Conditioned Inhibition of Heart Rate: The Effects of Ethanol on Three Inhibitory-Training Procedures

by

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INTRODUCTION

In his 1927 book, Conditioned Reflexes, I. P. Pavlov devoted approximately three times as many chapters to inhibition as he did to excitation. Moreover, Pavlov derived the basic laws of the functioning of the cerebral cortex largely from experiments on inhibition; properties of excitation were established after the mechanisms of inhibition had been elucidated (Konorski, 1948). Despite this early emphasis, Western psychologists have, for the most part, shunned study of the process of inhibition (Hearst, 1972). Part of this neglect can be traced to the fact that inhibitory phenomena were often difficult to detect and, therefore, fell under the rubric of unobservable behavior. Consequently, any consideration of this phenomenon was immediately criticized by some early American behaviorists.

Another factor hindering the study of inhibition was spelled out by Skinner (1938). The thrust of his argument was that inhibition actually referred to the direction of a change in response strength. Thus, virtually all the effects described as inhibitory may be more parsimoniously interpreted as mere reductions in a single factor, that of excitation. In spite of these early criticisms, inhibition has been the subject of numerous recent journal articles and books (Anokin, 1974; Boakes & Halliday, 1972; Gray, 1975; Konorski, 1967). As will be discussed later, specific and objective test procedures to identify inhibitory phenomena have become available in the last

10 to 15 years, and these procedures seem to have kindled much of the current interest in the area among American researchers.

Definitions of inhibition

In his work with classically conditioned salivary responses in dogs, Pavlov (1927) discussed two separate and distinct processes, excitation and inhibition. The repeated association of a previously neutral conditioned stimulus (e.g., a metronome) with an unconditioned stimulus (e.g., meat powder) that elicited an unconditioned salivary reaction was found to result in an increase in salivary secretions occurring to the metronome. This process was termed conditioned excitation, and the conditioned stimulus (CS), through its positive relation to the unconditioned stimulus (US), was thought of as having acquired the ability to elicit the conditioned response (CR). A stimulus having this ability was called a conditioned excitor.

In contrast to excitation, conditioned inhibition was posited by Pavlov (1927) to be a process that resulted in a decrease in salivary secretions. This process was thought to evolve when a negative relation existed between the CS and US. One example of such a relationship was having the CS and US not associated or paired with each other. The CS, through its negative relation to the US, was considered to have gained the ability to decrease salivary secretions and thus was termed a conditioned inhibitor.

Henceforth, a + sign will denote that a CS has been paired with a US or has a history of being paired with a US. A - sign will denote that a CS has not been paired with a US or has a history of not being

paired with a US. In addition, a + or - sign outside of parentheses will indicate that the enclosed CS or CS complex has been paired with or not paired with the US, respectively.

Pavlov (1927) classified inhibitory processes into two major categories. Both categories had in common a response decrement. The first major category was external inhibition. This process was not thought to be the result of learning but rather referred to those cases where a response decrement was maximal on the first occasion of a stimulus presentation. With repetition of the stimulus the magnitude of the decrement declined. The second major category was internal inhibition. By contrast, this process was thought to result from learning and to grow gradually with repeated stimulus presentations. Thus, these two processes, external and internal inhibition, were also referred to as unconditioned and conditioned inhibition, respectively.

Within the unconditioned-inhibition category, there were four groupings which included transmarginal inhibition of repetition, disinhibition, transmarginal inhibition of intensity, and external inhibition proper. Similarly, there were four groupings of conditioned inhibition--extinction, discrimination, inhibition of delay, and conditioned inhibition proper. Each of these groupings referred to a specific experimental procedure. Because of the relevance of conditioned-inhibition procedures to the present study, they will be further delineated below.

Pavlov postulated that extinction of an excitatory CR due to the omission of the US following the CS was an inhibitory process.

It was thought that active inhibition to the CS gradually built up and opposed the excitation that was present, thereby reducing the overall level of excitation. Thus, through extinction, a formerly excitatory CS was transformed into an inhibitory one.

Discriminative (or differential) conditioning consisted of having one stimulus (CS+) consistentently followed by the US and having another stimulus (CS-) never followed by the US. Under these circumstances, Pavlov believed that the CS- acquired the ability to control a tendency opposed to that of the CS+. Initially, the CS- may have possessed excitatory tendencies due to generalization from the CS+, but with further exposures this excitation declines.

Inhibition of delay referred to the observation that, with extended CS-US intervals, the onset of the CR was delayed until a point immediately prior to the onset of the US. Pavlov interpreted this finding to mean that the early portion of the CS acquired inhibitory tendencies. Initially, this early part of the CS elicited both excitation and inhibition, but with additional training it was felt that inhibitory tendencies became stronger than excitatory ones.

Conditioned inhibition proper referred to a procedure in which, like discrimination, two CSs were employed. One stimulus (CS_1+) was paired with the US. However, the other stimulus (CS_2) was presented in compound with the first stimulus, and that compound (CS_1+CS_2)- was not reinforced. At first, the compound elicited a response but with repeated nonreinforcement, a response decrement occurred.

Tests for inhibition

As mentioned above, specific tests for the presence of inhibitory phenomena have been advocated by a number of investigators (Gray, 1975; Pavlov, 1927; Rescorla, 1969). Four such tests are: (1) summation (combined cue), (2) retardation (reversal), (3) reaction of the reverse sign, and (4) induction. In a summation test, a known conditioned excitor (CS+), a stimulus with a history of having been positively related to the US, is presented in compound with a putative conditioned inhibitor (CS-), a stimulus that has been negatively related to the US. This combination of stimuli is given without reinforcement, and the expectation is that if the CS- has inhibitory potential, it will reduce responding to the CS+. For example, consider the situation in which one stimulus (CS+) reliably produces a CS (e.g., salivation) as a result of repeated CS–US pairings. Another stimulus (CS_2-) has not been paired with the US. If the CS_2- has inhibitory potential, then in the summation test, the compound (CS_1+CS_2^-) should result in reduced salivation relative to CS_1+ alone.

The retardation test consists of using the putative inhibitory stimulus (CS-) as the conditioned stimulus in paired CS-US excitatory-conditioning trials. The assumption is that the acquired inhibitory tendencies of the CS- will interfere with the new response that is being conditioned. Continuing with the example above, if the CS_2- has inhibitory potential, the use of this stimulus as the CS+ in paired CS--US presentations should result in slower (retarded) acquisition of the salivary CR as compared with novel stimuli or stimuli not having a history of being used in an inhibitory procedure.

The reaction of the reverse sign refers to a response tendency to the CS- that is opposite in direction to the response tendency elicited by the CS+. As pointed out by Gray (1975), much of the work dealing with the reverse-sign phenomenon has been done by Soviet investigators, although there have been some American studies bearing on this type of reaction and these will be discussed later. In this case, consider a situation in which the CS+ produces a CR that is a decrease in heart rate (HR). In such a response system, an inhibitory stimulus (CS-) would produce a response in the opposite direction, namely an increase in HR. It should be pointed out that the investigation of the reverse-sign phenomenon requires a response system with an above-zero baseline of activity.

Induction refers to the enhancement of the CR when the conditioned excitor (CS+) is presented just subsequent to a conditioned inhibitor (CS-). It has been argued (Pavlov, 1927) that removal of a putative inhibitor creates an excitatory after-effect which is opposed to the action of that inhibitor. The excitor combines with the after effect producing an enhanced CR. Considering again the HR example above, it would be predicted from the induction principle that presentation of a conditioned inhibitor would produce an after effect of HR decreases which would enhance the HR-decrease tendency of the conditioned excitor.

For the most part, the two most commonly employed tests for inhibition seem to have been summation and retardation. Moreover, it has been suggested that positive outcomes to these two tests

provide sufficient evidence to conclude that the stimulus in question is a conditioned inhibitor, given that the stimulus has had a history of being negatively related to the US (Rescorla, 1969 a, b).

Theories of inhibition

According to Pavlov (1927), conditioned inhibition and conditioned excitation developed at the primary afferent sites of the CS and irradiated gradually to include the whole cortex. In this way, inhibition and excitation were similar and parallel processes, although at the same time, they arose from separate centers and opposed each other. The two processes were not equal, however, since Pavlov thought of inhibition as being fragile, fading in time, and generally less stable than excitation.

In general, Pavlov believed that excitation accounted for orienting (investigative) responses (ORs) to novel stimuli, unconditioned reactions to the US, and the development of conditioned reflexes. Inhibition, on the other hand, was portrayed as being responsible for decrements in responding during habituation of the OR and extinction of the CR. Furthermore, the interaction and integration of excitatory and inhibitory processes was considered by Pavlov to be the basis by which all behavior could be explained.

A theory of inhibition and excitation which places more emphasis on the relationship between the CS and US is that offered by Konorski (1948). In his view, excitation resulted from the establishment of connections between a CS center and a US center, both of which

were located within the central nervous system. The presentation of the US was thought to activate the US center, and if the CS was administered at a time of increased activity in the US center, then the CS became an excitor. On the other hand, if the CS was administered during a time of decreased activity in the US center, then the CS became an inhibitor.

In contrast to what Pavlov believed concerning the role of inhibition during extinction, Konorski's theory predicted that omission of the US would not produce net innibition because response decrements cease when the US center is no longer activated. Moreover, simple presentation of a novel stimulus, as in OR habituation, would involve no activation of the US center, and thus neither excitation nor inhibition would result. In Konorski's view, the best procedure to produce inhibitory tendencies was backward conditioning in which the US preceded the CS.

The Pavlovian notion that nonreinforcement was involved in the development of inhibition is still a part of more contemporary theories of inhibition (Hearst, 1972; Rescorla, 1969a). At the same time, however, components of Konorski's theory also play a major role. According to these modern views (Rescorla, 1969a, 1975; Rescorla & Wagner, 1972), conditioned excitation results from a positive contingency or correlation between the CS and US, whereas conditioned inhibition is produced by a negative contingency between the CS and US. Thus, stimuli that provide information about reinforcement become excitatory and those generating information about

nonreinforcement become inhibitory. It should be pointed out that, in contrast to the Pavlovian notion of temporal contiguity between the CS and US, Rescorla's view suggests that ". . .the organism behaves as a relatively complex probability comparator" (Rescorla & Wagner, 1972). That is, if the probability that the US will occur in the presence of the CS [Pr (US/CS)] is larger than the probability of the US occurring in the absence of the CS [Pr (US/$\overline{CS}$)], then the condition for excitation is met. If the converse is true, [pr (US/CS) < Pr US/$\overline{CS}$)], then the condition for inhibition is met.

Under the Rescorla formulation, procedures such as discriminative conditioning, explicitly-unpaired presentations of the CS and US, backward conditioning, and conditioned inhibition are all examples of a negative relationship between the CS and US. That is, in each case the CS predicts the nonoccurrence of the US and, therefore, should lead to the development of conditioned inhibition. Such is not the case, however, for presentations of a novel CS and the process of extinction. Even though both of these procedures result in decrements in responding to the CS, which Pavlov attributed to inhibition, it has been argued by Rescorla (1975) that neither procedure produces inhibition. The reason for this is that extinction and presentations of a novel CS do not generate this necessary background state of excitation that is required for inhibition to develop.

In addition to their relevance to the study of inhibition, it should be noted that all of the above procedures except conditioned

inhibition have been employed as controls for nonassociative factors during excitatory conditioning. However, as discussed by Rescorla (1967), discriminative conditioning, backward conditioning, and explicitly-unpaired presentations of the CS and US are inappropriate control procedures because they introduce a new contingency between the CS and US. In each case, rather than signaling the presence of the US, the CS signals the absence of the US. Thus, the use of these procedures introduces a negative contingency which confounds interpretations about associative changes due to a positive contingency. In contrast, Rescorla suggested that the use of a truly-random procedure in which the [Pr (US/CS) = Pr (US/$\overline{CS}$)] does provide an appropriate control for nonassociative factors because neither a positive nor a negative contingency exists between the CS and US.

Most of the research utilizing the inhibitory procedures outlined above falls under the general classification of either classical-to-instrumental transfer studies or conditioned-emotional-response (CER) experiments. In the transfer studies, instrumental responding is first established, usually consisting of jumping a hurdle or barrier in a shuttlebox arrangement to avoid electric shock. In this situation, it is assumed that apparatus cues present in the shuttlebox are paired with shock and thus become conditioned excitors of fear (CS+). Then, classical conditioning is carried out involving a CS+ and a CS-. In a test phase, the CSs from classical conditioning are presented while the animals are performing the avoidance response. A reduction in avoidance responding in the

presence of CS- is taken as evidence of conditioned inhibition of fear. Conditioned excitation of fear is inferred when avoidance responding increases in the presence of CS+ during the test phase.

In CER studies, a stable level of operant responding (e.g., lever pressing) is first established for food or water reinforcement. Second, classical conditioning is carried out and the CSs are then given while the animals are lever pressing for food or water. It is assumed that the conditioned excitors of fear (CS+) from classical conditioning will supress the level of responding, whereas the conditioned inhibitors (CS-) will reduce this suppression.

In addition to these kinds of studies, more traditional classical-conditioning investigations involving a direct measure of the CR have also been published. The following sections are concerned with these three main sources of inhibition research.

Classical-to-instrumental transfer studies

Rescorla and LoLordo (1965) reported three experiments in which the effects of different CS-US relationships on avoidance resonding were studied. In each experiment, dogs were initially trained to jump a barrier in a two-way shuttlebox to avoid shock. A Sidman-avoidance paradigm was employed in which shock occurred every 10 sec but was postponed for 30 sec if the dog jumped the hurdle. Classical conditioning was carried out with the dogs confined to one side of the shuttlebox. The tone CSs used in the classical conditioning phase, were then superimposed on avoidance responding during a test phase.

In one experiment, a conditioned-inhibition procedure was used in which, for half the trials, one tone (CS_1+) was paired with shock, while for the other half of the trials the compound of the two tones (CS_1+CS_2)- was presented in the absence of shock. A control group was given an equal number of nonreinforced presentations of both tones. In the second experiment, conditioned inhibition and discrimination procedures were used. In the former, CS_1+ was paired with shock and the compound (CS_1+CS_2)- was given without reinforcement. In the latter procedure, CS_1+ was paired with shock and CS_2- was not. A control group was not exposed to either CS. In the third experiment, one CS (CS_2-) was presented, but never paired with shock (i.e., an explicitly-unpaired paradigm). A control group received presentations of CS_1 and CS_2 without reinforcement.

During test phases, it was found that the CS_1+, when given alone to both conditioned-inhibition groups and to the discrimination group, increased avoidance responding above baseline. The presentation of CS_2- to these three groups, on the other hand, decreased the rate of avoidance responding. The CS_2-, when given to the explicitly-unpaired group, also decreased responding. No differences in the rate of responding were found in the three control groups during the presentation of either CS. Rescorla and LoLordo suggested that the CS_1+ had developed excitatory properties (i.e., acquisition of fear) which enhanced avoidance responding. The acquisition of fear was thought to be due to a positive relationship between the CS and US. The authors also suggested that CS_2- had acquired fear-reducing tendencies (i.e., inhibition of fear) that derived from a negative relationship between the CS and US.

Using a Sidman-avoidance procedure similar to that employed by Rescorla and LoLordo (1965), Rescorla (1966) trained dogs to jump a hurdle to avoid shock. The rates of avoidance of dogs exposed to truly-random CS--US presentations, paired CS--US presentations, and explicitly unpaired presentations of the CS and US were compared in the test phase. The CS in each case was a tone and the US was electric shock. It was found that the CS produced no change in responding in the truly-random group, increased avoidance responding in the paired group, and decreased responding in the explicitly-unpaired group. It was suggested that stimuli which signaled increased probability of the US (i.e., CSs paired with US) became

elicitors of fear, resulting in increased avoidance rates, while stimuli which signaled decreased probability of the US (i.e., CSs explicitly unpaired with the US) became inhibitors of fear, resulting in decreased rates of avoidance. However, stimuli which signaled equally well the probability of presence or absence of the US, [Pr (US/CS) = Pr (US/$\overline{CS}$)], i.e., CS randomly paired with US, became balanced elicitors and inhibitors of fear, resulting in no change in avoidance rates. It should be pointed out that the above studies also satisfy the requirements of the reaction-of-the-reverse-sign test of inhibition in that a conditioned excitor led to an increase in responding, whereas a conditioned inhibitor led to a decrease in responding.

After establishing hurdle jumping in a Sidman-avoidance schedule, Moscovitch and LoLordo (1968) examined the effects of CSs from three different kinds of backward-conditioning sessions on the rate of avoidance responding in three different groups of dogs. In one group, onset of CS occurred 1 sec after termination of the shock US with an intertrial interval ranging from 2.0 to 3.0 min ($\overline{x}$ = 2.5 min). For the second group, onset of the CS occurred 15 sec after shock termination and was followed by the same intertrial intervals. In the third group, onset of the CS occurred 1.0 sec after shock termination but the intertrial intervals varied randomly from 0 to 15 min around a mean of 2.5 min. In each case, the CS was a tone.

Subsequent presentations of the CSs while the dogs were performing the avoidance response resulted in decreased jumping rates in the two groups with the narrower range of intertrial intervals, although the two groups

were not different from each other. The group with the wider range of intertrial intervals showed only slight decreases in responding. The authors interpreted their data to indicate that backward conditioning led to conditioned inhibition of fear because the CS signified the absence of US (i.e., the CS was a "safety" signal). These results support the position of Rescorla (1967) in which it was argued that backward conditioning can be thought of as a special form of the explicitly-unpaired procedure. That is, in each case the CS predicts a period free from the US, and the contingency between the CS and US is a negative one. Thus, in each case, the CS would be expected to develop inhibitory tendencies.

Using a discriminated-avoidance procedure, Bull and Overmier (1968) trained dogs to jump a barrier to avoid shock. In this procedure, a visual stimulus (S^D) was turned on, and if the dog did not jump the barrier within 10 sec, a shock was delivered and remained on until the dog crossed the barrier. If the dog responded within the 10-sec period, both the S^D and shock were terminated. Following this avoidance training, one group of dogs was given discrimination training, using two tones of different frequencies. One tone (CS+) was paired with the shock US, and the other tone (CS-) not paired. Another group was given truly random CS and US presentations. The authors reported that the CS+ increased jumping rates in the discrimination group, while the CS- decreased jumping rates when the CSs were presented while the dogs were performing avoidance responses. However, responding in the truly-random group was not

affected by presentation of the random CS. These outcomes were interpreted to mean that the additive and subtractive effects of fear-conditioned positive and negative stimuli on avoidance rates reflected the additive and subtractive properties of conditioned excitation and conditioned inhibition, respectively. In addition, the fact that the truly-random group did not display a change in response rate when the random CS was presented suggested that this CS was neutral.

Grossen and Bolles (1968) trained four groups of rats to avoid shock in a two-way shuttlebox on a Sidman schedule. The four groups were given separate classical conditioning training: (1) paired CS--US trials, (2) explicitly-unpaired CS--US trials, (3) random CS and US trials, and (4) trials in which USs but no CSs occurred. In each group the CS was a tone.

The various CSs were presented while the rats were performing the avoidance task. Administration of the CS+ to the paired group resulted in faster responding (i.e., decreased latency). The random CS had an effect on the avoidance responding of the random CS--US group. The authors argued that the effects of CS-, rather than inhibiting fear, may have represented a positive reinforcing effect of a safety signal.

Using a wheel-turning Sidman task, Weisman and Litner (1969) established avoidance responding in four groups of rats. This was followed by classical conditioning. One group received discriminative conditioning (CS_1+ paired with shock, CS_2- not paired).

The second group was given conditioned-inhibition training (CS_1+ paired with shock, (CS_1+CS_2)- not paired). The third group was given CS_1+ only (paired) trials and the fourth group, CS_2- (explicitly unpaired) trials. The CSs used were a tone and a light.

Test sessions, comprised of presenting the various CSs during avoidance responding, were interspersed at various stages of classical-conditioning training. It was found that at the end of training, the CS_1+ produced an increase in responding, while the CS_2- resulted in a decrease. However, the CS_1+ facilitation was present on the first test session, whereas the CS_2- depression developed gradually with training. It was suggested the conditioned inhibition of fear could not be acquired until after conditioned fear had first developed. This interpretation implies that inhibition of fear was a derivative process dependent on the prior acquisition of fear. An alternative explanation was that it may take more trials to establish inhibition of fear than to establish excitation of fear.

In a backward-conditioning study, Maier, Rapaport, and Wheatley (1976) trained separate groups of rats to perform a shuttlebox avoidance task on a Sidman schedule. After stable responding was acquired, one group was exposed to backward-conditioning sessions in which CS onset occurred 3 sec after the shock US, and another group received trials, in which CS onset occurred 30 sec after the US. For these two groups, the intertrial intervals were 2, 3, or 4 min. A third group received shocks 30 sec after the US but received CSs on a randomly varying schedule centering around 3.5 min. In all three

groups, the CS was a tone. During the test phase in which the CSs were superimposed on the avoidance task, it was found that both tne 3-sec CS and random-CS groups showed depressed responding but that the depression was greater in the 3-sec CS group. No depression was seen in the 30-sec CS group. Maier et al. argued that their results indicated that the temporal arrangement of the US and CS, as well as the informational aspects of the CSs, may have been involved in the development of conditioned inhibition in the backward-conditioning procedure.

CER studies

In a series of three separate studies (Hammond, 1966, 1967, 1968), the effects of CSs from discrimination training on ongoing operant responding were examined. In each study, two groups of rats were first trained to lever press for water reinforcement. One group then received discrimination classical conditioning training in which one stimulus (CS+) was paired with the US and another stimulus (CS-) was delivered alone. The CSs included a tone and a flashing light. The US was electric shock.

In the first study, the control group was given both tone and light presentations but no US presentations. During the test session, it was found that with respect to the control group, the CS+ given the discrimination group suppressed the level of on-going lever press behavior, while the CS- enhanced responding. This enhancement was only temporary, seemingly resulting from baseline changes, and did not appear to result from the CS- having acquired innibitory tendencies.

In the second study, the control group was given random presentations of the CS- that were interspersed with paired presentations of the CS+ and the US. In the test phase, the effects of the CS+, the CS-, and the compound of the two stimuli were examined. It was found that the CS+ suppressed responding and that the CS- led to no appreciable change. However, both CSs in compound produced a reduced level of suppression. Since these levels were compared against a random-control group, it was concluded

that the CS- had acquired active inhibitory tendencies that could not be attributed to stimulus generalization decrement.

In the final study in this series, the control group was given CS- presentations that were programmed to occur independently to CS+ trials. In the test phase, a retardation test was used. The outcomes of the retardation test demonstrated that the acquisition of conditioned suppression using the CS- of the discrimination group was slower than that of the random-control group. Thus, it was suggested that the CS- had developed conditioned inhibitory properties.

After establishing stable lever pressing for food reinforcement, Hendry (1967) exposed rats to a conditioned-inhibition procedure. On some trials, a white noise (CS_1+) was paired with a shock US and on others, CS_1+ was combined with a light stimulus (CS_2) to form a compound (CS_1+CS_2)- that was not reinforced. It was reported that lever press responding was suppressed when CS_1+ was presented alone but not when it was paired with CS_2-. However, no statistical support was cited for these results. Hendry interpreted the effect of CS_2- in terms of "disinhibition" since he felt that the suppression of lever pressing to CS+ was evidence of inhibition.

A conditioned-suppression paradigm was employed by Reberg and Black (1969) to study the effects of excitatory, novel, and inhibitory CSs. Discrimination training utilized a CS+ paired with shock and a CS- presented alone. The CSs were turning off of the houselight and white noise. Prior to discrimination conditioning, rats were trained to lever press for food. In the test phase, the CS+

suppressed responding and the CS- had no effect. However, the compound of CS+ and CS- produced less suppression than CS+ alone. The presentation of CS+ and a novel CS in compound produced a level of suppression not different from that produced by CS+ alone. These data were interpreted to indicate that the reduction in suppression that occurred to the compound of the CS+ and the CS- resulted from a combination of excitatory strengths tending to suppress lever pressing and inhibitory strengths tending to attenuate that suppression.

In two experiments, Rescorla (1969a) studied the effects of different negative relationships between the CS and US on baseline lever pressing of rats. For the first experiment, baseline lever pressing was established in six groups of rats. Two random-control groups (4-4 and 1-1) were given tonal CSs and shock USs that occurred at different frequencies (.4 or .1 per 2-min interval). Two negative contingency groups (0-4, 0-1) received the same treatment except that USs programmed to occur during the CS or during the succeeding 2-min were omitted. The final two groups were given a treatment identical to 0-4 and 0-1 groups except that a flashing-light CS was used instead of a tone. In the test phase, the groups were returned to the lever-press situation and the CSs were superimposed on this responding.

Rescorla found that all groups showed suppressed lever pressing during the test phase but that the degree of suppression varied. The 0-4 and 0-1 negative contingency groups showed retarded suppression as compared to their respective controls. Within the

two experimental groups, the 0-4 group showed less suppression. Rescorla suggested that these findings were consistent with the notion that the negative-contingency groups developed inhibitiory tendencies but that alternative explanations could not be ruled out.

In the second experiment, rats were first trained to lever press, then three separate groups of rats were given tonal CSs and shock USs that occurred at three different frequencies (.8, .4 or .1 per 2-min intervals. A fourth group was given the same number of CSs but no USs. Training also included presenting a flashing-light CS paired with a shock (i.e., a positive contingency).

Delivery of these CSs during on-going lever pressing showed that the positive CS+ (flashing light) suppressed responding in all groups equally well. However, when this CS+ was presented in compound with the negative tone CS- in a summation test, suppression was reduced. Moreover, the magnitude of this reduction was a function of the degree to which the CS was negatively related to the US such that the greater the negative contingency the greater the tone reduced the suppression to the light. In summarizing both experiments, Rescorla argued that the use of both retardation and summation test procedures are important in asserting that a stimulus is a conditioned inhibitor because positive outcomes to both tests help to rule out alternative interpretations for the effects such as increased or decreased attention.

Cappell, Herring, and Webster (1970) examined the effects of the compound of a positive and negative stimulus (CS+CS-) on conditioned suppression. After training one group of rats to lever press for food, discrimination conditioning, consisting of CS+ trials paired with a shock US and CS- trials unpaired with shock, was carried out. The CSs included a tone and two different-colored lights (red or white). In the test phase, which occurred during the acquisition of discrimination, CS+ gradually appeared to suppress responding, while CS- seemed to enhance responding but only during early presentations. When the compound CS+CS- event was given, the level of suppression, as compared to that observed to CS+, seemed to be reduced. It should be pointed out that data for individual Ss were presented and no statistical analyses were carried out, so little can be concluded from this study.

Hammond and Daniel (1970) exposed rats to two types of negative relationships, one in which the shock US never occurred during the CS or within 6-min of its presentation (i.e., an explicitly-unpaired CS) and another in which the US and CS occurred at random during each session (i.e., a random CS). For half the animals, a tone CS was used and for the other half, a flashing-light CS was used. Then both CSs were shifted to positive contingencies in which they were paired with the US (reversal training). Administration of the CSs- while the animals were lever pressing for water resulted in an increase in pressing rate in each group, although the increase was more prominent in the explicitly-unpaired group than in the truly-

random group. Furthermore, when these CSs- were used as CSs+ during reversal training, it was found that the rate of development of conditioned suppression (i.e., conditioned fear to CS+) was retarded more in the case of explicitly-unpaired CS- than in the case of the random CS-, suggesting that the former had developed inhibitory potential.

Using a backward-conditioning paradigm, Siegel and Domjan (1971) administered a tone CS and a shock US to five groups of rats. The investigation consisted of three phases: (1) lever-press training for food, (2) backward conditioning, and (3) superimposition of the CSs from Phase 2 on the baseline of lever pressing. An experimental group received 50 backward-conditioning trials in which the US and the CS were coincident but in which the CS outlasted the US (.5-sec US vs 2-min CS). Four control groups received either no CS or US presentations, CS alone, US alone, or truly-random CS and US presentations. In the test phase, it was reported that acquisition of suppression was retarded in the backward-conditioning group with respect to controls. The no-CS-no-US group showed the most suppression of the control groups and the other three controls showed equivalent intermediate amounts of suppression. These data support the notion that backward CS--US pairings endow the CS with inhibitory potential.

In a subsequent backward-conditioning study, Siegel and Domjan (1974) first trained five separate groups of rats to lever press for food reinforcement. Backward conditioning trials consisted of presenting a shock US for the first .5 sec of a 60-sec tone CS.

The various groups received 0, 5, 10, 25, or 50 trials. Superimposition of the CS during on-going lever pressing led to a retardation in the development of conditioned suppression which appeared to be linearly related to the number of previous backward trials. However, only the difference between the 0 and 50-trial groups was significant and appropriate statistical tests for linear trend were not carried out. Therefore, the assertion of a linear relationship between suppression and number of backward trials was not supported.

Direct-measure studies

In an experiment cited by Pavlov (1927), Frolov used a metronome as a reinforced stimulus (CS_1+) and an automobile horn as a nonreinforced stimulus (CS_2-) in a conditioned-inhibition paradigm to study conditioned salivary responses in a single dog. The CS_1+ was reinforced by a food US and the combination of CS_1 and CS_2 was presented without reinforcement. On its first application, the compound (CS_1CS_2-) did not affect the salivary responding, however, with repeated applications, the magnitude of the salivary CR was diminished. It was suggested that the CS_2- developed inhibitory properties as a result of systematic repetition without reinforcement.

As cited by Pavlov (1927), Kasheriniova also utilized a conditioned-inhibition paradigm [CS_1+ vs (CS_1+CS_2)-] in an experiment employing one dog. One CS was a tactile stimulus, and the other CS was a metronome. It was found that after 25 presentations, the compound of the two CSs evoked a secretory rate of only 3 drops/min, whereas CS_1+ alone produced a rate of 29 drops/min. In addition, when tested alone, the CS_2- evoked a rate of 8 drops/min. It was concluded that the compound of CS_1+ and CS_2- exerted a strong inhibitory influence, but at the same time, the CS_2- acquired some of the excitatory tendencies of the positive stimulus (CS_1+).

A series of experiments were performed by Konorski and Szwejkowska (1950, 1952a, 1952b) to examine possible mechanisms for transforming excitatory CRs into inhibitory ones. In the first experiment, dogs were given excitatory-conditioning trials using

two CSs (CS_1+ and CS_2+) both paired with a food US and salivary CRs were recorded. In the second experiment, conditioned paw flexion was established in dogs using an electric-shock US. The animals in both experiments were subsequently exposed to an extinction condition in which unreinforced presentations of (CS_1+)- were randomly interspersed among reinforced presentations of CS_2+ (food US in the first experiment, shock US in the second). Then reversal (reconditioning) training, using the same USs in each experiment, was initiated to assess the inhibitory potential of the extinguished CS (CS_1+).

The outcomes of both studies indicated that the process of extinction was long and gradual but that reversal conditioning to CS_1+ was quite rapid. It was suggested that the inhibitory properties developed by the extinction procedure were very weak since only a few reinforced trials were necessary to restore the salivary and paw-flexion CRs.

In the third experiment, nonreinforced presentations of a neutral stimulus (CS_0), rather than the conditioned excitor (CS_1+), were randomly interspersed among reinforced presentations of CS_2+. During subsequent reversal training, it was found that, in marked contrast to the reversal performance of CS_1+, the acquisition of CRs to the CS_0 was greatly retarded. Thus, the authors concluded that repeated nonreinforced presentations of a neutral CS endowed that stimulus with strong inhibitory properties.

Using the eyelid response as the dependent variable, Siegel and Domjan (1971) conditioned rabbits employing a tone CS and an electric-

shock US. Five separate groups were given either (1) 550 preexposures to the CS alone, (2) the US alone, (3) both the CS and US in a random manner, (4) both the CS and US in a backward manner, or (5) neither the CS nor the US. Following this preexposure session, 50 paired CS–US trials were administered. It was found that the groups did not differ during preexposure and none showed evidence of conditioned eyelid activity to the CS. However, during acquisition, the backward-pairings group showed the slowest development of the eyelid closure CR. The CS-alone and US-alone groups did not differ in acquisition performance but both acquired the CR faster than the backward-pairings and random-pairings groups and were slower than the no-CS-no-US group. It was concluded that stimuli negatively correlated with the US (in this case, a backward relationship) acquire active inhibitory tendencies which retard subsequent positive conditioning.

Both summation and retardation tests were utilized by Marchant, Mis, and Moore (1972) in analyzing the effects of CSs from conditioned-inhibition and special conditioning control procedures on nictitating-membrane responses in two groups of rabbits. Three CSs were used including a light, white noise or a tone. The US was electric shock. Initially, both groups were given paired CS--US presentations of all three CSs. Next, the conditioned-inhibition group received trials in which CS_1+ was paired with the US and trials in which CS_1+ and CS_3+ were given in compound $(CS_1+CS_3+)-$ and not reinforced. The control group was given a random sequence of CS_1+ and $(CS_1+CS_3+)-$ compound trials

in which both CSs were reinforced only half the time. In the summation test, nonreinforced presentations of the three CSs and each of the pairwise combinations (i.e., CS_1+CS_2+, CS_2+CS_3+, CS_1+CS_3+) were given to both groups. In the retardation test, all three CSs were reinforced.

The results of this study showed that in the conditioned-inhibition group CS_1+ when combined with CS_2+ led to a larger percentage (i.e., enhancement) of CRs than CS_2+ alone. When $(CS_3+)-$ was combined with CS_2+, the percentage of CRs was smaller (i.e., suppression) than that to CS_2+ alone. This enhancement held for the control group but the suppression effect did not. Thus, it was concluded that $(CS_3+)-$ had acquired inhibitory tendencies. In the retardation test, acquisition of CRs to $(CS_3+)-$ was slower than acquisition to CS_2+ or CS_1+ in the conditioned-inhibition group, whereas acquisition to all three CSs were approximately the same in the control group. This finding was interpreted to mean that the inhibition tendencies of $(CS_3+)-$ suggested by the summation test outcome were not the result of stimulus generalization, attention shift, or some other factor related to the novelty of the test compound.

In a followup to their earlier CER study, Siegel and Domjan (1974) examined the acquisition of eyelid conditioning in five separate groups of rabbits that had been preexposed to varying amounts of backward conditioning. The procedure of this study essentially matched the earlier study. That is, 0, 5, 10, 25, or 50 backward trials were administered to the various groups. In this study, a tone CS followed the shock US. It was reported that acquisition

of the eyelid CR was increasingly retarded with greater amounts of backward conditioning exposure. However, only the two groups on the extremes (0 and 50) were reliably different from each other.

Another backward-conditioning experiment was carried out by Plotkin and Oakley (1975). These authors studied backward pairings of a tone CS and a shock US in six groups of rabbits. The dependent variable was the nictitating-membrane response. Two experimental groups were given backward US--CS trials with an interstimulus interval of 200 msec or 500 msec. Four control conditions consisted of a passively-restrained group, a CS-alone group, a forward-conditioning group, and an explicitly-unpaired group. The two experimental groups both displayed similar retardation of the acquisition of the CR following the backward trials. The four control groups did not differ among themselves. The authors concluded that the retardation of acquisition in the backward groups may have resulted from conditioned inhibition being attached to the CS. They felt that the pattern of findings was not consistent with a latent inhibition or safety-signal explanation.

A discriminative-conditioning paradigm was used by Yamaguchi and Iwahara (1974) to study HR and movement activity in rats. Two CSs of different frequencies were employed, one CS (CS+) was paired with a tail-shock US while the other CS (CS-) was given alone. It was found that HR decreases occurred to CS+ and HR increases to CS-. No reliable differences were found between movement activity during the CS+ and CS-. It is conceivable that HR decreases in this

study reflected conditioned excitation and that HR increases reflected conditioned inhibition, although this was not specifically addressed or tested for by the authors.

Martin (1975) recorded HR and movement activity in rats receiving discriminative conditioning. Two tones, differing in frequency, were used as the CSs, and the US was electric shock. Unlike the Yamaguchi and Iwahara experiment, the results of this study indicated that HR decreases occurred to both the CS+ and CS-, although HR decreases to CS- were smaller than those to CS+. Moreover, only during the first third of the CS--US interval was there evidence of reliable differences between movement activity during the CS+ and CS-. In general, however, conditioned movement reactions were not systematically associated with learned changes in HR. It was suggested that HR responses elicited by the CSs were mediated by processes other than those controling skeletal-motor activity.

Cunningham, Fitzgerald, and Francisco (1977) compared heart-rate responses in two groups of rats that were exposed to explicitly-unpaired or truly-random control procedures. Initially in the paired phase, both groups were given excitatory (forward) conditioning trials in which one stimulus (CS+) was paired with the shock US. Next, in the "unpaired" phase, the explicitly-unpaired group was given 96 explicitly-unpaired trials with another CS (CS-) and the same US. The truly-random group received the same temporal sequence of CSs as the explicitly-unpaired group, but the US occurred randomly. The CSs were two tones of different frequencies. Following this

training, summation and retardation tests were carried out in both groups.

It was found that during the paired phase, heart-rate decreases (i.e., excitatory CRs) developed to CS+. In the "unpaired" phase, small heart-rate decreases were shown by the truly-random group, whereas heart-rate increases eventually developed in the explicitly-unpaired group. Even though this outcome satisfied the requirements of the reaction-of-the-reverse-sign test, the summation test with CS+CS- delivered together indicated no differences between the groups. In the retardation test, it was found that with respect to the truly-random group, the acquisition of the heart-rate CRs was slower in the explicitly-unpaired group. The authors suggested that the fact that the direction of the HR responses in the explicitly-unpaired group during the "unpaired" phase (HR acceleration) was opposite to that seen in the paired phase (HR deceleration), coupled with retarded reacquisition, gave credence to the proposition that the explicitly-unpaired CS was a conditioned inhibitor. However, the authors cautioned that a competing-response interpretation could not be overlooked as an alternative explanation of the results.

As cited by Rescorla and Wagner (1972), two groups of rabbits were trained by Wagner and Saaveda in an eyelid-conditioning situation using four CSs (A, B, C, and X). A and B were tones of different frequencies, C a flashing light, and X a vibratory stimulus. During excitatory training, varying associative strengths were established to A, B, and C by using different numbers of reinforced

trials. A+ was paired with a shock US 240 times, B+ 8 times, and C+ 548 times. In inhibitory training, X-, a novel CS, was compounded with A+ and B+ and not reinforced. These compound trials were interspersed among paired presentations of A+ and B+ alone. In the test phase, reinforced presentations of C+ alone and of the (C+X-)+ were given.

It was found that, during the excitatory and inhibitory-training phases, both groups showed a larger percentage of CRs to A+ than to B+. During the test phase, both groups responded equally well to C+ but responded differentially to the (C+X-)+ compound. The group trained with the relatively strong CS (A+) in compound with X showed a greater reduction in percentage of CRs than did the group trained with the relatively weak CS (B+) in compound with X-. These data suggested that the amount of inhibitory potential accrued to X- was a function of the associative strength that was established to A+ and B+ during excitatory conditioning.

In summary, the above three sections generally support the formulation that a negative relationship between the CS and US has different consequences than do neutral or positive relationships (Rescorla & Wagner, 1972). More specifically, such procedures as discrimination, conditioned inhibition, explicitly-unpaired presentations of the CS and US, and to a lesser extent, backward conditioning seem to produce CSs that have inhibitory tendencies owing to the fact that each procedure has in common a negative correlation between the CS and US. On the other hand, procedures in which the

CSs are given alone or in which random CS and US presentation are made appear to produce CSs that are relatively neutral. Furthermore, forward pairings of the CS and US tend to produce CSs with excitatory tendencies presumably resulting from a positive correlation between the CS and US.

Ethanol and inhibition

The purported ability of ethanol to "disinhibit" or "release from suppression" certain behaviors that are normally held in check has been a commonly-held belief in the lay community. Thus, it is not surprising that the investigation of this particular aspect of the drug be carried out in a scientific setting. The following section is concerned with studies dealing with the excitatory and inhibitory consequences of various doses of ethanol on behavior.

As cited in summaries by Andreyev (1934), perhaps some of the earliest reports dealing with the effects of ethanol came from Pavlov's laboratory. One such report was that of Zavadski (1908) in which doses ranging from 0.2 to 1.5 g/kg ethanol were given to dogs through a stomach tube. Although not immediately clear from the summary, it appears that, in a positive (i.e., forward pairing of the CS and US) conditioning procedure, the larger doses eliminated both salivary CRs and URs. This loss lasted for 30 to 60 min for URs and 2 to 4 hr for CRs. Smaller doses, on the other hand, reduced the magnitudes of the URs and only temporarily eliminated the CRs.

Subsequent to this study, another investigator from Pavlov's laboratory, Nikiforovski (1910), examined the effects of ethanol doses ranging from .15 to .50 g/kg administered rectally. In this experiment, a discrimination (CS+, CS-) paradigm was employed. Lower doses of ethanol were reported to affect inhibition such that responding to the CS+ was not influenced while responding to the CS- increased. Higher doses abolished responding to the CS+ and did not change responding to CS-. Thus, it was suggested that lower doses of ethanol demonstrated a "disinhibitory" effect.

Using a single dog, Andreyev (1934) first established conditioned salivary responses to a series of positive stimuli and established no responding to a series of negative stimuli. Then the effects of single doses of ethanol, administered through a gastric fistula, were examined. These doses ranged from .12 to 2.11 g/kg. The lower doses interrupted the established discrimination, leading to increased responding to CS- and decreased responding to CS+. Higher doses first augmented CS- responding then debilitated responding to both positive and negative stimuli. Administration of water in a control session led to no changes in responding. It was concluded that the most pronounced effect of ethanol was general depression of the central nervous system. Initially, ethanol moderately depressed both positive and negative responses, but in later stages, this depression was greatly increased such that all salivary responses were eliminated.

Five dogs were used by Gantt (1935) to study the effects of ethanol on motor and salivary conditioned responses. He employed three dose levels: (1) small, .4 g/kg; (2) moderate, 1.2 g/kg; (3) large, 2.4 to 3.2 g/kg. For each dose, solutions were diluted to 20% with milk and water and administered orally. Drug effects were evaluated by comparing CRs and URs of the dogs after administration of the drug with CRs and URs recorded after the dogs had consumed milk and water only. In general, dose-dependent decreases in CRs and URs occurred, with the CRs being influenced more than URs and with the largest doses showing the greatest depression. In at least one case, discrimination between positive and negative CSs was greatly impaired such that the reaction to the negative CS increased and matched that to the positive CS. Dworkin, Bourne, and Raginsky (1937) administered a series of ethanol dosages (2.0 to 4.0 g/kg) to dogs and cats that were trained to lift a lid for food in the presence of the CS+ and to withhold responding in the presence of the CS-. The lowest dose (2.0 g/kg) was found to produce ataxia and some loss of discrimination, whereas 2.4 and 2.8 g/kg doses led to total loss of discrimination (i.e., equal responding to CS+ and CS-). The highest dose (4.0 g/kg) abolished all responding to both CSs. The authors interpreted their results to indicate a "disinhibitory" effect of ethanol.

Two dose levels of ethanol (.8 and 2.4 g/kg) were used by Fitzgerald and Stainbrook (1977) to study the effects of that drug

on classically conditioned HR responses in rats. The lower dose decreased the magnitude of HR orienting responses but failed to affect HR CRs, whereas the higher dose eliminated both responses and changed the direction of the HR UR from a monophasic increase to a biphasic decrease-increase reaction. It was suggested that the lower dose may have interfered with neuronal pathways of the auditory system such that the subjective intensity of the tone CS was reduced. The higher dose was thought to have led to generalized depression of the central nervous system.

Stainbrook (1978) examined the effects of two doses of ethanol (.8 and 1.6 g/kg) on excitatory and inhibitory processes pertaining to classically conditioned HR and movement. Initially, three separate groups of rats were given excitatory conditioning trials to CS+. Then, employing a different CS (i.e., CS-), one group was given CS- alone trials, the second group received truly-random CS- and US presentations, and the third group was given explicitly-unpaired CS- and US presentations. The rats in the explicitly-unpaired group were partitioned into three equally-sized drug groups. The animals in these subgroups were given intraperitoneal injections of either saline, .8 g/kg, or 1.6 g/kg ethanol. Finally, two tests for inhibition were carried out, positive induction and retardation conditioning. During these tests, animals in each drug group were partitioned into three additional groups which received either saline, .8 g/kg, or 1.6 g/kg ethanol.

It was found that during inhibitory training, the directions of the HR responses of the three groups to CS- were not the same. The CS- alone group showed HR decreases, matching the HR decreases that occurred in all groups to CS+ during excitatory training. The truly-random group displayed variable reactions to CS-, consisting of relatively small HR increases and decreases. The explicilty-unpaired group, on the other hand, demonstrated HR increases to CS-. During the induction test, the presentation of the CS- immediately prior to the CS+ did not modify responding to the CS+ in any of the groups. In the retardation test, the explicilty-unpaired group showed slower acquisition of a decelerative HR CR to CS- than the truly-random or CS-alone groups. It was concluded that the explicitly-unpaired CS- may have developed inhibitory tendencies not present in the CS-alone or truly-random CS- conditions. At the same time, however, it was cautioned that alternative explanations of retarded acquisition to CS- (i.e., peripheral competing response hypothesis) were also tenable.

In terms of drug effects, it was found that both .8 and 1.6-g/kg doses of ethanol retarded the development of HR increases in the explicitly-unpaired group during inhibitory training. Reversal learning was more rapid in those animals that maintained the same drug state for both inhibition training and inhibition testing than in those animals that were switched from saline to ethanol or from ethanol to saline. These outcomes were interpreted in terms of ethanol's ability to disrupt discrimination responding and in terms of state-dependent learning factors.

Most of the above studies seem to suggest that ethanol, at low to moderate doses, possesses a selective effect on inhibition. In many cases this effect was indexed by the disruption of an established discrimination between positive and negative stimuli such that responding to the negative stimulus was no longer suppressed (i.e., a "disinhibitory" effect). It should be pointed out, however, that unlike the studies from the earlier sections (i.e., classical-to-instrumental transfer, CER, and direct-measure studies), many of these reports fail to provide any specific tests for inhibition such as summation or retardation. In addition, the use of the salivary response in many of the experiments makes the assessment of inhibitory tendencies difficult by not providing an above-zero baseline on which reactions of the reverse sign can be directly observed. Therefore, in light of these deficits, the proposition that ethanol has "disinhibitory" properties is not well supported.

Rationale

It has been proposed (Rescorla, 1975) that organisms are capable of learning not only that two events tend to go together (i.e., tend to co-occur), but also that two events tend not to go together (i.e., tend to occur apart). These two types of learning have been discussed in terms of conditioned excitation and conditioned inhibition, respectively. According to the Rescorla-Wagner model of conditioning (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972), conditioned excitation is specified in terms of changes in associative

strength of stimuli positively associated with a US. Hence, if a single stimulus (A) is paired with a US, then the changes in associative strength of A on each trial is determined by the following equation:

$$\Delta V_A = \alpha_A \beta_1 (\lambda - V_A).$$

In this equation, α_A and β_1 are rate constants related to the CS and US, respectively. The asymptotic level of conditioning that the US can support is expressed by λ. The difference between this asymptote and the instantaneous level of conditioning, V_A, determine changes in associate strength. Thus, associative strength changes whenever there is a discrepancy between the expected and actual US.

Rescorla and Wagner (1972) suggested that inhibitory-training procedures, such as explicitly-unpaired presentations of a CS and a US and discrimination training can be viewed as variations of the conditioned-inhibition [A+ vs (A+X)-] format. That is, in the case of the explicitly-unpaired situation, background cues (A+) are reinforced on US-alone trials, but are also present and given in compound (A+X)- with the explicitly-unpaired CS (X-) on CS-alone trials. Thus, this situation yields a format similar to conditioned inhibition, namely, A+ vs (A+X)-. For discrimination conditioning, background cues (A) are compounded with a discrete CS (B) and that compound (A+B+) is reinforced on paired trials. Another CS (X) is presented in combination with background cues (A+) and that combination (A+X)-

is not reinforced. Hence, discrimination training also produces a format similar to conditioned-inhibition, namely A+B+ vs (A+X)-.

In each of these paradigms, conditioned inhibition to AX- can be specified in terms of changes in associative strength of both stimuli resulting from nonreinforcement:

$$\Delta V_A = \alpha_A \beta_2 (0 - V_{AX}),$$

$$\Delta V_X = \alpha_X \beta_2 (0 - V_{AX}).$$

In the above equations, α_A and α_X are rate constants related to the two CSs, and β_2 is a rate constant related to the absence of the US. The asymptotic level of conditioning supported by nonreinforcement is assumed to be zero. V_{AX} is thought to be the sum of V_A and V_X.

Assuming X is neutral (i.e., $V_X = 0$) at the beginning of training and given that A has had some excitatory pretraining, V_{AX} will be positive since V_A is positive. Inasmuch as V_{AX} is subtracted from zero in the above equations, this quantity $(0 - V_{AX})$ will be negative. Therefore, the associative strength of V_A is reduced on (A+X)- trials but restrengthened on A+ trials. The associative strength of V_X is also reduced on (A+X)- trials but not affected on A+ trials. The associative strength of V_X is also reduced on (A+X)- trials but not affected on A+ trials. The associative strength of V_X will be repeatedly reduced and eventually become negative (i.e., inhibitory). Hence, the amount of inhibitory potential that X- gains is a function of the excitatory potential of A+.

In the conditioned-inhibition procedure, the associative strength of A is maintained and augmented by A+ trials. Since V_X depends on the strength of A+ and A+ is present on (A+X)- nonreinforced trials, then this procedure should be especially effective in establishing inhibitory tendencies to X-.

In the case of the explicitly-unpaired procedure, background cues (A+) are reinforced occasionally but never in the presence of the explicit CS (X-). Thus, background cues (A+) become excitatory, but the associative strength of A+ only is increased only on the US-alone trial. During the intertrial interval extinction of the association is assumed to occur. The effect of this is a weaker associative strength of A+ and an overall weaker inhibitory potential to X-.

It is anticipated that discrimination conditioning should produce the weakest inhibitory potential. In this procedure, one CS, X-, is never reinforced and all USs are preceded by a discrete CS+ (B). This tends to block or overshadow conditioning to background cues (A+) when both A+ and B+ are given in compound (A+B+) are reinforced. Because blocking virtually prevents increases in associative strength to A+, very little associative strength can accrue to A+ and this should allow X- to acquire very little inhibitory potential.

It will be recalled that most of the evidence for conditioned inhibition was provided by experiments concerned with skeletal-motor responses with but a single response being measured in each experiment. Hence, conclusions drawn from these sources may not necessarily

extend to autonomic-nervous system responses. Moreover, it is possible that autonomic responses may have also been conditioned in these studies. However, the failure to record such responses eliminated the opportunity to examine possible interactions of skeletal-motor and autonomic responses.

That some of the findings related to inhibitory tendencies in skeletal-motor responses also hold in autonomic reactions has been demonstrated by Cunningham et al. (1977). In their study, it was found that increases in HR occurred to explicitly-unpaired CSs, whereas decreases in HR occurred to paired CSs. Because of this opposing direction of the HR responses and the relative difficulty of establishing HR CRs to the explicilty-unpaired CS, the authors suggested that the explicitly-unpaired CSs may have developed inhibitory tendencies.

One purpose of the current investigation was to compare the effects of explicitly-unpaired presentations of the CS and US with those of two other procedures that are thought to generate inhibition. These were the conditioned inhibition paradigm and discrimination conditioning. Two response systems were examined, HR and skeletal-motor movement activity. The use of these two responses provided the opportunity to test for differential changes in the autonomic and skeletal-motor systems and for possible interactions between the two response systems. Moreover, the inclusion of HR holds the added advantage of providing an above-zero baseline on which both inhibitory and excitatory tendencies can be evaluated.

As discussed earlier, inhibitory tendencies are expected to have behavioral consequences which can be detected by using specific test procedures. Because it is assumed that a conditioned inhibitor will control response tendencies that are directionally opposite to those of a conditioned excitor, the presentation of a conditioned inhibitor should diminish the responses to a conditioned excitor in a summation test. During reversal (relearning) conditioning the development of a CR to a conditioned inhibitor should be retarded. Moreover, it has been suggested that positive outcomes in both of these tests are required to rule out alternative interpretations of inhibitory-like changes in responding (Rescorla, 1969a, b).

One such alternative interpretation involves shifts in attention (Hearst, 1972). It might be argued that a putative inhibitor exerts its decremental effect during summation testing by attracting attention. Presumably, increased attention to the inhibitor would produce decreased attention to the excitor and consequently reduce the excitatory response strength attached to the excitor. However, such an increment in attention, while leading to response decrements in the summation test, should be expected to facilitate, rather than retard, reversal learning. Therefore, increased attention to an inhibitor would satisfy summation criterion but fail the reversal criterion.

If, on the other hand, an inhibitor failed to be noticed, then it could be argued that this attentional deficit could lead to retarded reversal learning. However, not attending to an inhibitor would not be expected to affect responding to the excitor in a

summation test. Again, the criterion for one test is met, but the criterion for the second test is not. Since it is unlikely that an inhibitor will both be noticed during summation and ignored during reversal testing, positive outcomes to these two tests are generally considered to rule out attentional hypotheses.

At the same time, however, it should be pointed out that the combination of these summation and reversal tests fails to rule out the presence of competing responses, which could provide another alternative explanation for inhibition-test outcomes (cf., Gormezano & Kehoe, 1975). Under this hypothesis, it can be postulated that animals could readily learn peripheral responses during inhibitory training which would later compete with the performance of CRs.

A second purpose of the current investigation was to assess the effects of two doses of ethanol, 1.0 and 2.0 g/kg, on the inhibition-producing capabilities of the three inhibitory-training conditions. Ethanol was given during the summation and reversal test phases so that the effects of the drug on established inhibitory tendencies could be evaluated. As indicated earlier, ethanol has been purported to possess "disinhibitory" capabilities. These capabilities may be manifested by reducing decremental effects of inhibitors in a summation test or by facilitating the acquisition of excitatory CRs to inhibitors during reversal training.

METHOD

Subjects

The subjects were 90 hooded, Long-Evans, female rats purchased from Simonsen Laboratories and housed by the Department of Animal Care at the University of Oregon Health Sciences Center on a 12-hr, light-dark cycle. The rats ranged in weight from 250 to 300 g and were given food and water ad lib prior to training.

Apparatus

The rats were restrained in an inverted-U-shaped, Plexiglas holder purchased from Narco Bio-systems, Inc. Sliding plastic inserts were positioned in front of and behind each animal to inhibit movement. Under the floor of the holder was mounted a ceramic phonocartridge with a 10-cm long, 1.0-mm diameter, 1.6 g metal rod inserted into the needle housing. The cartridge was part of the system used to detect gross skeletal-motor activity. The holder was situated in a small-animal, Industrial Acoustic Corporation, sound-isolation chamber equipped with a 7.5-cm ventilation fan and an 8.3-cm wall-mounted speaker through which white noise (75 dB re .0002 μbars) was delivered to mask extraneous sounds.

The electrocardiogram (ECG) was recorded on a Grass Model-5 polygraph from two 20-ga hypodermic needles, inserted just under the skin, opposite each other on either side of the rat's thoracic cavity. A record of the heart beats occurring within each trial was provided by an automated system described previously (Fitzgerald, Vardaris & Teyler, 1968). This system contained a miniature,

low-force-lever switch (Robertshaw; 3a, 250v) that was mounted directly above the ECG polygraph pen in such a way that it was activated by the R wave of the QRS complex during each cardiac cycle. Each triggering of the switch was coded in a transistorized counting network, and at the end of selected time periods, the accumulated heart-beat totals were punched on a Tally paper-tape perforator. Periodically, the accuracy of the recording system was verified by substituting a 10-Hz signal for the incoming ECG signal.

Another channel of the Grass polygraph was used to record skeletal-motor (movement) activity. The output of the floor-mounted phonocartridge was fed into the bridge circuit of a Grass 5P1 preamplifier whose sensitivity was set at .5 mv/cm. The output of the preamplifier was fed into a Tektronix, AM 502, differential amplifier that magnified the signals between .1 and 30 kHz by a factor of 200. The spike-like activity was sensed by a Massey Dickinson resistive shift trigger and accumulated in a transistorized counting circuit. The accumulated contents of the counting circuit were punched on paper tape at the end of the same counting periods as those used for HR. The sensitivity of the system was such that most small movements other than those associated with respiration were detected.

There were three CSs, two auditory and one visual. The auditory CSs were 10.5-sec, 85-dB (re .0002 μbar) tones produced by Hewlett Packard oscillators. One tone (T_1) had a frequency of 500 Hz and was interrupted at a rate of 150-msec on, 150-msec off. The other

tone (T_2) had a frequency of 1.8 kHz and was continuous. The tones were delivered through separate, 10-cm Altec Lansing speakers located on a platform directly in front of the animal. The visual CS was a flashing light (L) that was produced by activating a miniature 28 v dc, 1-cm diameter lamp at a rate of 300-msec on, 300-msec off. The lamp was situated directly in front of the animal at eye level. The US was a .5-sec, 1.3-ma, 60-cycle ac electric shock produced by a Grason Stadler Shock Generator (B6070B) and delivered through the ECG electrodes. A relay was used to lock out the ECG signal during the .5-sec interval in which the US was presented. The intensity of the shock was monitored periodically by measuring the voltage drop across a fixed 100-ohm resistor in series with the animal.

Two rats were trained concurrently in two identically equipped chambers. Trials alternated between rats and were initiated by a film-tape programmer. Specific events within a trial, such as the duration of the CS and US and the duration of counting intervals, were programmed and timed by Massey Dickinson logic modules whose repeat accuracy was .05%.

Procedure

As shown in Table 1, the training procedure consisted of four phases that were carried out over four days. At the beginning of each day, the rats were allowed 15 min to adapt to the restrainer and sound-isolation chamber. In Phase 1, the excitatory or paired phase, all animals (N = 90) received 24 presentations of the 500-Hz, interrupted tone CS (T_1+) paired with the US in a delayed-conditioning paradigm. The CS--US interval was 10 sec, with the US occurring and overlapping the final .5 sec of the CS. The intertrial intervals were randomly varied over a range of 150, 180, and 210 ($\bar{x}$ = 180) sec.

In Phase 2, the inhibitory or "unpaired" phase, the animals were divided into groups of 30 animals each that received 192 training trials. One group (conditioned inhibition) was given 96 presentations of the 1.8-kHz, continuous tone CS (T_2+) paired with the US and 96 presentations of this CS coupled with the light CS (L) to form a compound CS (T_2+L)- that was not paired with the US. The second group (discrimination) received 96 trials with T_2+ being paired with the US and 96 trials with the light (L-) being presented alone. In each of these two groups, the trial types [T_2+ or (T_2+L)- and T_2+ or L-] were delivered in a semi-randomized sequence, with the restriction that no more than two consecutive trials of either type could occur in succession. As in Phase 1, the CS--US interval on paired trials was 10 sec and the US overlapped the final .5 sec of the CS. The intertrial intervals were randomly varied over a range of 150, 180, and 210 ($\bar{x}$ = 180) sec. The third group of animals was given an explicitly-

Phase 1 EXCITATORY TRAINING (Day 1)

Groups (N = 30)	Stimulus Conditions
"Conditioned Inhibition"	24 T_1+ trials
"Discrimination"	"
"Explicitly Unpaired"	"

+ = paired with or having a history of being paired with the US

- = not paired with or having a history of not being paired with the US

Phase 2 INHIBITORY TRAINING (Days 1, 2, and 3)

Groups (N = 30)	Stimulus Conditions
Conditioned Inhibition	96 T_2+ and 96 (T_2+L)- trials
Discrimination	96 T_2+ and 96 L- trials
Explicitly Unpaired	96 (T_2L)- and 96 US-alone trials

Phase 3 COMBINED CUE (Day 4)

Groups (N = 30)	Drug Treatment (N = 10)	Stimulus Conditions
Conditioned Inhibition	Saline	4 T_1+, 4 L-, and 12 T_1+ L- trials
Discrimination	1.0 g/kg ethanol	"
Explicitly Unpaired	2.0 g/kg ethanol	"

Phase 4 REVERSAL CONDITIONING (Day 4)

Groups (N = 30)	Drug Treatment (N = 10)	Stimulus Conditions
Conditioned Inhibition	Saline	48 (L-)+ trials
Discrimination	1.0 g/kg ethanol	"
Explicitly Unpaired	2.0 g/kg ethanol	"

Table 1. A summary of the experimental design, including the type of training, type of stimulus conditions, and drug treatments during each phase of the study.

unpaired procedure comprised of 96 trials with the T_2 and L compound CS (T_2L)- being given alone and 96 trials with the US alone. For this group, the two trials types [(T_2L)- and US alone] alternated, beginning with a US-alone trial. The time between successive USs was randomly varied among 150, 180, and 210 sec intervals ($\bar{x}$ = 180 sec). The intertrial intervals between a US and a CS were randomly varied over a range of 70, 90, and 110 ($\bar{x}$ = 90) sec.

In Phase 3, the three groups of 30 rats were divided into three subgroups of 10 rats each. Animals in these subgroups were injected intraperitoneally with the vehicle or one of the two ethanol doses (1.0 or 2.0 g/kg). Starting with a 95% ethanol solution, 13.3% and 26.6% ethanol solutions were prepared by dilution (v/v) with normal saline. Administration of these two drug concentrations, on the basis of 1 ml/100 g of body weight, yielded ethanol dosages of 1.0 g/kg and 2.0 g/kg, respectively. These dosages were given to animals in the two drug-treatment groups. An equivalent volume of the vehicle (normal saline) was given on the basis of 1 ml/100 g of body weight to animals in the drug-control group. After a 15-min absorption period, all rats received four nonreinforced (T_1+)- trials to check on the residual HR CR to the tone CS that was used in Phase 1, four nonreinforced (L-)- trials to provide an estimate of the capacity of the light by itself to produce a HR reaction and 12 nonreinforced combined-cue trials in which (T_1+)- and (L-)- were presented together in compound (T_1+L-)-. The ordered sequence of these trials was as follows:

$(T_1+)-$, $(T_1+)-$, $(L-)-$, $(L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+)-$, $(T_1+)-$, $(L-)-$, $(L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$, $(T_1+L-)-$.

During the final phase, Phase 4, all animals were given 48 reversal-conditioning trials in which the light (L-)+ CS was now paired with the US. As was true of Phases 1 and 2, the CS--US interval was 10 sec, the duration of the US was .5 sec, and the intertrial interval randomly fluctuated around a mean of 180 sec.

On the first day, 24 excitatory-conditioning trials (Phase 1) and 48 inhibitory-training trials were given (part of Phase 2). On each of the following two days, 72 additional inhibitory-training trials were given to complete Phase 2. On the fourth day, four $(T_1+)-$ alone, four $(L-)-$ alone, and 12 $(T_1+L-)-$ trials (Phase 3) and 48 (L-)+ reversal trials (Phase 4) were administered.

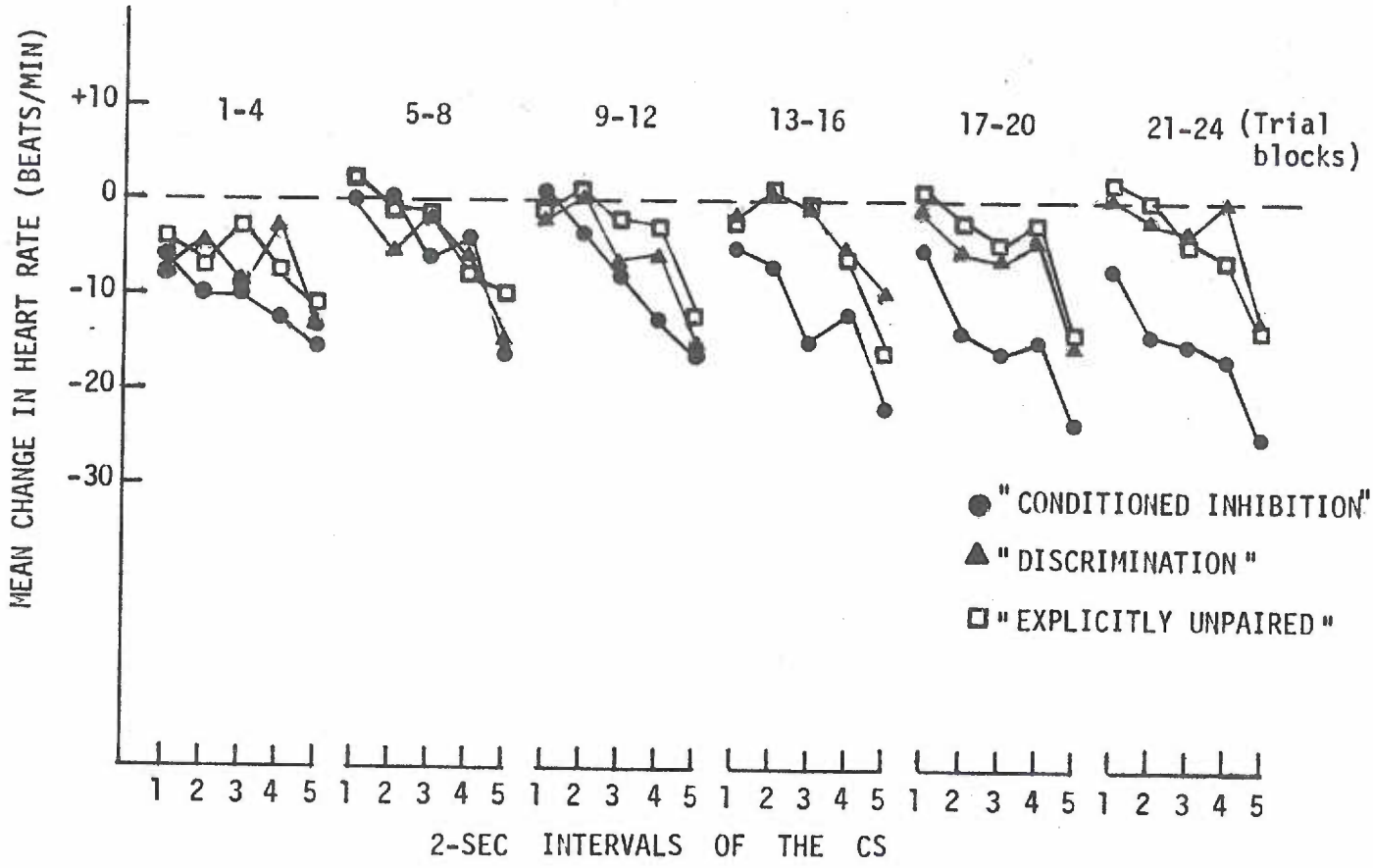
Heart rate and movement activity were recorded in seven successive counting intervals within each trial. The first interval was 10 sec in length and occurred immediately prior to the delivery of the CS. This interval provided baseline measures of HR and movement. The subsequent intervals were 2 sec in duration and occurred in succession beginning with CS onset. Heart rate and movement activity during the 10-sec interval was subtracted from the HR and movement activity in each of the 2-sec intervals to form difference scores for each trial.

RESULTS

Phase 1 - Excitatory Conditioning

During Phase 1, all animals were given 24 excitatory conditioning trials using T_1+ (interrupted 500-Hz tone) as the reinforced CS. The mean T_1+ minus pre-T_1+ HR responses of the "conditioned inhibition," "discrimination," and "explicitly-unpaired" groups (n = 30 in each case) in successive 2-sec periods of T_1+, averaged across six blocks of four trials each, are plotted in Figure 1. It can be observed in this figure that the directions of the HR CRs of each of the three groups to T_1+ were predominantly decelerative. The figure also shows that the CRs were larger toward the end of T_1+ or shortly before the presentation of the US than at the beginning of T_1+. Finally, the terminal level of the CR of the "conditioned-inhibition" group appeared to be slightly larger than that of the other two groups. The presence of a significant trial blocks effect, F (5, 435) = 2.73, $p < .05$, in a 3 by 6 by 5 ("inhibitory training" by trial blocks by counting periods) analysis of variance supports the conclusion that the overall magnitude of the CRs changed over trials. There was also a significant counting periods effect, F (4, 348) = 92.82, $p < .001$, and a significant Counting Periods by Trial Blocks interaction, F (20, 1740) = 1.88, $p < .05$, indicating that the overall change in HR across counting periods (i.e., topography) was reliable and that the topography changed reliably across trial blocks. In addition, there was an "Inhibitory Training" by Trial Blocks interaction,

Figure 1. Mean T_1+ minus pre-T_1+ HR reactions of the "conditioned inhibition", "discrimination", and "explicitly-unpaired" groups during successive 2-sec periods of the CS, averaged in six consecutive blocks of four excitatory conditioning trials each.


MEAN CHANGE IN HEART RATE (BEATS/MIN)
+10
0
-10
-20
-30
1-4
5-8
9-12
13-16
17-20
21-24 (Trial blocks)
"CONDITIONED INHIBITION"
"DISCRIMINATION"
"EXPLICITLY UNPAIRED"
1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5
2-SEC INTERVALS OF THE CS

F (10, 435) = 2.21, $p < .05$. This interaction was likely due to chance differences between the groups because no experimental manipulations of the groups had been made at this point in the experiment. Thus, the groups factor was a dummy variable.

Phase 2 - Inhibitory Conditioning

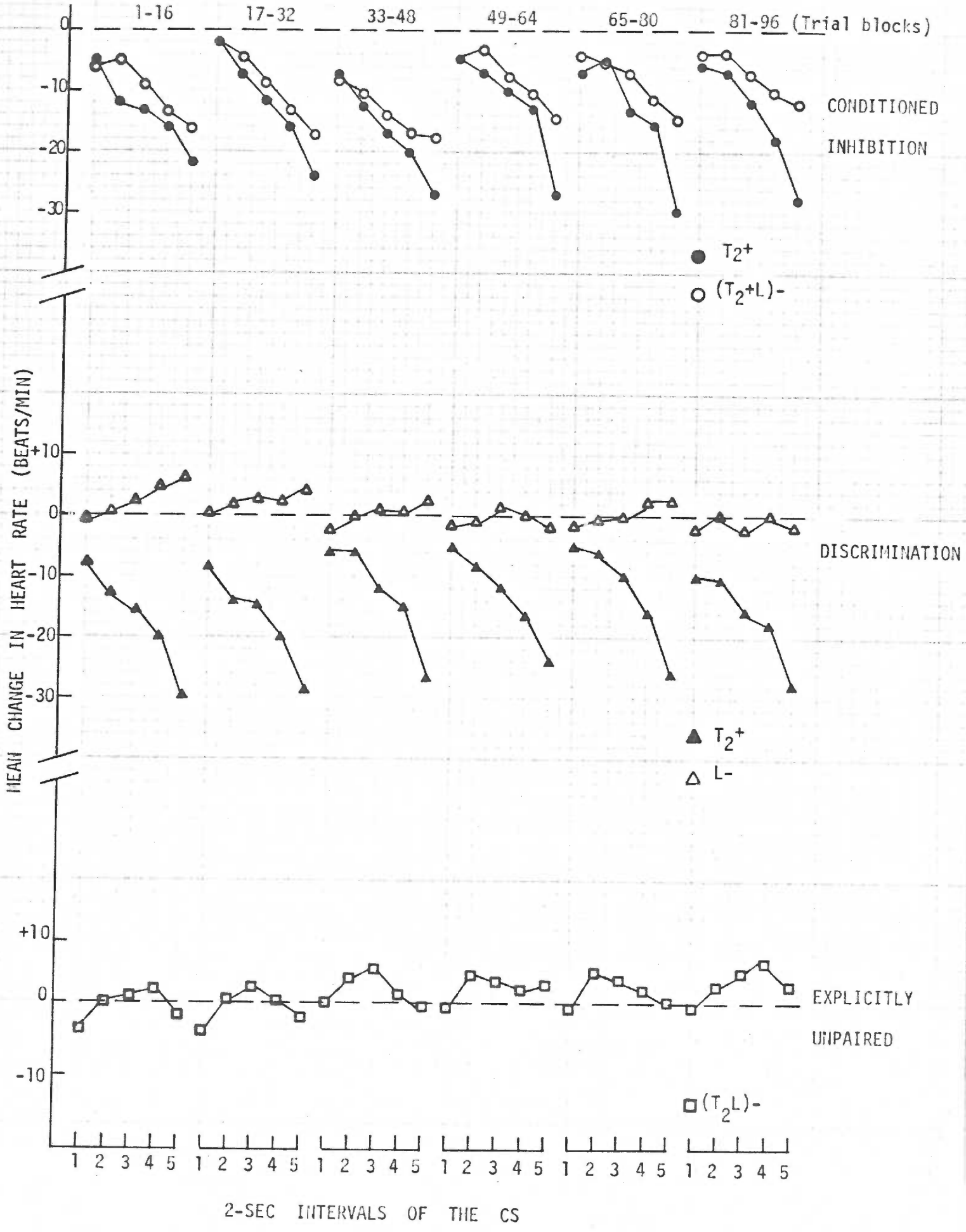
In Phase 2, there were three inhibitory treatment conditions that were given to three separate groups of animals. The three conditions were: conditioned inhibition, consisting of reinforced T_2+ (continuous 1.8-kHz tone) trials versus nonreinforced (T_2+L)- (tone plus interrupted light) trials; discrimination, consisting of reinforced T_2+ trials versus nonreinforced L- trials; and explicitly unpaired, comprised of nonreinforced (T_2L)- trials versus US-alone trials. The mean CS minus pre-CS HR responses to the various CSs within the three groups are shown in Figure 2. The reactions are plotted in successive 2-sec periods of the CSs averaged across six blocks of 16 trials each. Focusing first on the conditioned-inhibition group depicted at the top of the figure, it can be observed that the HR reactions to the reinforced (T_2+) and nonreinforced (T_2+L)- CSs were very similar. In both cases, the directions of the responses were cardiodecelerations. Moreover, the topographies of the reactions were such that maximum HR decelerations occurred toward the end of the CSs. The principal difference between the responses to the two CSs was in terms of response magnitude. In general, the magnitudes of the HR reactions to T_2+ were larger than those to (T_2+L)-, especially toward the end of the CSs. In addition, the responses to T_2+ became larger across trial blocks, while the responses to (T_2+L)- tended to

stay the same. By the final trial block, the HR change in the last 2 sec of T_2+ was -27 bpm and to (T_2+L)-, -12 bpm.

A 2 by 6 by 5 (type of CS by trial blocks by counting periods) analysis of variance performed on the data of the conditioned-inhibition group demonstrated a significant type of CS effect, F (1, 29) = 50.87, $p < .001$, indicating that the HR responses to T_2+ were reliably larger than those elicited by (T_2+L)-. There was a significant counting periods effect, F (4, 116) = 66.12, $p < .001$, and a significant Trial Blocks by Counting Periods interaction, F (20, 580) = 1.97, $p < .01$. These outcomes showed that the overall form or topography of the HR reactions was reliable and that this form changed reliably across trial blocks. The analysis also contained a significant Type of CS by Counting Periods interaction, F (4, 116) = 48.91, $p < .001$, and a significant Type of CS by Trial Blocks by Counting Periods interaction, F (20, 580) = 2.02, $p < .01$. The last two interactions demonstrate that the form of the HR responses to T_2+ and (T_2+L)- were reliably different from each other and that this difference developed across trial blocks.

Turning to the discrimination group shown in the middle of Figure 2, it can be noticed that the reinforced T_2+ CS produced HR reactions having the same decelerative direction and topography as those that occurred to T_2+ in the conditioned-inhibition group. In sharp contrast, however, to what occurred in the conditioned-inhibition group, the nonreinforced L- CS given to the discrimination

Figure 2. Mean CS minus pre-CS HR responses to the reinforced and nonreinforced stimuli of the conditioned inhibition, discrimination, and explicitly-unpaired groups in successive 2-sec periods of the CS, averaged across six consecutive 16-trial blocks of inhibition-training trials.


1-16
17-32
33-48
49-64
65-80
81-96 (Trial blocks)
0
-10
-20
-30
CONDITIONED
INHIBITION
T_2^+
$(T_2+L)^-$
MEAN CHANGE IN HEART RATE (BEATS/MIN)
+10
0
-10
-20
-30
DISCRIMINATION
T_2^+
L^-
+10
0
-10
EXPLICITLY
UNPAIRED
$(T_2L)^-$
1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5
2-SEC INTERVALS OF THE CS

group produced small acceleration-deceleration types of reactions. A modest HR acceleration to L-, evident on the first block of trials, rapidly decreased in size to a near zero level by the second or third trial block. One final point is that the overall magnitudes of the HR decelerations of the discrimination group to T_2+ did change appreciably over trials with maximum deceleration occurring within the first block of 16 trials.

A 2 by 6 by 5 (Type of CS by trial blocks by counting periods) analysis of variance produced a significant type of CS effect, F (1, 29) = 63.28, $p < .001$, and a significant Type of CS by Trial Blocks interaction, F (5, 145) = 4.61, $p < .001$, indicating that the overall mean HR reactions to T_2+ and L- were reliably different from each other and that this difference developed across trial blocks. There was also a significant counting periods effect, F (4, 116) = 107.81, $p < .001$, a significant Type of CS by Counting Periods interaction, F (5, 145) = 4.61, $p < .001$, and a significant Type of CS by Trial Blocks by Counting Periods interaction, F (20, 580) = 1.87, $p < .05$. These outcomes demonstrate that the forms of the responses to T_2+ and L- were reliably different, and that this difference developed across trial blocks.

To compare the HR responses of the conditioned inhibition and discrimination groups to the reinforced and nonreinforced stimuli, a 2 by 2 by 6 by 5 (inhibitory training by type of CS by trial blocks by counting period) analysis of variance was performed. Only outcomes related to the inhibition-training factor will be presented. There

was a significant effect of inhibitory training, F (1, 58) = 4.82, p < .05, a significant Inhibitory Training by Trial Blocks interaction, F (5, 290) = 2.34, p < .05, a significant Inhibitory Training by Type of CS interaction, F (1, 58) = 7.75, p < .01, and a significant Inhibitory Training by Type of CS by Counting Periods interaction, F (4, 232) = 2.48, p < .05. These outcomes demonstrate that the differentiation of the reinforced and nonreinforced stimuli was better in the discrimination group than in the conditioned-inhibition group and that this difference was reflecting in contrasting response topographies of the two groups to the nonreinforced stimulus.

At the bottom of Figure 2 are shown the HR responses of the explicitly-unpaired group to the nonreinforced (T_2L)- compound CS. In contrast to both of the other groups, the explicitly-unpaired group displayed HR responses to the nonreinforced CS that were mainly accelerative in direction. On the first two trial blocks of the unpaired phase, the responses of the explicitly-unpaired group contained both decelerative and accelerative components. However, on succeeding blocks of trials the reaction became more accelerative in nature. Although the overall magnitude of the HR acceleration was relatively small, averaging approximately +6.0 bpm in the final trial block, there was very little between-subject variability in its occurrence. Thus, on the last trial block, twenty of the thirty animals in the explicitly-unpaired group displayed a mean HR increase to the nonreinforced (T_2L)- compound. Moreover, separate t tests showed that the overall mean HR increases were significantly different from zero during the final three trial blocks of inhibition training (p < .05 in each case). One final

point has to do with the form of the HR response of this group. It can be noticed that throughout training, the response had a U-shaped form, with maximum HR accelerations occurring in the middle of the CS. This is in contrast to the forms of the HR responses shown by the other groups where maximum HR changes occurred near the end of the various CSs. A 6 by 5 (trial blocks by counting periods analysis of variance on the results of the explicitly-unpaired group established that the trial blocks effect was significant, F (5, 145) = 4.31, $p < .005$, establishing that the change in the direction of the response was reliable. The counting periods effect was also significant, F (4, 116) = 11.36, $p < .001$, indicating that the form of the reaction was reliable.

A comparison of the different HR responses shown by the three groups to the respective nonreinforced CSs in Figure 2 was made in a 3 by 6 by 5 (inhibitory training by trial blocks by counting periods) analysis of variance. This analysis provided a significant inhibitory training effect, F (2, 87) = 26.55, $p < .01$, indicating that the mean HR responses of the three groups were different. A subsequent Newman-Keuls analysis established that the conditioned-inhibition group was reliably different from the discrimination and explicitly-unpaired groups ($p < .05$). The two latter groups were not different from each other. The analysis of variance also demonstrated a significant Inhibitory Training by Trial Blocks interaction, F (10, 435) = 2.55, $p < .01$, indicating that the difference between the groups changed reliably as a function of trial blocks.

A significant counting periods effect, F (4, 348) = 13.10, $p < .001$, indicated that the change in HR within the CS was reliable. A significant Inhibitory Training by Counting-Periods interaction, F (8, 348) = 24.50, $p < .001$, demonstrated that this change was reliably different in the three groups.

To show how the response patterns of the three groups developed to the reinforced and nonreinforced CSs on the early trials of Phase 2, the first block of 16 trials was further divided into successive blocks of four trials. Figure 3 shows the HR responses on these smaller blocks, as well as on the very first trial with each CS. Beginning at the top of the figure, with the conditioned-inhibition group, it can be seen that on Trial 1, the HR reactions to the reinforced T_2+ CS and nonreinforced compound (T_2+L)- CS were quite dissimilar. That is, the direction of the response to T_2+ was a monophasic HR deceleration throughout the CS, while the response to (T_2+L)- consisted of a brief period of HR deceleration in the first counting period of the CS, followed by a more sustained period of HR acceleration in the remaining counting periods. With further exposures to the CSs, the HR reaction to the nonreinforced (T_2+L)- consisted of a brief period of HR deceleration in the first counting period of the CS, followed by a more sustained period of HR acceleration in the remaining counting periods. With further exposures to the CSs, the HR reaction to the nonreinforced (T_2+L)- CS rapidly changed and became more like the response produced by T_2+. Thus, responding to (T_2+L)- changed from a decelerative-accelerative pattern

to a uniformly decelerative pattern within the first four trials. These decelerative responses persisted throughout the next 12 trials, although the magnitudes of the reactions were generally smaller than those produced by T_2+.

A 2 by 4 by 5 (type of CS by trial blocks by counting periods) analysis of variance identical to the one used for the results displayed in Figure 2 was carried out on the blocks of 4-trial data for the conditioned-inhibition group. The outcomes of this analysis essentially matched those obtained for Figure 2. Thus, there was a significant type of CS effect, F (1, 29) = 7.72, $p < .01$, a significant effect of trial blocks, F (3, 87) = 5.62, $p < .05$, a significant counting periods effect, F (4, 116) = 33.93, $p < .001$, a significant Type of CS by Counting Periods interaction, F (12, 348) = 3.69, $p < .001$, and a significant Type of CS by Trial Blocks by Counting Periods interaction, F (12, 348) = 3.11, p .005.

As was true of the responses of the conditioned-inhibition group, the middle part of Figure 3 reveals that the HR reactions of the discrimination group to the reinforced T_2+ CS and nonreinforced L- CS on Trial 1 were also dissimilar. Thus, the direction of the response of the discrimination group on Trial 1 to T_2+ was consistently cardiodecelerative, whereas that to L- was consistently cardioaccelerative. Following these initial exposures, the HR acceleration to L- rapidly became smaller, such that, by the end of the 16 trials, HR changed very little to L-. On the other

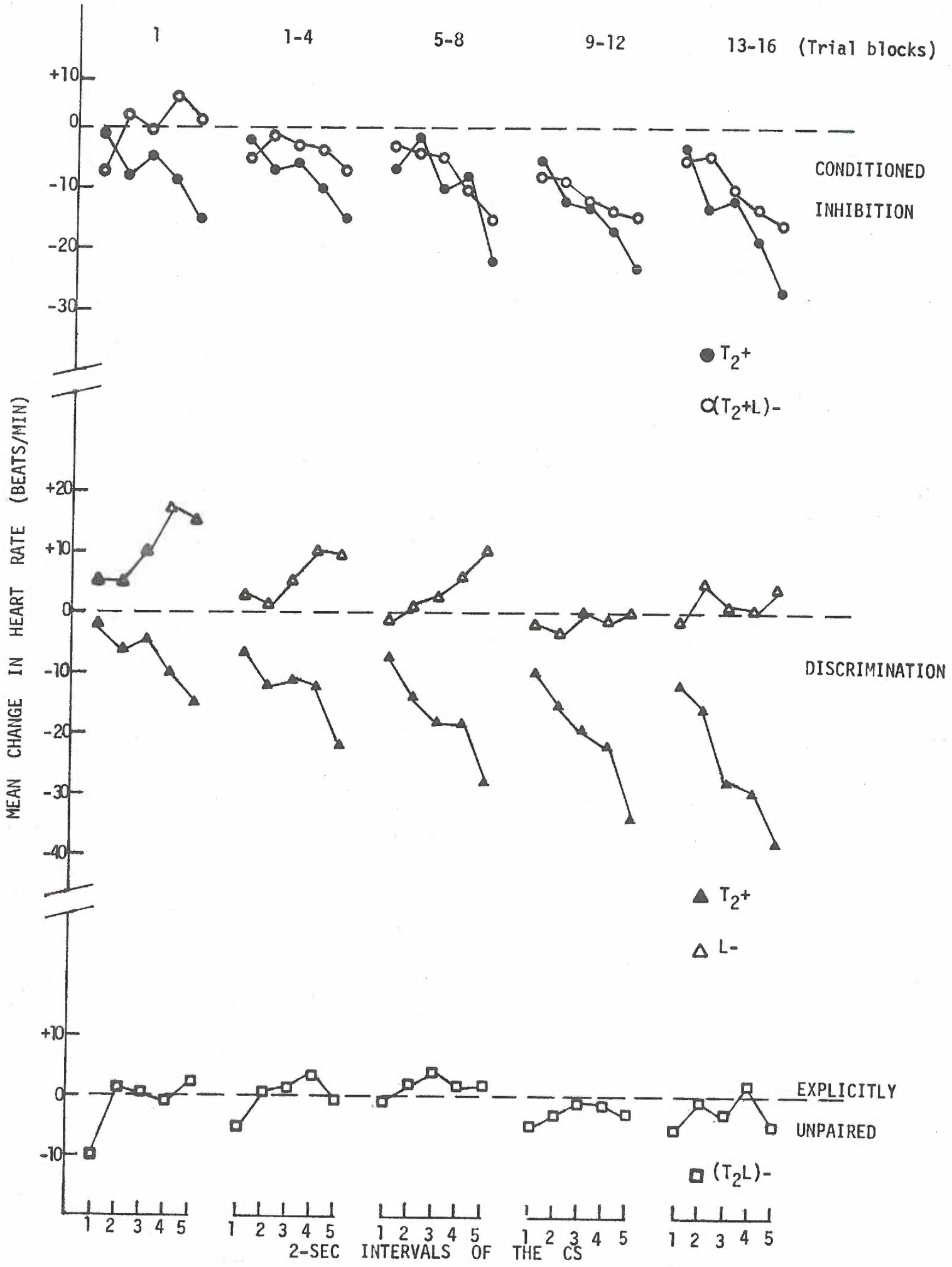
hand, the HR deceleration to T_2+ grew rapidly in size, with the level occurring at the end of the 16 trials being comparable to that which was present over the remaining 80 trials of Phase 2 (see Figure 2).

A 2 by 4 by 5 (type of CS by trial blocks by counting periods) analysis of variance performed on the 4-trial data of the discrimination group generated outcomes that again very nearly matched those pertaining to Figure 2. These outcomes yielded a significant type of CS effect, F (1, 29) = 75.28, $p < .001$, a significant trial blocks effect, F (3, 87) = 9.67, $p < .001$, a significant effect of counting periods, F (4, 116) = 17.22, $p < .001$, a significant Type of CS by Counting Periods interaction, F (4, 116) = 59.72, $p < .001$, and a significant Trial Blocks by Counting Periods interaction, F (12, 348) = 3.28, $p < .005$.

Considering the explicitly-unpaired group shown at the bottom of Figure 3, it can be seen that on Trial 1, the nonreinforced (T_2-) CS produced a biphasic deceleration-acceleration response which was highly similar to that produced by the same CS in the conditioned-inhibition group. On subsequent trials, the magnitude of the decelerative component in the first counting period quickly became smaller, with HR in the remaining periods being decelerative on some occasions and accelerative on others. There was little evidence of a consistent HR acceleration in any of the counting periods in these early trials.

A 4 by 5 (trial blocks by counting periods) analysis of variance corresponding to the one carried out for Figure 2, was performed on

Figure 3. Mean CS minus pre-CS HR responses to the reinforced and nonreinforced stimuli of the conditioned inhibition, discrimination, and explicitly-unpaired groups during the first 16 trials of inhibitory training in successive 2-sec periods of the CS averaged in four consecutive blocks of four trials each.


1
1-4
5-8
9-12
13-16 (Trial blocks)
+10
0
-10
-20
-30
CONDITIONED
INHIBITION
● T_2+
○ (T_2+L)-
MEAN CHANGE IN HEART RATE (BEATS/MIN)
+20
+10
0
-10
-20
-30
-40
DISCRIMINATION
▲ T_2+
△ L-
+10
0
-10
EXPLICITLY
UNPAIRED
□ (T_2L)-
1 2 3 4 5
2-SEC INTERVALS OF THE CS

the 4-trial data of the explicitly-unpaired group in Figure 3. The outcomes of this analysis were similar to those found for Figure 2. Thus, there was a significant trial blocks effect, F (3, 551) = 13.52, $p < .001$, and a significant counting periods effect (F (4, 551) = 7.00, $p < .001$. There was a significant Trial Blocks by Counting Periods interaction, F (12, 551) = 2.73, $p < .005$, which was not reliable in the Figure 2 analysis. This outcome indicates that the form of the response changed reliably in the first four blocks of inhibitory training.

Separate 2 by 5 (type of CS by counting periods) analyses of variance were carried out on just the Trial 1 results of the conditioned-inhibition and discrimination groups that are shown in Figure 3. For the conditioned-inhibition group, the analysis demonstrated a significant type of CS effect, F (1, 261) = 12.36, $p < .001$, and a significant interaction of Type of CS by Counting Periods, F (4, 261) = 3.00, $p < .05$. In the discrimination group, there was a significant CS effect, F (1, 261) = 34.32, $p < .001$. Thus, in the case of both groups the two CSs produced reliably different HR reactions.

CS from Phase 1 (T_1+)- and of the putative inhibitory CS from Phase 2 (L-)- were assessed. This was accomplished by presenting each of the CSs by themselves on each of four trials. The reactions on these trials were used to help evaluate the outcome of the combined-cue trials (T_1+L-)-. It may be recalled that the sequence of trials that was used was: (T_1+)-, (T_1+)-, (L-)-, (L-)-, (T_1+L-)-, (T_1+L-)-, (T_1+L-)-, (T_1+L-)-, (T_1+L-)-, (T_1+L-)-; followed immediately by the same sequence again. The mean CS minus pre-CS HR responses of the three subgroups in each of three inhibitory-training conditions are shown in Figure 4. These data are presented in successive 2-sec periods of the CSs averaged in blocks of four (T_1+)- and (L-)- trials and in two blocks of six (T_1+L-)- combined-cue trials.

The far left of Figure 4 shows that, in general, the directions of the HR responses of the various groups in each of the periods of (T_1+)- were decelerative. In contrast to Phase 1, the magnitudes of these decelerations were relatively small. In fact, only the 1.0 g/kg conditioned-inhibition and explicitly-unpaired groups showed substantial HR decreases to (T_1+)-. The figure also shows that in general, the forms of the HR responses to (T_1+)- in Phase 2 were such that maximal HR changes occurred in the middle of the CS, with HR returning to baseline by the end of the CS. This is in contrast to Phase 1 in which the HR responses of each group to (T_1+)- were uniformly maximal toward the end of the CS.

A 3 by 3 by 5 (inhibitory training by drug treatment by counting periods) analysis of variance carried out on the (T_1+)- data demonstrated a significant drug effect, F (2, 81) = 5.38, $p < .01$. A subsequent Newman-Keuls analysis indicated that the mean of the combined 1.0

Figure 4. Mean CS minus pre-CS HR reactions of the conditioned inhibition, discrimination, and explicitly-unpaired groups (including the various drug groups) to $(T_1+)-$, $(L-)-$, and $(T_1+L-)-$ in successive 2-sec periods of the CS averaged in blocks of four trials each for $(T_1+)-$ and $(L-)-$ and in two blocks of six trials each for $(T_1+L-)-$.

g/kg ethanol groups ($\bar{x}$ = -10.4) was significantly larger ($p < .05$) than the mean of the combined 2.0 g/kg ethanol groups ($\bar{x}$ = -1.6). The analysis of variance also included a significant counting periods effect, F (4, 325) = 3.47, $p < .01$, indicating that the overall form of the HR reactions to (T_1+) was reliable.

Figure 4 also indicates that the HR reactions of the various groups to (L-) (the panel next to $[T_1+]$) were variable in direction and that the magnitudes of the HR changes were relatively small. For example, the 1.0 g/kg discrimination groups displayed slight cardioaccelerations to (L-) while the 1.0 g/kg explicitly-unpaired group showed slight cardiodecelerations. In the case of the 1.0 g/kg conditioned-inhibition group, the HR change contained both accelerative and decelerative components. The forms of the responses to (L-) were also quite variable with the largest HR changes occurring in some cases in the beginning of (L-), in others in the middle of (L-), and in still others at the end of (L-). A 3 by 3 by 5 (inhibitory training by drug treatment by counting periods) analysis of variance carried out on the L- data provided no significant outcomes.

Turning to the combined-cue data displayed in Figure 4, it is clear that the directions of the reactions to the (T_1+L-) compound CS were variable. On the other hand, cardiodecelerations occurred in the 1.0 g/kg conditioned-inhibition and explicitly-unpaired group, but not in the 1.0 g/kg discrimination group. On the other hand, small decelerations also occurred in the 2.0 g/kg discrimination and explicitly-unpaired groups but not in the conditioned-inhibition group. At the same time, small cardioaccelerations were prevalent in the saline groups, especially in the second trial

block in the discrimination group and in both trial blocks in the explicitly-unpaired group. Similar to the reactions to (L-)-maximal HR reactions occurred near the end of (T_1+L-)-in some groups, and near the middle or the beginning in others.

The combined-cue data were subjected to a 3 by 3 by 2 by 5 (inhibitory treatments by drug treatments by trial blocks by counting periods) analysis of variance, which yielded a significant drug-treatment effect, F (2, 81) = 5.49, $p < .01$. A follow-up Newman-Keuls analysis revealed that the mean of the combined 1.0 g/kg ethanol groups ($\bar{x}$ = -6.4) was reliably larger ($p < .05$) than the mean of the combined saline group ($\bar{x}$ = +.7). The mean of the combined 2.0 g/kg ethanol groups ($\bar{X}$ = -2.8) was not different from those of the other combined groups. The analysis of variance generated a significant Drug Treatment by Counting Periods interaction, F (8, 324) = 2.55, $p < .05$, demonstrating that the combined drug-treatment groups had reliably different HR response topographies to (T_1+L-). In addition, there was a significant Trial Blocks by Counting Periods interaction, F (4, 324) = 2.51, $p < .05$, indicating that the overall form of the responses to (T_1+L-) changed reliably across the two trial blocks.

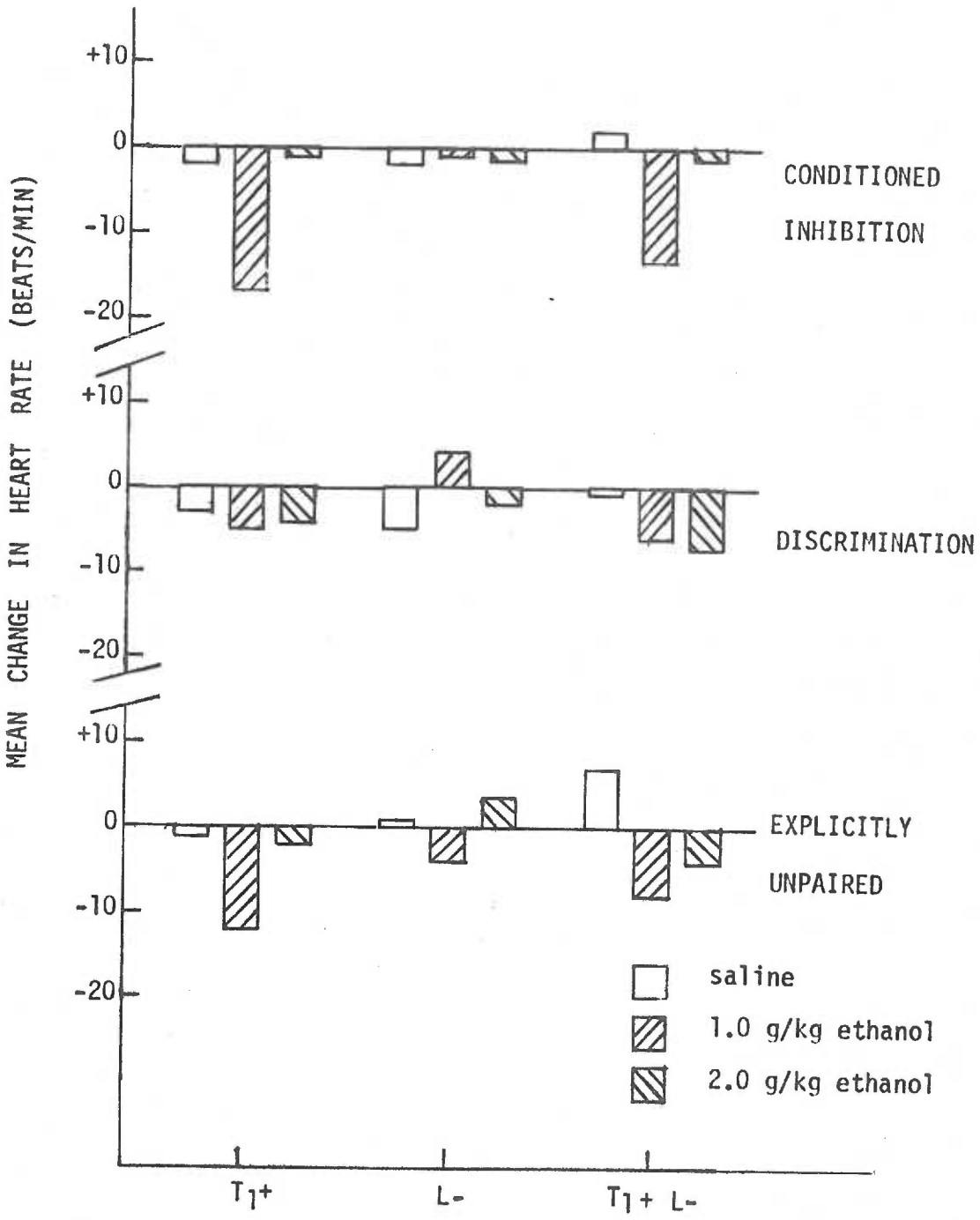
To facilitate comparisons of the overall magnitudes of the HR reactions of the groups to the three stimuli shown in Figure 4 (i.e., [T_1+], [L-], [T_1+L-]), the data of the nine groups comprising this figure were averaged over the five periods of the CSs and plotted in the form of bar graphs in Figure 5. These data represent the means of the four (T_1+) and (L-)-trials and the means of the first

block of six (T_1+L-)-trials. Paired *t* tests were used to evaluate the differences within groups.

Comparing (T_1+)-and (L-)-in the conditioned-inhibition groups, it can be observed that these stimuli elicited very modest HR changes in the saline and 2.0 g/kg groups. But, in the 1.0 g/kg group, (T_1+)-elicited relatively large HR decelerations while (L-)-elicited relatively small decelerations, t (9) = 2.24, $p < .05$. The 1.0 g/kg discrimination group displayed HR decreases to (T_1+)-and HR increases to (L-); t (9) = 2.65, $p < .05$. In the explicitly-unpaired groups, minimal differences between (T_1+)-and (L-)-were apparent in the saline group, whereas the ethanol groups showed marked differences. The overall HR decelerations to (L-)-were reliably smaller than the HR decelerations to (T_1+)-in the 1.0 g/kg group, t (9) = 3.36, $p < .05$. Moreover, the reactions of the 2.0 g/kg group to (T_1+)-and (L-)-, which were opposite in direction, were reliably different, t (9) = 2.50, $p < .05$.

Comparing (T_1+)-with (T_1+L-)-in the conditioned-inhibition groups reveals that the saline group showed reactions of opposite direction to these stimuli; in the 1.0 and 2.0 g/kg groups slightly larger HR decelerations seemed to occur to (T_1+L-)-than to (T_1+)-. However, paired *t* tests indicated that none of these differences reached significance. In the discrimination groups, the HR decelerations to (T_1+)-and to (T_1+L-)-were approximately the same. In the explicitly-unpaired saline groups, small decelerations occurred to (T_1+)-alone, and relatively larger accelerations occurred to the (T_1+L-)-compound. This difference was reliable, t (9) = 2.63, $p < .05$. In summary,

Figure 5. Overall magnitude of the HR responses of the conditioned inhibition, discrimination, and explicitly-unpaired groups (including the various drug groups) to (T_1+), (L-), and (T_1+ L-)-averaged in blocks of four trials each for (T_1+)-and (L-)-and in one block of six trials for (T_1+ L-)-.


MEAN CHANGE IN HEART RATE (BEATS/MIN)
+10
0
-10
-20
CONDITIONED
INHIBITION
DISCRIMINATION
EXPLICITLY
UNPAIRED
saline
1.0 g/kg ethanol
2.0 g/kg ethanol
T1+
L-
T1+ L-

the only evidence of inhibition, as indexed by HR reaction of opposite direction to (T_1+) and (T_1+L-) CSs occurred in the explicitly-unpaired saline group.

Phase 4 - Reversal training

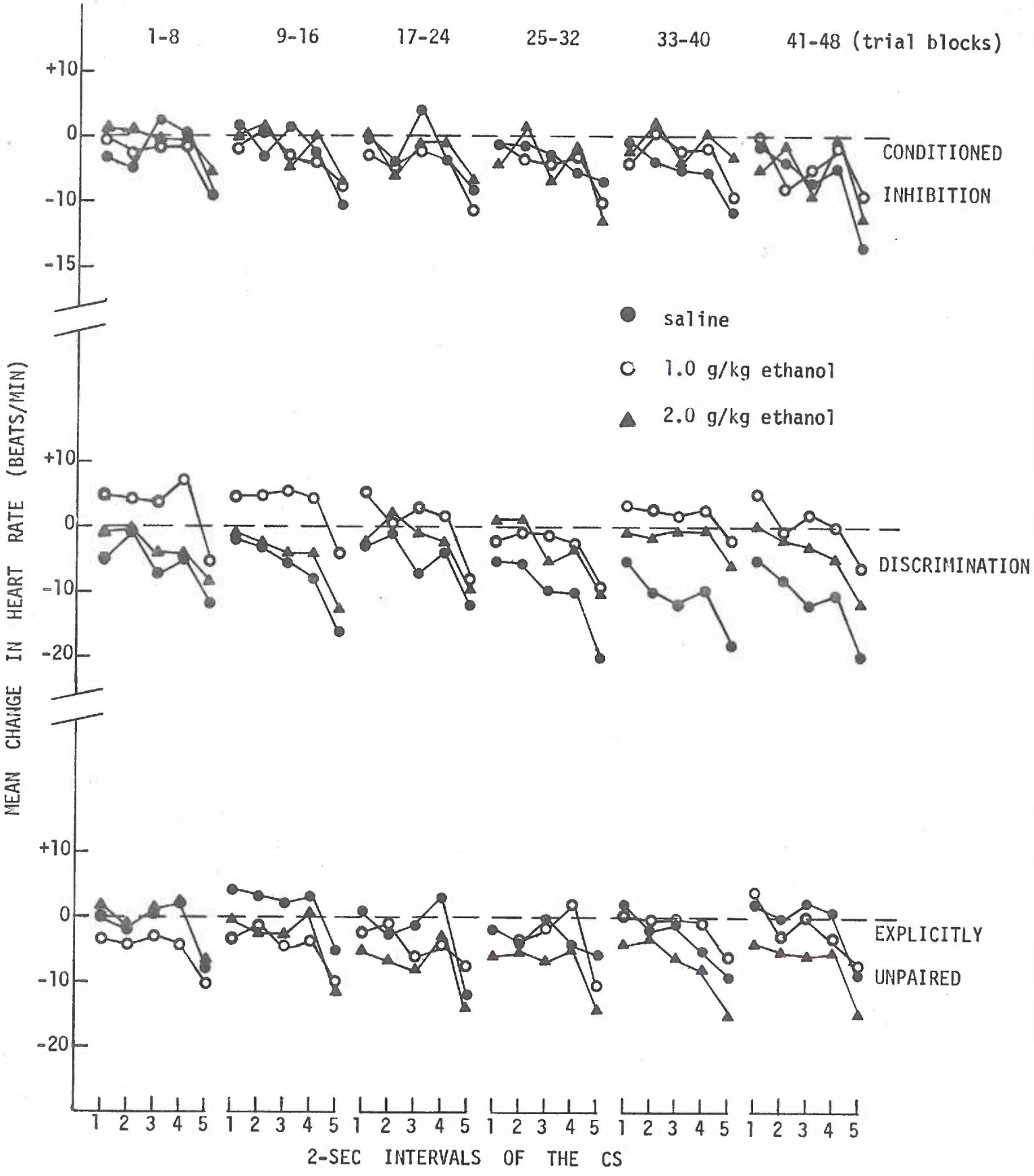
During Phase 4, all animals were given 48 reversal conditioning trials in which the previously nonreinforced stimulus (L-) was now paired with the US. The (L-)+ minus pre-(L-)+ reactions in successive 2-sec periods of the CS were averaged across six blocks of eight trials each and are plotted in Figure 6. Beginning at the top of the figure, it is apparent the directions of the HR CRs of the CS were averaged across six blocks of eight trials each and are plotted in Figure 6. Beginning at the top of the figure, it is apparent the directions of the HR CRs of all three conditioned-inhibition groups to (L-)+ were cardiodecelerations that developed gradually over trials. In addition, the forms of these responses matched those of Phase 1, with the magnitudes of the cardiodecelerations being maximal just prior to the delivery of the US. The overall magnitudes of the HR CRs that developed to (L-)+ in the three conditioned-inhibition groups were roughly equivalent.

As seen in the middle of Figure 6, the reactions of the discrimination groups to (L-)+ during reversal conditioning were not the same. The directions of the responses in the 1.0 g/kg ethanol group tended to be biphasic, consisting of initial cardioaccelerations followed by a single 2-sec period of cardiodeceleration. The directions of the responses of the 2.0 g/kg ethanol and saline groups were generally monophasic cardiodecelerations. In the saline discrimination group, HR decreases were present in each period of the CS and they became progressively larger in magnitude across trials. In general, the CRs of the saline group were larger than those of the other two groups.

Looking at the bottom of Figure 6, it can be pointed out that the CRs of the explicitly-unpaired groups were similar to those of the conditioned-inhibition groups. In general, the directions of the responses were cardiodecelerations in all three groups and the forms of the responses were similar to those of Phase 1. It should be noted, however, that the saline group, especially during early trial blocks, displayed a biphasic acceleration-deceleration response form that was similar to that of the 1.0-g/kg discrimination group.

A 3 by 3 by 6 by 5 (inhibitory training by drug treatment by trial blocks by counting periods) analysis of variance, carried out on the data from Figure 6, yielded a significant Inhibitory Training by Drug Treatment interaction, F (4, 81) = 4.78, $p < .005$. This interaction reflects the relative superiority of the discrimination saline group in the development of HR CRs to (L-)+. Subsequent Newman-Keuls analyses indicated that only among the discrimination groups were there reliable differences. In this case, the magnitudes of the HR decreases of the discrimination-saline group were significantly larger ($p < .05$) than those of the 1.0-g/kg and 2.0-g/kg ethanol discrimination groups. Furthermore, the HR responses of the discrimination saline group ($\bar{x} = -8.5$) were significantly ($p < .05$) larger than those of the conditioned-inhibition ($\bar{x} = -3.7$) and explicitly-unpaired ($\bar{x} = -1.9$) saline groups. The analysis of variance also provided a significant trial blocks effect, F (5, 405) = 3.10, $p < .01$, and a significant counting periods effect, F (4, 324) = 170.64, $p < .001$, indicating that the overall HR CR developed

Figure 6. Mean (L-)+ minus pre-(L-)+ HR reactions of the conditioned inhibition, discrimination, and explicitly-unpaired groups (including the various drug groups) in successive 2-sec periods of (L-)+, averaged across six consecutive blocks of eight reversal-training trials each.


1-8
9-16
17-24
25-32
33-40
41-48 (trial blocks)
+10
0
-10
-15
CONDITIONED
INHIBITION
saline
1.0 g/kg ethanol
2.0 g/kg ethanol
+10
0
-10
-20
DISCRIMINATION
MEAN CHANGE IN HEART RATE (BEATS/MIN)
+10
0
-10
-20
EXPLICITLY
UNPAIRED
1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5
2-SEC INTERVALS OF THE CS

across trial blocks and that the overall form of the HR response to (L-)+ was reliable.

Frequencies of trials showing increases, decreases, or no change in HR to positive and negative CSs

The present section provides additional information on the types of HR responses that occurred to the positive and negative CSs in the various phases of the experiment. This was accomplished by sorting the trials in each phase into three categories. The categories were trials in which HR increased to the CS, decreased to the CS, or did not change in the presence of the CS. The mean frequencies of these trials from Phases 1 and 2 and the corresponding percentages that they represent of the total trials are portrayed in Table 2. An inspection of this table reveals that, during excitatory conditioning in Phase 1, the T_1+ CS was more likely to produce HR decreases than increases or no changes in HR. This pattern was apparent for all three groups. Wilcoxon matched-pair signed-rank tests indicated that the HR-decrease category was a significantly larger than the HR increase or no change in HR categories in all three groups (significance level of at least $p < .05$ in each case). In the "discrimination" and "explicitly-unpaired" groups, the HR-increase categories were significantly larger ($p < .05$) than the no change in HR category. There were no significant differences between the groups in any of the three HR categories.

Table 2 also shows that during Phase 2, the conditioned inhibition and discrimination groups displayed the same basic pattern of responding to the reinforced T_2+ CS as was shown to T_1+ in Phase 1.

Phase	HR ↑	HR ~	HR ↓	
1 (T_1+)	4.7(20%)	3.7(15%)	15.6(65%)	CONDITIONED INHIBITION GROUP
2(T_2+)	15.8(16%)	10.2(11%)	70.0(73%)	
2(T_2+L)-	21.5(22%)	13.1(14%)	61.4(64%)	
1(T_1+)	6.4(27%)	3.6(15%)	13.9(58%)	DISCRIMINATION GROUP
2(T_2+)	15.7(15%)	8.3(9%)	72.0(75%)	
2(L-)	35.0(36%)	19.0(20%)	42.0(44%)	
1(T_1+)	6.9(29%)	3.7(15%)	12.4(56%)	EXPLICITLY UNPAIRED GROUP
2(T_2L)-	39.0(41%)	16.7(17%)	40.3(42%)	

Table 2. Mean number of trials from the conditioned inhibition, discrimination, and explicitly unpaired groups on which HR increased, did not change, or decreased during Phases 1 and 2. Also, the corresponding percentages of the total trials that each number represents are shown.

That is, the HR-decrease category was reliably larger ($p < .05$) than the other two categories, and the HR-increase category was reliably larger ($p < .05$) than the HR-no-change category. Once again, there were no group differences in the three response categories.

Turning to the nonreinforced CS during Phase 2, it can be seen that the dominant response to $(T_2+L)-$ in the conditioned-inhibition group was a HR decrease with 65% of the trials being of this type. This percentage was significantly different from the no-change percentage as was the 22% increase category ($p < .05$ in each case). In the discrimination and explicitly-unpaired groups, by contrast, the percentage number of HR decreases and increases to the nonreinforced CS was approximately the same. Thus, in the discrimination group, HR decreases occurred on 44% of the L- trials and HR increases on 36% of the L- trials. In the explicitly-unpaired group, HR decreases were present on 42% of the $(T_2L)-$ trials and HR increases on 41% of the $(T_2L)-$ trials. Mann-Whitney-U tests revealed that the numbers of HR-increase trials to the nonreinforced CS in Phase 2 shown by both of these groups were significantly larger than those shown by the conditioned-inhibition group ($p < .05$ in each case). Also, the numbers of HR-decrease trials displayed by these groups to the same CS were significantly smaller than those shown by the conditioned-inhibition group.

Table 3 depicts for each group, the mean frequencies and the corresponding percentages of trials in which HR increased,

Phase(CS)	HR ↑	HR ~	HR ↓	
		CONDITIONED-INHIBITION GROUPS		
3(T_1+ L-)	4.9(41%)	1.8(15%)	5.3(44%)	Saline
3(T_1+ L-)	1.4(12%)	2.1(18%)	8.5(70%)	1.0 ethanol
3(T_1+ L-)	2.7(22%)	4.3(36%)	5.0(42%)	2.0 ethanol
4(L+)	13.7(26%)	9.9(19%)	28.4(55%)	saline
4(L+)	12.7(24%)	11.4(22%)	27.9(54%)	1.0 ethanol
4(L+)	10.1(10%)	14.5(28%)	27.4(53%)	2.0 ethanol
		DISCRIMINATION GROUPS		
3(T_1+ L-)	4.7(39%)	1.4(12%)	5.9(49%)	saline
3(T_1+ L-)	3.7(31%)	1.9(16%)	6.8(53%)	1.0 ethanol
3(T_1+ L-)	1.7(14%)	3.9(16%)	6.4(53%)	2.0 ethanol
4(L+)	7.7(15%)	9.2(18%)	35.1(68%)	saline
4(L+)	17.9(34%)	11.1(21%)	23.0(44%)	1.0 ethanol
4(L+)	10.1(19%)	13.6(26%)	28.3(55%)	2.0 ethanol
		EXPLICITLY-UNPAIRED GROUPS		
3(T_1+ L-)	5.7(48%)	1.2(9%)	5.1(43%)	saline
3(T_1+ L-)	2.1(18%)	3.1(26%)	6.8(56%)	1.0 ethanol
3(T_1+ L-)	2.9(24%)	4.1(34%)	5.0(42%)	2.0 ethanol
4(L+)	15.5(30%)	9.2(17%)	27.3(53%)	saline
4(L+)	11.8(22%)	12.9(25%)	27.3(53%)	1.0 ethanol
4(L+)	10.6(20%)	10.9(21%)	30.5(59%)	2.0 ethanol

Table 3. Mean frequencies of trials for the conditioned inhibition, discrimination, and explicitly-unpaired groups (including the various drug groups) in which HR increased, did not change, or decreased from baseline during the 12 combined-cue trials in Phase 3 and during the 48 reversal trials from Phase 4. Also shown are the percentage number of the total HR trials in Phases 3 and 4 that correspond to the three categories.

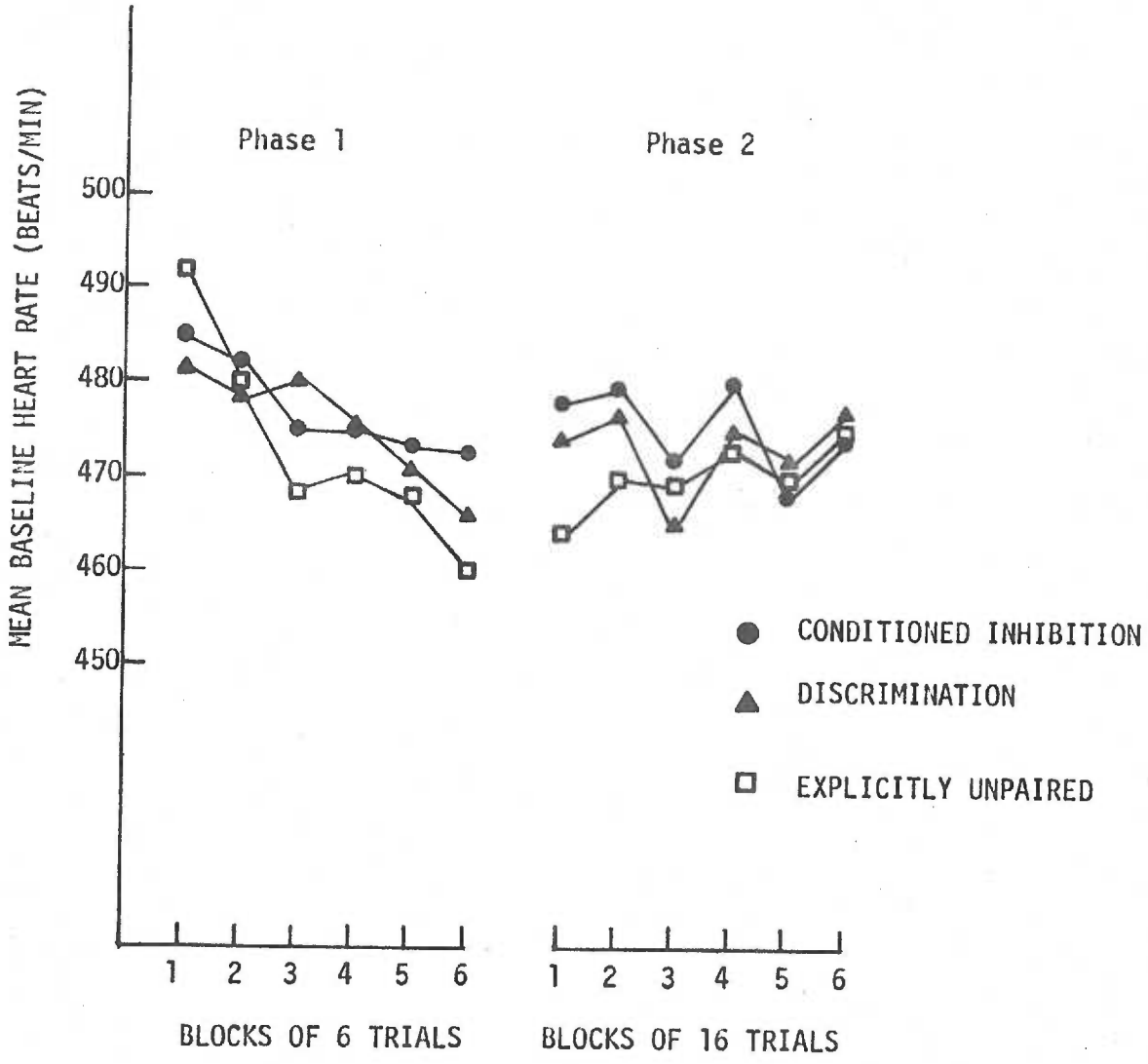
did not change, or decreased during the combined-cue (T_1+ L-) trials in Phase 3 and during the reversal conditioning (L-)+ trials in Phase 4. Starting with Phase 3 it can be seen that each of the saline groups showed approximately the same number of HR increases and decreases to T_1+ L- on the combined-cue trials. The actual percentages were: Saline combined-cue, 41% HR increase and 44% HR decrease; saline discrimination, 39% HR increase and 49% HR decrease; and saline explicitly-unpaired, 48% HR increase and 43% HR decrease. In each case, the percentages were significantly different from the no change in HR category ($p < .05$) but not from each other. In contrast to the saline groups, the ethanol groups displayed more HR decreases to T_1+ L- on the combined-cue trials than HR increases. Separate Wilcoxon matched-pair signed-rank tests established that the differences between the two types of trials were significant for all of the ethanol groups.

Inspection of the Phase 4 results in Table 3 shows that the pattern of responding to the (L-)+ CS resembled very closely that which occurred to the reinforced stimuli from Phases 1 and 2 (i.e., T_1+ and T_2+). Namely, it was more likely for HR to decrease to (L-)+ than to increase or not change. This was true of the saline and ethanol groups alike ($p < .05$ in each case).

Baseline HR

The mean HR baselines of the conditioned inhibition, discrimination, and explicitly-unpaired groups are shown in Figure 7 averaged in six consecutive blocks of four trials each for Phase 1

Figure 7. Mean baseline HR levels of the conditioned inhibition, discrimination, and explicitly-unpaired groups averaged in six consecutive blocks of six trials each for Phase 1 and in six consecutive blocks of 16 trials each for Phase 2.

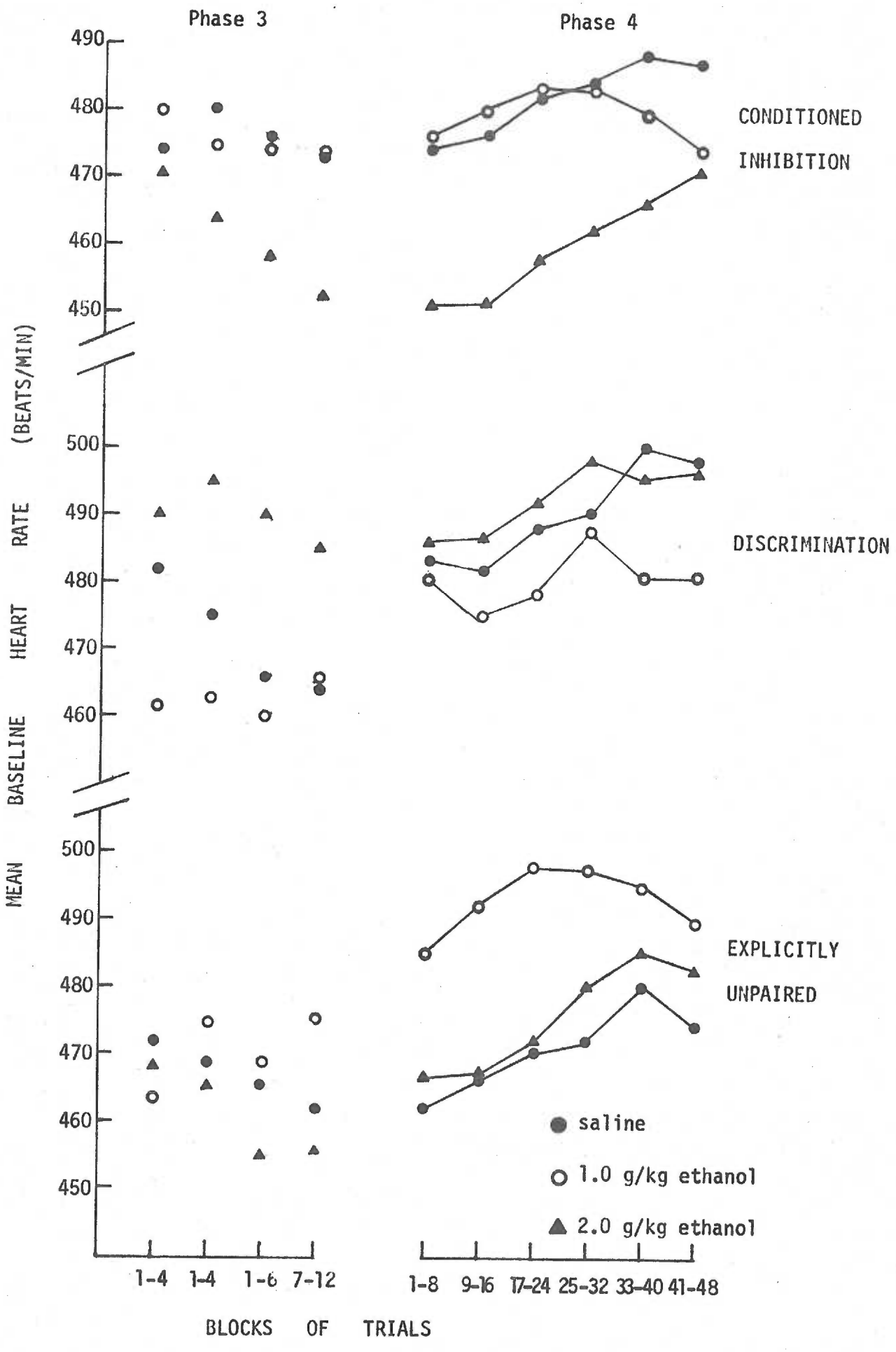

MEAN BASELINE HEART RATE (BEATS/MIN)
Phase 1
Phase 2
500
490
480
470
460
450
1 2 3 4 5 6
BLOCKS OF 6 TRIALS
1 2 3 4 5 6
BLOCKS OF 16 TRIALS
CONDITIONED INHIBITION
DISCRIMINATION
EXPLICITLY UNPAIRED

and in six consecutive blocks of 16 trials each for Phase 2. As seen on the left, it is apparent that in Phase 1, all three groups displayed similar baseline HR levels and that in each group these levels declined across trial blocks. The reliability of this decline was supported by a significant trials blocks effect, F (5, 435) = 14.03, $p < .001$, in a 3 by 6 (inhibitory training by trial blocks) analysis of variance.

The data for Phase 2 shown on the right of the figure were based on HR responding prior to the nonreinforced CS. A preliminary 2 by 2 by 6 (inhibitory training by type of CS by trial blocks) analysis of variance indicated no significant effects of type of CS on baseline HR during Phase 2. Inspection of Figure 7 shows that the baseline HRs of the three groups were highly similar during most of Phase 2. The only significant effect was that of trial blocks, F (5, 435) = 3.07, $p < .05$.

Figure 8 contains the mean HR baseline levels of the various groups during Phases 3 and 4. For Phase 3, the data were averaged in blocks of four trials each prior to T_1+ and L- and in two blocks of six trials each prior to T_1+ L-. For Phase 4, the baseline data were averaged in six consecutive blocks of eight trials each. Separate 3 by 3 (inhibitory training by drug treatment) analysis of variance performed on the baseline levels of T_1+ and L- trials yielded no significant outcomes. The figure shows that, in general, baseline HR of the various groups on T_1+ L- trials tended to decrease across the two trial blocks. A 3 by 3 by 2 (inhibitory training by drug

Figure 8. Mean baseline HR levels of the conditioned-inhibition, discrimination, and explicitly-unpaired groups (including the various drug groups) averaged in blocks of four trials each prior to T_1+ and L- and two blocks of six trials each prior to T_1+ L- for Phase 3 and in six successive blocks of eight trials each for Phase 4.


Phase 3
Phase 4
MEAN BASELINE HEART RATE (BEATS/MIN)
490
480
470
460
450
500
490
480
470
460
500
490
480
470
460
450
CONDITIONED
INHIBITION
DISCRIMINATION
EXPLICITLY
UNPAIRED
saline
1.0 g/kg ethanol
2.0 g/kg ethanol
1-4
1-4
1-6
7-12
1-8
9-16
17-24
25-32
33-40
41-48
BLOCKS OF TRIALS

treatment by counting periods) analysis of variance demonstrated a significant trial blocks effect, F (1, 81) = 8.11, $p < .05$, reflecting the reliability of the decrease in baseline across trials. There was also a Drug Treatment by Trial Blocks interaction, F (2, 81) = 5.53, $p < .005$.

As seen on the right of Figure 8, there was a tendency in Phase 4 for baseline HR in all the groups to increase across trial blocks. Also, the effect of ethanol on HR was not the same within each of the inhibitory training conditions. Thus, baseline HR in the 2.0-g/kg conditioned-inhibition group appeared to be suppressed relative to the two other conditioned-inhibition groups. Also, the 1.0-g/kg explicitly-unpaired group showed higher baseline HR levels than the two remaining explicitly-unpaired groups. A 3 by 3 by 6 (inhibition training by drug treatment by trial blocks) analysis of variance verified the reliability of the increase in HR across trials in providing a significant effect of trial blocks, F (5, 405) = 18.29, $p < .001$. That HR levels changed differentially across trial blocks was indicated by a significant Drug Treatment by Trial Blocks interaction, F (10, 405) = 4.02, $p < .001$.

Movement

As was true of the HR responses, movement activity prior to the CS (i.e., baseline movement) was subtracted from movement activity during each of the 2-sec periods of the CS to yield a series of difference scores. An examination of the raw data of individual animals indicated that movement rarely occurred, either in the presence or absence of the CS.

Thus, the distribution of movement data was skewed and it was reasoned that the median, rather than the mean, would provide a more appropriate measure of movement activity. In each phase, medians were computed over the same size trial blocks as were used for HR. An inspection of these data revealed that, with the exception of a few isolated cases, median baseline and CS difference scores were zero in all groups during each of the four phases of the study.

To determine the extent to which movement that was present on a particular trial influenced HR, all trials during Phases 1 and 2 were sorted on the basis of whether overall HR increased or decreased during the CS. Within each of these HR categories, trials were again sorted on the basis of whether movement increased, did not change, or decreased from baseline. Thus, in all six separate classifications were formed. The six columns in Table 4 show the mean HR changes of each group on the respective types of trials in Phases 1 (i.e., T_1+) and 2 (i.e., T_2+, $[T_2+L]-$, $L-$). Also shown below the means in parentheses are the percentage number of trials in which movement increased, showed no change, or decreased within each of the two HR categories. For both HR categories, the mean HR responses when movement increased or decreased were compared with the mean HR responses when movement did not change using paired *t* tests. Only those animals that provided data for each trial type were used in the tests. Significant outcomes of these tests are indicated by an asterisk.

Phase(CS)	HR↑ trials MV↑	MV~	MV↓	HR↓ trials MV↑	MV~	MV↓	
1(T_1+)	21.3* (24%)	13.2 (36%)	13.0 (40%)	-14.6* (12%)	-17.9 (35%)	-18.0 (53%)	
2(T_2+)	16.0 (27%)	15.2 (36%)	14.0 (37%)	-17.0 (10%)	-19.4 (37%)	-22.4* (53%)	CONDITIONED-INHIBITION GROUP
2(T_2+L-)	18.9 (36%)	16.3 (29%)	16.9 (35%)	-16.6 (13%)	-18.7 (35%)	-20.8 (53%)	
1(T_1+)	22.9* (42%)	12.1 (23%)	22.9* (25%)	-14.8 (20%)	-17.2 (30%)	-16.1 (50%)	
2(T_2+)	17.7* (40%)	12.3 (24%)	12.9 (36%)	-14.6 (10%)	-20.1 (39%)	-22.7* (51%)	DISCRIMINATION GROUP
2(L-)	19.1* (42%)	12.1 (25%)	17.1* (33%)	-9.9 (17%)	-11.2 (40%)	-15.2* (43%)	
(T_1+)	18.1* (28%)	11.7 (30%)	13.1 (42%)	-12.7 (12%)	-15.7 (34%)	-17.7 (54%)	EXPLICITLY-UNPAIRED GROUP
2(T_2L-)	17.9* (48%)	13.5 (23%)	14.8 (29%)	-10.9* (21%)	-13.2 (36%)	-14.6 (43%)	

*p<.05

Table 4. Mean HR increases and decreases (in bpm) of the conditioned-inhibition, discrimination, and explicitly-unpaired groups when movement (MV) increased, did not change, or decreased from baseline during the reinforced and nonreinforced CSs presented in Phases 1 and 2. For each mean, the percentage number of the total HR increase or decrease trials is also shown. An asterisk indicates when the HR means corresponding to increases or decreases in movement were significantly ($p<.05$) different from HR mean corresponding to no change in movement.

Concentrating first on the HR-increase category on the left side of Table 4, it can be observed that for each group and for both excitatory (i.e., T_1+, and T_2+) and inhibitory (i.e., T_2+L- and L-) CSs, the magnitudes of the HR increases were larger when movement increased than when movement did not change. In six out of the possible eight tests (i.e., 75%), these differences were significant ($p < .05$). Looking at the HR-decrease category on the right, there is a tendency for the HR responses of the groups to each CS to be larger when movement decreased, than when movement did not change. It should be noted, however, that this tendency was not as strong as that observed in the case of HR and movement both increasing. Only three out of the possible eight (38%) differences between the movement-decrease and the movement-no-change classifications were significant ($p < .05$). In general, Table 4 shows that there was a tendency for the magnitudes of the HR responses to be facilitated when the direction of movement activity matched that of HR, although this tendency seemed to be stronger for HR increases than for HR decreases.

DISCUSSION

The principle findings of this study were: (1) the HR CRs that were established to the reinforced T_1+ CS during the excitatory paired phase were decelerative in direction with the form of the responses being such that maximum decelerations occurred toward the end of the CS; (2) during the following inhibitory unpaired phase the responses shown to the respective nonreinforced CS- events by the three inhibitory-training groups were not the same. The conditioned-inhibition group displayed monophasic decelerative HR responses to (T_2+L)- compound throughout the "unpaired" phase. The discrimination group showed HR accelerations to the L- CS at the very start of the "unpaired" phase, but with further trials the magnitude of the responses decreased to a near-zero level. In contrast to both of these groups, the explicitly-unpaired group reacted to the nonreinforced (T_2L)- compound with HR accelerations that developed over the course of the "unpaired" phase. The explicitly-unpaired and discrimination groups demonstrated more frequent HR accelerations to their respective CSs- than did the conditioned-inhibition group; (3) during combined-cue testing, the explicitly-unpaired saline group showed HR accelerations to the (T_1+L)- compound that were reliably different from the HR decelerations displayed to T_1+ alone; (4) in the reversal conditioning test, acquisition of HR CRs to L- was retarded in both the explicitly-unpaired and the conditioned-inhibition saline groups when compared

to the discrimination saline group; (5) in the discrimination condition the 1.0 and 2.0-g/kg ethanol groups demonstrated poorer reversal conditioning than the saline group; (6) baseline HR of the three inhibitory-training groups decreased during original excitatory conditioning in Phase 1 and during parts of inhibitory training in Phase 2. During reversal conditioning, baseline HR increased in all groups; and (7) in general, very little skeletal-motor activity occurred during any of the four phases of the experiment. When movement activity was present, it generally tended to facilitate increases in HR, and sometimes impaired decreases in HR. On the other hand, decreases in movement failed to affect increases in HR, but sometimes facilitated decreases in HR.

The development of HR decelerations to the T_1+, T_2+ and (L-)+ CSs that were positively correlated with the US is consistent with the outcomes of a large number of prior studies (Fitzgerald & Hoffman, 1975; Fitzgerald, Martin, & Hoffman, 1974; Fitzgerald, Martin, & O'Brien, 1973; Holdstock & Schwartzbaum, 1965). Also in accordance with these reports was the fact that the forms of topographies of the excitatory HR CRs in the various groups were such that the magnitudes of the HR changes increased during successive intervals of the CS and were maximal just before the US was introduced. This particular response form has been described as resembling Pavlovian inhibition of delay (Fitzgerald, Martin, & O'Brien, 1973).

The observation in the present investigation that it was more likely for HR to decrease than to increase or show no change to the

T_1+ CS during original excitatory conditioning to the T_2+ CS during inhibitory training, and to the (L-)+ CS during reversal training is in agreement with findings reported by Martin (1975). He used a discrimination conditioning procedure and found that HR decreases to CS+ occurred on 70% of the trials. This percentage is comparable to the 65%, 58%, and 56% of trials in which HR decreased to T_1+ in the conditioned inhibition, discrimination, and explicitly-unpaired groups, respectively, in the current study.

One possible explanation for the appearance of decelerative HR CRs to CS events that are positively correlated with an aversive US involves the notion of species-specific defense reactions. As outlined by Bolles (1970), three types of such behaviors can be demonstrated in rats: (1) fleeing, (2) freezing, and (3) attack. Bolles suggested that under certain conditions (i.e., being exposed to electric shock), normal exploratory and grooming behaviors are replaced by one or more of these species-specific defense reactions. Furthermore, these defense reactions are not thought of as random behaviors, but rather are controlled by the particular environment in which the rat is confined. Thus, it is not unreasonable to assume that rats, exposed to the classical conditioning situation in the present experiment in which they were tightly restrained and unable to escape shock, would develop reactions related to natural freezing behavior.

The observation that freezing behavior and HR decelerations often co-occur in rats exposed to an inescapable danger signal was reported

by Hofer (1970). He found that, in six different species of rodents, behavioral freezing consisting of prolonged immobility ranging from 2 to 60 min was accompanied by reduced HR and a high incidence of cardiac arrhythmias. Restrained rats in the present laboratory have occasionally demonstrated arrhythmias similar to those described by Hofer. It is possible that similar processes may be operating in both situations and that restrained rats receiving classical conditioning may develop decelerative HR CRs that are part of general behavioral freezing.

Along these lines, Obrist, Sutterer, and Howard (1972) proposed that HR decelerations might be initiated by a central process that inhibits ongoing skeletal-motor responses. Fitzgerald and Teyler (1970) argued that decelerative HR CRs in restrained rats may result from a preparatory central state that becomes active when the rat learns that skeletal-motor responses will not lead to the escape from or avoidance of the shock US. Once it is learned that the US inevitably follows the CS, the central state produces a cessation in motor responses (i.e., behavioral freezing) and a deceleration in HR. This behavioral freezing notion is supported by the fact that restrained rats in the current experiment displayed very little skeletal-motor activity.

In contrast to decelerative HR reactions to a CS positively correlated with the US, the present study demonstrated that accelerative HR reactions can occur to a CS negatively correlated with the US. Moreover, consistent with prior studies (Cunningham,

Fitzgerald, & Francisco, 1977; Stainbrook, 1978), accelerative HR responses to the explicitly-unpaired CS, in this case (T_2L)-, developed across trials, suggesting that they were learned reactions. In the conditioned-inhibition group, uniformly smaller decelerative HR responses occurred to (T_2+L)- as compared to T_2+. Since the CSs were negatively related to the US in both groups, the respective HR responses suggest that factors other than CS--US correlation are needed to account for the HR changes.

Rescorla (1969a) originally defined a conditioned inhibitor as a stimulus that comes to control a tendency which is opposite to that controlled by a conditioned excitor. Under this definition, the explicitly-unpaired (T_2L)- qualifies as a conditioned inhibitor because the HR responses to this stimulus were accelerative and opposite in direction to the decelerative HR responses that occurred to the paired CSs. More recently, a slightly different definition was offered by Rescorla (1975) in which it was suggested that a conditioned inhibitor interferes with the effects which would normally be produced by a conditioned excitor. In this case, the directionality of the effects was not mentioned. In this context, the L- in the conditioned-inhibition group can also be thought of as a conditioned inhibitor which attenuated the magnitude of the HR decelerations to the (T_2+L)- compound during inhibition training.

Perhaps both of these definitions can be incorporated into a single viewpoint. It could be hypothesized that inhibitory tendencies summate with excitatory tendencies such that the HR response that occurred on any given trial was determined by the algebraic difference between the two tendencies. The initial decelerative HR reactions

shown by the groups to T_2+, $(T_2L)-$, and $(T_2+L)-$ at the beginning of the "unpaired" phase presumably resulted from excitatory tendencies attached to T_1+ generalizing to T_2. With respect to the discrimination and explicitly-unpaired groups, these generalized excitatory tendencies would be expected to weaken due to extinction since T_2 was not reinforced in these groups. In the case of the explicitly-unpaired group, extinction of excitatory potential to T_2 would allow the background cues to accrue excitatory strength since they were paired with the US. This excitatory background could have provided the necessary conditions for the development of inhibition to the non-reinforced $(T_2L)-$ compound CS in this group.

To carry this viewpoint further, it might be argued that the reduced level of decelerative HR responding to $(T_2+L)-$ in the conditioned inhibition group represented the algebraic summation of excitatory (T_2+) and inhibitory (L-) strengths with the excitatory potential being somewhat larger to T_2+ because of pairings with the US. Although the reduction in magnitude of HR decelerations to $(T_2+L)-$ across trials is consistent with a progressive increase in inhibitory strength to L- under this assumption, the outcomes of L- test trials did not support the presence of inhibition. It would be expected that HR accelerations should occur to L- alone when T_2+ was removed. However, the L- alone trials indicated that HR decelerations occurred when these trials were administered the day after completion of the "unpaired" phase. Perhaps L- test trials would have more accurately assessed inhibitory strength to L- if they had

been interspersed among inhibitory-training trials during the "unpaired" phase. The inclusion of such nonreinforced trials must be done with care, however, as such trials would be expected to lead to extinction of potential inhibitory properties. For conditioned inhibitors, however, this may not be a major problem as Zimmer-Hart and Rescorla (1974) found that 96 nonreinforced presentations of an inhibitor did not extinguish its power.

Yamaguchi and Iwahara (1974) reported that rats exposed to discrimination conditioning using tone CSs, displaced HR accelerations to the nonreinforced CS-. Evidence of persistent HR increases to L- in the discrimination group was not obtained in the current investigation. Rather, after the first few trials, very little HR responding to L- was apparent in the discrimination group, either accelerative or decelerative. In an earlier discrimination study in which two tones of different frequencies served as CS+ and CS-, Martin (1975) observed rather substantial HR decelerations to CS-, although they were smaller than those occurring to CS+. Perhaps the reason that similar decelerative responses to CS- were not obtained in the current experiment is because the light CS- that was used was very different from the tone CS+.

There was evidence of HR accelerations to L- during early presentations of that CS in the "unpaired" phase. These initial accelerations dissipated with additional training and were absent after approximately eight trials. This suggests that the HR increases represented an unconditioned or orienting response to the

light stimulus. Moreover, due to the fact that the early HR accelerations to L- were so quickly lost, it cannot be forcefully argued that an unconditioned accelerative tendency to the light stimulus was responsible for the appearance of HR accelerations to (T_2L)- in the explicitly-unpaired group or for the reduced-magnitude HR decelerations to (T_2+L)- in the conditioned-inhibition group.

It should be pointed out that, in many of the transfer and CER studies from which much of the evidence for conditioned inhibition has been derived (e.g., Bull & Overmier, 1968, Rescorla, 1966; Rescorla & LoLordo, 1965), no responses were recorded during inhibitory-training trials. Thus, there was no opportunity to observe the development and expression of inhibitory potential. It will be recalled that early experiments from Pavlov's (1927) laboratory showed evidence of a gradual development of inhibition. In general, Pavlov used a conditioned-inhibition procedure in which the putative inhibitor was tested at various stages of training by presenting the inhibitor in compound with an excitor. In those experiments, however, the expression of inhibitory potential was obviously limited by the response system that was chosen. Once the delivery of the CS- produced a zero level of salivary secretions, increments in inhibitory potential resulting from additional CS- trials could not be observed. Because HR generates an above-zero baseline, this response system has the advantage of allowing uninterrupted observations to be made of the development of inhibitory tendencies to CS-. For example, judging by the decreasing magnitude

of HR decelerations to $(T_2+L)-$ across trials in the conditioned-inhibition group, it is conceivable that with additional training the responses to $(T_2+L)-$ would have become accelerative and matched those in the explicitly-unpaired group.

The Rescorla-Wagner model of conditioning (Rescorla & Wagner, 1972) predicts that, in addition to the development of inhibitory tendencies to a CS- negatively correlated with the US, different degrees of inhibitory strength accrue to the CS- depending on the associative strength of excitatory tendencies to CS+. Because T_2+ was repeatedly reinforced on paired T_2+ trials and presented with L- on compound $(T_2+L)-$ trials, the conditioned-inhibition procedure was expected to produce the strongest inhibitory tendencies. The explicitly-unpaired procedure was anticipated to lead to somewhat weaker inhibitory tendencies, since the excitatory CS+ received only partial reinforcement. In this case, the background cues would be expected to serve as the CS+ and would be reinforced during US-alone trials and extinguished during the intertrial interval. The weakest innibitory potential was expected to result from discrimination conditioning because increments in excitatory associative strength to the CS+ background cues should be blocked by specific excitatory conditioning to T_2+ when that CS is paired with the US.

The outcomes of the special tests for inhibition in the current investigation were not consistent with these predictions, in that the strongest evidence of inhibition was found in the explicitly-unpaired saline group. In this group, the compound of the putative

inhibitor, L-, with the excitor, T_1+, resulted in HR reactions during the summation test that were opposite in direction from those occurring to T_1+ alone. Thus, T_1+ L- produced HR acceleration and T_1+ alone resulted in HR deceleration. In the conditioned inhibition and discrimination groups, the HR responses on the summation test were not different from those occurring to T_1+ alone.

During reversal conditioning, the acquisition performance of both the explicitly-unpaired and conditioned-inhibition-saline groups was poorer than that of the discrimination-saline group. In addition, during tne "unpaired" phase, the explicitly-unpaired group displayed more accelerative HR trials than did the conditioned-inhibition group. Thus, the combination of positive outcomes on both summation and reversal tests and the acquisition of HR accelerations to the nonreinforced (T_2L)- compound suggest that the explicitly-unpaired procedure led to the strongest inhibitory potential. Since the conditioned-inhibition-saline group displayed more retarded acquisition of HR CRs to L- during the reversal conditioning test than the discrimination saline group, it would appear that the conditioned-inhibition procedure elicited moderate inhibitory potential and discrimination conditioning the weakest potential.

The magnitudes of the cardioaccelerations to (T_2L)- in the explicitly-unpaired group in the present study appeared to be somewhat smaller than those that have occurred in prior studies (Cunningham, Fitzgerald, & Francisco, 1977; Stainbrook, 1978). This apparent discrepancy may have been related to procedural

differences between the studies. For example, in both prior studies, the explicitly-unpaired CS- was a single stimulus and consisted of a tone of a frequency different from the CS+ used in excitatory training. In the present experiment, the explicitly-unpaired CS- was a compound stimulus consisting of a tone (T_2) plus a light (L-). However, it does not seem likely that this difference in the CSs can account for the discrepant outcomes of the studies, for it can be argued that generalization of excitatory decelerative HR changes would take place more readily when CS+ and CS- were both tones than when CS+ was a tone and CS- a tone plus light. Hence, on the basis of stimulus generalization alone, it should have been more difficult to obtain HR accelerations in the Cunningham et al. and Stainbrook studies than in the current investigation.

A second procedural factor relates to when inhibitory training was initiated. In the Stainbrook study, the "unpaired" phase began 24 hr after the paired phase, whereas in the Cunningham et al. study, the "unpaired" training began immediately after the completion of the paired phase. It will be recalled that, as was true of the Cunningham et al. study, nearly one third of the "unpaired" trials were given in the present study immediately following excitatory conditioning. Thus, time since excitatory conditioning cannot easily explain the smaller HR accelerations that were obtained here.

A third procedural factor has to do with the schedule of explicitly-unpaired trials that was used. In both the Cunningham et al. and Stainbrook studies, the CS-alone and US-alone trials in

the explicitly-unpaired paradigm occurred in a semi-randomized sequence with the restriction that no more than three trials of either type could occur in a row. In the current study, explicitly-unpaired CS-alone and US-alone trials alternated. Conceivably, the alternating schedule provided more information about the occurrence of the US than did the random schedule and this somehow reduced the overall magnitude of the accelerative HR reactions to the nonreinforced CS.

While some of the effects that were obtained in the current study can be interpreted in terms of the development and presence of inhibitory tendencies, a number of alternative explanations can also be offered. For example, the relatively poor performance of the conditioned-inhibition-saline group during reversal conditioning, coupled with the absence of response decrements during combined-cue trials, could be attributed to the animals in this group having attentional deficits with respect to (L-)+ (Hearst, 1972; Mackintosh, 1973). That is, it could be argued that animals in the conditioned-inhibition group learned to "ignore" (i.e., decreased attention) the light stimulus during the "unpaired" phase. Consequently, it was more difficult to develop HR CRs to (L-)+ when it was paired with the US during reversal conditioning. However, it cannot be argued that the conditioned-inhibition group learned to "ignore" the light completely, for if that were the case, equal responding to T_2+ and (T_2+L)- should have occurred. The fact that it didn't indicates that L- was noticed. Moreover, it is difficult to understand how a

change in attention could account for the HR accelerations that occurred to (T_2L)- in the explicitly-unpaired group during the "unpaired" phase and to T_1+ L- in the explicitly-unpaired saline group during the combined-cue test. There is no reason to expect that attention per se should change the direction of the HR response to a CS.

Another alternative conception to that of inhibition is based on the notion of competing responses. It can be postulated that animals learn particular responses to a nonreinforced CS- that compete with the responses to CS+ during combined-cue testing and that interfere with the development of excitatory CRs to CS- during reversal training. Along these lines, several contemporary theorists (Black, 1971; Gormezano & Kehoe, 1975; Trapold & Overmier, 1972) have suggested that unrestrained animals could easily learn instrumental responses during inhibitory training which would subsequently compete with avoidance responding in a classical-to-instrumental transfer situation. In the present study, however, the animals were restrained, and there was no evidence that well defined skeletal-motor responses were learned.

The possibility remains, however, that other responses that were not measured such as respiration, blood flow, and blood pressure may have been learned in the "unpaired" phase, and that these responses competed with the reactions to CS+. In this regard, a recent experiment by Hoffman & Fitzgerald (1978) examined HR and blood-pressure reactions of rats exposed to a classical-conditioning

paradigm in which CS+ was positively related to a shock US. Although conditioned changes in HR and BP were demonstrated in that study, it was shown that the HR CRs did not depend upon the blood-pressure CRs. In terms of responses that were measured in the current study, it might be argued that the HR accelerations themselves actually served to interfere with the expression of opposing HR decelerative responses during the test phases. This argument is difficult to discount on the basis of the present findings. However, other experiments have shown that the occurrence of accelerative HR changes to a CS did not subsequently interfere with the development of decelerative HR CRs to that CS (Fitzgerald & Hoffman, 1976; Fitzgerald, Stainbrook, Francisco & Hoffman, 1978).

A final alternative hypothesis to inhibition would be to postulate that HR accelerations simply resulted from reductions in excitation (Hearst, 1972). In the case of the explicitly-unpaired group, it might be suggested that the pairing of background cues with the US on US-alone trials during the "unpaired" phase led to a persistent excitatory state of conditioned HR deceleration. Continual maintenance of such an excitatory state would be expected to result in reduced pre-CS baseline HR levels. Subsequent presentations of (T_2L)- may have released the tonic excitatory state that was present with the net result being HR accelerations. However, support for the view that excitatory conditioning to background cues occurred in just the explicitly-unpaired group and not in the other groups was not found since the pre-CS baseline HR levels of the explicitly-

unpaired group were not different from those of the conditioned-inhibition and discrimination groups.

While conditioning to background cues cannot account for differences between groups, it perhaps can help to explain baseline HR changes that occurred across trials for all groups. During the original excitatory phase, baseline HR declined across trials in all three groups. This has often been observed in other classical conditioning studies involving restrained rats (Burgoyne, Pote, & Freedman, 1967; Fitzgerald & Hoffman, 1976; Fitzgerald & Martin, 1971; Martin, 1975). A close inspection of the "unpaired"-phase data indicated that there was also a tendency for baseline HR to decrease within each day. These generalized HR decreases could reflect conditioning to background cues or more simply, habituation to the stresses associated with handling and restraint that one triggered at the beginning of each day.

In the case of reversal training, baseline HR increased across trials in the various groups. Such an increase has not been reported before in HR conditioning studies involving restrained rats. Conceivably, the use of a light as the CS contributed to the increases as auditory CSs were employed in almost all of the earlier experiments. Ethanol can be ruled out as a contributing factor as the saline groups showed baseline increases that matched those occurring in the ethanol groups.

It will be recalled that in the current study, 1.0 and 2.0-g/kg doses of ethanol were administered just prior to the summation and

reversal tests for inhibition. In general, there were very few consistent effects of ethanol either during either of these test procedures. The only effect of the drug was seen in the discrimination groups during reversal training. In this phase, with respect to the saline group, both the 1.0 and 2.0-g/kg ethanol groups tended to show debilitated acquisition of HR CRs to (L-)+. This effect is contrary to what would be expected on the basis of a proposed "disinhibitory" effect of ethanol (Dworkin, Bourne, & Reginsky, 1937; Nikiforovski, 1934). If ethanol acted as a "disinhibitor," then reversal performance of groups in which L- had developed inhibitory potential (most notably the explicitly-unpaired group) should have been facilitated.

In a recent study by Fitzgerald and Stainbrook (1978), it was found that a .8-g/kg dose of ethanol did not affect the development of HR CRs during excitatory conditioning. In the present investigation, a dose of 1.0 g/kg did interfere with the acquisition of HR CRs during reversal training in one group. This could mean that the effects of ethanol may be different in original as opposed to reversal classical conditioning.

Consistent with a study by Martin (1975), the findings of the current experiment indicated that very little skeletal-motor activity occurred, either during the intertrial intervals or during the vario CSs. When movement was present and matched the direction of t change, the HR change was generally magnified. Thus, HR were generally larger when movement increased. To

HR decelerations tended to be larger when movement decreased. Martin (1975), however, demonstrated that conditioned changes in movement were restricted to the early part of the CS and were not systematically related to conditioned changes in HR. Thus, there are scant data to suggest that the decelerative and accelerative HR responses in the current study were mediated by changes in skeletal-motor activity.

SUMMARY AND CONCLUSIONS

The present investigation was conducted to compare the effects of conditioned inhibition, discrimination conditioning, and explicitly-unpaired presentations of a CS and a US on HR and movement responses of restrained rats. All of these procedures shared the condition that the CS was negatively correlated with the US, and each procedure has been implicated by contemporary theorists as being capable of generating inhibitory tendencies. To assess the putative "disinhibitory" effects of ethanol, both 1.0 and 2.0-g/kg doses of ethanol were administered after inhibitory training had been completed.

The experiment involved 90 rats and consisted of four phases that were carried out over four days. In Phase 1 (the paired phase), all animals received 24 excitatory conditioning trials in which one tone (T_1+) was paired with an electric-shock US. Phase 2 (the "unpaired" phase) consisted of administering the three inhibitory procedures to separate groups of rats (N = 30 in each case). The conditioned-inhibition group received 96 paired presentations of a second tone (T_2+) and with the US and 96 presentations of T_2+ and a light in compound (T_2+L)- with the US. The discrimination group was given 96 paired T_2+ trials and 96 unpaired L- trials. The explicitly-unpaired group received 96 unpaired (T_2L)- trials and 96 US-alone trials. Following inhibitory training the animals were divided into three sub-groups (N = 10) and were given intraperitoneal injections of either

saline, 1.0, or 2.0 g/kg ethanol. In Phase 3 (the combined-cue phase), all groups were given 4 T_1+, 4 L-, and 12 T_1+ L- trials without reinforcement. In Phase 4 (the reversal phase), all groups received 48 reversal-training trials on which the putative inhibitory L- CS was paired with the US.

It was found that decelerative HR CRs developed to the reinforced T_1+ during the paired phase. The forms of the HR CRs were consistent with the phenomenon of Pavlovian inhibition of delay. During the "unpaired" phase, the directions of the HR responses to the nonreinforced CS were not the same. The conditioned-inhibition group showed HR decelerations to (T_2+L)- that were smaller in magnitude than HR decelerations to T_2+. In the discrimination group, HR accelerations occurred to L- on early trials, but these reactions were reduced in magnitude as training progressed. In contrast, the explicitly-unpaired group showed HR accelerations to (T_2L)- that increased in magnitude across trials. Significantly more HR accelerations occurred to the CS- in the explicitly-unpaired and the discrimination groups than in the conditioned-inhibition group. The explicitly-unpaired saline group displayed HR decelerations to T_1+ alone and HR accelerations to T_1+ L- on combined-cue trials. During the reversal test, both the conditioned-inhibition and the explicitly-unpaired-saline groups showed retarded acquisition of HR CRs to (L-)+ with respect to the discrimination-saline group. In the discrimination condition, the 1.0 and 2.0-g/kg groups showed retarded acquisition of HR CRs to (L-)+ as compared to the saline group.

In general, movement did not occur, but when it was present, HR accelerations were larger when movement increased, and HR decelerations were occasionally larger when movement decreased. Baseline HR decreased during the paired and "unpaired" phases but increased during reversal training.

It was suggested that conditioned inhibitory tendencies may have developed in the explicitly-unpaired group, and to a lesser extent, in the conditioned-inhibition group. Although in the latter case, the expression of inhibitory tendencies could have been overshadowed by concurrent excitatory tendencies. The least evidence of conditioned inhibition was found in the discrimination group. It was noted that alternative explanations such as attentional deficits and competing responses could not easily account for the effects attributed to inhibition. A "disinhibitory" effect of ethanol was not observed.

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