acquisition measures, since subjects learn different things during acquisition depending upon sequence.

At the heart of the present approach, however, is the rapid internal stimulus changes which are proposed to occur on N or R trials. These are conceptualized as memories or as internal stimuli produced by a mechanism which follows the laws of memory. Memories depend critically upon external stimulation and are less time-dependent, i.e. they are retrieved when the situation which initiated them is re-presented. In contrast to r_g's which are presumably fairly insensitive to prevailing external stimuli, and gradually build up over trials, memories are not independent of external stimuli but depend critically upon external stimulation (i.e. retrieval cues). Capaldi states:

> "In contrast to r_g, memories are not learned; classical conditioning is considered to be far too conservative a mechanism for altering the animals internal stimulus environment." (Capaldi, 1971, p. 119).

Thus, within a learning framework many trials are required for anticipations to develop, but in the memory model they occur from the onset of training. An animal will remember a single nonreward on being placed again in the apparatus, or if he is rewarded, he will retrieve this event on a subsequent trial.

Since the study to be reported was conducted with a 24 hour ITI, it is appropriate to discuss the internal non-reward-related stimuli associated with spaced trials.

Capaldi, Berg and Sparling (1971) attempted to determine to what extent internal non-reward-related stimuli associated with massed trials (S^{MA}) differed from those associated with spaced trials (S^{SP}). The only

hypothesis available at that time was that of Glass, Ison and Thomas (1969) who speculated that the frustrative properties of nonreward declined as the intertrial interval (ITI) increased, i.e. S^{SP} was hypothesized to be less frustrative than S^{MA}. Capaldi et al (1971) found that a shift from a short (3 - 4 min.) ITI in acquisition to a long ITI (24-hr) in extinction produced substantial resistance to extinction in a PRF trained group while the opposite shift in ITI greatly reduced or eliminated the PREE unless extinction occurred under amobarbital. These findings suggested that the stimulus complex S^{MA} is wider or more extensive than S^{SP}, that S^{SP} is contained within S^{MA}, and that the wider portions of S^{MA} are at least in part frustrative. Capaldi et al (1971) concluded that S^{SP} is much less frustrative than S^{MA} and not much more frustrative than the stimulus associated with reinforced trials, i.e. S^{SP} is not very frustrative. Thus the Capaldi et al (1971) study supported the Glass, Ison and Thomas (1969) hypothesis; the aversive or frustrative characteristics of nonreward abate with time.

In addition, Ziff and Capaldi (1971) obtained a PREE following limited acquisition under amobarbital thereby suggesting that S^{MA} consists in part of nonfrustrative stimulus components. In addition, Capaldi and Waters' (1970) observation of a PREE following N-R transitions but no PREE following R-N transitions, also support this notion. For greater numbers of acquisition trials, nonreward is assumed to become frustrating (Amsel, 1958; Ziff and Capaldi, 1971) and contains both frustrative and nonfrustrative stimulus components (Capaldi and Sparling, 1971).

Capaldi et al (1971) also point out that there is sufficient data to suggest that S^{SP} is substantially frustrative at a 24-hr ITI (Wagner, 1961). By assuming that the wider portions of S^{MA} (i.e. $S^{MA} - S^{SP}$) consist of both

frustrative and nonfrustrative stimulus components which fade as ITI increases, these authors tried to reconcile the previous findings that S^{SP} was substantially frustrative at a 24-hr ITI with their findings that S^{SP} was much less frustrative than S^{MA}. It was suggested that perhaps unique nonreward-related stimuli associated with a given ITI might comprise part of the S^{SP} complex but would not be contained in the S^{MA} complex. Whether or not such unique stimuli would be frustrative or nonfrustrative, could not be determined from the data reported by Capaldi et al (1971). Finally, it was suggested that, all factors being equal, nonreward may be less frustrating at long ITI's than at short ITI's.

In his earlier writings, Capaldi (1966, 1967) emphasized a hard principle of reinforcement. Recently (Capaldi, 1974) this reinforcement assumption of the sequential model has been replaced by that of <u>reinforcement level</u>. Capaldi suggests that strength of conditioning is always determined by the relationship between the reward that is expected and the reward that is obtained. This relationship determines reinforcement level. Capaldi states:

> "...if obtained reward is greater than expected reward, then available stimuli will acquire greater capacity than otherwise to elicit the reaction. However, if obtained reward is smaller than expected reward, then available stimuli will suffer a decrease in their capacity to elicit the reaction (unconditioning)" (Capaldi, 1974, p. 958).

Thus, within the framework of this revised hypothesis, one would expect eventual weak conditioning for a CRF group because the value of expected reward eventually becomes identical to the value of obtained reward. For a PRF group, however, the value of the expected reward stabilizes between two values of obtained reward, that available on rewarded trials and that

available on nonrewarded trials. There is strong conditioning on rewarded trials and unconditioning on nonrewarded trials. The above reinforcement level hypothesis was introduced to explain interactive schedule effects found in extinction, negative contrast and positive contrast (Capaldi, 1974).

1.5 Some Behavioral effects of Sodium Amobarbital

Sodium amobarbital is a barbiturate classified as a short or intermediate acting sedative-hypnotic (Goth, 1970). The barbiturates produce all degrees of depression of the central nervous system, ranging from mild sedation to coma. When barbiturates are taken repeatedly at short intervals, tolerance (both metabolic and cellular) develops, and may contribute to the decreased duration and intensity of the response to a given dose (Goodman and Gilman, 1970).

Miller (1961) described some of the earlier studies of the effects of sodium amobarbital on fear and conflict behaviors. Grinker and Spiegel (1945a, b) demonstrated that sodium amobarbital is useful in the therapy of combat neuroses and some civilian disorders in which fear and conflict play a prominent role. Bailey and Miller (1952) found a fear reducing effect of the drug in cats. Miller and Barry (1960) reported a failure to suppress responding in an automated conflict situation, by rats injected with sodium amobarbital. Control rats readily showed response suppression in this situation. Miller (1961) demonstrated that the "fear-reducing" effects of this drug were not produced indirectly by drug induced changes in the stimulus situation, nor was the effect due to a greater effect of the drug on the more recently established habit or response. In addition, no generalization from the drugged to normal state seemed to occur for sodium amobarbital.

On the basis of dose-response effects of amobarbital on speed to approach food, and to avoid or escape shock, Miller (1964) selected a dose that seemed to be maximally effective in conflict situations. This dose-response curve is depicted in Figure 3. Miller used the optimum dose of 20 mg/kg body weight in most of his other studies. These demonstrated a decrease in conditioned suppression for drug injected animals in a conditioned suppression paradigm and increased approach responding in the telescope alley test. In addition, fear conditioned rats learned to press a bar to inject themselves with amobarbital and extinguished this response after the fear-eliciting stimuli were turned off (Miller, 1964).

Kamano, Powell, Martin and Ogle (1967) trained rats to avoid shock in a shuttle box. Immediate extinction of avoidance responses followed injection of 20 mg/kg amobarbital. The authors attributed this to the amobarbital blocking the expression of the conditioned avoidance response (CAR). They also found increased freezing behavior occurring with the precipitous extinction of the CAR, thus excluding a fear-reduction explanation for the effectiveness of amobarbital. Kamano (1973) gave rats unsignalled shuttle box avoidance training and separately Pavlovian fear conditioning. This was followed by avoidance extinction sessions under amobarbital and Pavlovian fear conditioning without drug on alternate days. The CS+ was then presented in two post-avoidance extinction sessions to determine the effect of the CS+ on the avoidance response extinguished under amobarbital. It was found that amobarbital facilitated extinction of unsignalled shuttle box avoidance, but failed to block the reaction to the CS+ in the same setting. This finding suggested that rather than having a global effect, amobarbital acts selectively on processes mediating expression of avoidance behavior.

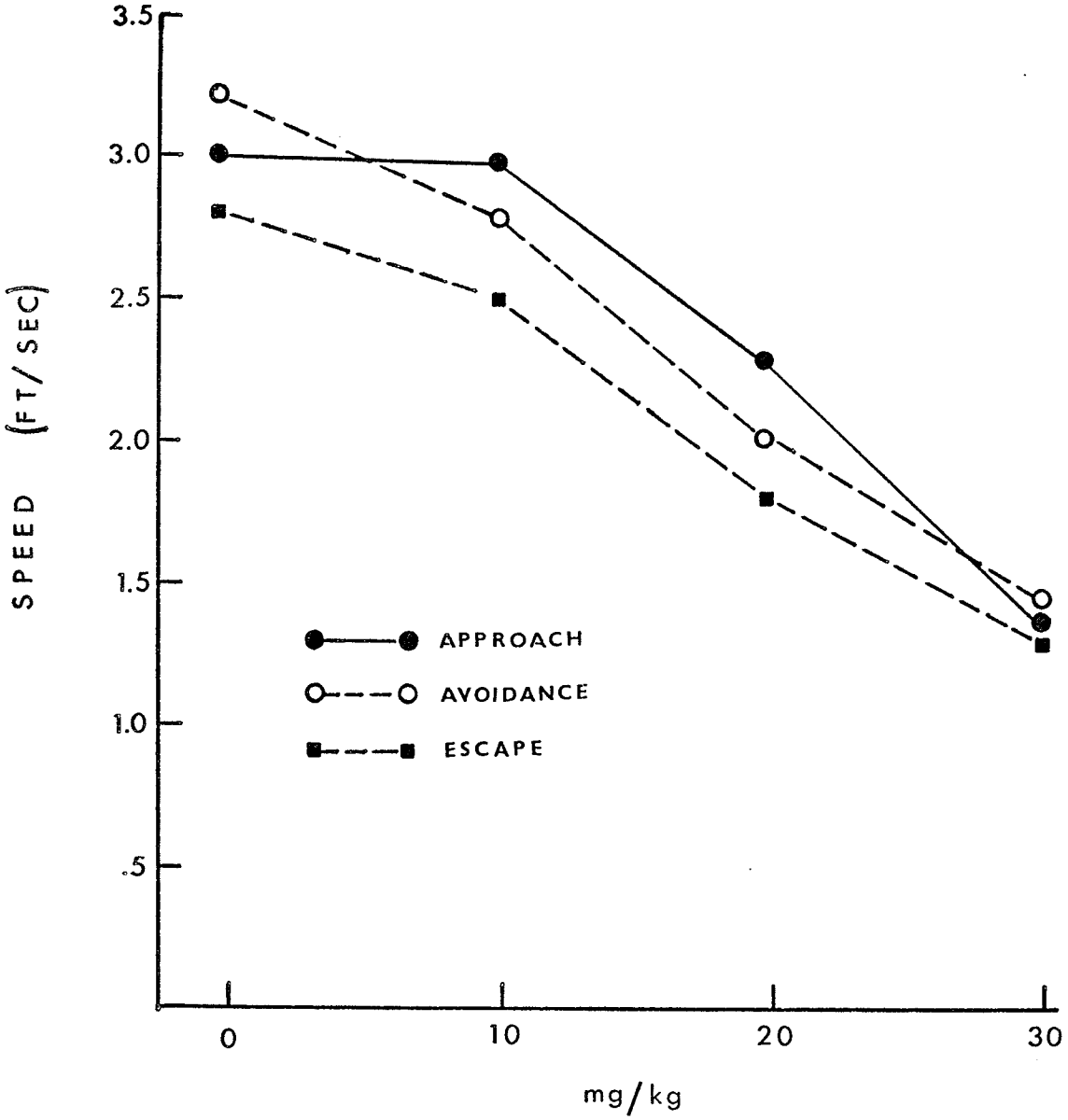


Fig. 3. The seperate dose-response effects of amobarbital on the speed to approach food, and to avoid or escape shock. (from Miller, 1964)

An experiment on the effects of sodium amobarbital on the extinction of a positively reinforced response was conducted in a 2 x 2 x 2 design (Barry, Wagner and Miller, 1962). Two groups of rats were given 40 training trials in a six-foot runway for continuous food reinforcement (CRF). No drug effect on terminal running speeds was apparent. Two groups were then subdivided into two subgroups. One subgroup was given nonreinforced trials following injection of amobarbital and the other group following an injection of isotonic saline. The drug injected groups extinguished more slowly than the saline injected groups. A further subdivision of the groups was made with one continuing on extinction with the drug and the other without. The results were the same as those for the second stage, drug injected groups extinguished more slowly. These results suggested that sodium amobarbital may reduce the emotional effects of nonreward frustration in the same way it reduced "fear" established by electric shock.

Ison and Northman (1968) and Rosen, Glass and Ison (1967) found that sodium amobarbital eliminated the rapid performance decrement usually found after a reduction in reward, but found no effect when reward magnitude was increased. Similarly, Ridgers and Gray (1973) found that the operant depression effect was significantly reduced by amobarbital, but the operant elation effect was unaffected. These results were taken as support for the view that amobarbital impairs behavioral responses to departures from expected magnitude of reward only when the departure is in an unfavourable direction.

1.6 Sodium Amobarbital and the Partial Reinforcement Effect

Following the demonstration that sodium amobarbital attenuated performance decrements in extinction, Stretch, Houston and Jenkins (1964) and Gray (1967) reported a reduced PREE under amobarbital. Both studies

reported only overall runway times and both used an incomplete factorial design in which rats were either trained under saline and extinguished under amobarbital (Stretch et al, 1964) or trained under amobarbital and extinguished under saline (Gray, 1967). Wagner (1963) observed that amobarbital eliminated the PRAE (Goodrich, 1959) in which PRF groups run faster in the initial sections of a runway than CRF groups under conditions of appetitive reward.

The theoretical analysis of the attenuation of the PREE by amobarbital, was based on frustration theory. It was assumed that the drug decreased anticipatory frustration (Ison, 1968; Miller, 1964). This decrease in anticipatory frustration decreased the PREE because this effect depends on the presence of anticipatory frustration responses in both acquisition and extinction. The elimination of the PRAE (Wagner, 1963) could also be explained in terms of reduction in anticipatory frustration as a result of drug injection.

Ison and Pennes (1969) gave rats 40 acquisition trials under CRF or PRF (50%) reinforcement followed by 28 extinction trials. Within each reinforcement condition one group received injections of saline prior to the daily block of four acquisition trials, and one group received injections of amobarbital (20 mg/kg). In extinction, these groups were subdivided such that half received extinction under saline and half under amobarbital. The usual attenuation of the PREE was observed for the group receiving amobarbital during acquisition and saline in extinction. However, the group receiving amobarbital in both acquisition and extinction did <u>not</u> show an attenuation of the PREE, as would be predicted if amobarbital decreased anticipatory frustration. Ison and Pennes (1969) suggested that instead of decreasing anticipatory frustration, amobarbital suppressed (dissociated) the normal

responses associated with $r_f - s_f$. This hypothesis was termed the suppression hypothesis.

Gray (1967) in addition to demonstrating the attenuation of the PREE in animals trained with a PRF acquisition schedule and receiving amobarbital injections, tested the effect of amobarbital in Amsel's double runway frustration situation. In this situation one usually finds an increased speed of running in a second runway after nonreinforced trials in the goal box of the first runway, relative to speeds following reinforced trials. No effect of amobarbital on Amsel's double runway frustration effect could be established. Gray (1967) attempted to account for this finding on the basis of strain differences and indicated the subsequent pilot studies did find an effect of amobarbital on the Amsel double runway frustration effect. In addition, Gray suggested that unconditioned frustration (primary) is more resistant to the effects of the drug (though not entirely resistant). Subsequent studies by Gray (1969) did not support the contention that primary frustration was more resistant to the effects of amobarbital. The author concluded that both the unconditioned frustration and conditioned (anticipatory) frustration were equally affected by sodium amobarbital.

To test the effects of amobarbital on the small trial PREE, Ziff and Capaldi (1971) gave rats limited acquisition training (3 or 6 trials) followed by 12 extinction trials. They found that groups receiving amobarbital during acquisition ran faster than saline controls in acquisition but slower in extinction (no injections were given to any group in extinction). A PREE was obtained following amobarbital injections during acquisition which was as large as that obtained for groups receiving saline injections. The authors pointed out that the amobarbital was indeed reducing emotionality in their

study as demonstrated by faster acquisition speeds for amobarbital injected animals (apparatus and general situational cues evoke less fear and thus faster running for amobarbital subjects) and a marked decrease in running speed by the amobarbital group when shifted to no drug conditions in extinction (no habituation to apparatus cues would be possible in this group during acquisition since no emotional responses occurred). Ziff and Capaldi (1971) concluded that since amobarbital reduced emotionality, the small trial PREE requires a conditioning interpretation which assumes that the stimuli conditioned are cognitive or neutral rather than frustrative or emotional.

Capaldi and Sparling (1971) trained two groups of rats on a PRF schedule which contained either N - R transitions (nonreward followed by reward) or R - N transitions. One group received injections of amobarbital on days when N - R transitions occurred and saline when R - N transitions occurred. The other group was assigned to the opposite schedule, amobarbital on R - N days and saline on N - R days. The results indicated that amobarbital only affected the group receiving the drug on N - R transitions in terms of the PREE. The R - N group demonstrated the PREE under saline extinction, the N - R group did not. Both groups showed the PREE under amobarbital extinction. This study emphasises the importance of trial sequence and indicates that it is emotional (frustrative) cues occurring on the N trial which are being conditioned to R_I on the subsequent R trial which produces the greater Rn.

In an attempt to get at the physiological basis of the partial reinforcement effect, Gray (1970) and Gray and Ball (1970) investigated the relationship between hippocampal theta rhythm, the partial reinforcement effect and amobarbital action. It was found that the hippocampal theta rhythm in

freely moving rats showed frequency-specific correlations with behavior and reinforcement contingencies. A frequency of approximately 7.5 - 8.5 Hz was seen during exploration of an alleyway and in response to nonreward. When running along an alley toward a known reward, the frequency rose to 8.5 - 10 Hz and there was also a marked increase in amplitude. When consuming reward, the frequency fell again to about 6 - 7.5 Hz. Amobarbital was found to selectively raise the threshold for septal driving of the hippocampal theta rhythm at the frequency of 7.7 Hz, the same frequency occurring in response to nonreward. In addition, Gray (1970) found that septal driving of hippocampal theta at 7.7 Hz during acquisition or extinction had <u>opposite</u> effects on behavior to amobarbital; applied during extinction it facilitated extinction, applied during acquisition it created a "pseudo partial reinforcement extinction effect".

Gray (1969) also reported several interesting correlations between hippocampal theta frequency at the end of acquisition and Rn. Theta frequency was found to be higher on nonrewarded trials and during extinction than on rewarded trials. Additionally it was found that the higher the theta frequency during extinction, the more rapidly the subject extinguished ($r = -.64$) and conversely, the higher the theta frequency at the end of acquisition the more slowly the subject extinguished ($r = +.69$). Gray suggested that the way in which partial reinforcement training increases resistance to extinction is by raising the theta frequency on rewarded trials from 6 - 7 Hz (which the CRF animals displayed) to something closer to the 7.5 - 8.5 Hz which occurred normally during extinction. The author further suggested that:

> "...theta frequency behaves very much like a physiological analogue of Amsel's (1962) construct of "frustration". If it is high in extinction, the speed of extinction is great; if it is raised at the end of acquisition, whether by septal stimulation, or by the occurrence of nonrewarded trials, or as a result of individual differences, the speed of extinction is retarded." (Gray, 1970, p. 476).

It was further proposed that there is a septo-hippocampal system which mediates the behavioral effects of frustrative nonreward and punishment and that amobarbital acts on behavior by antagonizing this system.

1.7 Statement of the Purpose of the Study

The finding that the PREE under saline extinction does not occur when subjects receive amobarbital injections prior to N - R transitions (Capaldi and Sparling, 1971) prompted the present study. There has been no attempt to answer the question of whether sodium amobarbital directly attenuates emotional (frustrative) responses on nonreinforced trials or whether the drug prevents the conditioning of the emotional cues (memories) to the instrumental response on reinforced trials. Secondly, the degree to which internal nonreward-related stimuli associated with spaced (24-hr ITI) training are frustrative has not been adequately determined. This study was designed to answer these questions by factorially manipulating sodium amobarbital on the reinforced and/or nonreinforced trials, of acquisition under widely spaced training and testing conditions (24-hr ITI's).

In addition, the study was designed to test certain aspects of frustration theory. For example, if amobarbital is given on N trials during acquisition, one would expect rapid extinction since primary frustration would be blocked by the drug. If, however, the drug also blocks anticipatory frustration, then a group receiving amobarbital on R trials during

acquisition should show fairly rapid extinction as well, due to the fact that r_F would not occur (or occur weakly) and thus not become conditioned to the instrumental response.

In addition to the main hypotheses, the present study was designed to provide information on the following:

a) to replicate the finding of the PREE at an ITI of 24-hrs.

b) to replicate the finding of the PRAE at an ITI of 24-hrs.

c) to replicate the finding that amobarbital injections during acquisition eliminate the PRAE.

d) to determine if acquisition differences exist between a group receiving amobarbital on N trials and a group receiving amobarbital on R trials.

e) to determine if the main extinction effects of amobarbital given in acquisition occur on the N trials, R trials or on both N and R trials during acquisition training.

Chapter 2 Method

2.1 Subjects

Forty experimentally naive male rats (Ratus norvegicus albinus) of the Sprague-Dawley strain, were used in the experiment. The rats were from 210 to 270 gms in weight at the start of the experiment and were purchased from North American Laboratory Supply Ltd. in Manitoba. The animals were housed individually in a colony room kept on a 8:16 hr light-dark cycle and 72° F. The subjects were assigned to one of five groups of 8 subjects each. Each group was balanced with the others for weights of the animals.

2.2 Apparatus

The apparatus consisted of a single straight alley runway painted black. The dimensions were 15 cm high x 10 cm wide x 183 cm long. The sides and floor were made of wood and the alley was covered by a clear plexiglass sheet attached to one of the sides by hinges. The runway was divided into a 30 cm start section, a 122 cm run section, and a 30 cm goal section; all sections being separated by aluminum guillotine doors. A glass coaster painted bright yellow and placed in the middle of the far end of the goalbox served as a foodcup. The foodcup contained the 15 food pellets on all rewarded trials during acquisition training and the block of CRF trials.

The movement of the animals down the runway was monitored by three 0.01 sec Standard timers. By raising the first guillotine door a microswitch (located at the top of the start box door) was closed and started the first timer. The interruption of a photocell beam located 10 cm into the runway stopped the first timer, which registered the start time, and started the second timer. Interruption of a second photocell beam located 10 cm in

front of the goalbox (102 cm from the first photocell) stopped the second timer, which registered the run time, and started the third timer. The last timer was stopped when a third photocell beam (7.5 cm into the goal box) was interrupted, and this timer registered the goal time measure.

Start, run and goal speed measures were calculated by reciprocating the start, run and goal times (in sec) respectively. Total speeds were obtained by summing the start, run and goal times and reciprocating this sum value. The guillotine doors were manually operated and the timers reset after each trial.

2.3 Drug Conditions

Sterile sodium amobarbital purchased from Eli Lilly and Company (Canada) Ltd., was dissolved in boiled, physiological (isotonic) saline solution. The saline was boiled for 15 min before use, in order to "boil off" as much CO_2 from the saline solution as possible. This boiling off of the CO_2 was done in order to decrease hydrolyzation. One ampoule of 250 mg of sodium amobarbital was dissolved in 25 cc of warm isotonic saline solution, giving a concentration of 10 mg/ml of solution. At this concentration the sodium amobarbital was injected in a volume of 2.0 ml/kg body weight for a dosage of 20 mg/kg. Equal volumes of isotonic saline solution served as the vehicle control. Injections were given intraperitoneally (i.p.) at the lower part of the abdomen. Solutions of the sodium amobarbital were prepared every second day, one-half of the quantity being used immediately after preparation, the other half being stored overnight at approximately 8^{o} C.

2.4 Preliminary Training Procedure

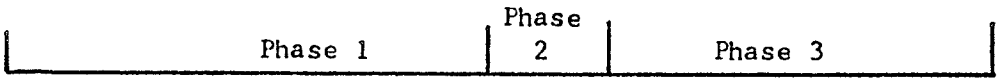
For 10 days after arrival in the laboratory, the animals were allowed free access to food and water. On Day 1 of the training procedure

the animals were weighed, assigned to one of the five groups, tail marked, and placed on a 23 hr food deprivation schedule (Days 1 - 11). During food deprivation conditions the subjects were fed a daily ration of 12 gm of Purina Laboratory Chow at 24 hr intervals and kept on an ad libitum water schedule. By Day 15 the subjects had reached approximately 85% of their initial body weight.

Starting on Day 4 the rats were handled for 1 min/day in squads of two for 3 days. From Day 7 - 13 the animals were handled for 2 min/day. On Days 12 and 13, the rats were given experience eating 0.045 gm Noyes food pellets in the home cage (approximately 20 pellets each day). On Days 14 and 15, the animals each received two goalbox placement trials per day in which they were placed directly into the enclosed goal area and allowed to consume 6 pellets (12 pellets/day). On these days, as on all subsequent days, the animals were fed 30 min after being returned to their home cages.

2.5 Experimental Training Procedure

The experimental procedure consisted of three phases: Phase 1 - 20 trials of acquisition training; Phase 2 - 5 trials of continuous reinforcement training; Phase 3 - 16 trials of extinction (trials were never reinforced). The temporal paradigm of the experiment may be diagrammed as follows:



On Day 16 spaced (24 hr inter-trial-interval (ITI.) acquisition training (Phase 1) began. One group (CRF) were trained on a 100% reinforcement schedule. The other four groups (NRS, NRA, NS, and NA) were trained on

a 50% partial reinforcement schedule. The twenty trials of partial reinforcement acquisition training were given in the following schedule sequence:

R R N R N N R N N R N N R N R R N N R R

This sequence of reinforced and nonreinforced trials provided for six transitions from an N trial to an R trial (N-R transition) and six transitions from an R trial to an N trial (R-N transition). There were a total of 10 N trials and 10 R trials.

The schedule of drug injections for the four partial reinforcement (PRF) groups and the continuous reinforcement (CRF) group, is shown in Table 1. The CRF group was divided into 2 groups with one group receiving saline injections on trials corresponding to N trials in the PRF schedule and the other group receiving amobarbital injections. On the trials corresponding to R trials in the PRF schedule the reverse injection schedule was in effect.

Starting on Day 36 (Phase 2), all groups received five trials of continuous reinforcement, again at spaced intervals of 24 hr. During this block of CRF trials none of the animals were injected.

Starting on Day 41 (Phase 3) all groups received 16 trials of nonreward at 24 hr ITI. Again none of the rats were injected during this extinction phase.

The daily running procedure consisted of injecting the appropriate animals with either sodium amobarbital (20 mg/kg) or an equal volume of saline, 15 min prior to being placed in the experimental apparatus. The animals were transported in squads of 5 (one animal from each of the five groups) to the experimental room in a carrying cage (painted black). The running order of the squads was the same each day, however the intra-squad

Table 1. Schedule of drug injections for the five groups used in the experiment.

GROUP	REINFORCEMENT SCHEDULE	TYPE OF INJECTION ON R TRIALS	TYPE OF INJECTION ON N TRIALS
CRF *	CRF (100%)	½ gr. saline ½ gr. amobarbital	½ gr. amobarbital ½ gr. saline
NRS	PRF (50%)	saline	saline
NRA	PRF (50%)	amobarbital	amobarbital
NS	PRF (50%)	amobarbital	saline
NA	PRF (50%)	saline	amobarbital

* The injection schedule for the CRF group corresponded to the R and N trials for the PRF schedule even though these animals experienced only R trials.

order of running was randomly varied each day (i.e. the order in which the groups were run was randomly varied) and was consistent for all squads on a given day.

The animals were placed in the start box, both guillotine doors were raised, and the animal was allowed to traverse the length of the runway. As soon as the rat was in the goalbox, the guillotine doors were lowered to prevent the rat from leaving the goalbox. On reinforced trials the animal was removed from the goalbox as soon as the 15 Noyes pellets had been consumed. On nonreinforced trials the animal was detained in the goalbox for a 30 sec time period. If an animal took longer than 60 sec to traverse any part of the runway, then a score of 60 sec was assigned to that section as well as to all remaining sections for that trial. In this case the rat was removed from the runway and placed directly into the goalbox.

The animals were weighed every four days during the acquisition training period (Phase 1) in order to calculate new injection volumes for each subject.

2.6 Statistical Analysis of the data

The results were analyzed using a mixed design repeated measures analysis of variance. The following analyses were completed for all four dependent measures (start, run, goal and total speeds):

Acquisition results -

1) Overall acquisition performance;
   groups (5) x trials (20)
2) Test for the Partial Reinforcement Acquisition Effect (PRAE); groups (3) x blocks of 2 trials (10)

3) Performance on N and R trials for groups NS and NA; groups (2) x reinforced trials (10) x nonreinforced trials (10)

CRF Block results -

1) Overall performance (last day of acquisition plus CRF block); groups (5) x trials (6)

2) Effect of drug administration during acquisition on performance in the CRF block;
groups receiving drug on acquisition R trials (2) x groups receiving drug on acquisition N trials (2) x trials (6)

Extinction results -

1) Overall extinction effect (last 2 days of CRF block plus extinction trials);
groups (5) x blocks of 3 trials (6)

2) Effect of drug administration during acquisition on extinction performance;
groups receiving drug on acquisition R trials (2) x groups receiving drug on acquisition N trials (2) x blocks of 3 trials (6)

In addition, terminal acquisition performance (last 2 trials of the CRF block plus the 1st extinction trial) was analyzed with a single-factor analysis of variance to test for terminal acquisition performance differences.

Post-hoc comparisons were made with the Scheffe method for comparisons among class means (Scheffe, 1959).

Chapter 3 Results

3.1 Mortality and Body Weights

One subject from group NRA died during Phase 1 of the study and one subject from group NS was sacrificed during Phase 2 because of middle ear disease. Subsequently, the data of one animal from each of the remaining groups (CRF, NRS, NA) were randomly discarded in order to retain symmetry for reasons of statistical analyses. Thus the size of each group was seven subjects (N = 7/ group).

A repeated measures design analysis of variance (5 groups x 6 days) on body weights of the animals during the pretraining and acquisition training periods, indicated no significant group effect (F (4,30) = 0.68), a significant days effect (F (5,20) = 145.1, $p < .001$) (demonstrating a reduction in body weights following the start of food deprivation) and no significant group x days interaction (F (20,150) = 1.18). The failure to find group differences indicates that subsequent performance differences could not be attributed to differences in body weights.

3.2 Acquisition (Phase 1)

A 5 (groups) x 20 (trials) repeated measures analysis of variance on the twenty acquisition trials indicated a significant group effect only for the run and goal measures (F (4,30) = 2.702, $p < .05$; F (4,30) = 3.901, $p < .025$, respectively) (Table 2). The Scheffe test indicated no significant differences between any of the group means for the run measure and a significant difference only between groups CRF and NS ($p < .05$) for the goal measure. A significant trial main effect was found for each of the four dependent measures (F (19,570) =

Table 2. Analysis of Variance on the twenty acquisition trials for the four speed measures:

START

Source	df	MS	F	p
Groups	4	45246208.	1.409	n.s.
Error 1	30	32119408.		
Trials	19	38751232.	15.702	<.001
Group x Trials	76	4473505.	1.813	<.005
Error 2	570	2467841.		

RUN

Source	df	MS	F	p
Groups	4	918267.5	2.702	<.05
Error 1	30	339884.7		
Trials	19	926470.7	47.670	<.001
Groups x Trials	76	55245.5	2.843	<.001
Error 2	570	19434.9		

Table 2. continued....

GOAL

Source	df	MS	F	p
Groups	4	6637652.	3.901	<.025
Error 1	30	1701459.		
Trials	19	6174677.	33.496	<.001
Group x Trials	76	571349.	3.099	<.001
Error 2	570	184339.		

TOTAL

Source	df	MS	F	p
Groups	4	399278.3	2.450	n.s.
Error 1	30	162947.7		
Trials	19	380661.1	40.604	<.001
Group x Trials	76	28953.5	3.088	<.001
Error 2	570	9374.9		

15.702, 47.670, 33.496, 40.604, $p < .001$ for start, run, goal and total times respectively). Inspection of Figure 4 reveals that the significant trial effect represents an increase in performance over the twenty trials, i.e. acquisition of the instrumental response occurred. The group by trial interaction was significant for all measures (start, F (76,570) = 1.813, $p < .005$) run, goal and total F (76,570) = 2.843, 3.099, 3.088, $p < .001$, respectively). The significant interactions indicated that the various groups were acquiring the running response at different rates. Inspection of Figure 4 indicates that groups CRF and NRS had the fastest terminal acquisition performance, group NRA was intermediate and groups NS and NA were the lowest in performance.

In addition, Figure 4 indicates that groups NS and NA exhibited a "pattern" discrimination from about trials 3 to 14, however the pattern for group NS was inverse (mirror image) to that of group NA. That is, group NS showed better performance on R trials relative to N trials whereas group NA showed better performance on the N trials relative to R trials. Additionally, when a sequence of 2 consecutive N trials occurred, group NS showed a further decrease in performance from the first N trial to the second N trial. Group NA, on the other hand, showed increased performance from the first to the second N trial (trials 5 to 6 and 11 to 12).

From trials 14 to 20 both groups (NS and NA) exhibited the same pattern, i.e. groups NS and NA showed performance decrements on N trials and performance increments on R trials.

In order to better ascertain the effects of the drug injections on N and R trials for groups NS and NA, a repeated measures analysis of variance (2 groups x 2 reinforcement conditions x 10 trials) was performed on the R and N trials of acquisition for these two groups (Table 3). No

Fig. 4 Mean total speeds as a function of acquisition trials (Phase 1) and CRF trials (Phase 2) for all five groups.

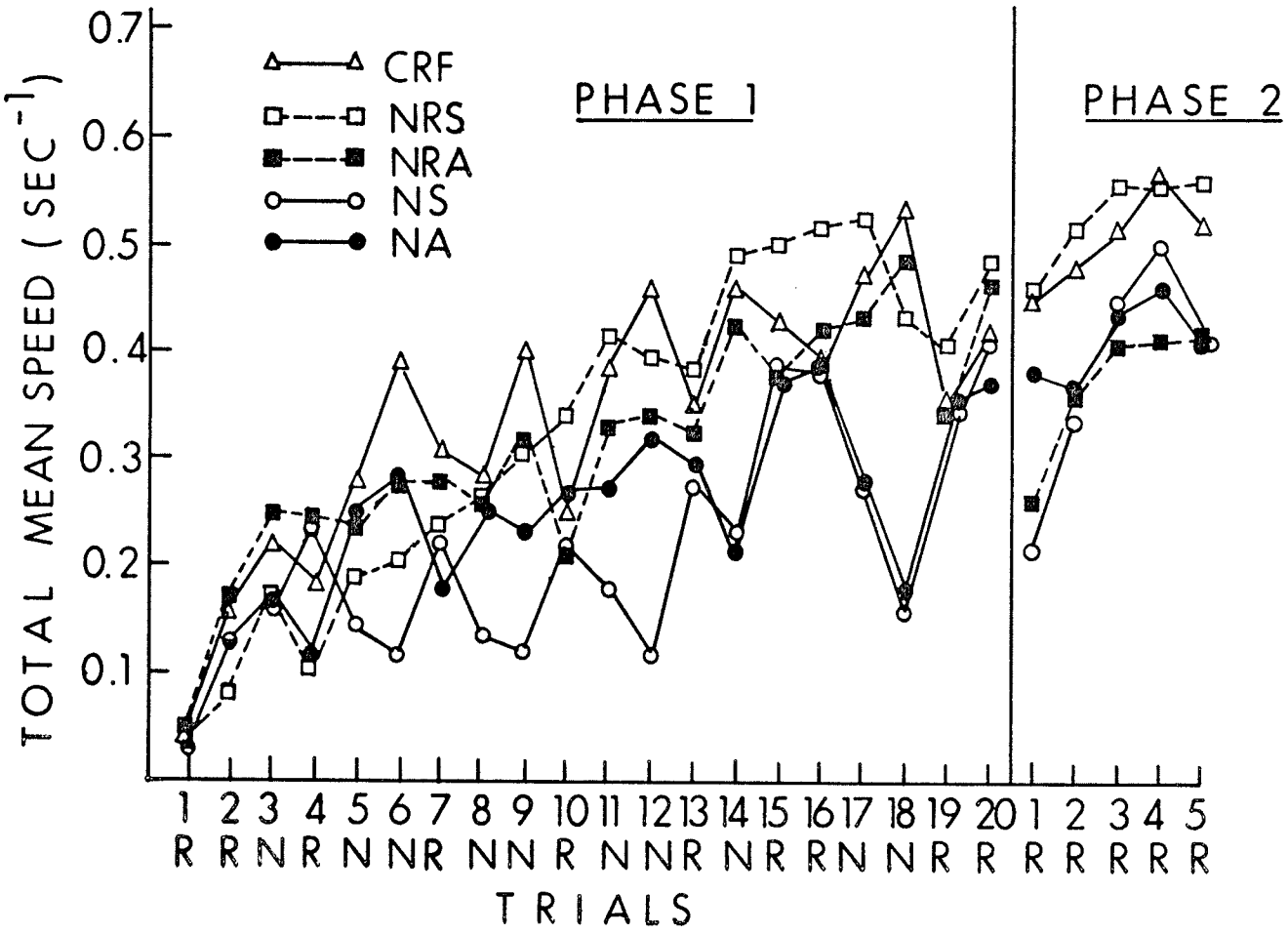

TOTAL MEAN SPEED (SEC^{-1})
0.7
0.6
0.5
0.4
0.3
0.2
0.1
CRF
NRS
NRA
NS
NA
PHASE 1
PHASE 2
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 1 2 3 4 5
R R N R N N R N N R N N R N R R N N R R 1 R R R R R
TRIALS

Table 3. Analysis of Variance on the reinforced and nonreinforced trials during acquisition for groups NS and NA.

Start

Source	df	MS	F	p
Group	1	4242332.	0.389	n.s.
Error 1	12	10899021.		
Reinforcement Cond. (RFM)	1	12076855.	6.880	$<.025$
Group x RFM	1	18232976.	10.387	$<.01$
Error 2	12	1755398.		
Trials	9	13317751.	10.063	$<.001$
Groups x Trials	9	1164291.	0.880	n.s.
Error 3	108	1323385.		
RFM x Trials	9	8180133.	6.338	$<.001$
Groups x RFM x Trials	9	2804213.	2.173	$<.05$
Error 4	108	1290558.		

RUN

Source	df	MS	F	p
Group	1	86576.4	0.377	n.s.
Error 1	12	229429.5		
Reinforcement Cond. (RFM)	1	321866.6	10.628	$<.01$
Group x RFM	1	82780.7	2.733	n.s.
Error 2	12	30284.5		
Trials	9	400690.1	20.131	$<.001$
Group x Trials	9	28279.2	1.421	n.s.
Error 3	108	19903.8		
RFM x Trials	9	164845.6	9.112	$<.001$
Group x RFM x Trials	9	39302.2	2.172	$<.05$
Error 4	108	18092.0		

Table 3. continued.....

GOAL

Source	df	MS	F	p
Group	1	1720558.	0.873	n.s.
Error 1	12	1970340.		
Reinforcement Cond. (RFM)	1	2618098.	5.192	$<.05$
Group x RFM	1	4563335.	9.049	$<.025$
Error 2	12	504298.		
Trials	9	2945708.	16.040	$<.001$
Group x Trials	9	442137.	2.408	$<.025$
Error 3	108	183648.		
RFM x Trials	9	1536407.	7.417	$<.001$
Group x RFM x Trials	9	187226.	0.904	n.s.
Error 4	108	207151.		

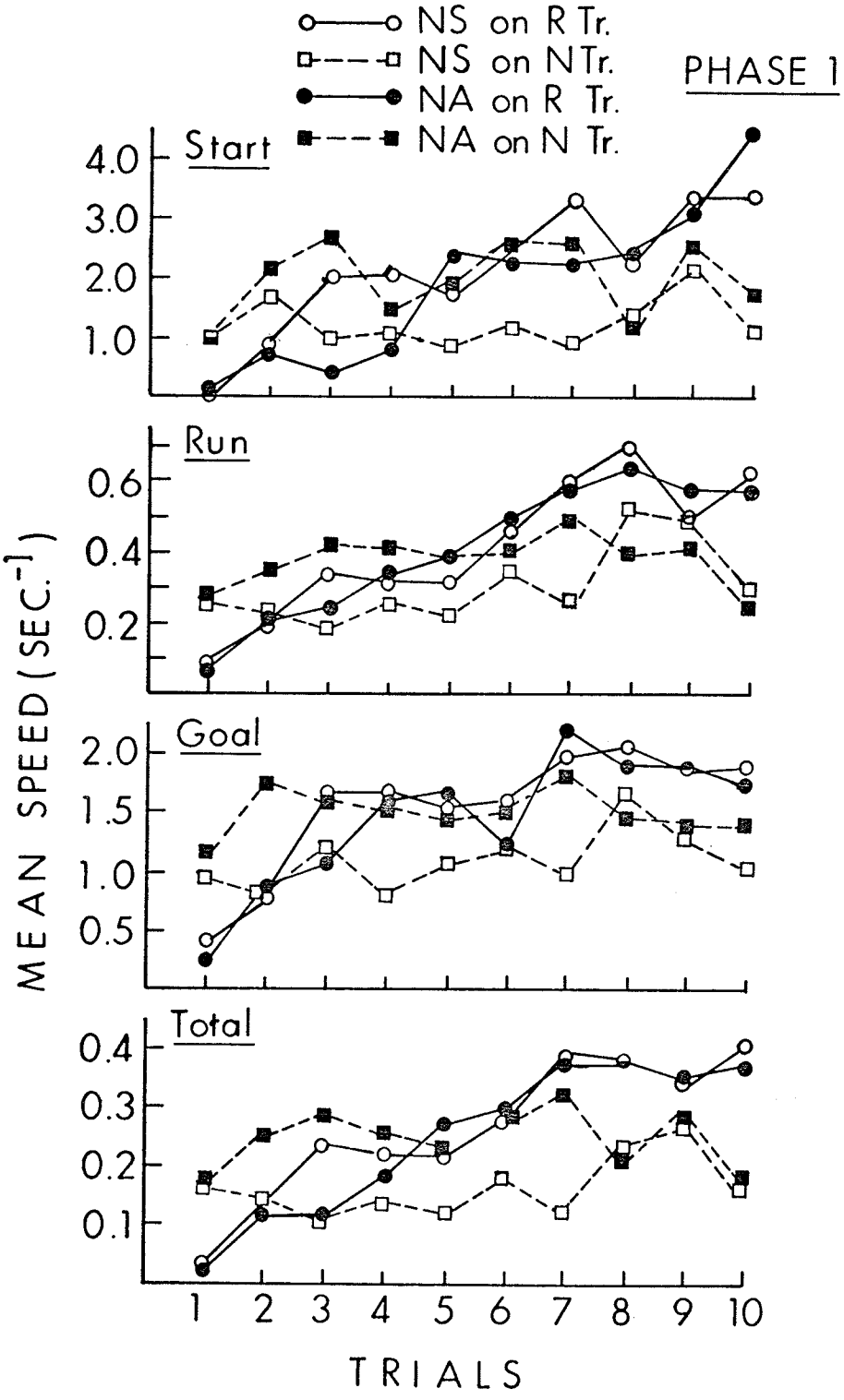
TOTAL

Source	df	MS	F	p
Group	1	80360.0	0.745	n.s.
Error 1	12	107907.8		
Reinforcement Cond. (RFM)	1	184933.0	11.276	$<.01$
Group x RFM	1	150108.0	9.153	$<.025$
Error 2	12	16400.0		
Trials	9	145167.5	16.320	$<.001$
Group x Trials	9	9688.0	1.089	n.s.
Error 3	108	8895.1		
RFM x Trials	9	85306.0	10.935	$<.001$
Group x RFM x Trials	9	18128.5	2.324	$<.05$
Error 4	108	7801.4		

significant group main effect was obtained on any of the dependent measures (F (1,12) = 0.389, 0.377, 0.873, 0.745, for start, run, goal and total times respectively). A significant difference between performance on R trials and N trials was found for all measures (start, F (1,12) = 6.880, $p<.025$; goal, F (1,12) = 5.192, $p<.05$; run and total, F (1,12) = 10.628, 11.276, $p<.01$ respectively). In addition, a significant group by reinforcement condition interaction was found for three measures (start, F (1,12) = 10.387, $p<.01$; goal and total, F (1,12) = 9.049, 9.153, $p<.025$ respectively). Inspection of the means indicated that performance was better on R trials than on N trials. The significant interaction indicated differential performance changes for the two groups during the two reinforcement conditions. Figure 5 reveals little difference between the two groups on R trials, however, group NS shows overall performance decrements on N trials relative to group NA from trials 2 to 8 of the N trials. The significant reinforcement condition by trials interaction for all four measures (F (9,108) = 6.338, 9.112, 7.417, 10.935, $p<.001$ for start, run goal and total times respectively) indicated that there were differential performance changes over the ten R trials as compared to the ten N trials. Figure 5 again shows that performance improved more rapidly on R trials than on N trials.

When the means for blocks of 2 trials were plotted over the acquisition phase for groups CRF, NRS and NRA (Fig. 5) for the start and goal measures, the relationship depicted suggested that group NRS showed the PRAE relative to the CRF group. Group NRA did not show the initial slower performance in the start or goal sections but did show the same terminal performance as group NRS, although group NRS demonstrated faster performance than group

Fig. 5 Mean start, run, goal and total speeds as a function of reinforced and nonreinforced acquisition trials (Phase 1) for groups NS and NA.


NS on R Tr.
NS on N Tr.
NA on R Tr.
NA on N Tr.
PHASE 1
Start
Run
Goal
Total
MEAN SPEED (SEC.$^{-1}$)
TRIALS
1 2 3 4 5 6 7 8 9 10

CRF much earlier than group NRA in the start measure. A repeated measures analysis of variance (3 groups x 10 blocks of trials) revealed no significant group main effect (F (2,18) = 0.220, 0.771, for start and goal respectively). However, a significant block effect was obtained (F (9,162) = 15.409, 45.870, $p < .001$ for start and goal respectively) indicating acquisition of the instrumental response. No significant group by block interaction was found in the start measure (F (18,162) = 1.228) but a significant interaction was found in the goal measure (F (19,162) = 3.088, $p < .001$). Thus, only the PRAE goal box effect was demonstrated by group NRS relative to the CRF group. Group NRA did not show this effect (Figure 6).

3.3. CRF Block (Phase 2)

In order to analyze performance differences during the block of CRF trials (Phase 2) the results of the last acquisition trial were included to yield a 5 (group) x 6 (trial) repeated measures analysis of variance. No significant group main effect was found for any of the four measures (F (4,30) = 0.597, 1.562, 0.584, 1.452, for start, run, goal and total times respectively), but the trial main effect was significant for all measures (start, F (5,150) = 2.875, $p < .025$; run goal and total, F (5,150) = 13.603, 10.580, 10.347, $p < .001$, respectively) (Table 4). The trial effect reflected performance gains over trials (See Figure 7). The group by trials interaction was non-significant for all four measures (F (20,150) = 1.171, 1.621, 1.084, 1.457, for start, run, goal and total times respectively).

When the effect of receiving drug injections on N or R trials during acquisition was taken into account, the only significant effect were the trial main effect (start, F (5,120) = 2.593, $p < .05$; run, goal and total, F (5,120) = 11.427, 8.420, 8.807, $p < .001$, respectively) and a drug on R

Fig. 6 Mean start and goal speeds as a function of blocks of two acquisition trials (Phase 1) for groups of CRF, NRS and NRA.

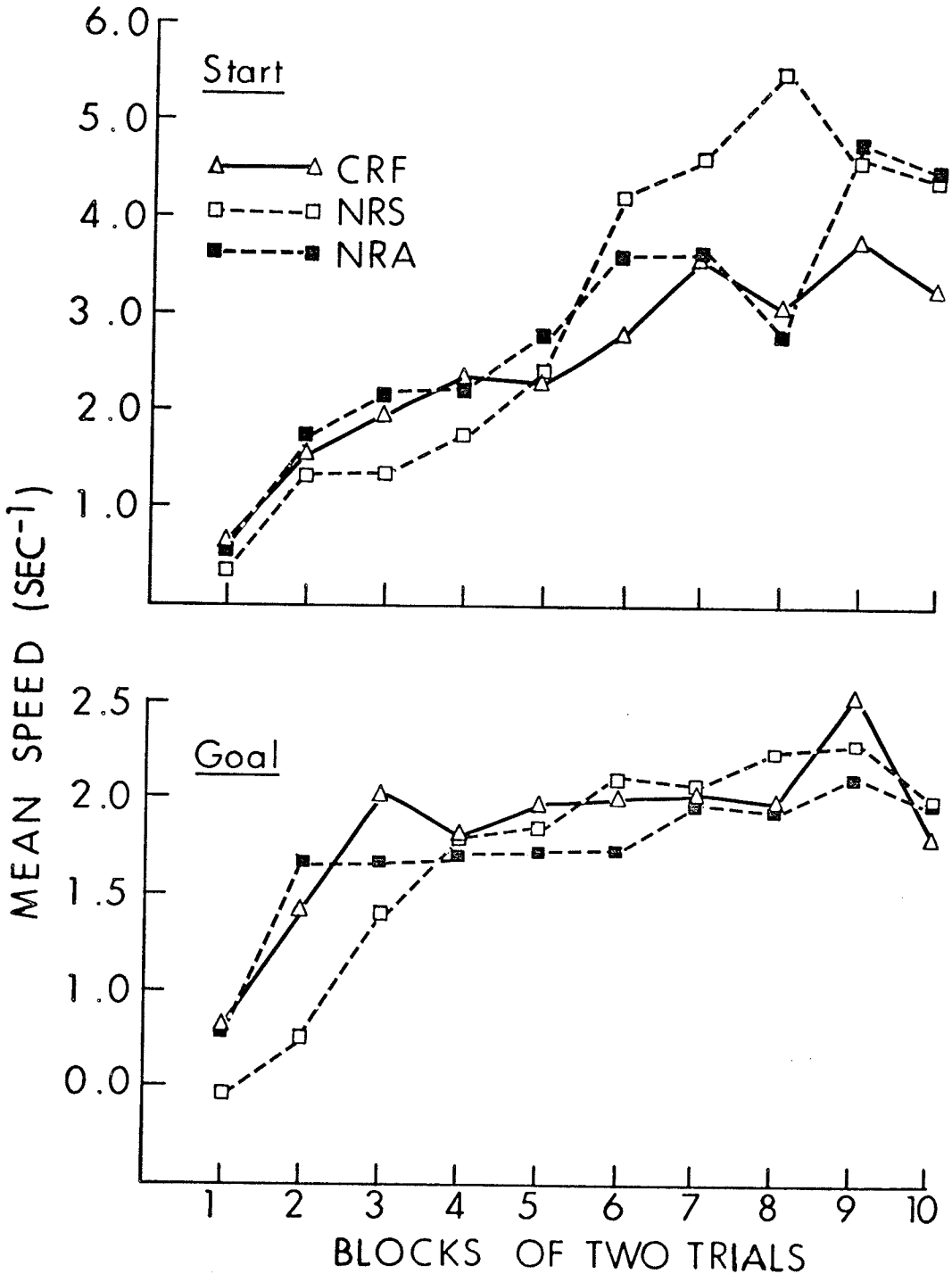

6.0
5.0
4.0
3.0
2.0
1.0
Start
CRF
NRS
NRA
MEAN SPEED (SEC⁻¹)
2.5
2.0
1.5
1.0
0.0
Goal
1
2
3
4
5
6
7
8
9
10
BLOCKS OF TWO TRIALS

Table 4. Analysis of Variance on the last acquisition trial and the five crf trials for the five groups and the four speed measures:

START

Source	df	MS	F	p
Group	4	8909184.	0.597	n.s.
Error 1	30	14922043.		
Trials	5	8141212.	2.875	<.025
Group x Trials	20	3316300.	1.171	n.s.
Error 2	150	2831441.		

RUN

Source	df	MS	F	p
Group	4	429644.0	1.562	n.s.
Error 1	30	274975.9		
Trials	5	228834.4	13.603	<.001
Group x Trials	20	27262.2	1.621	n.s.
Error 2	150	16822.5		

Table 4. continued....

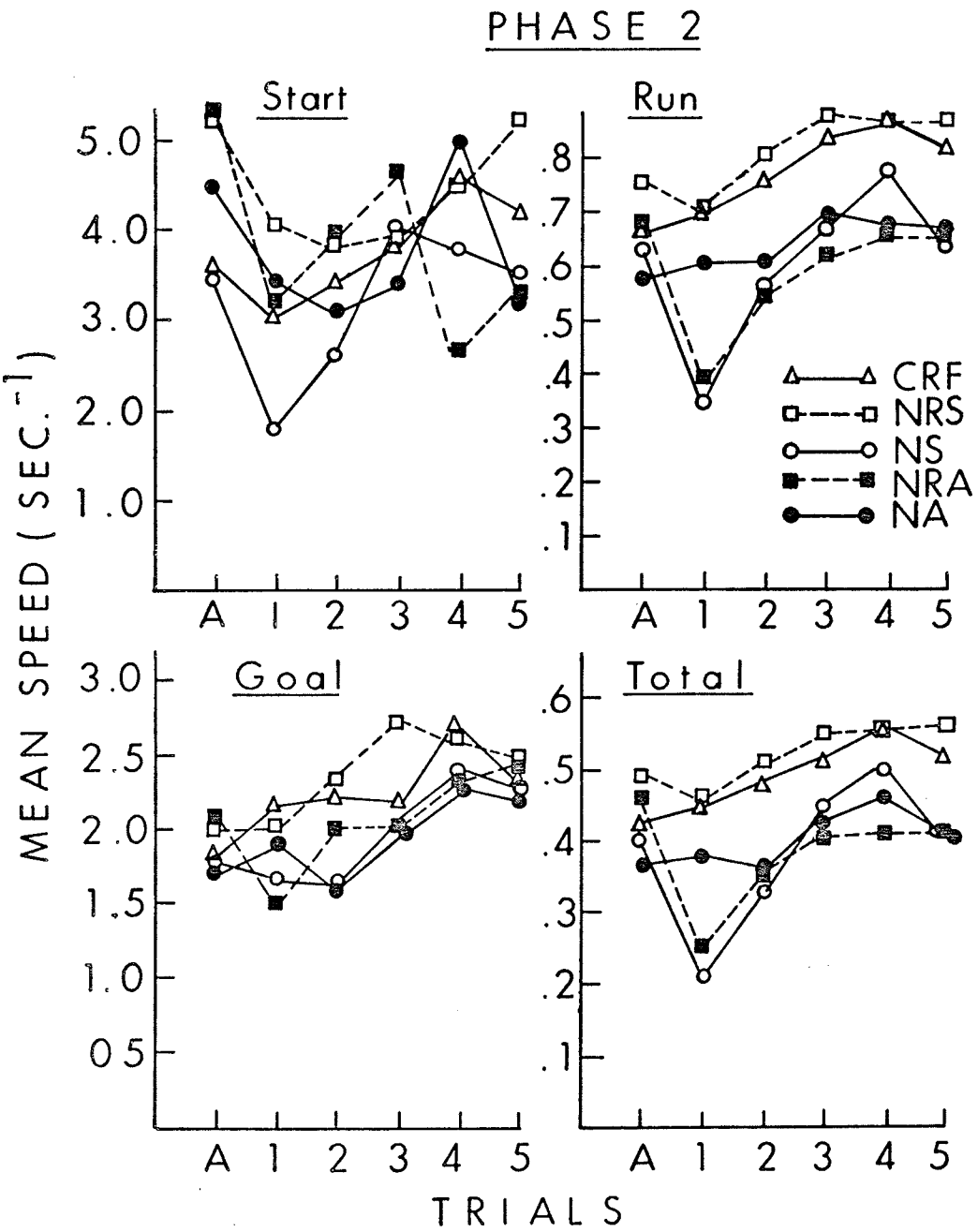
GOAL

Source	df	MS	F	p
Group	4	1218468.	0.584	n.s.
Error 1	30	2084760.		
Trials	5	2173915.	10.580	<.001
Group x Trials	20	222771.	1.084	n.s.
Error 2	150	205475.		

TOTAL

Source	df	MS	F	p
Group	4	168705.7	1.452	n.s.
Error 1	30	116177.4		
Trials	5	93180.6	10.347	<.001
Group x Trials	20	13124.4	1.457	n.s.
Error 2	150	9005.9		

Fig. 7 Mean start, run, goal and total speeds as a function of CRF trials (Phase 2) for all five groups. Trial A is the last trial of acquisition.


PHASE 2
Start
Run
Goal
Total
MEAN SPEED (SEC.$^{-1}$)
TRIALS
A 1 2 3 4 5
5.0
4.0
3.0
2.0
1.0
.8
.7
.6
.5
.4
.3
.2
.1
3.0
2.5
2.0
1.5
1.0
0 5
CRF
NRS
NS
NRA
NA

trials by trials interaction on two measures (run, F (5,120) = 3.758, $p < .005$; total, F (5,120) = 2.876, $p < .025$) (Table 5). The significant interaction reflects performance decrements for groups NS and NRA from the last acquisition trial to the first CRF block trial in the run and total speed measures (Figure 7). This temporary decrement disappears by the later part of Phase 2 such that by the last three trials in Phase 2 (last three acquisition trials) no significant difference existed between the groups on any measure as indicated by a factorial analysis of variance for the 5 groups (F (4,30) = 0.86, 0.97, 0.16, 1.00 for start, run, goal and total speeds respectively) (Table 6).

3.4 Extinction (Phase 3)

The last two trials in phase 2 (CRF block) plus the 16 extinction trials were partitioned into 6 blocks of 3 trials with the first block representing the terminal acquisition performance. The repeated measures analysis of variance on the five groups over the six blocks of trials, revealed a significant group main effect for all four measures (start, F (4,30) = 2.760, $p < .05$; run and goal, F (4,30) = 4.281, 4.103, $p < .01$ respectively; total, F (4,30) = 5.189, $p < .005$) (Table 7). Post hoc comparisons with Scheffe's test indicated that in the start measure both group NRS and NS showed significantly superior performance to groups NRA and NA (all $ps < .01$) and to the CRF group ($p < .05$, $p < .01$) for group NS and NRS, respectively. Similarly in the run and total measures groups NRS and NS exhibited significantly superior performance to groups NRA, NA and CRF (all $p < .01$). However, for the goal measure groups NRS, NS, and NRA were superior to groups CRF and NA (all $p < .01$); neither the former or the latter groups differed from each other. (Table 8).

Table 5. Analysis of Variance on the last day of acquisition and the five crf trials for the groups receiving amobarbital on reinforced and groups receiving amobarbital on non-reinforced trials, for the four dependent measures.

START

Source	df	MS	F	p
Drug on N trials (N)	1	24192.	0.001	n.s.
Drug on R trials (R)	1	16160816.	0.930	n.s.
N x R	1	19441296.	1.118	n.s.
Error 1	24	17382112.		
Trials	5	7701232.	2.593	<.05
N x Trials	5	2647591.	0.892	n.s.
R x Trials	5	4749696.	1.599	n.s.
N x R x Trials	5	4100912.	1.381	n.s.
Error 2	120	2969578.		

RUN

Source	df	MS	F	p
Drug on N trials (N)	1	346619.4	1.126	n.s.
Drug on R trials (R)	1	707850.5	2.300	n.s.
N x R	1	266165.5	0.865	n.s.
Error 1	24	307755.4		
Trials	5	202892.7	11.427	<.001
N x Trials	5	12590.3	0.709	n.s.
R x Trials	5	66731.6	3.758	<.005
N x R x Trials	5	8626.1	0.486	n.s.
Error 2	120	17755.5		

Table 5. continued....

GOAL

Source	df	MS	F	p
Drug on N trials (N)	1	1259699.	0.534	n.s.
Drug on R trials (R)	1	734447.	0.311	n.s.
N x R	1	2178079.	0.922	n.s.
Error 1	24	2361112.		
Trials	5	1852813.	8.420	<.001
N x Trials	5	107101.	0.487	n.s.
R x Trials	5	271840.	1.235	n.s.
N x R x Trials	5	266381.	1.211	n.s.
Error 2	120	220036.		

TOTAL

Source	df	MS	F	p
Drug on N trials (N)	1	143800.1	1.083	n.s.
Drug on R trials (R)	1	240249.2	1.809	n.s.
N x R	1	144956.1	1.091	n.s.
Error 1	24			
Trials	5	84382.6	8.807	<.001
N x Trials	5	6258.7	0.653	n.s.
R x Trials	5	27553.3	2.876	<.025
N x R x Trials	5	8178.5	0.854	n.s.
Error 2	120	9580.9		

Table 6. Analysis of Variance on the last three acquisition trials for the five groups on the four dependent measures:

START

Source	df	MS	F	p
Group	4	2740643.	0.86	n.s.
Error	30	3174557.		

RUN

Source	df	MS	F	p
Group	4	55672.2	0.97	n.s.
Error	30	57399.3		

GOAL

Source	df	MS	F	p
Group	4	77426.6	0.16	n.s.
Error	30	481319.6		

TOTAL

Source	df	MS	F	p
Group	4	25047.4	1.00	n.s.
Error	30	24989.9		

Table 7. Analysis of Variance on the last block of three acqusition trials plus the five blocks of three extinction trials for the four speed measures:

START

Source	df	MS	F	p
Group	4	52451280.	2.760	<.05
Error 1	30	19004448.		
Trials	5	12537209.	6.372	<.005
Group x Trials	20	2400346.	1.220	n.s.
Error 2	150	1967436.		

RUN

Source	df	MS	F	p
Group	4	878153.0	4.281	<.01
Error 1	30	205151.2		
Trials	5	1051351.0	54.649	<.001
Group x Trials	20	44000.7	2.287	<.005
Error 2	150	19238.2		

Table 7. continued....

GOAL

Source	df	MS	F	p
Group	4	4777076.	4.103	<.01
Error 1	30	1164346.		
Trials	5	17615168.	85.406	<.001
Group x Trials	20	352192.	1.708	<.05
Error 2	150	206253.		

TOTAL

Source	df	MS	F	p
Group	4	433530.0	5.189	<.005
Error 1	30	83552.2		
Trials	5	620460.0	85.592	<.001
Group x Trials	20	25706.8	3.546	<.005
Error 2	150	7249.0		

Table 8. Post hoc comparisons for group means in extinction with Scheffe's test. ($^{*}p<.05$, $^{**}p<.01$)

START		NRS	NS	CRF	NA	NRA
	NRS			**	**	**
	NS			*	**	**
	CRF					
	NA					
	NRS					

RUN		NRS	NS	NRA	NA	CRF
	NRS			**	**	**
	NS			**	**	**
	NRA					
	NA					
	CRF					

GOAL		NRS	NS	NRS	NA	CRF
	NRS				**	**
	NS				**	**
	NRA				**	**
	NA					
	CRF					

TOTAL		NRS	NS	NRA	NA	CRF
	NRS			**	**	**
	NS			**	**	**
	NRA					
	NA					
	CRF					

A significant trial main effect was observed (start, F (5,150) = 6.372, $p < .005$; run, goal and total, F (5,150) = 54.649, 85.406, 85.592, $p < .001$, respectively) which indicated that extinction produced a decrement in responding (Figure 8). The group by trials interaction reached an acceptable level of significance only for the run, goal and total measures (F (20,150) = 2.287, 1.708, 3.546, $p < .005$, $p < .05$, $p < .005$ respectively).

To determine the effects of drug injections on N or R trials during acquisition, this factor was included in a repeated measures analysis of variance (drug condition on N trials (2) x drug condition on R trials (2) x blocks of trials (6)) on the extinction performance for the four groups involved (Table 9). A significant effect of drug injections on N trials (N) during acquisition was obtained in all runway segments except goal (start, F (1,24) = 8.536, $p < .01$; run and total, F (1,24) = 9.947, 11.588, $p < .005$ respectively). From Fig. 9 it can be seen that the groups receiving amobarbital on N trials during acquisition (NRA and NA) were inferior in extinction performance to groups receiving saline injections on N trials during acquisition (NRS and NS).

No effect of drug injection on R trials (R) and no N x R interactions were found to be significant. An interaction of drug injections on N trials with blocks of extinction trials (N x BLK) was found to be significant for three measures (start, run and total, F (5,120) = 2.380, 2.801, 4.086, $p < .05$, $p < .025$, $p < .005$ respectively) and gave additional indication that the groups receiving the drug on N trials during acquisition extinguished their instrumental response more quickly than the groups receiving saline on N trials during acquisition (Figure 9). No other interactions were found to be statistically significant.

Fig. 8 Mean start, run, goal and total speeds as a function of blocks of three extinction trials (Phase 3) for all five groups. Block R represents the last two trials in the CRF phase plus the first extinction trial.

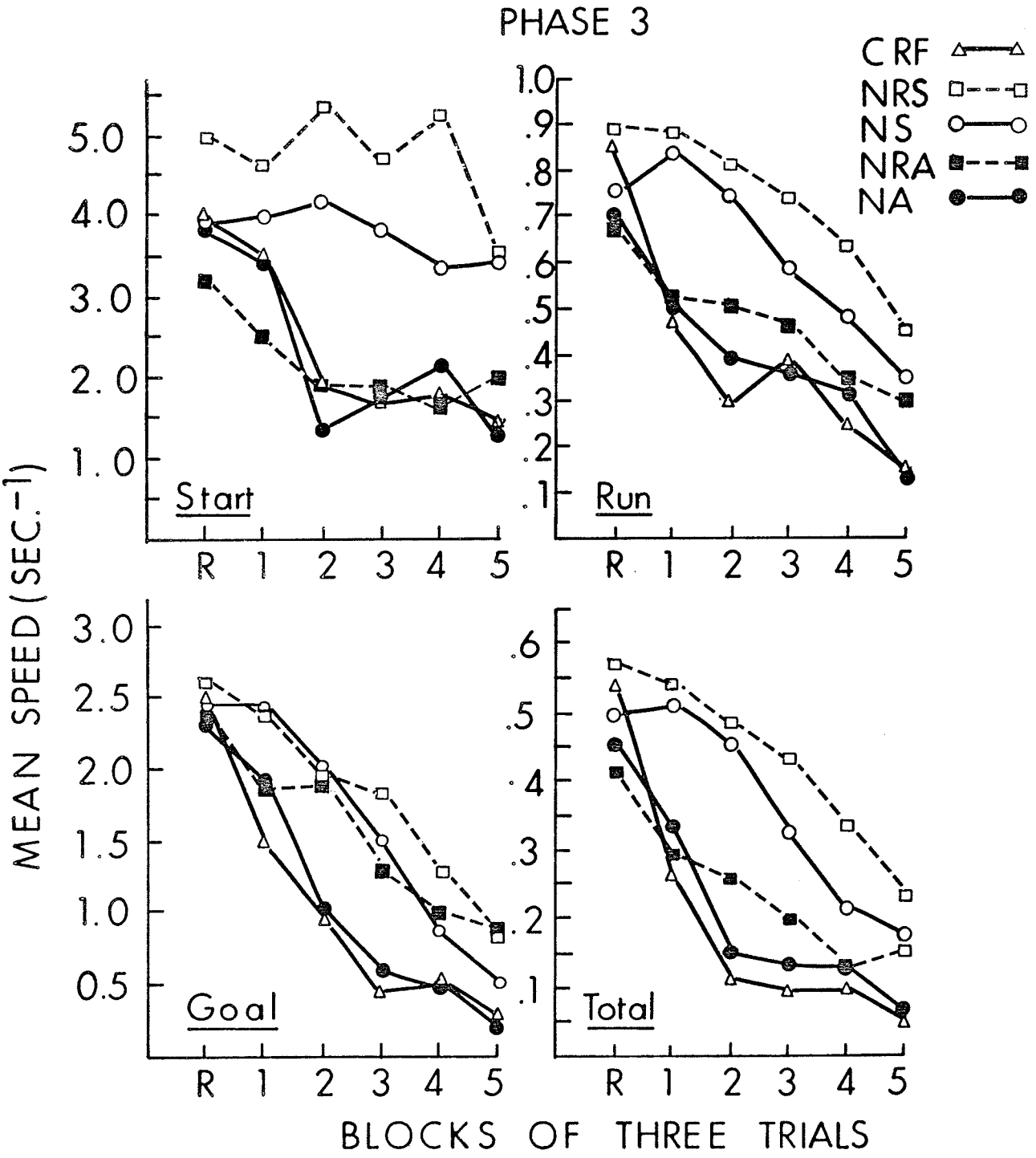

PHASE 3
CRF
NRS
NS
NRA
NA
MEAN SPEED (SEC.-1)
Start
Run
Goal
Total
R 1 2 3 4 5
BLOCKS OF THREE TRIALS

Table 9. Analysis of Variance on the last block of 3 acquisition trials and the five blocks of 3 extinction trials for the groups receiving amobarbital on reinforced trials and groups receiving amobarbital on nonreinforced trials.

START

Source	df	MS	F	p
N	1	165445616.	8.536	<.01
R	1	12505584.	0.645	n.s.
N x R	1	6171764.	0.318	n.s.
Error 1	24	19381280.		
BLK	5	6799564.	3.596	<.005
N x BLK	5	4500944.	2.380	<.05
R x BLK	5	2035556.	1.077	n.s.
N x R x BLK	5	914348.	0.484	n.s.
Error 2	120	1890774.		

RUN

Source	df	MS	F	p
N	1	2386188.	9.947	<.005
R	1	23541.	0.098	n.s.
N x R	1	303450.	1.265	n.s.
Error 1	24	239881.		
BLK	5	740326.	41.951	<.001
N x BLK	5	49424.	2.801	<.025
R x BLK	5	12536.	0.710	n.s.
N x R x BLK	5	9811.	0.556	n.s.
Error 2	120	17648.		

Table 9. continued....

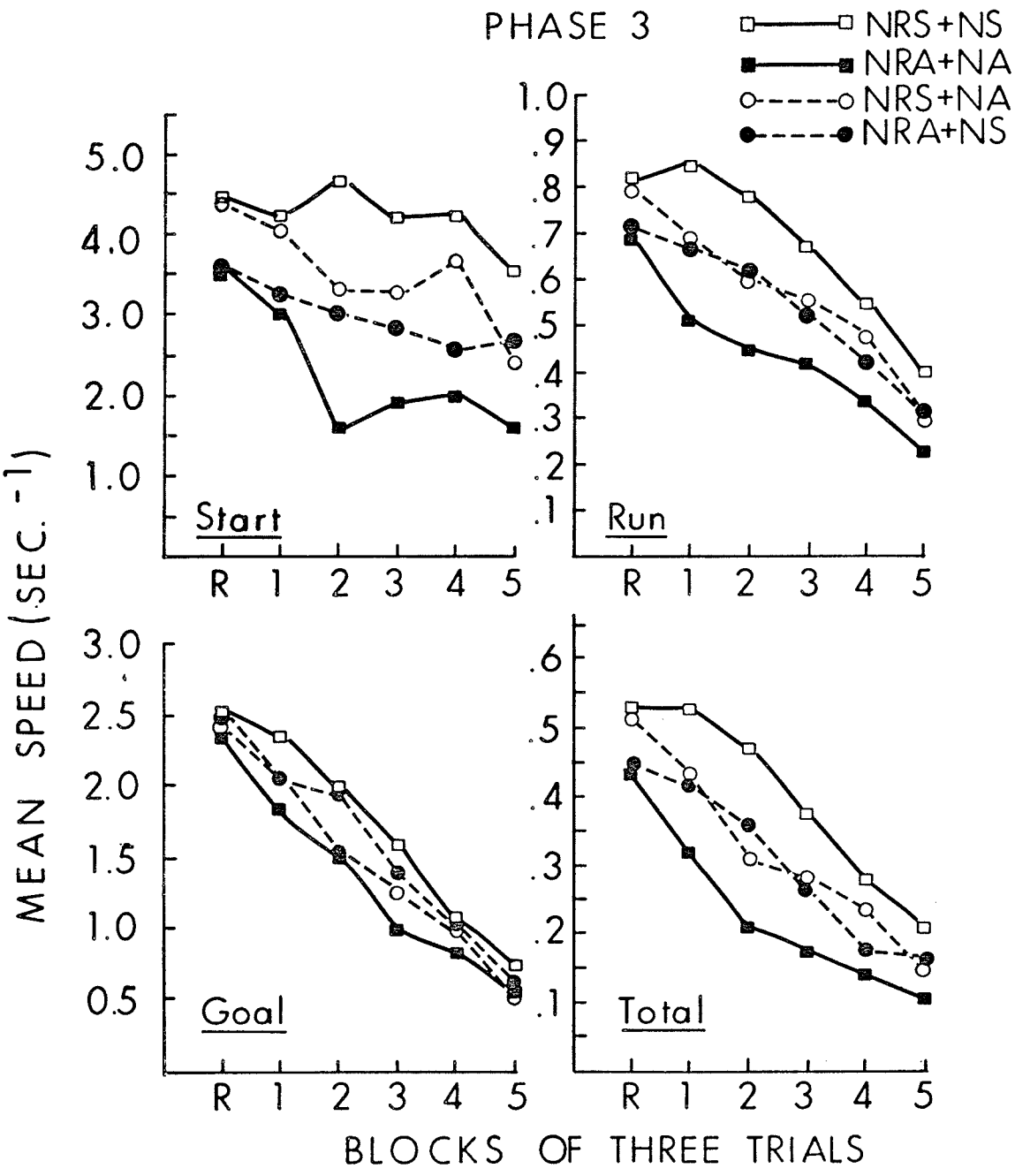
GOAL

Source	df	MS	F	p
N	1	5918248.	4.143	n.s.
R	1	721548.	0.505	n.s.
N x R	1	4288641.	3.002	n.s.
Error 1	24	1428400.		
BLK	5	13375910.	58.966	<.001
N x BLK	5	284258.	1.253	n.s.
R x BLK	5	199616.	0.880	n.s.
N x R x BLK	5	314574.	1.387	n.s.
Error 2	120	226841.		

TOTAL

Source	df	MS	F	p
N	1	1167330.0	11.588	<.005
R	1	11434.3	0.114	n.s.
N x R	1	107212.3	1.064	n.s.
Error 1	24	100737.8		
BLK	5	441189.6	55.972	<.001
N x BLK	5	32206.9	4.086	<.005
R x BLK	5	12136.8	1.540	n.s.
N x R x BLK	5	6920.0	0.878	n.s.
Error 2	120	7882.3		

Fig. 9 Mean start, run, goal and total speeds as a function of blocks of three extinction trials (Phase 3) for groups receiving amobarbital on N trials (NRA + NA) or R trials (NRA + NS), or saline on N trials (NRS + NS) or R trials (NRS + NA) during acquisition. Block R represents the last two trials in the CRF phase plus the first extinction trial.


PHASE 3
NRS+NS
NRA+NA
NRS+NA
NRA+NS
MEAN SPEED (SEC.$^{-1}$)
Start
Run
Goal
Total
R 1 2 3 4 5
BLOCKS OF THREE TRIALS

Chapter 4 Discussion

4.1 Major Findings of the Study

The major acquisition and extinction findings of this study can be summarized as follows:

I Acquisition

a) The groups that received sodium amobarbital injections exclusively on N or R trials exhibited patterned running in Phase 1 of the experiment. The tendency to run slower on N trials than on R trials was seen early in subjects experiencing saline on N trials (Group NS), however this effect was observed only following considerable training in subjects receiving amobarbital on N trials (Group NA). Moreover, the latter group performed better on N trials than the former group.

b) Amobarbital eliminated the reverse PRAE (in early training) and the "goal box effect".

c) The groups receiving amobarbital on R trials (Groups NS and NRA) temporarily declined in performance when drug injections were discontinued in Phase 2.

II Extinction

a) A robust spaced trials PREE was observed following PRF training under saline conditions, however, sodium amobarbital administered on all trials of acquisition eliminated the effect (except in the goal section).

b) In the groups that received amobarbital on half of the acquisition trials it was found that amobarbital injections prior to N trials eliminated the PREE whereas amobarbital administered prior to R trials did not

have the corresponding effect. Thus, subjects receiving amobarbital only on N trials (Group NA) performed similarly to subjects experiencing the drug on all trials (Group NRA) and conversely, subjects not given the drug on N trials (Group NS) performed as subjects who had never received the drug (Group NRS).

4.2 Acquisition Findings

The finding that groups NS and NA exhibited "pattern" discrimination responding is most readily interpreted in terms of 2 factors: a) drug state dependent cues which would provide information regarding nonreinforcement - reinforcement events (Overton, 1964, 1969) and b) emotional responses elicited initially by the runway situation and incompatible with the instrumental response (such as "freezing" responses interfering with running responses).

Early in acquisition when the runway situation is still eliciting emotional responses, the drug would attenuate these responses (thus group NA would run faster on N trials and group NS on R trials) whereas the saline injections would not, thus the "pattern" of responding would follow the pattern of the drug injection for a given group. Since the drug injection schedule for groups NS and NA were opposite in pattern, the "pattern" of responding would be inverse (mirror image) for these two groups, early in acquisition. After considerable training, however, the drug state dependent cues would have allowed the animals to form a discrimination between N and R trials on the basis of these cues. Viewed in this manner, the presence or absence of the drug functions much like black and white alleys signalling reinforcement and nonreinforcement respectively, as in a successive

discrimination paradigm. Since presumably N trials are more aversive than R trials, the animals would run faster on R trials relative to N trials. The formation of a discrimination on the basis drug state dependent cues would require group NA to eventually reverse their pattern since initially they would be running faster on N trials but ultimately slower on N trials relative to R trials. Group NS would show the same pattern throughout acquisition. The acquisition "pattern" discrimination results found in this study are consistent with the above analysis.

Further support for the cue function of amobarbital was observed in Phase 2. The finding of a decrement in performance from the last acquisition trial to the first trial in Phase 2 (CRF block) for group NS indicates that this group was discriminating N from R trials on the basis of drug state dependent cues. Group NRA exhibited the decrement from the last acquisition to the first CRF trial as well. Overton (1964, 1969) demonstrated that no transfer of training occurred between the nondrug and drug state for sodium pentobarbital (25 mg/kg), which suggests that the decrement observed for group NRA in this study, represented a failure of transfer from the acquisition phase (when all trials were preceded by drug injections in group NRA) to the CRF phase (when no injections were given).

Although both groups NS and NA ran faster on R trials than on N trials in the later part of acquisition, group NA ran faster than group NS on N trials. This superior performance on N trials for group NA relative to NS, could be interpreted by considering the emotion attenuating function of the drug. That is, amobarbital is assumed to reduce emotional responses occasioned by nonreward in group NA but not in group NS. The finding that

group NS and NA did not differ significantly on R trials is consistent with several other studies. For example, Ison and Northman (1968) and Rosen, Glass and Ison (1967) found that amobarbital eliminated the rapid performance decrement usually found after a reduction in reward, but had no effect when reward magnitude was increased. Thus, the attenuation of the performance decrement following reward reduction (R to N trials) by amobarbital was demonstrated on a trial to trial basis (group NA) in this study. In addition, the failure to find effects of the drug following increases in reward magnitude (N to R trials) was also observed (group NS).

The observation that the PRF group receiving saline injections in acquisition on all trials (NRS) was initially slower than the CRF group in goal speeds, whereas the PRF group receiving the drug on all trials (NRA) in acquisition, was not, can perhaps best be understood in terms of nonreward produced emotional responses being attenuated or eliminated by amobarbital. The failure to confirm the "cross over" PRAE and the superior final acquisition performance for the PRF group, was probably a result of insufficient acquisition trials since Weinstock (1953) found the "cross over" effect only after 50 trials for a 50% PRF trained group.

4.3 Extinction Findings

The robust PREE demonstrated by group NRS confirms Weinstock (1958) who also observed a PREE at a 24 hr ITI. The finding that a block of CRF trials interpolated between the acquisition and extinction phases did not eliminate the PREE is consistent with Theios (1962) and extends those findings to situations involving widely spaced training and testing.

The elimination of the PREE in group NA but not in group NS would

seem to present difficulty for Amsel's frustration hypothesis which will be discussed in detail below. On the other hand, these data are consistent with Capaldi's sequential hypothesis, which will also be discussed.

4.4. The Extinction Findings and Amsel's Frustration Theory

Amsel (1958) indicated that in order for S_F to become conditioned to R_I, a moderate number of acquisition trials are required. He suggested that for the PREE to occur, approximately 80 acquisition trials are required. It would seem from the results of this study that at spaced trials (which are presumably less frustrative than massed trials) 20 acquisition trials are sufficient to produce a robust PREE. Indeed, Capaldi, Berg and Sparling (1971) found a PREE at 24 hr ITI acquisition after only sixteen acquisition trials. The more recent position of Amsel, Hug and Surridge (1968) that S_F can build up fairly quickly when multiple pellet rewards are used, partly circumvents this difficulty.

The finding that amobarbital injections during all acquisition trials (group NRA) eliminates the PREE is also readily incorporated by frustration theory. Both F (primary frustration) and S_F (anticipatory frustration) would be eliminated by the amobarbital thus preventing S_F from being conditioned to R_I. However, difficulty arises when one considers the extinction performance of groups NS and NA. Since amobarbital presumably eliminates primary frustration (and thus prevents S_F from occurring), injections of the drug on N trials would eliminate F and thus group NA would not show the PREE, which in fact was observed. However the studies by Gray (1967, 1969, 1970) indicated that amobarbital also eliminated S_F. Following the assumption of Gray, amobarbital injections on R trials preceded by N trials would be expected

to eliminate S_F and prevent S_F from being conditioned to R_I. However, group NS demonstrated a robust PREE suggesting that either the drug was not affecting S_F or that S_F did not occur on R trials and that some other mechanism was responsible for the observed results. The finding by Ison and Pennes (1969) that animals trained and extinguished under amobarbital showed a PREE together with the results of this study would seem to suggest that in fact some other mechanism than frustration may have been operating.

4.5 The Extinction Findings and Capaldi's Sequential Theory

Capaldi's sequential theory suggests that nonreward related internal cues occurring on N trials (S^N) are reinstated (as memories) on R trials and become conditioned to R_I. If, S^N consists in part of frustrative or emotional components and amobarbital attenuates those components (Capaldi et al, 1971), the subjects experiencing the drug on N trials should have a nonfrustrative S^N (i.e. S^N without the frustrative components) conditioned to R_I. However, S^N in extinction was by definition frustrating (since the drug was not administered) and thus the rapid extinction of groups NA and NRA may be accounted for in terms of generalization decrement (i.e. a shift from a less to a more frustrative or emotional S^N).

Furthermore, since the internal stimulus complex S^N is assumed to be reinstated on R trials as a memory (Capaldi, 1971), the amobarbital most likely would have no effect (or very little) on this memory stimulus and thus the memory of S^N (in this case containing frustrative components) would be conditioned to R_I. In this case there would be no generalization decrement (or very little) since S^N in acquisition and early extinction would be frustrative to a similar degree and thus group NS would exhibit a PREE,

which was confirmed in this study.

Indeed, the argument just presented would predict that amobarbital should have its main effects on the N trials in acquisition (when primary frustration occurs), a result again supported by the present study. On the other hand, Amsel's Frustration Theory would have predicted that amobarbital should exert its effects on both N and R trials in acquisition (since F occurs on N trials and S_F occurs on R trials) since amobarbital presumably antagonizes both of these responses.

The robust PREE for groups NRS and NS and the almost complete attenuation of the PREE in groups NRA and NA at a 24 hr ITI, suggests that the internal nonreward-related stimuli for spaced trials (S^{SP}) are more emotional than other investigators have assumed (cf. Capaldi, Berg and Sparling, 1971) since the emotional components were presumably eliminated by the amobarbital. Thus it could be argued on the basis of the present data that S^{SP} may be substantially more frustrative or emotional than previously assumed by investigators.

4.6 The Extinction Findings and Gray's Physiologically Based Hypothesis

Gray (1970) indicated that perhaps one could view the theta rhythms in the hippocampus as a physiological analogue of Amsel's (1962) construct of frustration. However, as pointed out earlier, the data from the present study provide difficulty for Amsel's Frustration Theory. A modification of Gray's (1970) position would lead to a more satisfactory explanation of the data. Rather than viewing the theta rhythm in the hippocampus as an analogue of frustration, it could be viewed as being involved in, or epiphenomenal to, memory processes (cf. Bennett, 1971). Since amobarbital presumably has little effects on memory at the dosage used (20 mg/kg), the

theta ryhthm frequencies could algebraically average out over the acquisition trials as a result of many occurrences of N and R trials with their corresponding memories. The correlations found by Gray (1970) and Gray and Ball (1970) between theta frequency during extinction and R_n as well as during acquisition and R_n, would be predicted from this hypothesis. However, it should be noted that the foregoing is purely speculative and that the role of the hippocampus in memory processes is by no means clear (Adey, 1970; Bennett, 1971).

4.7 Conclusions

The results of this study seem to be most consistent with Capaldi's sequential theory. Amsel's Frustration Theory would only be viable in this instance if one assumed that sodium amobarbital had little effect on anticipatory frustration, an assumption not consistent with previous research (Gray, 1967, 1969, 1970). The present study suggests that a memory mechanism as hypothesized by Capaldi (1971) in which the occurrence of primary frustration on an N trial is remembered on a following R trial and becomes conditioned to R_I, is involved in the partial reinforcement extinction effect. Gray's (1970) attempts to find the physiological basis for the partial reinforcement effect should be pursued in the light of Capaldi's hypothesis. Perhaps by selectively inducing retrograde amnesia following either N or R trials in a partial reinforcement alleyway study similar in design to the present one, one could eliminate the consolidation of the memory of nonreinforcement following N trials and in this manner shed some light on the mechanism involved.

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