FACTORS AFFECTING THE DIRECTION OF CLASSICALLY CONDITIONED HEART-RATE RESPONSES OF RATS: INFLUENCE OF INSTRUMENTAL CONTINGENCIES, DEGREE OF RESTRAINT, AND SKELETAL-MOTOR ACTIVITY

by

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INTRODUCTION

The broad objective of the present research was to provide new information that will hopefully lead to a better understanding of those processes contributing to classically conditioned changes in heart rate. An attempt was made to examine the effects of instrumental contingencies on the direction of the conditioned heart-rate responses of unrestrained and restrained rats and to determine if conditioned changes in skeletal-motor activity comparable to those shown by heart rate occurred during conditioning. As the review of the literature presented later will indicate, both of these factors have been frequently mentioned in theories attempting to account for the complex nature of the conditioned heart-rate response.

Despite an outpouring of experimental and theoretical contributions in the last decade concerning the physiological and psychophysiological mechanisms underlying classically conditioned changes in heart rate, it has not been possible to formulate a unified conceptual framework that embraces the diverse and often conflicting findings reported in the literature. There has been an all-too-frequent absence of appropriate control groups in this work, and an occasional misuse of statistical tests. However the major reason for the apparent lack of consistency in the experimental results may be attributable to the failure to distinguish between studies using traditional classical conditioning procedures with those failing to adhere strictly to these procedures, or with those employing a combination of classical and instrumental paradigms. In the present treatment of the literature, a distinction was made

between these various kinds of investigations in the hope that this would help identify some of the variables that may have contributed to the divergent findings that have been reported.

Traditionally, classical conditioning has been defined as a procedure in which a relatively neutral conditioned stimulus (CS) is paired with an unconditioned stimulus (US) that reliably elicits an unconditioned response. After a number of pairings the CS evokes a conditioned response which usually involves a change in activity in the same response system as that of the unconditioned response. Rarely, however, does the magnitude of the conditioned response approach that of the unconditioned response.

An important aspect of the classical conditioning procedure is that the presentation of the US should not be contingent upon the presence or absence of the conditioned response. In other words, the occurrence of the US should be independent of the behavior of the subject, and at least in principle the latter should not alter the effectiveness or modify the consequences of the US. In attempting to meet these requirements, investigators employing an electric shock as the US have usually restrained the subjects and attached the shock electrodes directly to the subject's body on the assumption that this would minimize response-contingent modifications of the US.

Aside from the formal procedural aspects of classical conditioning it is also necessary to employ adequate control groups to assess unlearned changes in behavior to the CS. This is especially important in the case of responses such as heart-rate changes in that, prior to conditioning, the CS may elicite an unlearned orienting response that is similar to the conditioned response. Although

preconditioning trials with the CS alone are usually given to habituate this original response, controls are nevertheless needed to evaluate the extent to which it might recover following the application of the US.

Most major learning theorists have emphasized either a "stimulus substitution" (Konorski, 1967; Pavlov, 1927) or "stimulus response" (Guthrie, 1959; Hull, 1943; Spence, 1956) view of the classical conditioning process. In stimulus substitution formulations an association is presumably formed between the CS and the US while in the stimulus-response treatments the association is between the CS and the unconditioned response. Regardless of the association that is assumed to be formed, both types of theories lead to the expectation of a relatively close resemblance between the conditioned and unconditioned responses. Such a resemblance has been demonstrated in hundreds of classical conditioning studies involving a wide variety of responses including salivation, leg flexion, eyeblink, and nictitating-membrane closure. It has also been recognized, however, that the conditioned response rarely duplicates the unconditioned response (Kimble, 1961).

In apparent contradiction to this vast literature, investigators who have studied classically conditioned changes in the cardiovascular systems of dogs, rats, rabbits, cats, pigeons, monkeys, and humans have found that conditioned heart-rate reactions were frequently grossly dissimilar to the unconditioned responses. In fact, in some species the directions of the conditioned responses have been exactly opposite to that of the unconditioned responses, that is, the unconditioned responses may be increases in heart rate

while the conditioned responses prove to be decreases. Furthermore, the direction of the conditioned heart-rate response of a given species has varied with the particular situation in which conditioning was carried out.

In dogs, classically conditioned changes in heart rate may take the form of an acceleration (Black, Carlson, & Solomon, 1962; Church & Black, 1958; Fitzgerald, Vardaris, & Teyler, 1966) or a deceleration (Black, Carlson, & Solomon, 1962) while the unconditioned response has consistently been an acceleration. Similarly, in the rat both heart-rate accelerations (Black & Black, 1967; Duncan, 1972; Fehr & Stern, 1965; McDonald, Stern, & Hahn, 1963; Miller, Banks, & Caul, 1967; Werboff, Duane, & Cohen, 1964) and decelerations (Fitzgerald & Martin, 1971; Fitzgerald & Teyler, 1970; Fitzgerald, Martin, & O'Brien, 1973; Holdstock, 1970; Holdstock & Schwartzbaum, 1965; Teyler, 1971) have been reported with the unconditioned response generally being an acceleration in heart rate. Instances of decelerative unconditioned responses in rats within certain portions of the post-US period have also been reported (Fitzgerald & Teyler, 1970; Stainbrook, 1975; Teyler, 1971).

Similar discrepancies between the direction of the conditioned and unconditioned heart-rate reactions have been found in rabbits (Schneiderman, 1970), monkeys (Brady, Kelly, & Plumlee, 1969; Smith & Stebbins, 1965) and humans (Obrist, Sutterer, & Howard, 1972; Obrist, Wood, & Perez-Reyes, 1965; Wilson, 1969).

In an initial attempt to account for the direction of the conditioned responses of rats, Fitzgerald and Teyler (1970) noted that in those studies in which the response was heart-rate acceleration

the shock US was delivered through a grid floor to the foot pads of unrestrained rats (Black & Black, 1967; Feher & Stern, 1965) whereas in those studies in which the response was a deceleration (Fitzgerald, Vardaris, & Brown, 1966; Holdstock & Schwartzbaum, 1965; Vardaris, 1968) the shock US was presented through electrodes attached to the tail or chest of restrained subjects. They postulated that shock to the sensitive foot pads may have been more painful than shock delivered via the tail or chest and that this difference may have contributed to the conditioned heart-rate reactions. This suggestion was prompted in part by previous findings indicating that at high intensities of shock the conditioned heart-rate response shown by dogs was predominantly an acceleration in heart rate, but that at low intensities the conditioned response was frequently a deceleration (Lang & Black, 1963). To test their hypothesis, Fitzgerald and Teyler (1970) employed restrained rats receiving one of six intensities of the shock US; ranging from 0.4 ma up to 5.0 ma. Contrary to expectation they found that the conditioned responses consisted of deceleration at every intensity.

In discussing their results, Fitzgerald and Teyler (1970) pointed out that unrestrained rats are able to make instrumental escape responses, such as jumping off the electrified grid floor, which may provide temporary relief from the foot shock. It was suggested that this escape contingency might lead to a state of readiness or heightened excitement that could result in heart-rate acceleration as the conditioned response. This excitatory state would prepare the rat to make short-latency escape responses to the shock. On the assumption that the opportunity to make jumping

responses can only provide escape from shock when the shock is applied to the feet, it was reasoned that unrestrained animals shocked through electrodes attached to their body would show conditioned deceleration in heart rate.

To test this hypothesis, Teyler (1971) examined the effects of degree of restraint (restrained vs. unrestrained), location of shock (chest-shock vs. foot-shock) and US intensity on classically conditioned heart-rate changes in rats. Contrary to what had been reported in previous studies (Black & Black, 1967; Fehr & Stern, 1965; McDonald et al., 1963) the unrestrained foot-shock group on the whole failed to show reliable conditioning since one-half of the subjects developed decelerative reactions and the other half showed accelerative reactions. In the three remaining groups, the direction of the conditioned response was uniformly decelerative, the magnitude of the heart-rate response being larger in the restrained groups than in the unrestrained chest-shock group. Teyler noted that the conditioned decelerations shown by the unrestrained chest-shock group were consistent with the notion that in restrained rats a state of behavioral inhibition may develop which augments conditioned decelerative reactions based on inescapable shock. This state was viewed as being opposite to the excitatory state that Fitzgerald and Teyler (1970) proposed might develop in unrestrained rats receiving potentially escapable shock.

In summary, the above studies offer some support for the position that development of decelerative conditioned heart-rate reaction in restrained rats may be related to a central inhibitory state that is acquired when the rat learns that it cannot escape

from the shock US. The results also suggest that if escape from the shock is possible an excitatory condition may be fostered that triggers heart-rate accelerations to the CS.

Alternative explanations of the heart-rate responses that are seen in both classical conditioning and other more complex learning situations have placed considerable emphasis on changes in skeletal-motor activity. These formulations were prompted in part by the observation that increases or decreases in somatic activity are sometimes accompanied by comparable changes in heart rate. In order to account for this relationship it has been suggested that the somatic and cardiac response systems may be coupled together centrally (Black and deToledo, 1972; Obrist et al., 1972).

In outlining their position, Obrist et al. (1972) described the results of several classical heart-rate conditioning experiments that did not seem to agree with traditional theories of classical conditioning. For example, the conditioned heart-rate decelerations that have been reported in humans were consistently opposite to the accelerative unconditioned responses in humans which were always accelerative (Notterman, Schoenfeld, & Bersh, 1952; Zeaman, Deane, & Wegner, 1954). Furthermore, contrary to the results of most classical conditioning studies, US intensity failed to influence the magnitude of the conditioned response (Obrist et al., 1965). Finally, manipulations of the direction of the unconditioned response did not alter the direction of the conditioned reaction (Hastings & Obrist, 1967; Obrist, 1968; Wood and Obrist, 1968).

As an alternative to traditional theories of conditioning,

Obrist et al. (1972) proposed that learned modifications in skeletal-motor activity may trigger changes in heart rate that are appropriate to meet the metabolic demands of the activity, even before the occurrence of increased metabolic requirements. Within this conceptual framework, learning paradigms that yield increases in motor activity are likely to produce cardioaccelerations while those resulting in decreases in motor responding may lead to cardiodecelerations. In their view, heart-rate reactions are mediated not by peripheral feedback from skeletal muscles but by central nervous system processes controlling the initiation of skeletal-motor activity. In short, they argue that changes in heart rate obtained in classical conditioning situations and in other learning paradigms may reflect changes in somatic activity in preparation for the presentation of the US.

A second hypothesis that has been offered as an explanation of conditioned heart rate is that of Black and deToledo (1972). In outlining this hypothesis emphasis was also placed on the apparent coupling of skeletal-motor activity and heart rate. According to Black and deToledo, during classical conditioning, the CS comes to elicit a centrally located motivational state. Moreover, the neural centers controlling this state are assumed to be connected to central-motor structures that are in turn linked directly to cardiovascular response centers. On the basis of this model, increases in heart rate to the CS should be associated with elicitation of those motivational states leading to skeletal-motor activity whereas decreases in heart rate to the CS might be expected given the elicitation of a motivational state that inhibits motor responding. The question of

whether conditioned changes in heart rate could occur in the complete absence of motor activity was left open. Black and deToledo also argued that specific measures of motor responding such as bar pressing, gross movements, and EMG activity would not be correlated with heart rate unless they reflect the overall general activity of the organism.

The above formulations derive some support from neurophysiological and behavioral studies demonstrating a central linkage between autonomic and motor reactions. Electrical stimulation of restricted central pathways in both anesthetized and unanesthetized animals has in general resulted in short latency changes in heart rate. Hsu, Hwang, and Chu (1942) reported that stimulation of the motor cortex in dogs often resulted in a 20% increase in heart rate. Landau (1952) found that heart rate accelerated upon stimulation of the pyramidal tract in cats. Kaada (1960) showed that electrical stimulation of limbic structures frequently elicited a cessation of motor activity accompanied by cardiac deceleration. Similarly, Lofving (1961) stimulated limbic structures in unanesthetized cats and found a generalized inhibition of spontaneous motor activity and a depression of respiration. Abrahams, Hilton, and Zbrozyna (1960) presented evidence that the hypothalamus participated in the control of muscle blood flow in preparation for movement. They stimulated hypothalamic areas in free-moving cats and elicited defense reactions consisting of an alerting response followed by either rapid movements or attack. The concomitant cardiovascular effects were characteristic of vigorous muscular exercise.

In addition to the neurophysiological evidence, several behavioral studies have furnished support for the position that heart rate and motor activity systems may overlap in the central

nervous system. Rushmer, Smith, and Lasher (1960) found that the heart-rate response of dogs was sustained tachycardia beginning two or three beats after the start of an unsignalled period of enforced exercise (i.e., too soon to result from the metabolic demands of exercise). Similar changes in heart rate occurred during a stimulus signalling imminent exercise. The latency of heart-rate changes, prior to any vigorous muscular activity led the authors to suggest that higher nervous centers controlled both heart rate and motor activity.

Results comparable to those reported by Rushmer et al. (1960) have been found in humans. Petro, Hollander and Bouman (1970) demonstrated that cardiac acceleration occurred during the first interbeat interval following the onset of a short isometric contraction of the flexor muscles of the arm. Their results suggested that the accelerations were mediated either by decrease in vagal tone initiated by the motor cortex, or by afferents from muscle spindles. Freyschuss (1970), using selective pharmacological blockade of autonomic functioning in humans, demonstrated that heart-rate acceleration at the onset of muscle contraction was predominately elicited by a decrease of vagal tone. Further studies, in which neuromuscular block was used, indicated that the increase in heart rate did not originate in reflex involvement of muscle spindle afferents but was primarily of central origin.

In summary, the results of the above studies indicate that there are several areas in the brain capable of initiating simultaneous changes in heart rate and motor activity. However, it remains unclear what role if any this apparent coupling plays in mediating classically conditioned changes in heart rate.

In surveying the literature, it is possible to identify two basic groups (types) of experiments that have provided specific evidence bearing on the question of the degree to which changes in skeletal-motor activity are associated with classically conditioned heart rate. The first classification includes traditional classical conditioning studies as well as studies in which modified versions of the classical conditioning procedure have been employed. Experiments of this general type would seem to offer the most direct approach to obtaining information on the possible linkage between classically conditioned heart rate and movement.

The second classification contains those investigations in which a combination of classical conditioning and instrumental learning paradigms has been employed. Many of these experiments involved the use of the conditioned-emotional-response (CER) or conditioned-suppression paradigm. Basically, this procedure consists of the presentation of aversive classical conditioning trials when subjects are engaged in a food-reinforced instrumental task such as lever pressing. In the case of rats, the general outcome of these experiments has been that after relatively few conditioning trials, the CS elicits a deceleration in heart rate and a reduction in rate of lever pressing.

In considering experiments of this second type, it is important to note that certain problems may exist in using the CER paradigm to provide an estimate of classically conditioned heart rate. First, CS-US intervals of 2 to 3 minutes have routinely been employed on the conditioning trials. Such intervals far exceed those that have been shown to produce behavioral effects in traditional classical conditioning

studies (Kimble, 1961; Mackintosh, 1974). Although Pavlov (1927) was apparently able to maintain conditioned salivation in dogs with CS-US intervals of several minutes the conditioned response was initially established using much shorter intervals of about 5 sec. after which extensive training was given with progressively longer intervals. In CER studies, comparable training procedures have not been employed and yet behavioral changes to the CS have been observed after only four to eight conditioning trials.

Second, the use of both food and shock in the same situation provides an opportunity for aversively reinforced changes in heart rate to compete, or interact with, those heart-rate changes that are reinforced by food. Schoenfeld, Matos, and Snapper (1967) reported that conditioned accelerations in heart rate develop in rats receiving food reinforcement as the US. It has also been observed (Ehrlich & Malmo, 1967; Sutterer, Petrella, Orlick, & Gutman, 1974) that changes in heart rate accompany the delivery of food and the lever-press response.

A third point is that with the exception of one study (Roberts & Young, 1972), the rats in CER paradigms have been unrestrained with the shock US delivered to the foot pads through an electrified grid floor. As was noted earlier, this arrangement fails to meet the usual definition of classical conditioning in that instrumental escape responses can be made to the shock. A final point is that the heart-rate reactions to the CS occurred simultaneously with

marked shifts in skeletal-motor activity. In summary, the CER paradigm involves a number of features including unusually long CS-US intervals, introduction of food rewards, possible confounding instrumental contingencies, and lack of control over skeletal-motor activity all of which make it exceedingly difficult to determine if the heart-rate changes observed using these procedures reflect classical conditioning processes.

While some authors have carefully avoided the use of the term "conditioned" in referring to the changes in heart rate that are found in CER procedures others have not. In fact, in a recent treatment of the heart-rate conditioning literature (Mackintosh, 1974), results obtained with the CER paradigm were used uncritically to support the view of a connection between classically conditioned heart rate and skeletal-motor activity. The issue of whether the heart-rate responses were in fact the result of classical conditioning processes or simply due to the reduction in skeletal-motor activity associated with the cessation of lever pressing was not directly addressed.

The results of two recent experiments have provided revealing information bearing directly on this question. Duncan (1972) first trained rats to press a lever for food and then gave them six classical conditioning trials consisting of a tone CS paired with a foot-shock US on each of 6 days. Four of the trials were presented with the lever present in the chamber while the subjects were actively responding. The two remaining trials were given with the lever removed from the chamber, thereby preventing lever pressing. The results revealed that with the lever in the chamber the heart-

rate response to the first CS presentation was a deceleration whereas on later trials it was an acceleration. However, when the lever was not in the chamber the CS consistently elicited cardiac accelerations that were always greater in magnitude than those occurring with the lever available. Thus, direction of the heart-rate change to the CS was determined in part by the presence or absence of ongoing lever pressing suggesting that base-line motor activity may have been an important factor controlling not only the ongoing heart rate, but also conditioned changes in rate.

Further evidence in support of this possibility was provided by Borgealt, Donahoe and Weinstein (1972). In the first phase of the experiment, rats were trained over a period of days to lever press for a food reinforcement. In the second phase the lever was retracted from the chamber and 10 classical conditioning trials with a foot-shock US were given on each of 15 days. The conditioned response measured during this phase was cardiac acceleration which is consistent with what unrestrained rats receiving foot shock have shown in other experiments (Black & Black, 1967; Duncan, 1972; Fehr & Stern, 1965; McDonald et al., 1963). However, when the lever was inserted into the chamber allowing lever-press responses to occur the CS elicited decelerations in heart rate and suppression of lever pressing. The authors suggested that this heart-rate deceleration was directly related to the suppression of lever pressing and that its occurrence may have masked the accelerative reaction that was found in the absence of lever pressing. The results of these two experiments reinforce the serious doubts raised above regarding the usefulness of the CER procedure to study classically

conditioned heart rate in rats.

The results of several recent studies indicate that heart-rate responses during conditioned suppression training may at times be relatively independent of changes in lever-pressing rate. Brady et al. (1969) recorded heart rate and blood pressure during CER training in five monkeys. They found that in four of the subjects partial suppression of lever pressing developed early in training without the occurrence of concomitant autonomic responses. During the next few trials almost complete suppression was observed along with decelerations in heart rate. In the remaining subject suppression developed more slowly, unaccompanied by decelerative heart-rate reactions. Later in conditioning, the predominate heart-rate response of four of the subjects was cardioacceleration. However, one monkey with an extremely high lever-pressing rate showed persistent cardiac deceleration to the CS throughout acquisition. It was also noted that following extinction heart-rate decelerations were not observed during early re-conditioning trials.

Brady et al. (1969) interpreted their results as furnishing examples of both dependence and independence of heart rate and lever pressing. Since cardiodecelerations always occurred in the presence of substantial decreases in lever-pressing rate they suggested that these heart-rate reactions were dependent on changes in skeletal-motor activity. However, some independence of the two responses was demonstrated by the fact that suppression preceded the development of the heart-rate reactions and extinguished more rapidly. In addition, once suppression was well established or re-conditioned cardiac acceleration was obtained in spite of decreased gross-motor activity.

Lack of correlation between changes in heart rate and lever pressing was also found by deToledo and Black (1966) during differential CER training rats. They obtained reliable suppression of lever pressing during the second conditioning session whereas significant heart-rate decelerations did not develop until the sixth conditioning day. Cardiodeceleration, when it occurred, was greatest during the first minute of the CS with heart rate then gradually returning toward pre-CS base level. The authors noted that this pattern was similar to that which would occur if the heart-rate changes were simply artifacts of decreases in bar pressing. However, this possibility was rejected because the heart-rate reactions did not develop until after lever-press suppression was established.

Two studies have been reported in which heart rate was measured in rabbits receiving CER training (Sampson, Francis, & Schneiderman, 1974; Swadlow, Hosking, & Schneiderman, 1971). In both investigations conditioned heart-rate decelerations accompanied lever-press suppression. In the Sampson et al. study, it was found that reliable cardiodeceleration developed prior to suppression of lever pressing, suggesting that the two reactions might be independent of each other. Further, in contrast to what has been shown for unrestrained rats the deceleration persisted when the lever was removed from the test chamber. However, magnitude of cardiodeceleration was larger when accompanied by suppression of lever pressing (lever-available condition) perhaps indicating that an autonomous heart-rate reaction was further augmented by the cessation of skeletal-motor activity.

A number of other studies have furnished more detailed information on the relation between heart rate and somatic activity during CER

training. Black and deToledo (1972) recorded heart rate, electromyographic (EMG) activity, gross-motor activity, and bar pressing in a series of CER studies in unrestrained rats. They found that the magnitude of the correlations between decelerations in heart rate and decreases in EMG activity or bar pressing varied with the type of CS as well as with the US intensity and the stage of acquisition. However, there was always a high correlation between the amount of decrease in gross-motor responding and the magnitude of heart-rate deceleration elicited by the CS. The authors interpreted their findings in terms of a model incorporating a central linkage between conditioned changes in heart rate and skeletal-motor activity. An alternative explanation may be that the decelerations were simply augmented by simultaneous reductions in general activity, i.e., lever pressing.

A comprehensive analysis of electrodermal activity, movement, and heart rate in a differential CER paradigm was provided by Roberts and Young (1971). The rats were secured to a restraining platform with the electrodes delivering shock attached to the rats' tails. Voltage fluctuations in a wire grid laced through the restraining collar served as an indicator of movement activity. Heart-rate difference scores were computed on both CS+ and CS- trials from the final 10 sec. of the 3-min. CSs while lever pressing and movement suppression ratios were based on the last 20 sec. of the CSs. These scores revealed that suppression of lever pressing developed on the second day of conditioning accompanied by a decrease in movement and by a deceleration in heart rate.

The relation between gross-motor behavior and heart rate was investigated in detail by tabulating heart-rate responses that occurred on CS+ trials and then noting whether movement increased, decreased, or did

not change. Decrease-in-movement trials were scored if movement during the 3-min. CS was discriminably lower than a 3-min. pre-CS control period. Increase-in-movement trials were tabulated if movement increased for at least 10 sec. from a totally suppressed pre-CS base line. No-change-in-movement trials required that movement activity remain completely suppressed during both pre-CS and CS periods except for small bursts of movement that occurred during the first 2 sec. of the CS.

The results of this sorting procedure demonstrated that decreases in movement occurred on 65% of the 400 conditioning trials while increases or no change in gross movement were tallied on 6% and 20% of the trials, respectively. Fifteen trials were selected randomly from each category and corresponding heart-rate and gross-motor changes were tabulated during the first 30 sec. of CS+. This time interval was selected because movement increases were confined primarily to this period. These results demonstrated that heart rate decreased 49 beats per minute when movement was suppressed by the CS and that heart rate increased 36 beats per minute on those rare occasions when increased movement was elicited by the CS. The authors concluded that the heart-rate reactions were determined primarily by movement responses to CS+. However, whether heart rate was influenced directly by classical conditioning processes could not be evaluated.

Stebbins and Smith (1964) examined the relation between heart rate, blood flow, and gross-motor activity in monkeys during classical conditioning trials superimposed upon a food reinforced key-pressing task. They reported reliable suppression of the instrumental response accompanied by an increase in heart rate and blood flow. In addition, a considerable amount of gross-motor responding occurred during the CS as

measured via a strain gauge attached to the primate restraining chair. Nevertheless, large increases in heart rate generally preceded the movement activity indicating that the cardioaccelerative reactions were not secondary to changes in skeletal-motor responding or necessarily linked to such responding.

Recently, Sutterer and Obrist (1972) studied the relation between heart rate and gross-motor behavior in five dogs exposed to several different learning paradigms. These paradigms were designed to produce either increases and/or decreases in general activity and included (1) differential classical conditioning, (2) presentation of signalled weak and signalled strong shock on a differential reinforcement for low rates of responding base line, and (3) conventional CER training with CS+ and CS- trials. Gross movement was assessed by means of strain gauges attached to the floor of the conditioning chamber.

The results showed that if a given procedure produced an increase in gross-motor responding to CS+ then heart-rate accelerations were observed. Similarly, when decreases in general activity occurred to CS+ decelerative heart-rate reactions resulted. Although gross-motor activity and heart rate changed in the same direction, panel pressing was not consistently related to changes in either heart rate or general activity. Presumably, because of the small number of subjects employed, statistical tests comparing CS+ and CS- responding to establish that reliable conditioning had in fact occurred were not carried out. Rather, separate t-tests based on the performance of individual subjects were used to evaluate the statistical significance of the outcomes. While this method of analyzing the data may have been adequate to support the authors' conclusions, that their results were consistent with a cardiac-somatic

coupling model, the method was not appropriate to establish that the behaviors in question were conditioned.

In the following section are presented those studies in which skeletal-motor activity was measured during traditional classical conditioning procedures. These experiments provided the most direct method of assessing the relation between classically conditioned changes in heart rate and conditioned motor activity.

A systematic examination of classically conditioned heart rate in pigeons has been provided by Cohen (1969). The basic procedure involved the use of restrained pigeons with electrodes for delivering a shock US attached to one leg of the pigeon. The conditioned response, which was uniformly heart-rate acceleration, was generally paralleled by conditioned increases in respiratory rate (Cohen, 1967; Cohen & Durkovic, 1966; Cohen & Pitts, 1968; Durkovic & Cohen, 1969). Cohen and Durkovic (1966) reported that increases in gross-motor activity accompanied the cardioaccelerative responses in 4 of 12 pigeons. In his review, Cohen (1969) described unpublished observations indicating that increases in EMG activity may sometimes occur along with the accelerative heart-rate responses. He suggested that somatic activity augments cardioacceleration in pigeons but that the two responses were not necessarily linked.

Conditioned heart-rate reactions in restrained rabbits have almost always been found to be decelerative (Elliott & Schneiderman, 1968; Manning, Schneiderman, & Lordahl, 1969; Meredith & Schneiderman, 1967; Schneiderman, Smith, Smith, & Gormezano, 1966). Elster, VanDercar, and Schneiderman (1970) investigated differential heart-rate conditioning in rabbits using electrical stimulation of either the midbrain, subthalamus, or hypothalamus as the US. Stimulation of all three locations produced reliable

heart-rate conditioned responses. The direction of the conditioned responses generally depended upon the direction of the unconditioned heart-rate reactions elicited by the US. In US locations that elicited cardiac accelerations, a strong positive relationship was noted between diffuse movements elicited by the US and the subsequent development of conditioned heart-rate acceleration.

In an unpublished study, Schneiderman (1974) reported that intracranial microinjections of acetylcholine as the US resulted in conditioned cardiac acceleration, while equivalent injections of norepinephrine led to conditioned cardiac decelerations. Somatic activity was monitored during these injections via a phonocartridge attached to the test chamber. It was found that movement responses occurred more often after injection of acetylcholine which elicited heart-rate acceleration than after injections of norepinephrine which elicited heart-rate decelerations. These results provided an indication that brain regions related to unconditioned cardiac acceleration may also be linked to areas controlling skeletal activity. However, the exact relationship between gross movement and heart rate remained unclear in that unconditioned movement reactions did not always occur on every trial. Furthermore, when gross-motor behavior was elicited, it followed rather than preceded unconditioned cardiac acceleration.

Results suggesting that heart-rate accelerative reactions in rabbits may be related to increased somatic activity have been provided by Powell and Joseph (1974). These investigators attempted to determine the extent to which corneo-retinal potentials (a somatic response constellation including eye lid closure, nictitating membrane extension, and retraction of the eyeball), hippocampal theta, gross movement, EMG activity, and heart rate were related during aversive classical conditioning.

In Experiment I, rabbits were given 64 differential classical conditioning trials on each of 30 days with a reinforced trial consisting of a 1-sec. tone CS paired with an eye-shock US. Four test trials without the US were given on each of the 30 days. During early acquisition the conditioned heart-rate responses were consistently decelerative. These decelerations were associated with conditioned increases in theta activity and conditioned corneo-retinal potential responses. As acquisition progressed an accelerative heart-rate reaction to the offset of the CS developed. At this time, the occurrence of both corneo-retinal potentials and hippocampal theta activity were maximal. This later finding prompted the investigators to suggest that the heart-rate accelerations to CS offset toward the end of acquisition may have been related to increased somatic activity.

A second experiment was performed to determine if skeletal-motor activity as indexed by gross movement and EMG activity accompanied the accelerative component of the heart-rate reactions. Once again, heart-rate decelerations following the offset of the CS changed to heart-rate accelerations during the final conditioning sessions. Heart-rate reactions during the CS were consistently decelerative. A composite movement score computed from the gross movement and EMG responses during the 1-sec. CS demonstrated that reliable conditioned movement occurred and that the magnitude of this reaction was largest when the accelerative heart-rate component appeared. The authors interpreted these results as providing evidence that cardiac accelerations may be related to increased somatic activity. However, as the investigators themselves pointed out, conditioned increases in gross-motor activity accompanied conditioned heart-rate decelerations throughout the 30 days of

conditioning. Moreover, increased motor responding always preceded the accelerative heart-rate reactions that occurred to the offset of the CS late in acquisition. In contrast to the interpretation offered, the results of these two experiments provided very little support for the position that conditioned heart rate in traditional classical conditioning situations is related to skeletal-motor activity.

The relationship between heart rate and skeletal activity during classical conditioning has been investigated in several studies in which cats were used as subjects. Bruner (1969) measured heart rate and hind-leg tremor in cats receiving conditioned leg-flexion training in a canvas hammock that allowed the animals to maintain a normal standing position. During the course of acquisition cardiac deceleration developed to the CS paralleled by a clear reduction in tremor of the hind leg. It was also noted, however, that occasionally heart-rate reactions occurred without any changes in tremor and that decreases in tremor occurred in the absence of heart-rate responses.

In a similar study, Hein (1965) recorded a number of physiological responses including heart rate and EMG activity during differential leg-flexion conditioning in the cat. The subjects were trained to stand without restraint in the testing chamber prior to the beginning of conditioning. The flexor and extensor muscles of the foreleg were used as sites from which EMG activity was recorded. Although all subjects showed reliable decelerative heart-rate changes, consistent changes in EMG activity were not observed. However, if EMG responses were present just prior to the beginning of a trial, they generally decreased or disappeared in the presence of the CS. Only rarely were increases in EMG activity observed to CS+ and as conditioning progressed background

EMG was reduced to a minimum. The administration of a muscular blocking agent eliminated all overt skeletal activity (the animals were artificially respirated) without any apparent effect on the decelerative heart-rate reactions.

Recently, Howard, Obrist, Gaebelein, and Galosy (1974) examined the relationship between heart rate, gross-motor responses, EMG activity, and respiration in cats. A control group receiving unpaired presentations of the CS and US was not employed to assess nonassociative changes in behavior. Furthermore, separate statistical tests were carried out on the raw data of each subject. These two factors make meaningful evaluation of their results difficult. In Experiment I, four unrestrained cats were given 15 trace conditioning trials daily on each of 6 to 17 days. Gross-somatic responses were recorded via strain gauges attached to the floor of the conditioning chamber while EMG activity was monitored from muscles in the region of the neck. Trials were initiated manually so that the beginning of a trial could be delayed if the animal was violently active. The results that were reported were based on scores obtained during the last 2 or 3 conditioning days. The authors found consistent cardiodecelerations in two animals and biphasic responses (accelerations followed by decelerations) in the remaining two subjects. In all cases, the patterns of gross-motor responding closely paralleled the heart-rate reactions in that monophasic heart-rate decelerations were accompanied by monophasic decreases in movement activity. Furthermore, biphasic heart-rate changes were associated with biphasic changes in skeletal-motor activity. Electromyographic activity failed to show any consistent relation to the direction of the heart-rate changes. There were positive correlations between heart rate and general activity while EMG responses

and respiration were unrelated to heart rate, to each other, and to general activity.

To provide another measure of somatic responding, a second experiment was carried out in which multiple-unit activity in the pyramidal tract was recorded in five cats. Once again, a conditioning control group was not used. Consistent with what was observed in the first experiment cardiac decelerations were accompanied by decreases in somatic activity with both gross-motor activity and pyramidal tract responses being reliably correlated with heart rate and each other. Electromyographic activity was unrelated to the heart-rate reactions. Howard et al. (1972) suggested that heart rate adjusts to the total somatic activity of the organism at any given moment. Thus, increases in overall skeletal activity result in cardioaccelerations while decreases in somatic activity trigger cardiodecelerations. The lack of a consistent relationship between heart rate and EMG changes was interpreted as a failure of EMG responses to reflect overall ongoing motor activities.

Obrist and Webb (1967) examined the relationship between heart rate and somatic activity in seven dogs secured in a hammock-type sling. The experimenter was in the room with the dogs during the course of conditioning and trials were delayed if the dogs were struggling or showing respiratory irregularity such as panting. Nonassociative effects of the shock US were evaluated in six of the subjects using unpaired presentations of the CS and US. The unpaired trials were given, either at the beginning of each conditioning session or following the completion of the study. Two of the dogs so tested showed heart-rate accelerations that were smaller than those observed during regular conditioning. The authors also stated that a differential conditioning procedure involving CS+ and

CS- trials was tried and abandoned because the dogs did not show differential responding. The data from the main part of the experiment were analyzed by means of separate within-subject correlation tests performed on the heart rate and movement scores of each dog. The main findings of the study were that: (1) heart rate accelerated in the presence of increased gross-motor activity with the magnitudes of the two reactions being positively correlated; and (2) cardiac changes did not occur or were greatly reduced in the absence of somatic activity.

Obrist (1968) studied the relation between changes in heart rate and striate skeletal-muscle activity during aversive classical conditioning in humans. Electromyographic activity was measured from muscles in the chin, the neck, and the left arm. Two CS-US intervals were employed which produced either accelerations in heart rate (1.0 sec.) or decelerations in heart rate (7.0 sec.). Heart-rate decelerations were accompanied by inhibition of EMG activity while heart-rate accelerations were accompanied by bursts of EMG activity. An unpaired control group was not employed to establish that conditioning occurred.

In an attempt to extend the generality of the above results, Obrist, Webb, and Sutterer (1969) measured heart rate and somatic activity during a simple reaction-time task. They hypothesized that the decelerative heart-rate reactions typically found with this procedure would be related to inhibition of somatic-motor activity. Skeletal involvement was evaluated by recording EMG responses from the chin and neck muscles. The results of Experiment I revealed that a decrease in EMG activity and cardiac deceleration occurred together just before and during the behavioral response. The magnitude of the somatic and cardiac responses were positively correlated with each other in that the largest decreases in gross-

motor activity accompanied the largest heart-rate decelerations. An additional experiment replicated the above results and extended the somatic measures to both eye movement and eye blinks.

One of the few experiments in which skeletal-motor activity and heart rate were compared in rats receiving traditional classical conditioning was reported by Holdstock and Schwartzbaum (1965). They employed restrained rats and measured EMG responses from the thigh muscles of one leg. A group receiving unpaired presentations of the CS and US was included in the design of the study. The experimental group demonstrated conditioned decelerations in heart rate and essentially no changes in EMG activity. On the other hand, the control group showed accelerative heart-rate reactions that were accompanied by reliable increases in EMG activity. Thus, sensitized heart-rate accelerations appeared to be associated with increased skeletal-motor responding. However, the fact that the conditioning trials were always initiated in the absence of pre-CS EMG activity, precluded the possibility of obtaining decreases in EMG activity in the experimental group.

A comprehensive study of conditioned heart rate involving restrained rats was carried out by Fitzgerald and Teyler (1970). They employed a 2 x 6 factorial design to compare the effects of trace and delayed conditioning procedures at six US intensities ranging from 0.4 to 5.0 ma. All subjects were given 20 pre-test, 30 acquisition, and 30 extinction trials. In addition to heart rate, gross-movement activity was measured via a phonocartridge attached to the restraining device. The sensitivity of the movement detector was set just above that necessary to record respiration. Conditioned movement reactions were not found to accompany the conditioned heart-rate decelerations shown by the rats.

In a subsequent study, Teyler (1971) measured general activity and heart rate in 12 experimental groups representing a 2 x 2 x 3 factorial design in which the factors were degree of restraint (restrained or unrestrained), locus of shock (foot shock or chest shock), and US intensity (0.8, 1.6, or 3.0 ma). Since US intensity did not influence the heart-rate reactions the results were collapsed across the shock dimension. With the exception of the unrestrained foot-shock group, all of the groups demonstrated conditioned decelerations in heart rate. In the case of the unrestrained chest-shock group the decelerations were positively correlated with decreases in gross-motor activity.

In another study involving freely-moving rats, Duncan (1972) measured heart rate and motor activity using a classical conditioning procedure in which a tone CS was paired with a foot-shock US. A pseudo-conditioning control group was not included to evaluate the presence of nonassociative changes in behavior. He found that the CS elicited cardiac accelerations that were augmented in the presence of gross-movement activity. However, heart rate still accelerated on movement-free trials. Furthermore, on movement trials the heart-rate increases preceded movement by several seconds.

Although obviously complex, the preceding review of the literature provides evidence suggesting that given the appropriate conditions, heart-rate reactions occurring in learning situations may be linked to changes in skeletal-motor activity. In general, unrestrained rats receiving CER training typically showed decelerations in heart rate to the CS that were accompanied by decreased rates of lever pressing. However, with the lever removed from the conditioning chamber, thereby lowering the background level of motor activity the same rats showed heart-rate acceleration to the CS. Similar reactions were found in unrestrained rats

receiving a foot shock US in the absence of any CER training. Restrained rats actively pressing a lever for food demonstrated predominantly decelerative changes in heart rate to the CS. These decelerations were associated with decreases in general activity and with the cessation of lever pressing. Evidence that heart-rate decelerations revealed by unrestrained rats in the CER paradigm may at times be independent of lever pressing was indicated by the fact that the suppression of lever pressing can develop prior to a change in heart rate. Whether the same relation is possible in the case of gross-motor activity has not been established.

Consistent with what has been found for rats, CER training was shown to produce heart-rate decelerations in restrained rabbits. Although removing the response manipulandum did not change the direction of the heart-rate responses of rabbits, the decelerations were found to be magnified in the presence of a decrease in the rate of lever pressing. Nevertheless, some degree of independence between motor activity and heart rate in rabbits was revealed by the occurrence of conditioned heart rate before the development of reliable suppression of lever responding. Finally, restrained monkeys working within the constraints of the CER procedure have shown cardiodecelerations accompanied by pronounced decreases in lever pressing on early training trials. On later trials, increased heart rate was commonly observed in spite of decreased lever responding. Evidence was also found that suppression of lever pressing occurred in monkeys prior to the development of the heart-rate response.

In summary, given the complexity of the CER procedures and recognizing the host of factors that can influence heart rate, it has not been possible to determine whether the cardiac reactions occurring in CER training paradigms are a reflection of classical conditioning processes,

of changes in skeletal-motor activity associated with lever pressing, of changes in gross-movement activity, or of a combination of these events.

In traditional classical conditioning paradigms, much less evidence of a dependent relation between heart rate and skeletal-motor activity has been found. In restrained rats, there was no indication that decreases in heart rate were associated with decreased movement. During classical conditioning in rabbits, increases in somatic activity actually accompanied conditioned cardiac decelerations. Although motor activity sometimes appeared to augment the cardiac reactions of rabbits, heart-rate changes frequently preceded or occurred in the absence of movement. In the case of dogs and humans, evidence was presented that heart rate and skeletal activity may co-vary in classical conditioning situations. Unfortunately, however, appropriate control groups necessary to rule out nonassociative changes were frequently absent in these studies and in several instances, questionable statistical procedures were used to evaluate the results. In general, then, evidence linking classically conditioned changes in heart rate to conditioned changes in skeletal-motor activity is less than convincing. On the other hand, in nonclassical conditioning situations, motor activity as well as various uncontrolled instrumental contingencies may exert a pronounced effect on heart-rate responses.

The present investigation was designed to examine the effects of providing rats with the instrumental opportunity to control the duration of the shock US on the direction of anticipatory heart-rate reactions to a CS signalling the shock. It was predicted that a signalled-escape procedure would lead to heart-rate accelerations in both restrained and unrestrained rats whereas heart-rate decelerations would occur in rats receiving yoked classical conditioning training. In an attempt to

determine the role of somatic activity in the development of the heart-rate reactions in these situations, both gross movement and EMG activity were recorded. It was hoped that the present investigation would help illuminate those factors that control the direction of the classically conditioned heart-rate responses of rats.

METHODS

Subjects

Fifty-six female Long-Evans hooded rats ranging in weight from 220-320 gms served as subjects. The rats were purchased from Charles River Laboratories and were housed under conditions of free access to food and water in the facilities provided by the Department of Animal Care at the University of Oregon Health Sciences Center.

Apparatus

The conditioning chamber consisted of a 40-cm high x 25-cm wide x 30-cm deep aluminum box with a Plexiglas door across the front. A mercury-pool swivel commutator and two 10-cm speakers were mounted on the ceiling of the enclosure. The floor of the chamber rested on four 1.25-cm vertical compression springs located in the corners of the chamber. An Astatic (#24) phonocartridge with a 10-cm long, 1.0-mm diameter metal rod inserted in the needle housing was mounted along the edge of the floor to monitor gross-motor activity. A 1.25-cm wide x 20-cm long slot in the center of the floor parallel to the rear wall was used to hold the tails of unrestrained subjects.

The chamber was equipped with a response wheel constructed of two 6.9-cm diameter brass end plates held 10 cm apart by 2.32-mm stainless-steel spacer rods set at 2.19-mm intervals around the circumference. The wheel assembly was mounted in an opening in the middle of the side wall 1.25 cm above the floor and protruded 1.25 cm into the enclosure. An aluminum shield rotating on the same axis as the wheel was remotely raised or lowered by a belt drive attached to a 110-V.

reversible motor mounted on the outside of the chamber. A photoelectric cell and light source were positioned so that the light beam passed through holes spaced at 90° intervals around the perimeter of the end plates of the wheel. A single interruption of the light beam triggering appropriate programming logic constituted a wheel-turning response. To prevent unwanted auditory signals from reaching the subjects, the conditioning chamber was located in a small animal IAC sound isolation room equipped with an air supply, a 60-w. house light, and a 10-cm speaker attached to the ceiling directly above the chamber. The speaker was used for the continuous presentation of white noise at 75 dB (measured on the C scale of a Scott sound pressure meter) to provide additional masking of external sounds.

The restrained subjects were held in a heavy-gauge wire mesh cage measuring 4.5-cm wide x 6.0-cm high x 18-cm long with a small slot above the subject's head. The cage was secured to the floor of the conditioning chamber and positioned so that the subject could rotate the wheel when the aluminum shield was raised. A removable vertical series of metal slats at the rear of the restrainer were adjusted to hold the subject tightly in the restrainer.

The CSs were a 1-kHz and a 5-kHz tone generated by an audio-oscillator, and presented through the speakers mounted on the top of the conditioning chamber. The stimuli were counterbalanced such that one half of the subjects received the 1-kHz tone as CS+ whereas the other half received the 5-kHz signal as CS+. The intensities of the CSs were adjusted to 15 dB above the white noise background (using the C scale of a Scott sound pressure meter) measured 4 cm in front of the response wheel.

The US was a 20 pps train of 0.1 msec., 240 volt pulses provided by a Massey Dickinson constant wattage shocker. The US was delivered to the base of the rat's tail through #6 round-head machine screws attached to two 1.25-cm pieces of rubber tubing that fit snuggly on the rat's tail. A liberal amount of electrode paste was applied to the tail to help maintain constant electrode resistance throughout conditioning. To prevent biting at the shock electrodes, the tail of the unrestrained rat was inserted through the slot in the floor of the conditioning chamber and held there by means of a plastic washer. The rat's tail was inserted through the washer and a piece of adhesive tape was placed below the washer to keep the rat from pulling its tail back through the slot. The shock electrodes were then positioned on the tail below the washer and anchored there with an additional band of tape. Although this arrangment did reduce, to some extent, the kinds of activities that the unrestrained subjects could make, it was relatively easy for them to move around the conditioning chamber.

Heart rate was recorded by means of a punched paper tape system that provided an on-line tabulation of the number of heart beats occurring in successive time intervals within each trial. This system, much of which has been described in detail elsewhere (Fitzgerald, Vardaris, & Teyler, 1968), operated in the following manner. The electrocardiogram (ECG) was amplified by a solid state differential amplifier and then written out on one channel of a Grass polygraph. A lever-type Microswitch was positioned such that a switch closure was produced when the oscillograph pen was deflected by the R wave of the QRS complex. The switch closure triggered a pulse shaper that supplied a pulse to a BCD counter for each heart beat. An end-of-count command occurred at

the end of each counting interval transferring the contents of the counter to storage thus freeing the counter to accept incoming signals within 30 μ sec. The heart-beat total was then punched out on paper tape and the storage mode reset for the next counting interval. To provide a visual check on the reliability and accuracy of the counting circuit, the output of the pulse shaper activated another oscillograph pen. These spikes represented the heart beats that were actually counted. The accuracy of the heart-rate circuit was periodically checked by substituting a 10-Hz signal for the ECG signal.

The EMG signal was measured using a Tektronix 561A oscilloscope equipped with a 2A61 differential amplifier. The amplifier's 60-Hz notch filter and main filters (set to pass 6 Hz to 600 Hz) were used in order to eliminate unwanted noise. The output of the differential amplifier fed a Massey Dickinson Schmitt trigger that provided a pulse for each EMG spike that exceeded .05 mv. The output of the Schmitt trigger was connected to a BCD counting network identical to that used to tabulate heart beats.

Movement activity of the subjects was monitored by amplifying the output of the floor-mounted phonocartridge with a Grass low level D.C. preamplifier set at .05 mv/cm sensitivity. The output of the amplifier was fed to an integrating device (Bitterman, 1966) that provided 50-msec. pulses, the frequency of which was proportional to the input voltage. These movement events were accumulated in a third circuit identical to those used for heart beats and EMG activity.

Latencies of wheel-turning escape responses were printed out to the nearest 0.1 sec. at the end of each trial using a Massey Dickinson counter-printer.

Trials were initiated automatically by a film-tape programmer, while events within a trial were programmed and timed using Massey Dickinson transistorized logic modules. Two identical conditioning chambers, each housed in sound isolation enclosures, allowed two subjects to be conditioned concurrently.

ECG and EMG electrodes

The ECG electrodes consisted of 1.2-cm stainless-steel lock washers soldered to 7.5-cm pieces of number 26-ga. plastic insulated wire. The EMG electrodes were constructed of 0.125-mm diameter teflon coated stainless-steel wires. Each recording wire was soldered to an Amphenol Reliatac pin and inserted into a 5-pin Amphenol "Tiny Tim" head-post connector. Two electrodes were used for both ECG and EMG recording, while a fifth 2.5-cm bare #30 stainless-steel wire served as a ground. All five electrode wires were contoured to lie flat along the base of the head post and were insulated with a small amount of dental acrylic.

Electrode implantation

All subjects were anesthetized with a 40 mg/kg dose of sodium pentobarbital. Additional amounts of Penthrane (methoxyflurane) were administered as needed to maintain proper depth of anesthesia. Surgery was carried out under "clean" conditions with aseptic procedures not necessary due to the rat's high resistance to infection. A 5-cm dorsal-midline incision was made starting 1.25 cm in front of the eyes. A second 1.25-cm ventral-midline incision was made above the sternum. After the skull was scraped clean, the head post was anchored in place with dental acrylic and two machine screws.

One ECG electrode was sutured at the posterior end of the dorsal incision while the other lead was tunneled under the skin and secured in a similar manner above the sternum. Both EMG wires were tied in a knot leaving a small loop for anchoring to the surrounding tissue. Each of the EMG leads was inserted 3.1 mm into a 25-ga. needle and bent back in line with the needle shaft. The needle was thrust into the region of the trapezius muscle and then withdrawn leaving the EMG wire embedded in the muscle tissue. All subjects were given at least four days to recover from the surgery before being tested in the conditioning procedure.

Procedure

The basic design of the experiment was a 2 x 2 factorial with the two dimensions being the conditioning paradigm (signalled escape training vs. yoked classical conditioning) and the degree of restraint imposed (unrestrained vs. restrained). Fourteen subjects were used in each group.

All subjects were given a differential conditioning procedure in which CS+ was paired with the US and CS- was not paired with the US. For one-half of the subjects CS+ was a 1-kHz tone and CS- a 5-kHz tone. The other half of the subjects in each group received a 5-kHz tone as CS+ and a 1-kHz tone as CS-. All subjects received a 15-min. period of adaptation to the conditioning chamber followed by 24 CS-alone trials consisting of 12 CS+ and 12 CS- presentations. Next, 80 acquisition trials were given with 40 CS+ and 40 CS- trials. The two types of trials were presented in a random series of 10 trials (CS+, CS-, CS+, CS+, CS-, CS+, CS-, CS+, CS-, CS+, CS-, CS-)repeated eight times. All trials were presented with an intertrial interval of either 80, 120, or 160 sec ($\bar{X}$=120 sec.). An escape subject and a yoked classical

subject were conditioned at the same time with trials alternating between the subjects.

All trials began with the shield in the down position blocking the animal's access to the wheel. On reinforced-escape trials CS+ was followed 6 sec. later by the raising of the shield. When the wheel was sufficiently exposed to permit a response, the shock was delivered with both the CS and shock remaining on until the wheel was turned. If no response occurred within 60 sec., the CS and shock were terminated automatically. Reinforced-classical trials were exactly the same as escape trials except that the response wheel was locked in position and the subjects were unable to terminate the US. The durations of the CS and US for a yoked classical subject on any given trial were identical to those received by the escape subject on that trial. Nonreinforced trials for both the escape and classical subjects consisted of a 7-sec. presentation of CS- without the US or the raising of the shield. The duration of CS- was chosen to overlap the first 0.5 sec. of shock that would necessarily occur on all escape trials.

Heart beats, movements, and EMG activity were counted in a 6-sec. pre-CS interval, in three subsequent 2-sec. intervals during the CS and in a 6-sec. interval following the US. Each score was converted to a response-per-minute index. These scores were then corrected for base-line activity by subtacting the rate during the 6-sec. pre-CS period. The resulting difference scores were positive when there was an increase in heart rate, movement, or EMG activity and negative when there was a decrease in responding. All transformations and data analyses were performed on a Wang 720 minicomputer system.

RESULTS

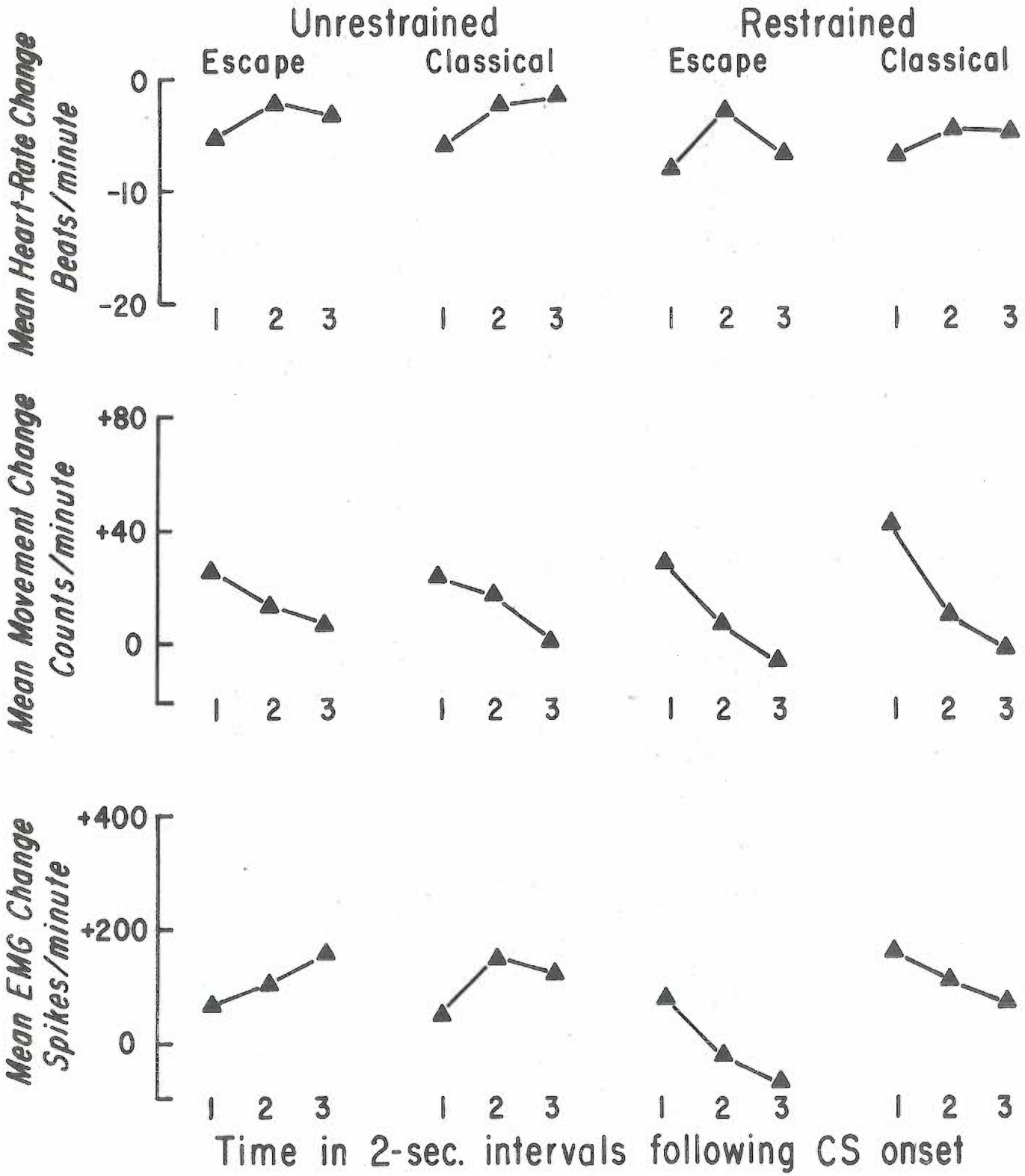
Pre-test CS alone

Mean heart-rate, movement, and EMG difference scores in the three 2-sec. intervals of the CS period averaged over all of the pre-test trials are shown for each group in Figure 1. The responses elicited by the two CSs were averaged together after separate analyses of variance demonstrated no significant differences between CS+ and CS-.

Heart rate. The top portion of Figure 1 shows the heart-rate reactions of each group on the pre-test trials. It can be seen that the CS elicited decelerative changes in heart rate in all of the groups with the magnitudes of the changes being slightly larger in the first counting interval than in the last counting interval. A three-way analysis of variance (unrestrained vs. restrained x escape vs. classical x counting intervals) demonstrated that the change in heart rate across the counting intervals was reliable ($F=9.76$, $df=2/104$, $p<.01$). There were no significant effects of degree of restraint or conditioning procedure.

Movement activity. Mean movement difference scores of the four groups are plotted in the middle section of Figure 1. Inspection of this part of the figure indicates that the CS was accompanied by an overall increase in movement in all of the groups with the onset of the CS eliciting the greatest change. By the end of the CS period, movement was at or near pre-CS levels in all groups. Comparison of the upper and middle sections of the figure reveals that heart rate decelerated in spite of increased movement activity and that the largest decelerations occurred at the same time as the biggest movement responses. A three-way analysis of variance established that the reduction in movement over the counting intervals was reliable ($F=16.05$, $df=2/104$, $p<.01$).

Figure 1. Mean CS minus pre-CS heart rate, movement, and EMG responses of the unrestrained-escape, unrestrained-classical, restrained-escape and restrained-classical groups during the three 2-sec. intervals of the CS averaged over all of the pre-test trials.


Unrestrained
Restrained
Escape
Classical
Escape
Classical
Mean Heart-Rate Change
Beats/minute
0
-10
-20
Mean Movement Change
Counts/minute
+80
+40
0
Mean EMG Change
Spikes/minute
+400
+200
0
1 2 3
Time in 2-sec. intervals following CS onset

Once again there were no significant differences as a result of the degree of restraint or conditioning procedure.

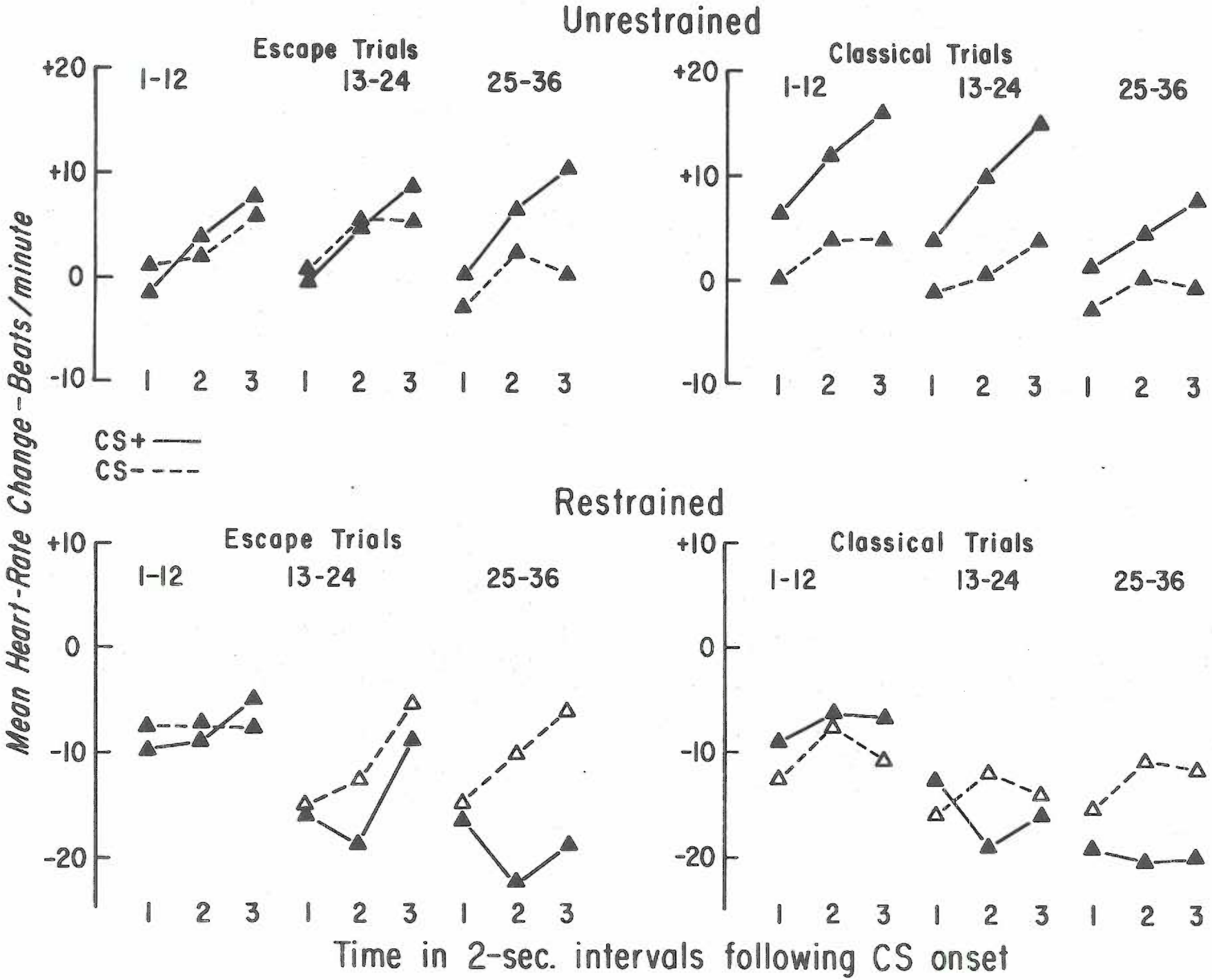
EMG activity. The bottom section of Figure 1 depicts the EMG responses of the four groups. Data of five subjects in each group were lost due to recording difficulties. With the exception of the restrained-escape group, EMG activity, like movement, increased overall to the CS in each group. However, the unrestrained groups showed an increase in EMG activity over the three counting intervals while the restrained groups showed a decrease. Thus, in the unrestrained groups the pattern of EMG activity was opposite to that of movement whereas in the restrained groups the patterns were similar. An analysis of variance revealed a significant unrestrained vs. restrained x counting intervals interaction ($F=4.83$, $df=2/64$, $p < .01$) reflecting the reliability of the difference in the patterns of EMG responses of the unrestrained and restrained groups.

Acquisition

Heart rate. Mean heart-rate responses to CS+ and CS- during the three 2-sec. periods of the CS-US interval averaged over successive 12-trial blocks of acquisition are plotted in Figure 2. The top half of the figure shows the results of the unrestrained groups and the bottom half those of the restrained groups. Comparison of the two parts of the figure reveals that the heart-rate reactions of the unrestrained groups to both CSs were predominately accelerative while those of the restrained groups were consistently decelerative.

More detailed examination of the upper left-hand portion of Figure 2 indicates that the unrestrained-escape group showed little evidence of conditioning until the last 12 trials of acquisition. During the first 24 trials both CSs elicited comparable accelerative heart-rate responses that increased in magnitude across the three

Figure 2. Mean CS minus pre-CS heart-rate responses of the unrestrained-escape, unrestrained-classical, restrained-escape, and restrained-classical groups in each 2-sec. interval of both CS+ and CS- as a function of 12-trial blocks during acquisition.


Unrestrained
Escape Trials
Classical Trials
1-12
13-24
25-36
+20
+10
0
-10
1 2 3
CS+
CS-
Restrained
+10
-20
Mean Heart-Rate Change-Beats/minute
Time in 2-sec. intervals following CS onset

counting intervals. On the final block of trials a similar pattern of heart-rate acceleration continued to occur to CS+ with responding to CS- being diminished. Separate 2 x 3 analyses of variance (CS+ vs. CS- x counting intervals) were performed on each 12 trial block of acquisition. During the final 12 acquisition trials there was a reliable difference in the heart-rate responses to CS+ and CS- ($F=10.42$, $df=1/65$, $p<.01$) establishing that conditioning occurred. There was also a reliable counting interval effect in all phases of acquisition (Block one: $F=8.59$, $df=2/56$, $p<.01$; Block two: $F=3.69$, $df=2/65$, $p<.05$; Block three: $F=5.42$, $df=2/65$, $p<.01$).

The unrestrained-classical group shown in the upper right-hand portion of Figure 2 revealed evidence of conditioning in all phases of acquisition. However, the magnitude of the difference between CS+ and CS- was somewhat smaller in the last 12 trials than in the first 24 trials. The basic form of the conditioned response, consisting of a progressive increase in the magnitude of heart-rate acceleration in the presence of the CS, was established in the first 12 acquisition trials, and remained virtually unchanged through the remainder of acquisition. Separate 2 x 3 analyses of variance indicated that responding to CS+ and CS- was significantly different in each block of acquisition, Block one: $F=21.33$, $df=1/65$, $p<.01$; Block two: $F=11.82$, $df=1/65$, $p<.01$; Block three: $F=7.34$, $df=1/65$, $p<.01$. The heart-rate change over counting periods was reliable in the first ($F=4.37$, $df=2/65$, $p<.05$) and second ($F=3.64$, $df=2/65$, $p<.05$) blocks.

Although there was some suggestion that the unrestrained-classical group conditioned faster than the escape group a 2 x 2 x 3 analysis of variance (escape vs. classical x CS+ vs. CS- x trial blocks) carried out on the mean heart rate of the three counting intervals failed

to provide any significant differences between the two groups. There was, however, a reliable difference between the overall responses to CS+ and CS- ($F=7.54$, $df=1/26$, $p<.05$) for both groups combined.

Mean heart-rate difference scores of the restrained-escape group are plotted in the lower left-hand section of Figure 2. It can readily be seen that decelerative heart-rate responses occurred to both CS+ and CS- and that the largest difference between the two responses reflecting conditioning occurred during the last 12 trial block of acquisition. The most pronounced difference in the forms of the responses also appeared to occur during the last block of acquisition. At this point heart-rate deceleration to CS+ was greatest during the second and third 2-sec. periods whereas the response to CS- was maximal during the first 2-sec. interval. Analyses of variance (CS+ vs. CS- x counting intervals) computed at each stage of acquisition demonstrated that only during the final 12 trials was there a significant difference between the responses to CS+ and CS- ($F=27.31$, $df=1/64$, $p<.01$). The CS+ vs. CS- x counting intervals interaction was also significant ($F=5.50$, $df=2/65$, $p<.01$) indicating that the forms of the two responses were reliably different on these trials. During the second block of trials there was a reliable change in heart rate across the counting intervals ($F=7.46$, $df=2/65$, $p<.01$).

The lower right-hand portion of Figure 2 indicates that like the restrained-escape group the restrained-classical group also demonstrated decelerative heart-rate responses to both CS+ and CS-. The form of the heart-rate reactions to both CSs varied considerably over the first two blocks of acquisition. By the final 12 trials, there were uniform decelerations in heart rate over the three counting intervals of CS+ which were larger in magnitude then those elicited by CS-. Evidence of conditioning in terms of a significant difference

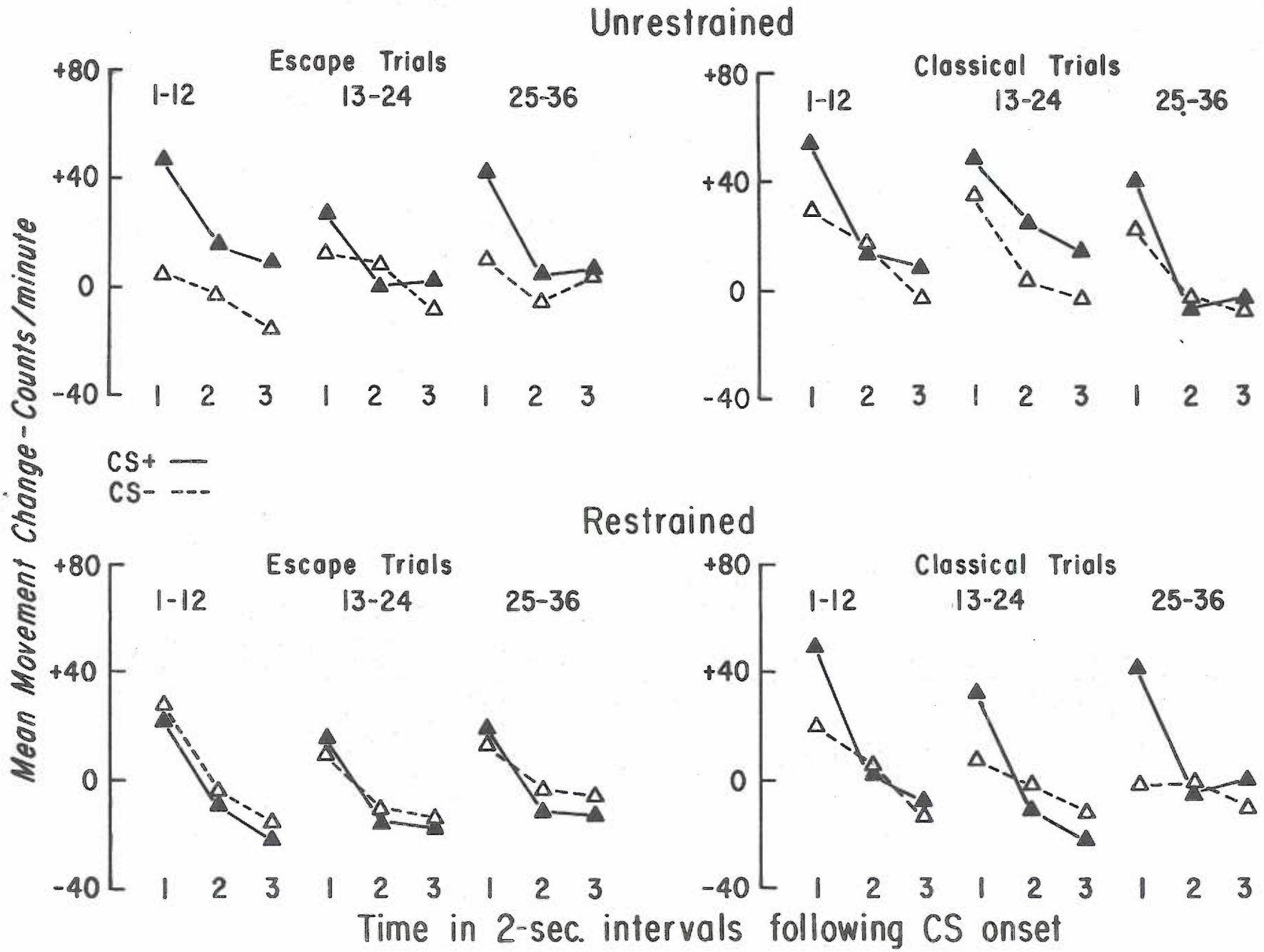
in the magnitude of heart-rate deceleration to CS+ and CS- occurred during the last 12 trials of acquisition ($F=11.46$, $df=1/65$, $p<.01$). No significant effects were obtained in the other trial blocks.

The mean responses of the two restrained groups were compared using a 2 x 2 x 3 analysis of variance (escape vs. classical x CS+ vs. CS- x trial blocks). This test established that the groups were not significantly different from each other. There was a reliable increase in the magnitudes of the heart-rate responses over the three blocks of acquisition ($F=9.46$, $df=2/52$, $p<.01$) and a reliable CS+ vs. CS- x trial blocks interaction ($F=9.88$, $df=2/52$, $p<.01$) indicating that the responses to CS+ and CS- diverged over the three trial blocks.

Movement. Mean movement responses of the groups in each 2-sec. interval of CS+ and CS- are plotted in Figure 3 as a function of 12 trial blocks during acquisition. Examination of the upper half of the figure reveals that the onset of both CSs produced a burst of activity in the unrestrained groups that was slightly larger in magnitude to CS+ than to CS- during each phase of acquisition. These onset reactions were then followed by gradual reductions in movement across the three measurement intervals to near or in several instances slightly below pre-CS levels.

Individual 2 x 3 (CS+ vs. CS- x counting intervals) analyses of variance performed at each stage of acquisition provided a significant overall difference between CS+ and CS- during the first 12 trials in the escape group ($F=15.27$, $df=1/65$, $p<.01$). In this block there was also a reliable CS+ vs. CS- x counting periods interaction ($F=5.15$, $df=2/65$, $p<.01$) reflecting the difference in the changes in movement over the counting intervals of the CSs. In

Figure 3. Mean CS minus pre-CS movement responses to CS+ and CS- during the three 2-sec. periods of the CS-US interval averaged over successive 12-trial blocks of acquisition for the unrestrained-escape, unrestrained-classical, restrained-escape and restrained-classical groups.


Unrestrained
Escape Trials
Classical Trials
1-12
13-24
25-36
+80
+40
0
-40
1 2 3
CS+
CS-
Restrained
Mean Movement Change - Counts/minute
Time in 2-sec. intervals following CS onset

addition, the overall changes in movement during the counting intervals of the CSs were reliable in each 12 trial block of acquisition: Escape group (Trial block one: $F=5.17$, $df=2/65$, $p<.05$; Trial block two: $F=3.19$, $df=2/65$, $p<.05$; Trial block three: $F=5.65$, $df=2/65$, $p<.01$), Classical group (Trial block one: $F=6.94$, $df=2/65$, $p<.01$; Trial block two: $F=4.16$, $df=2/65$, $p<.05$; Trial block three: $F=12.60$, $df=2/65$, $p<.01$). None of the outcomes pertaining to differences in the topography of movement activity to the two CSs were significant. As a further test of the presence of conditioning, the movement responses in the first counting intervals of CS+ and CS- were compared for both groups using separate 2 x 3 (CS+ vs. CS- x trial blocks) analyses of variance. For the unrestrained escape group, these tests resulted in a reliable effect of CS+ vs. CS- ($F=9.87$, $df=1/65$, $p<.01$) demonstrating an overall learned movement reaction to the onset of CS+. In the unrestrained classical group the CS+ vs. CS- effect fell just short of statistical significance ($F=3.79$, $df=1/65$, $p>.05$; $F=3.98$ needed for $p<.05$) indicating that there was also a tendency for conditioned movement to occur to the onset of CS+ in this group.

Finally, to determine if the conditioning procedure affected the movement activity shown by the unrestrained groups a 2 x 2 x 3 (escape vs. classical x CS+ vs. CS- x trial blocks) analysis of variance was carried out using the movement reactions averaged over the three CS counting intervals as the raw data. This test demonstrated that the two groups were not significantly different from each other. There was, however, a significant difference in the overall responses to CS+ and CS- ($F=5.14$, $df=1/26$, $p<.05$) providing additional evidence of learned increases in movement to CS+ in the unrestrained treatment condition.

The lower half of Figure 3 shows that the burst of activity to the CSs found in the unrestrained groups was also generally present in the restrained groups. However, in the latter case there was a clear tendency for movement to decrease below pre-CS levels by the second or third counting interval. Of the two restrained groups, only the group receiving classical-conditioning training provided visually apparent differential responding to CS+ and CS- with that occurring mainly in the first counting interval of the CSs.

To test for the presence of conditioned movement in the restrained groups separate 2 x 3 (CS+ vs. CS- x counting intervals) analyses of variance were performed on the trial block data shown in the lower portion of Figure 3. The only significant outcomes were those relating to overall changes in movement to the CSs across the three counting intervals: Escape group (Trial block one: $F=4.78$, $df=2/65$, $p<.05$; Trial block two: $F=3.29$, $df=2/65$, $p<.05$; Trial block three: $F=6.81$, $df=2/65$, $p<.01$), Classical group (Trial block one: $F=10.10$, $df=2/65$, $p<.01$; Trial block two: $F=6.10$, $df=2/65$, $p<.01$). None of the factors reflecting possible differences in responding to CS+ and CS- were significant. As a further assessment of conditioning, the responses in the first counting intervals of CS+ and CS- for both groups were compared by means of individual 2 x 3 (CS+ vs. CS- x trial blocks) analyses of variance. The only significant outcome of these tests was that of CS+ vs. CS- ($F=8.84$, $df=1/65$, $p<.01$) in the restrained classical group demonstrating that learned movement activity occurred during the first counting interval of CS+ in this group.

Finally, to assess the influence of conditioning procedure on the movement responses of the restrained groups a 2 x 2 x 3 (escape vs.

classical x CS+ vs. CS- x trial blocks) analysis of variance was carried out using the mean movement scores averaged over the three intervals of the CSs. This test failed to show that there was a reliable difference between the groups. The analysis did demonstrate that the slight reduction in movement across blocks of acquisition was reliable ($F=3.20$, $df=2/52$, $p < .05$).

One final point to be made with respect to the data shown in Figure 3 is that the unrestrained groups showed a higher overall level of movement to both CSs than did the restrained groups. This visually apparent finding was shown to be reliable in a 2 x 2 x 3 (unrestrained vs. restrained x CS+ vs. CS- x trial blocks) analysis of variance ($F=4.53$, $df=1/54$, $p < .05$). None of those factors relating to conditioning of movement were reliable.

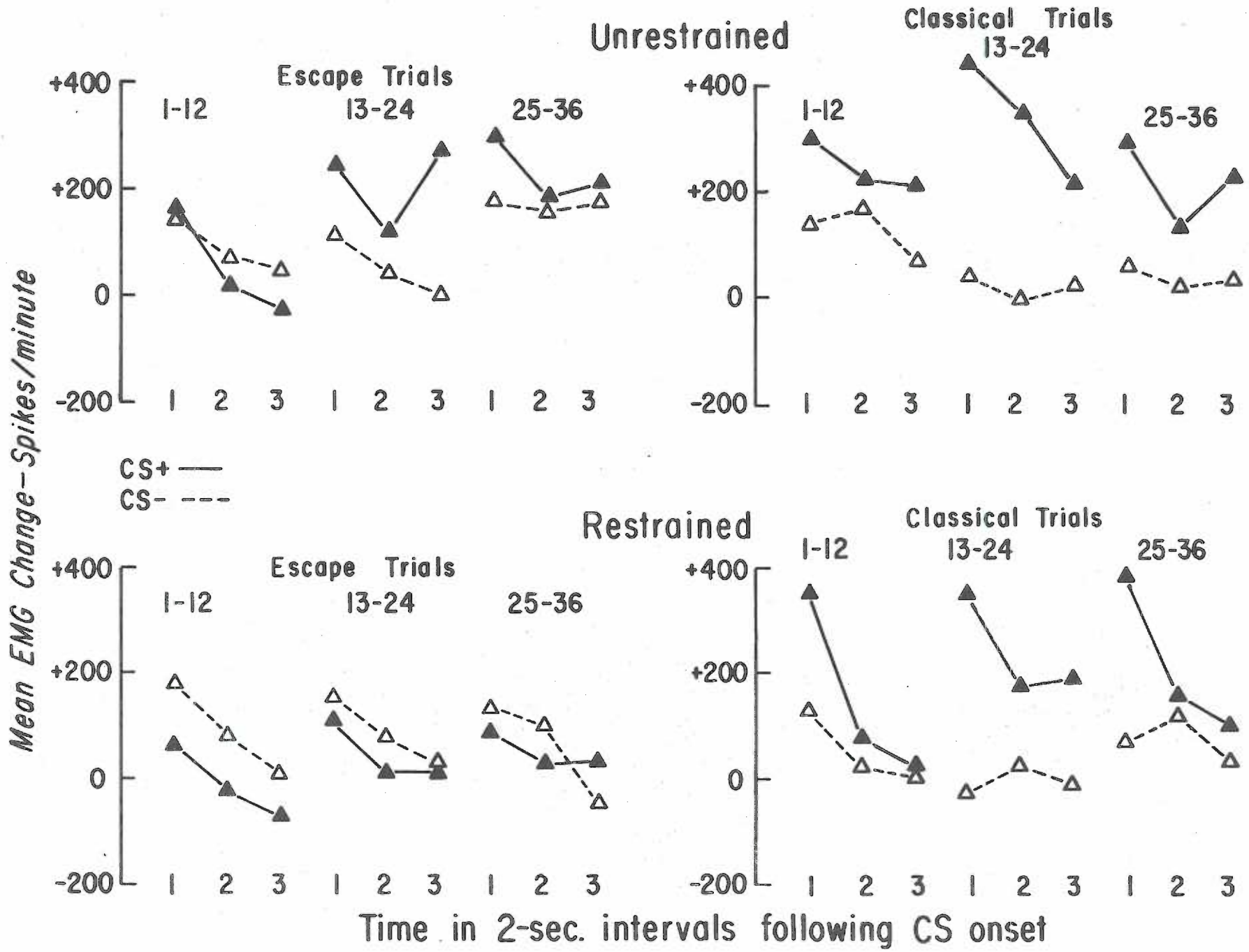
In summary, the analyses carried out in this section provided some indication that conditioned movement occurred in both the unrestrained and restrained groups. However, in both cases it was restricted primarily to the first third of the CS-US interval. Only in the unrestrained-escape group were the overall responses to CS+ and CS- reliably different and this occurred during the first block of acquisition trials. There were no differences between the escape and classical groups in terms of amount and pattern of movement activity shown. It may be pertinent to note that when compared to movement in the pre-test trials (see Figure 1) the conditioned movement responses in the first counting interval shown by the unrestrained-escape and restrained-classical groups seemed to be due to a reduction in movement to CS- rather than to an increase in movement to CS+. Thus, the reactions to CS+ during acquisition were highly similar in magnitude to those elicited during pre-test. On the other hand, CS- reactions appeared

to be inhibited during acquisition.

Comparison of Figures 2 and 3 reveals that there was virtually no temporal relationship between conditioned changes in movement activity and conditioned changes in heart rate. The learned movement responses shown by the unrestrained escape group in the first block of acquisition were accompanied by the complete absence of learned heart rate. Furthermore, the conditioned increases in movement to the onset of CS+ occurred in this group at the same time that the smallest differences in the accelerative heart-rate reactions to CS+ and CS- were found. In fact, the heart-rate differences in the first counting intervals of the CSs were close to zero in each of the three phases of acquisition. In the case of the restrained-classical group, heart rate decelerated to the onset of CS+ in spite of conditioned increases in movement during this period of time. However, it was true that the magnitude of the heart-rate differences between CS+ and CS- were generally smaller during the first counting interval than during subsequent intervals.

EMG activity. Mean EMG activity of the groups in each 2-sec. interval of CS+ and CS- are depicted in Figure 4. It can be seen that, in general, all groups showed an overall increase in EMG activity during the CSs. Although somewhat more variable than movement, the EMG reactions also tended to be larger during the first counting intervals of the CSs than during the second and third intervals. With the exception of the restrained-escape group shown in the lower left-hand portion of the figure, all of the groups demonstrated more EMG activity in the first counting interval of CS+ than in the same interval of CS-. Due largely to the responses of

Figure 4. Mean CS minus pre-CS EMG responses of the unrestrained-escape, unrestrained-classical, restrained-escape and restrained-classical groups in each 2-sec. interval of CS+ and CS- for each 12-trial block during acquisition.


Unrestrained
Classical Trials
Escape Trials
1-12
13-24
25-36
+400
+200
0
-200
1 2 3
CS+
CS-
Restrained
Mean EMG Change–Spikes/minute
Time in 2-sec. intervals following CS onset

one subject, the restrained-escape group showed just the opposite relationship. Contrary to what was observed for movement, the EMG reactions of the restrained groups generally failed to fall below pre-CS levels in the second and third counting intervals.

In general, Figure 4 shows that there was a larger difference between the responses to CS+ and CS- in the classical groups than in the escape groups. The differences between CS+ and CS- were significant for the unrestrained classical group in trial blocks two ($F=22.19$, $df=1/40$, $p < .01$) and three ($F=8.37$, $df=1/40$, $p < .01$) and for the restrained classical group in trial block two ($F=9.21$, $df=1/40$, $p < .01$). Although Figure 4 provides some visually apparent differences between the EMG responses of the escape and classical groups in the restrained and unrestrained conditions none of these were significant according to appropriate analyses of variance.

In a further attempt to ascertain the extent to which conditioned changes in EMG activity occurred, separate 2 x 3 (CS+ vs. CS- x trial blocks) analyses of variance were performed on the responses of all four groups in the first counting intervals of CS+ and CS-. The results of these tests were consistent with those performed at each stage of acquisition in that there was a reliable conditioned EMG reaction in the first interval of CS+ only in the unrestrained ($F=10.56$, $df=1/40$, $p < .01$) and restrained ($F=10.90$, $df=1/40$, $p < .01$) classical groups.

To summarize, it is important to note that the reliable conditioning effects shown by the two classical groups reflect the fact that EMG activity increased more to CS+ than to CS-. Although the conditioned increases in the unrestrained-classical group was temporally

coincident with the conditioned increases in heart rate shown by this group (see upper right-hand section of Figure 2) the topographies of the two reactions were quite different. Thus, the magnitudes of the EMG differences between CS+ and CS- became smaller over the three counting intervals at the same time that the magnitudes of the accelerative heart-rate differences became larger. The absence of a positive relationship between EMG and heart-rate changes is even more obvious in the restrained-classical group. In this case, the two responses changed in opposite directions with EMG increasing to CS+ and heart rate decreasing to CS+.

Trial by trial analysis of heart rate and movement

The acquisition results provided virtually no evidence of a relation between conditioned changes in heart rate and conditioned changes in motor activity. The single instance of an overall learned movement reaction shown by the unrestrained-escape group in the first block of acquisition was accompanied by the absence of a learned heart-rate response. Also, in the unrestrained-escape and restrained-classical groups the learned bursts of movement activity during the first counting interval were not temporally coincident with learned heart-rate changes. That is, the heart-rate change to the two CSs was essentially zero for the unrestrained group during the first counting interval while the restrained group showed substantial nondifferential decelerations to both CSs during the same period.

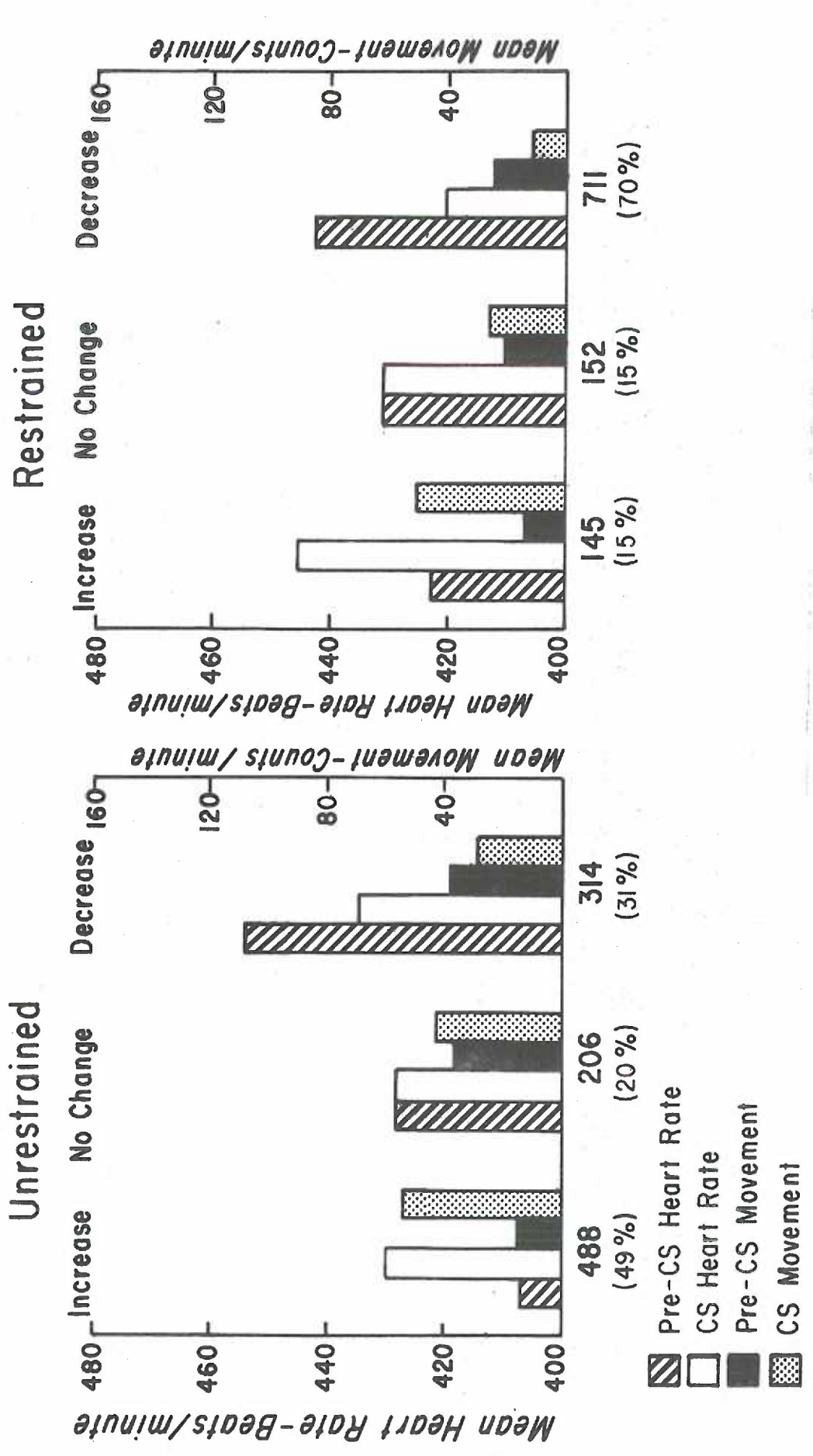
Disregarding learned changes and simply focusing on the form of movement and heart-rate reactions failed to provide any clear evidence of a correspondence between the two measures. All

groups showed a similar burst of movement activity to the onset of CS+ with the amount of activity then declining toward pre-CS base-line levels during the final two counting intervals. In contrast to this characteristic movement response, the heart-rate reactions tended to peak in the final counting interval just prior to the presentation of the US. In the case of the unrestrained groups this movement was maximal at the onset of the CS whereas heart-rate acceleration was maximal at the end of the CS. For the restrained groups increased movement at the beginning of the CS was associated with cardiodeceleration. Only the decreases in movement during the final two counting intervals shown by the restrained groups furnished a correspondence between decelerations in heart rate and reductions in movement activity.

It is conceivable, however, that the above analysis based on averages over blocks of 12 trials may have obsecured common variations between heart rate and movement that may have been present from trial to trial during acquisition. In order to obtain information bearing on this possibility all CS+ trials were sorted into three categories on the basis of whether heart rate increased, showed no change, or decreased. Movement activity on the three types of trials was then tabulated.

Figure 5 shows mean absolute heart rate and movement during the pre-CS+ and CS+ periods in each of the three categories of trials. Since there was no statistical difference between the results of the escape and classical groups in the unrestrained and restrained conditions, the two groups were combined in each condition. The results of the combined unrestrained group are presented on the left side of the figure

Figure 5. Mean heart rate and movement of the unrestrained and restrained groups during the pre-CS+ and CS+ periods on those trials in which heart rate either increased, showed no change or decreased. The total number and percentage number of each type of trial are shown below each set of histograms.


Unrestrained
Restrained
Increase
No Change
Decrease
Mean Heart Rate-Beats/minute
Mean Movement-Counts/minute
480
460
440
420
400
160
120
80
40
488 (49%)
206 (20%)
314 (31%)
145 (15%)
152 (15%)
711 (70%)
Pre-CS Heart Rate
CS Heart Rate
Pre-CS Movement
CS Movement

while those of the combined restrained group are depicted on the right side. The absolute numbers and percentage numbers of each type of trial are shown at the bottom of each category in the figure.

The left side of the figure reveals that there was considerable variability in the direction of the heart-rate responses of the unrestrained group. Heart rate increased to the CS+ on 49% of the trials and decreased on 31% of the trials. On the remaining 20% of the trials there was no change in heart rate. That heart-rate accelerations were the predominant responses was shown by the fact that the percentage number of trials in the increase category was significantly larger than either the percentage number of decrease ($t=2.14$, $df=29$, $p < .05$) or no change ($t=2.45$, $df=29$, $p < .01$) trials. Furthermore, decreases in heart rate occurred more frequently than no change ($t=2.89$, $df=29$, $p < .01$) in heart rate.

On trials when heart rate accelerated (see far left of figure) a relatively large increase in movement resulted as compared to the slight movement change in both the no change and decrease categories. Only on accelerative trials was the change in movement reliably different from zero ($t=4.87$, $df=27$, $p < .01$) indicating that heart-rate accelerations tended to be associated with increased movement while decelerations were relatively independent of reliable changes in movement.

The left side of Figure 5 also reveals that pre-CS+ heart rate was lower on trials in which cardioacceleration occurred to CS+ than on trials in which cardiodeceleration occurred with the rates being 407 and 454 beats per minute, respectively. Although

this difference was significant ($t=8.68$, $df=26$, $p < .01$) there were no reliable correlations between the magnitudes of the conditioned heart-rate changes and basal heart rates.

Inspection of the right panel of Figure 5 reveals that the restrained group showed decelerative responses on 70% of the trials. This proportion of trials was significantly larger than either the no change ($t=12.31$, $df=29$, $p < .01$) or the increase category ($t=10.02$, $df=29$, $p < .01$), each of which comprised only 15% of the trials. The proportions of trials in the latter two categories were not reliably different from each other. The large number of trials (711 out of 1,008) in the decrease category attests to the consistency of the heart-rate reactions in the restrained as compared to the unrestrained groups.

Examination of the movement responses of the restrained groups indicates that there was a small overall decrease in movement on decelerative heart-rate trials and an increase on accelerative trials. The change in movement to CS+ was reliably different from zero in both cases (decrease $t=2.73$, $df=27$, $p < .01$; increase $t=2.70$, $df=21$, $p < .01$). The slight increase in movement in the no-change-in-heart-rate category was not reliably different from zero. These results suggest that there was a tendency for the heart rate and movement responses of the restrained group to go together.

Similar to what was found in the unrestrained condition base-level heart rate was significantly higher on heart-rate decelerative trials than on heart-rate accelerative trials and this difference was significant ($t=4.67$, $df=21$, $p < .01$). However, there were again no reliable correlations between base-level heart rates and the

magnitudes of either accelerative or decelerative heart-rate reactions to CS+.

The above analysis provided an overall assessment of mean movement activity on trials when heart rate either increased, decreased, or showed no change. However, this sorting procedure did not allow a determination to be made of the actual numbers of trials that movement changed in a given direction within each heart-rate category. This information was obtained by sorting the trials within each of the three heart-rate categories on the basis of whether an increase, no change, or a decrease in movement occurred. The results of this procedure for both unrestrained and restrained groups are presented in Table 1. The absolute numbers of trials in the respective movement classifications and the percentage number of such trials along with mean pre-CS+heart rates, mean heart-rate changes to CS+, mean pre-CS+ movements, and mean movement changes to CS+ occurring on these trials are presented in columns one through six, respectively. When appropriate, the values in each no-change-in-movement grouping were tested separately against those in both the increase and decrease in movement classes using paired t-tests. These tests were based on varying number of rats since not all rats contributed data to all movement categories.

Responses of the unrestrained group are presented in the upper portion of Table 1. Inspection of the increase_in_heart-rate section shown at the top of the table reveals that of the 488 trials in which cardioacceleration occurred to CS+, movement increased on 263 or 54% of the trials with the average being +71.40 counts,showed no change on 204 or 42% of the trials, and decreased on 21 or 4% of

Table 1. The number of CS+ trials in which heart rate increased, showed no change, or decreased are shown separately for the unrestrained and restrained groups in the first column. The number of trials within each of these heart-rate categories when movement increased, showed no change, or decreased is depicted in the second column. The percentage number of these trials, along with mean pre-CS+ heart rates, mean heart-rate changes to CS+, mean pre-CS+ movements, and mean movement changes to CS+ are presented in columns three through seven, respectively. The heart rate and movement data in each of the no-change-in-movement groupings were tested against those that were found in the increase-and decrease-in-movement classifications using paired t-tests.

UNRESTRAINED

	Total Mov. Trials	% Total	Pre-CS HR	CS-HR	Pre-CS Mov.	CS-Mov.
↑Heart-rate Trials (488)	↑ 263	54	408.66**	27.60***	9.40*	71.40
	~ 204	42	405.76	18.74	.46	.00
	↓ 21	4**	473.25***	17.94*	191.90***	-117.20
~Heart-rate Trials (206)	↑ 67	33	436.79*	.00	13.10*	59.90
	~ 119	58	416.48	.00	.04	.00
	↓ 20	9**	473.21***	.00	184.20***	-104.10
↓Heart-rate Trials (314)	↑ 115	37	456.78*	-17.17	25.30	43.70
	~ 128	41	445.52	-19.05	2.50	.00
	↓ 71	22*	477.69***	-23.29**	124.90***	-99.10
Total	1008					

RESTRAINED

	Total Mov. Trials	% Total	Pre-CS HR	CS-HR	Pre-CS Mov.	CS-Mov.
↑Heart-rate Trials (145)	↑ 73	50	431.11*	23.09**	4.40	96.20
	~ 54	37	423.19	13.52	1.60	.00
	↓ 18	13	443.00	15.50	110.00***	-46.50
~Heart-rate Trials (152)	↑ 59	39*	430.82	.00	5.30	33.80
	~ 80	53	429.86	.00	.25	.00
	↓ 13	8**	457.77	.00	237.70*	-156.10
↓Heart-rate Trials (711)	↑ 139	19**	444.26	-18.78	2.00*	26.40
	~ 431	61	435.63	-20.72	.34	.00
	↓ 141	20***	464.30***	-30.58***	82.20***	-69.70
Total	1008					

Increase ↑
No Change ~
Decrease ↓

$p < .05$ *
$P < .01$ **
$p < .001$ ***
(one tailed)

the trials with the average decrease of -117.20. The difference between the percentage number of no change and increase trials was not reliably different indicating that movement was equally likely to either increase or show no change in the presence of heart-rate acceleration to CS+. Although they rarely occurred decreases in movement were sometimes accompanied by increases in heart rate.

The mean heart-rate acceleration of 27.60 beats per minute shown by the unrestrained group on movement increase trials was reliably higher than the 18.74 beats-per-minute acceleration occurring on movement-free trials. In those instances when decreases in movement occurred heart-rate accelerations were slightly but, nevertheless, reliably reduced. When compared to the no-change-in-movement category, pre-CS+ heart rate and movement were reliably elevated in both the movement increase and decrease groupings with the differences being larger in the case of the decrease category. In summary, these results indicate that the accelerative heart-rate reactions of the unrestrained group occurred in the absence of movement but that the magnitudes of the accelerations varied with the direction of motor activity elicited by CS+. Also, pre-CS+ heart-rate levels were positively related to base-level movement activity.

From an examination of the trials comprising the cardiodeceleration category shown in the lower portion of the top half of Table 1 it can be seen that movement increased on 37% of the trials with the average being +43.70 counts, showed no change on 41% of the trials and decreased on 22% of the trials with the average being -99.10 counts. The increase and no change percentages were not reliably different from one another. However, the percentage of decrease-in-movement trials was reliably smaller than the percentage of trials

in which there was no change in movement. Thus, cardiodeceleration in the unrestrained group occurred more frequently on no-change and increase-in-movement trials. In addition, the heart-rate decelerations on movement increase trials were comparable to those that occurred on movement-free trials. However, when movement decreased, decelerations in heart rate were slightly but significantly larger than those obtained on movement-free trials. Compared to that which was found on no-change trials, pre-CS+ heart rate was reliably elevated on movement-increase and movement-decrease trials as was pre-CS+ movement on decrease trials. Consistent with what was found in the heart-rate increase section, the cardiodecelerations of the unrestrained groups occurred in the complete absence of motor activity on a substantial number of trials. However, the magnitude of the deceleration was augmented when movement decreased.

The middle no-change-in-heart-rate section shows that movement trials were approximately equally distributed between the no-change and increase categories with the percentages of each type of trial not being reliably different from one another. The percentage of decrease trials was significantly lower than that shown in the no-change grouping. Once again, pre-CS+ heart rates and motor movements in the increase and decrease categories were elevated above the levels observed in the no-change classification. As a final point, it should be noted that the magnitudes of the movement changes on the increase and decrease trials were comparable to those shown in the two other heart-rate sections and yet corresponding changes in heart rate did not occur on these trials.

The three heart-rate categories of the restrained group sorted on the basis of whether movement increased, showed no change, or decreased are presented in the bottom half of Table 1. Focusing on the lower panel it can be seen that of the 711 decelerative trials, movement showed no change to CS+ on 431 or 61% of the trials, increased on 139 or 19% of the trials with the average being +26.40 counts,and decreased on 141 or 15% of the trials with the average being -69.70 counts. The percentage of no-change-in-movement trials was reliably larger than either the percentage of trials in the increase- or decrease-in-movement groupings. The percentages of trials in which movement either increased or decreased were essentially identical. Thus, in the restrained group decelerative heart-rate reactions were equally likely in the presence of an increase or a decrease in movement to CS+ and in fact decelerations occurred most of the time in the absence of any change in movement.

The lower panel of Table 1 also shows that magnitude of the heart-rate deceleration (31 beats per minute) occurring on movement decrease trials was reliably larger than that shown on no-change-in-movement trials (21 beats per minute). Cardiodecelerations on the increase- and no-change-in-movement trials did not differ from each other. When compared to the no-change category, both pre-CS+ heart rate and movement were reliably elevated in the decrease-in-movement classification, while only pre-CS+ movement was reliably higher in the increase-in-movement category. In summary, these results indicate that in the restrained group heart-rate decelerations occurred without any corresponding movement change or even when movement increased. When movement changed in the same direction as heart-rate cardiodecelerations were augmented. This

finding is similar to that found in the accelerative and decelerative heart-rate categories of the unrestrained group.

Examination of the cardioaccelerative trials shown in the upper section of the bottom half of Table 1 reveals that movement increased on 50% of the trials averaging +96.20 counts,showed no change on 37% of the trials and decreased on only 13% of the trials averaging -46.50 counts. Like heart-rate decelerations, cardioaccelerations of the restrained group occurred in the absence of any movement and were reliably augmented when movement changed in the same direction as heart rate. However, the heart-rate accelerations on movement decrease trials were comparable to those that occurred on movement-free trials. As compared to the no-change-in-movement grouping, pre-CS+ heart rate was reliably elevated when movement increased while pre-CS+ movement was reliably elevated when movement decreased.

Inspection of the middle, no-change-in-heart-rate section reveals that the percentage of no-change-in-movement trials (53%) was reliably larger than the percentage of trials in which movement increased (39%) with an average of +33.80 counts,or the percentage of trials in which movement decreased (8%) with an average of -156.10 counts. On the extremely few decrease-in-movement trials pre-CS+ movement was reliably higher than on movement-free trials. Once again, sizeable increases and decreases in movement occurred on trials in the absence of any heart-rate change on these trials.

The total number of trials in which movement increased, showed no change, or decreased was calculated separately for the unrestrained and restrained groups. This analysis indicated that in the unrestrained group movement increased on a total of 445 trials (44%)

showed no change on 451 trials (45%) and decreased on 112 trials (11%). In the restrained group movement increased on 271 trials (27%), did not change on 565 trials (56%) and decreased on 172 trials (17%).

Separate t-tests comparing these percentages demonstrated that there were more movement-increase trials in the unrestrained group ($t=2.63$, $df=54$, $p < .01$) than in the restrained group. Furthermore, there were more decrease-in-movement trials in the restrained group ($t=2.45$, $df=54$, $p < .01$) than in the unrestrained group. No-change-in-movement trials occurred equally often in the two groups.

Variability of heart-rate reactions

The results of the previous section demonstrated that the heart-rate reactions of the unrestrained group to CS+ were considerably more variable than those of the restrained group. In the unrestrained condition, increases, no change and decreases in heart rate constituted 49, 20, and 31 percent of the trials respectively, while in the restrained condition heart rate decreased on 70 percent of the trials. Examination of the mean difference score heart-rate responses to CS+ of the combined unrestrained groups during the last 12 acquisition trials revealed that approximately one-half of the subjects showed accelerative heart-rate reactions while the remaining subjects showed decelerative reactions. In the combined restrained group only two out of 28 subjects failed to show a mean decrease in heart rate during the final block of acquisition.

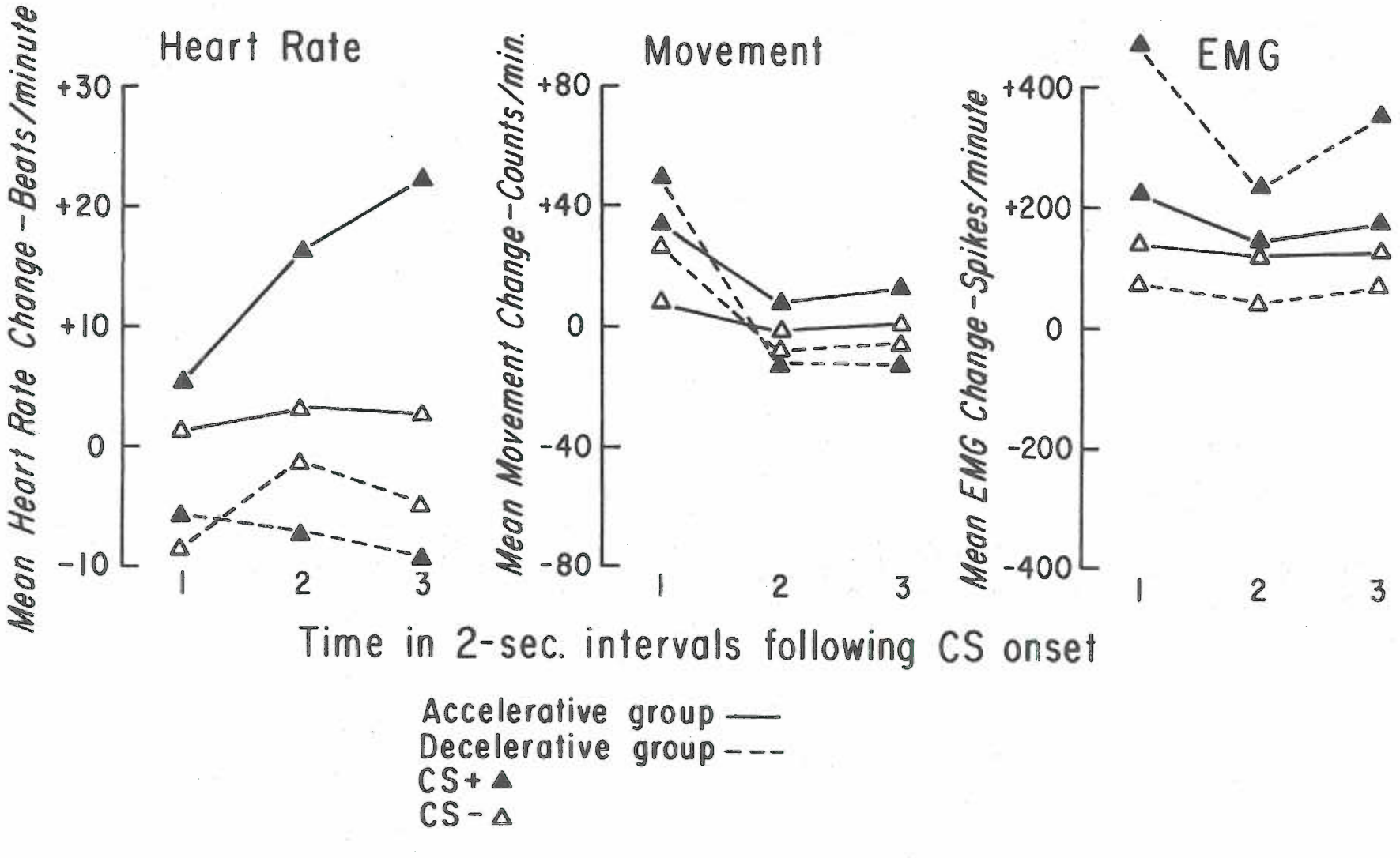
In order to examine further the relationship between heart rate and movement activity in the unrestrained condition, those subjects showing accelerative reactions (n=16) were combined into one group, while those showing decelerative responses (n=12) were combined into

another group. Heart rate, movement, and EMG responses of the two groups in successive 2-sec. intervals of CS+ and CS- adjusted for base level and averaged over the last 12 trials of acquisition are plotted in Figure 6. From inspection of the left-hand portion of the figure it is apparent that the accelerative group developed a well differentiated conditioned response. Cardioacceleration increased in magnitude to CS+ over the three counting intervals while the response to CS- was near zero. The reliability of these visually apparent findings were established in a 2 x 3 (CS+ vs. CS- x counting intervals) analysis of variance. The outcomes of this test provided a significant effect of CS+ vs. CS- ($F=53.75$, $df=1/75$, $p < .001$) a significant effect of counting intervals ($F=11.02$, $df=2/75$, $p < .01$) and a significant CS+ vs. CS- x counting period interaction ($F=8.00$, $df=2/75$, $p < .01$) reflecting the difference in the heart-rate changes to the two CSs.

In contrast to the obvious difference between CS+ and CS- in the accelerative group, there was little evidence of differentiation between the two CSs in the decelerative group. The only indication of a conditioned heart-rate reaction was the slight increase in magnitude of the heart-rate change to CS+ over the three counting intervals coupled with the small decline in response magnitude to CS-. A 2 x 3 analysis of variance resulted in a reliable CS+ vs. CS- x counting intervals interaction ($F=3.88$, $df=2/55$, $p < .05$) demonstrating that the heart-rate changes to CS+ and CS- were in fact different. None of the other factors were significant.

To determine whether differences in base-level heart rates might have contributed to the opposing heart-rate response shown by these two

Figure 6. Mean CS minus pre-CS heart rate, movement and EMG responses to CS+ and CS- during each 2-sec. interval of the CSs for those unrestrained subjects (n=16) showing heart-rate acceleration to CS+ and those unrestrained subjects (n=12) showing heart-rate deceleration to CS+ averaged over the last 12 acquisition trials.


Heart Rate
Movement
EMG
Mean Heart Rate Change - Beats/minute
Mean Movement Change - Counts/min.
Mean EMG Change - Spikes/minute
+30
+20
+10
0
-10
+80
+40
0
-40
-80
+400
+200
0
-200
-400
1
2
3
Time in 2-sec. intervals following CS onset
Accelerative group
Decelerative group
CS+
CS-

groups, pre-CS+ heart rates were tabulated for both groups over the last 12 acquisition trials. These tabulations revealed that the rates for the accelerative and decelerative groups were 413 and 408 beats per minute, respectively. A t-test established that this difference was not significant.

The middle panel of Figure 6 shows the movement responses of the two groups. Both groups displayed an increase in movement to the onset of the CSs followed by a decline in movement over the remaining two counting intervals. However, only the accelerative group showed any indication of a conditioned movement response. A 2 x 3 (CS+ vs. CS- x counting intervals) analysis of variance performed on the movement results of the accelerative group demonstrated that the difference between CS+ vs. CS- was reliable ($F=8.29$, $df=1/74$, $p < .05$). An identical analysis carried out for the decelerative group revealed that the overall change in movement during the CSs was the only significant factor ($F=14.45$, $df=2/55$, $p < .01$). It may be pertinent to note that movement activity of the unrestrained decelerative subjects in this analysis was similar to that shown for the restrained groups in the bottom half of Figure 3. Thus, whether restrained or unrestrained, those rats showing cardiodeceleration to the CSs displayed a common pattern of movement activity. Movement responses of the accelerative subjects were also comparable to those shown for the unrestrained groups in the top half of Figure 3.

The EMG responses of both groups are presented in the right-hand section of Figure 6. The reactions of the accelerative group were similar to their movement responses, but in this case the small dif-

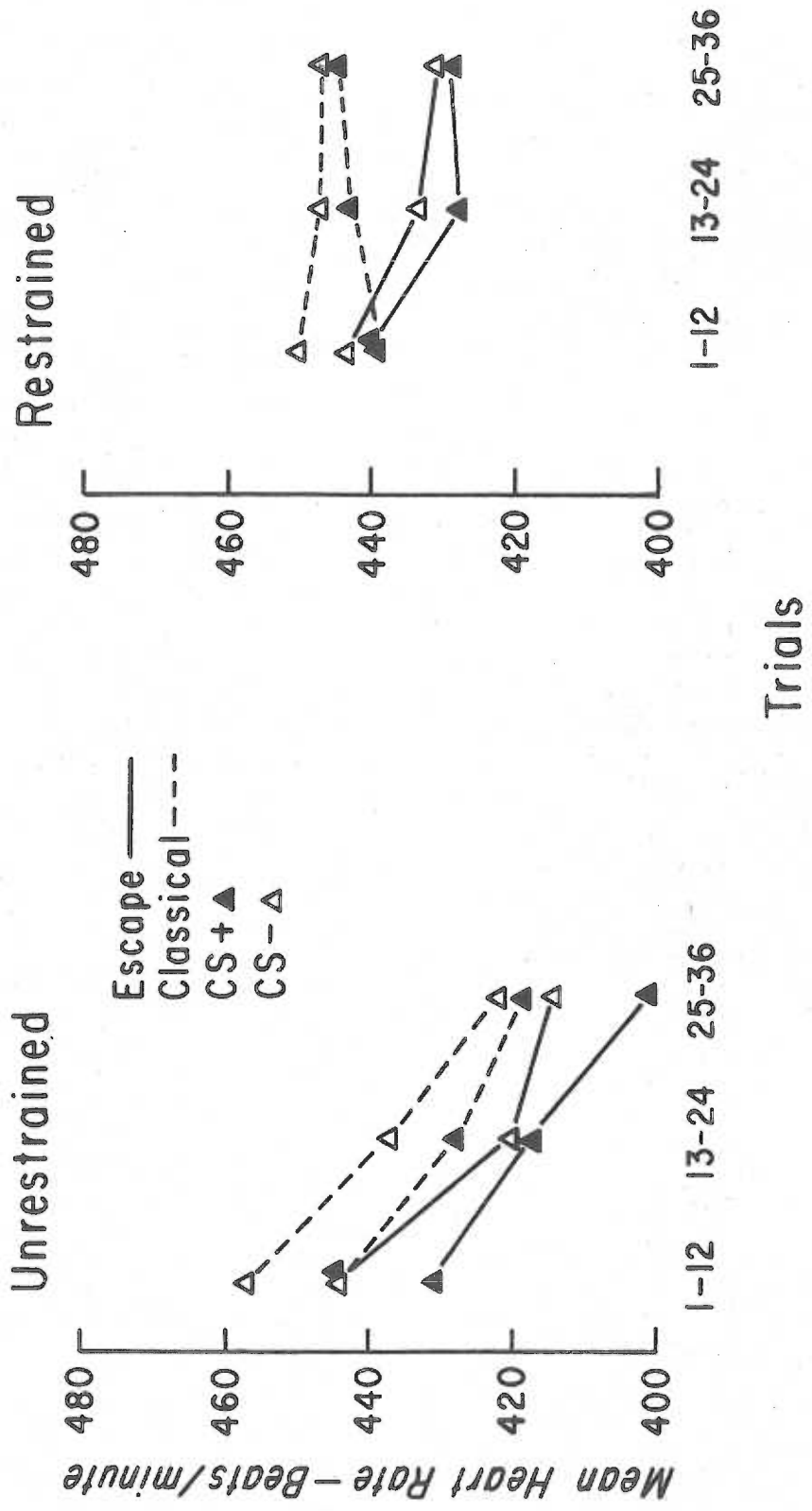
ference between the two CSs was not significant. In contrast to both their heart-rate and movement reactions the decelerative group showed a substantial conditioned EMG response. The reliability of this response was established by a significant effect of CS+ vs. CS- ($F=7.33$, $df=1/20$, $p < .05$). Comparison of the right and left-hand portions of Figure 12 indicates that this conditioned increase in EMG activity was associated with nonsignificant cardiodeceleration to CS+

Base level heart-rate, movement and EMG activity

An estimate of base-level responding was provided by measuring the amount of activity in a 6-sec. interval of time located just prior to onset of the CS. This pre-CS measure was subtracted from activity shown during the CS to form a difference score adjusted for base level.

Heart rate. Pre-CS heart rate on CS+ and CS- trials of all four groups are presented in Figure 7 in three successive 12-trial blocks of acquisition. Inspection of the left side of the figure reveals that pre-CS heart rates of the unrestrained groups decreased substantially over acquisition with those of the escape group generally being lower than those of the classical group. Both groups also showed a slightly higher heart rate on CS- trials than on CS+ trials. In an analysis of variance the change in heart rate across acquisition trials was significant ($F=39.96$, $df=2/52$, $p < .001$) as was the difference between CS- and CS+ ($F=66.95$, $df=1/26$, $p < .001$). The difference between the escape and classical groups was not significant. The analysis also provided a significant CS+ vs. CS- x trial blocks interaction ($F=3.77$, $df=2/52$, $p < .05$) indicating that

Figure 7. Mean pre-CS heart rate on CS+ and CS- trials averaged over successive 12-trial blocks of acquisition for the unrestrained-escape, unrestrained-classical, restrained-escape, and restrained-classical groups.


Unrestrained
Restrained
Escape ——
Classical - - -
CS+ ▲
CS− △
Mean Heart Rate — Beats/minute
480
460
440
420
400
1-12
13-24
25-36
Trials

the difference between CS+ and CS- was not constant across acquisition and a significant escape vs. classical x CS+ vs. CS- x trial blocks interaction ($F=5.02$, $df=2/52$, $p < .05$) indicating that pre-CS heart rate on CS+ and CS- trials changed differently over acquisition in the two groups.

Examination of the right side of Figure 7 shows that at the beginning of acquisition pre-CS heart rates of the restrained-escape and classical groups were similar. Thereafter, their rates diverged with the escape group showing a slight decline and the classical group remaining fairly constant. Like the unrestrained groups, base-level heart rate of the restrained groups was slightly higher on CS- trials than on CS+ trials although in the case of the classical group this difference was larger at the beginning than at the end of acquisition. Analysis of variance provided a significant effect of CS+ vs. CS- ($F=15.38$, $df=1/26$, $p < .01$) and a significant excape vs. classical x CS+ vs. CS- x trial blocks interaction ($F=3.76$, $df=2/52$, $p < .05$). The significant triple order interaction reflects the fact that the difference between CS+ and CS- changed over acquisition and that this change was not the same in the escape and classical groups.

Perusal of the two sides of Figure 7 reveals that while the pre-CS heart rates of the unrestrained and restrained groups were comparable at the beginning of acquisition, by the end of acquisition the rates of the unrestrained groups declined to a point considerably below those of the restrained groups. Analysis of variance collapsed across the CS+ vs. CS- and classical vs. escape dimensions provided a significant effect of trial blocks ($F=25.11$,

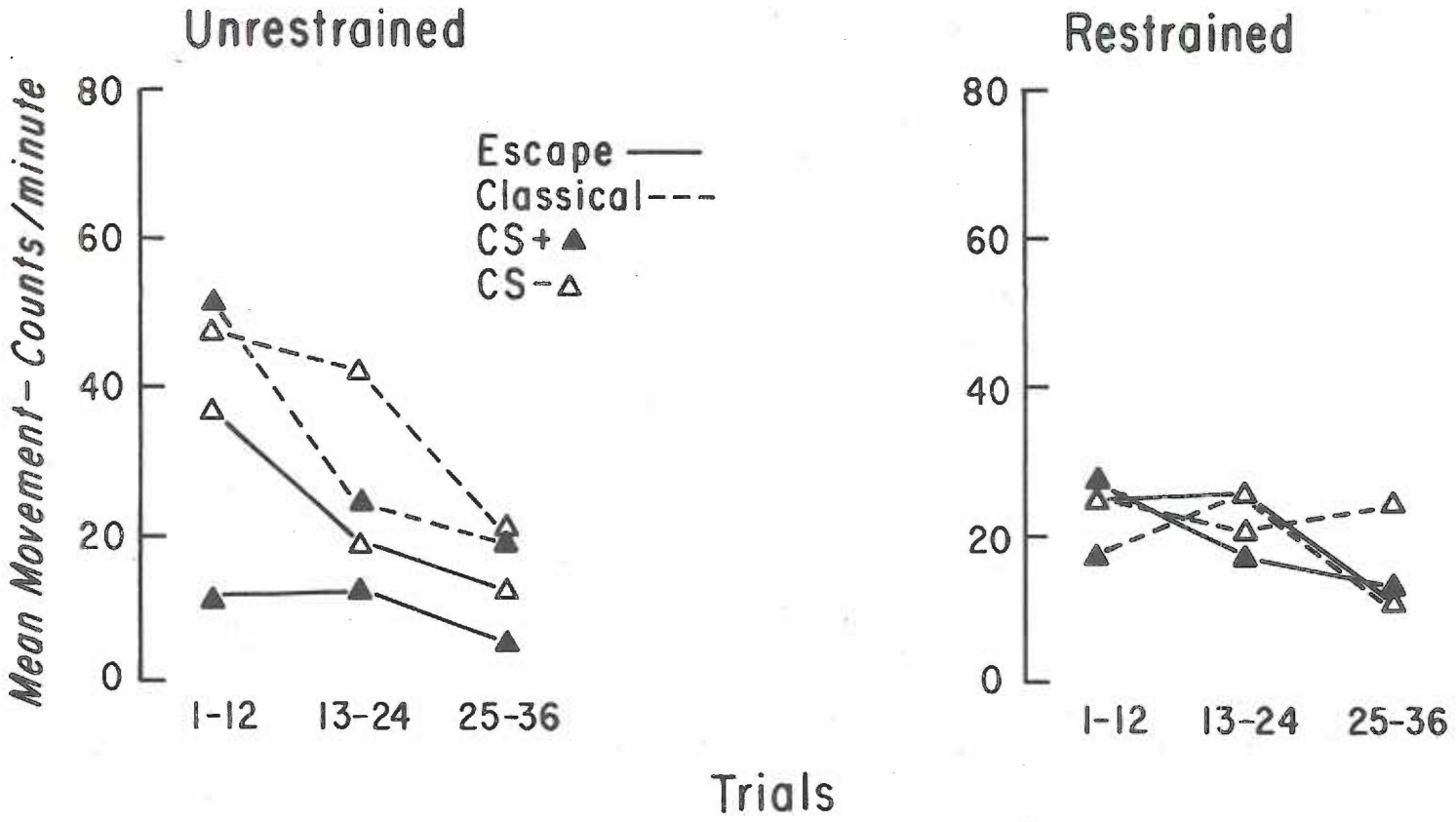
df=2/108, $p < .001$) and a significant unrestrained vs. restrained x trial blocks interaction ($F=11.06$, df=2/108, $p < .001$) establishing that the differential change in heart rate over acquisition was reliable. A comparable test carried out on just the final block of trials demonstrated that base-level heart rate of the combined restrained groups was significantly higher than that of the combined unrestrained groups at that point ($F=4.28$, df=1/54, $p < .05$).

Movement activity. Pre-CS movements to CS+ and CS- exhibited by each group are plotted separately in Figure 8. Examination of the left side of the figure reveals that the amount of movement shown by the unrestrained groups decreased over acquisition ($F=10.43$, df=2/52, $p < .01$) with more movement occurring prior to CS- than to CS+ ($F=8.07$, df=1/26, $p < .01$). Although it is visually apparent that movement in the escape group was consistently less than that in the classical group this difference was not significant.

The right side of Figure 8 fails to show any consistent differences in base-level movement of the restrained groups. Although not easy to see, there was a small decrease in movement over acquisition which was significant ($F=3.67$, df=2/52, $p < .05$).

Comparison of the left sides of Figure 7 and 8 provides visual evidence of a reasonably close relationship between heart rate and movement in the unrestrained condition. Although not perfect, the relative differences between pre-CS+ and pre-CS- heart rate and the decrease in heart rates over trials were similar to that found for movement. To check on the reliability of this relationship the decreases in heart rate between trials 1-12 and trials 25-36 collapsed across the two CSs were compared to the decreases in movement by

Figure 8. Mean pre-CS movement activity of the unrestrained-escape, unrestrained-classical, restrained-escape and restrained-classical groups on both CS+ and CS- trials as a function of 12-trial blocks during acquisition.


Unrestrained
Restrained
Mean Movement – Counts/minute
80
60
40
20
0
Escape ——
Classical---
CS+ ▲
CS- △
1-12
13-24
25-36
Trials

means of separate Spearman rank correlation tests for the classical and escape groups. The magnitudes of the heart rate and movement decreases were reliably correlated in the escape group ($r=.52$, $p < .05$, one tailed), but not in the classical group ($r=.40$ $p > .05$, one tailed).

EMG activity. Mean base levels of EMG activity to CS+ and CS- of the four groups are plotted separately in Figure 9. In general, base-level EMG activity paralleled movement in the unrestrained and restrained groups. In the unrestrained groups, EMG activity decreased over acquisition and appeared to be higher on CS- than on CS+ trials. An analysis of variance provided a significant effect of trial blocks ($F=5.73$, $df=2/32$, $p < .01$), a significant effect of CS+ vs. CS- ($F=6.08$, $df=1/16$, $p < .05$) and a significant escape vs. classical x CS+ vs. CS- x trial blocks interaction ($F=5.50$, $df=2/32$, $p < .01$).

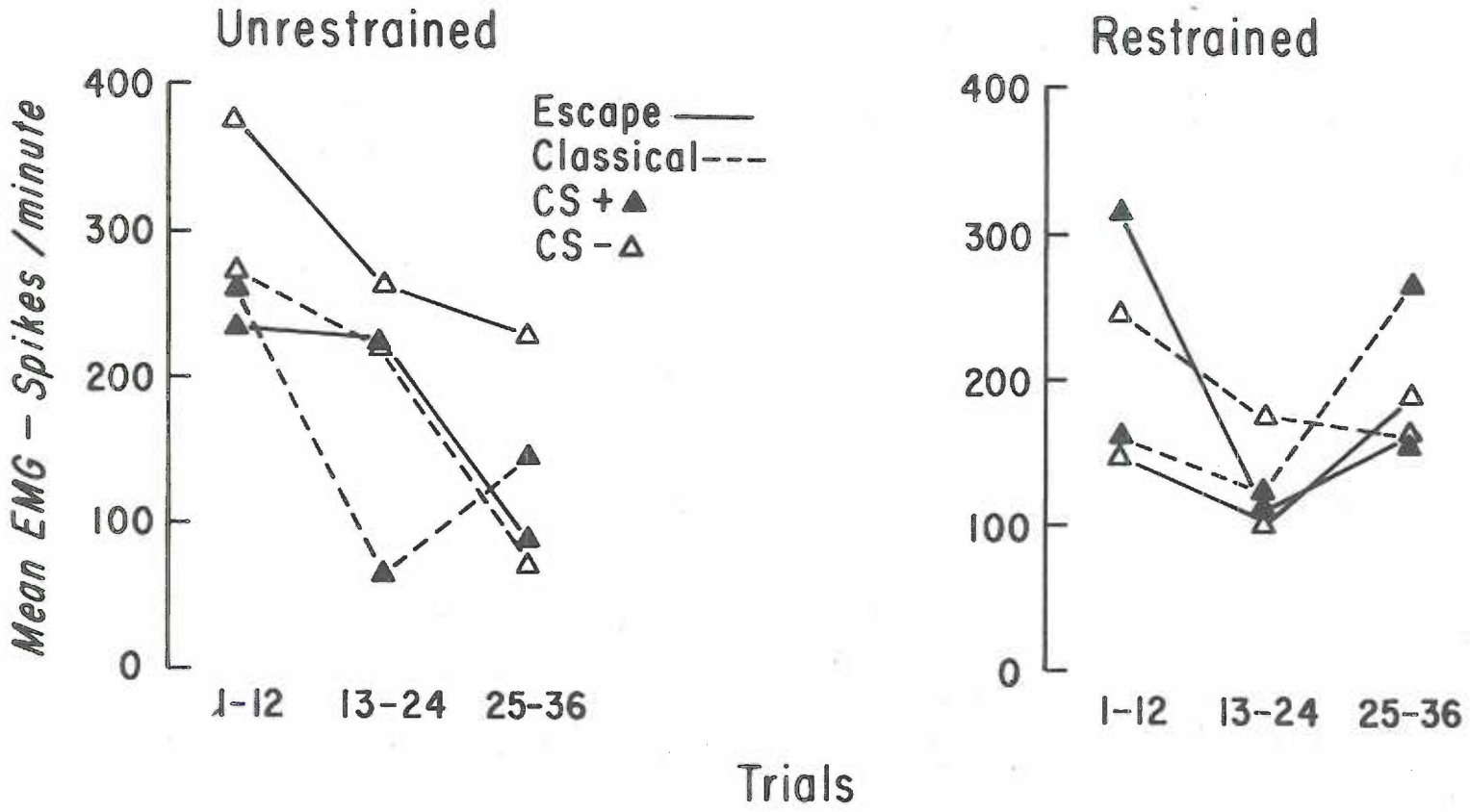
For the restrained groups, there was no evidence of systematic differences in EMG activity and an analysis of variance confirmed this impression.

Unconditioned responses

Unconditioned heart rate and movement were measured in a 6-sec. interval on each trial beginning 3 sec. after the termination of shock. In the case of EMG activity the 6-sec. period began 5 sec. after shock. These numbers, corrected for base level by subtracting the 6-sec. pre-CS measure from each of them, were then converted to a rate-per-minute index. The adjusted heart-rate responses of the four groups averaged over three successive blocks of 12 trials are plotted in the left-hand side of Figure 10.

Heart rate. It is obvious that the unconditioned heart-rate reactions of all groups were accelerative with the magnitudes of the

Figure 9. Mean pre-CS EMG activity for both CS+ and CS- trials averaged over 12-trial blocks during acquisition for the unrestrained-escape, unrestrained-classical, restrained-escape, and restrained-classical groups.


Unrestrained
Restrained
Mean EMG – Spikes / minute
400
300
200
100
0
Escape ——
Classical---
CS +▲
CS -△
1-12
13-24
25-36
Trials

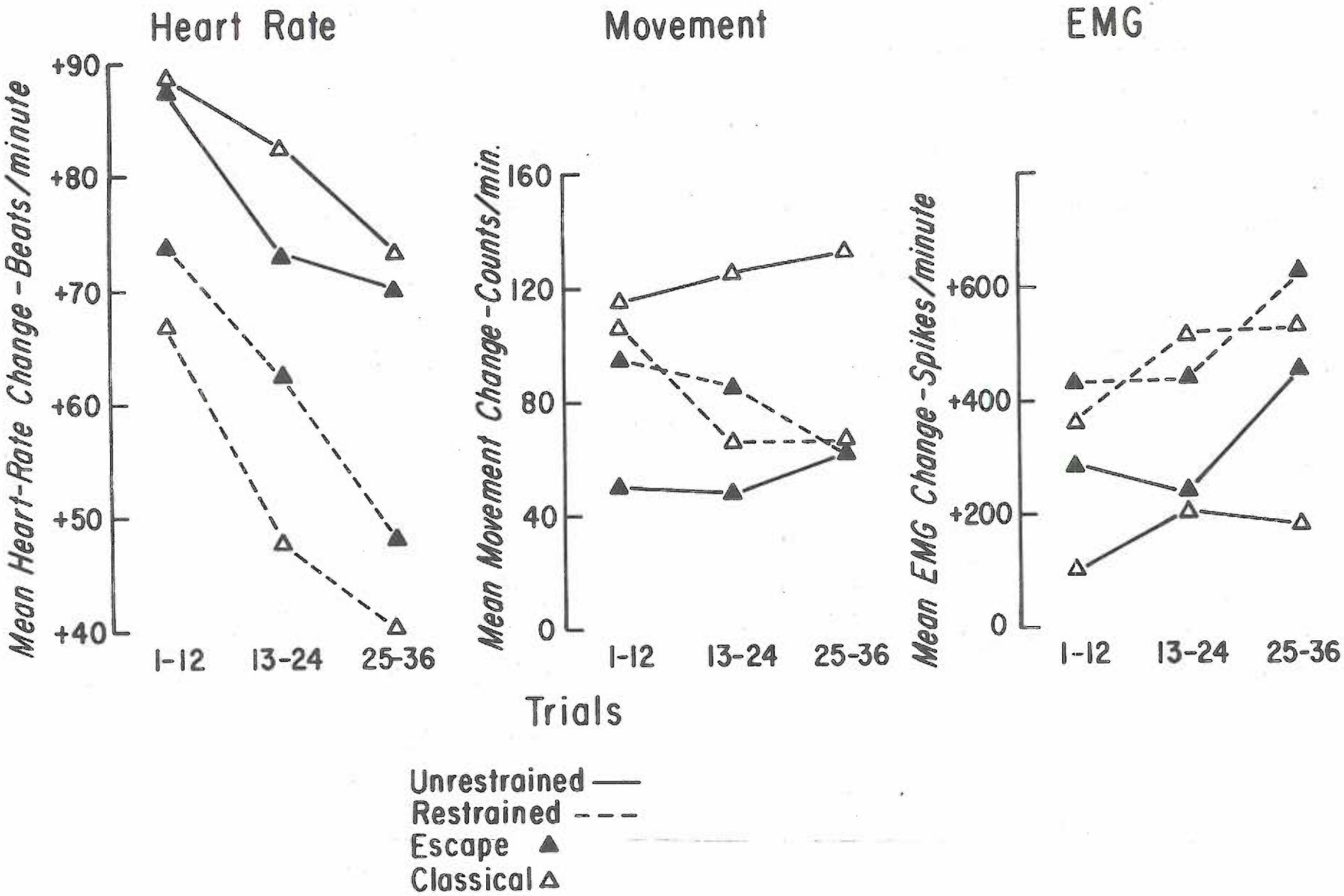
reactions decreasing systematically over acquisition ($F=22.30$, $df=1/52$, $p < .001$). As might be expected, the shocks elicited bigger accelerations in the unrestrained groups than in the restrained groups ($F=14.16$, $df=1/52$, $p < .01$). The unrestrained-escape group that was required to rotate the wheel to terminate shock appeared to show a slightly smaller heart-rate reaction than did the unrestrained-classical group that could not manipulate the wheel. Just the opposite relationship seemed to be present in the restrained condition. However, neither of these differences were significant.

Movement activity. Inspection of the middle section of Figure 10 reveals that unconditioned movement activity of the unrestrained groups increased slightly over acquisition while the amount of this activity in the restrained groups decreased slightly. Within the unrestrained treatment, the classical group appeared to demonstrate more movement than did the escape group. However, the only significant outcome of an analysis of variance was that of the restrained vs. unrestrained x trial blocks interaction ($F=4.54$, $df=2/104$, $p < .05$) reflecting the fact that the change in the amount of movement of the two groups across acquisition was reliably different.

Consistent with what was found for conditioned heart rate, movement and unconditioned heart rate appeared to show some independence. Thus, in the restrained groups, sizeable decreases in the magnitudes of the heart-rate unconditioned responses were associated with rather small decreases in movement. In the unrestrained groups, heart rate and movement changed in opposite directions.

EMG activity. Examination of the right-hand portion of Figure 10 suggests that the restrained group showed slightly more unconditioned

Figure 10. Mean unconditioned heart rate, movement and EMG responses of the unrestrained-escape, unrestrained-classical, restrained-escape, and restrained-classical groups for each 12-trial block during acquisition. Unconditioned heart rate and movement responses were measured in a 6-sec. interval beginning 3 sec. after termination of the shock. In the case of EMG activity the 6-sec. period began 5 sec. after the shock. All three response measures were corrected for base level by substracting from each the level of responding that was present during a 6-sec. pre-US period.

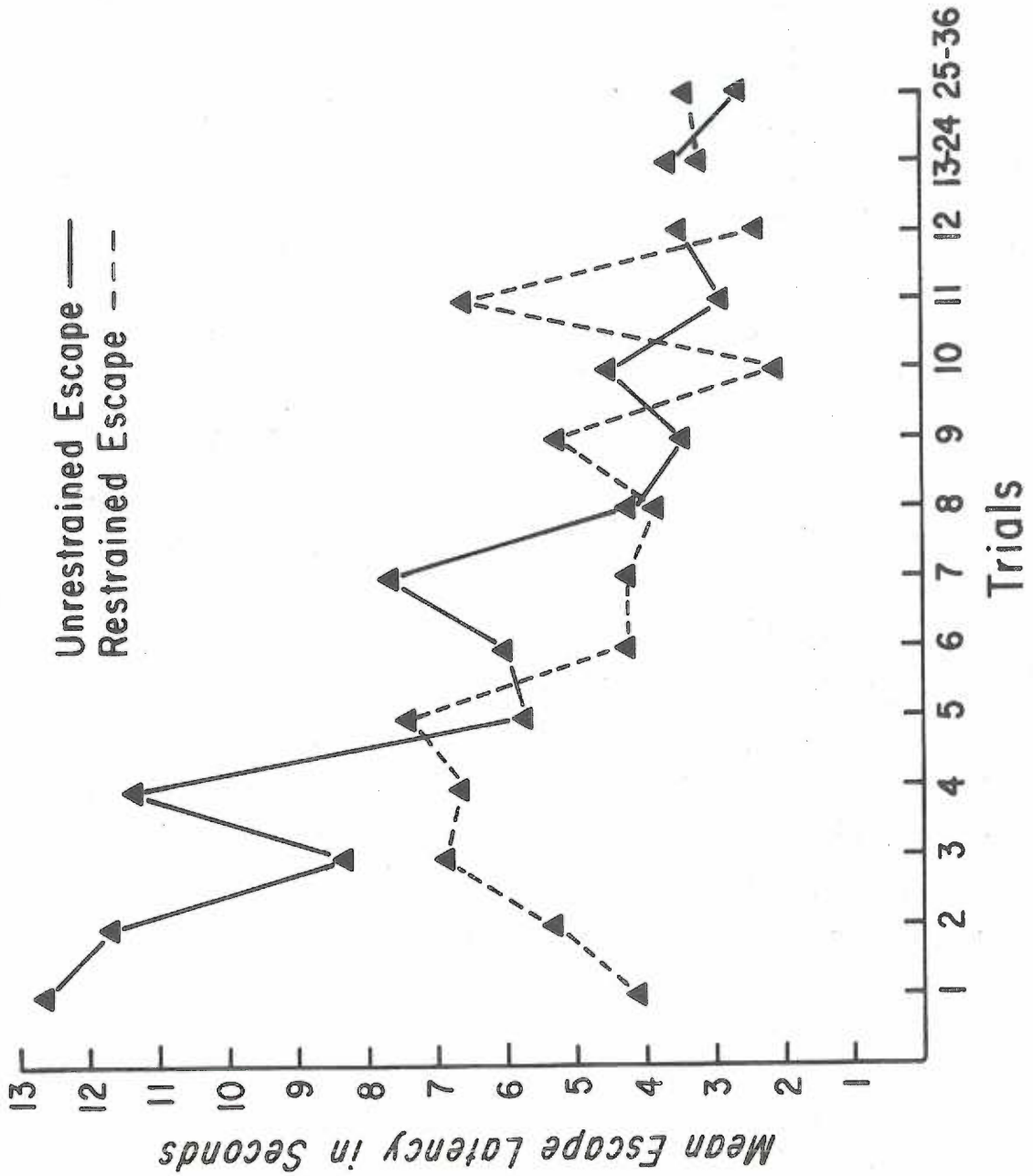

Heart Rate
Movement
EMG
Mean Heart-Rate Change - Beats/minute
+90
+80
+70
+60
+50
+40
Mean Movement Change - Counts/min.
160
120
80
40
0
Mean EMG Change - Spikes/minute
+600
+400
+200
0
1-12
13-24
25-36
Trials
Unrestrained
Restrained
Escape
Classical

EMG activity than did the unrestrained group and that the amount of activity may have increased across acquisition for all groups. However, none of the factors of an analysis of variance carried out on these data were significant.

Wheel-turning escape latencies

It will be recalled that the duration of shock on each conditioning trial received by the subjects in the escape groups was determined by how long it took the subjects to rotate the wheel once the shock was presented. The latencies of these wheel-turn responses also controlled the duration of shock presented to the yoked classical subjects. These response latencies of the two escape groups on each of the first 12 trials of acquisition and on the remaining two blocks of 12 trials are presented in Figure 11. Examination of this figure reveals that during the earliest part of conditioning the latencies of the wheel-turning responses of the unrestrained groups were appreciable longer than those of the restrained groups. By the end of the first 12 trials both groups turned the wheel within 2.5 to 3.5 sec. after the onset of shock. No further improvement was evident on the remaining 36 conditioning trials. A 2 x 2 analysis of variance carried out on the data shown in Figure 11 resulted in a reliable effect of trials ($F=2.92$, $df=13/388$, $p < .01$). The unrestrained vs. restrained x trials interaction was just short of the 0.5 level of significance ($F=1.70$, $df=13/338$, $p < .10$). These results indicate that during most of conditioning the durations of shocks experienced by the unrestrained and restrained groups and by the yoked classical groups were comparable.

Figure 11. Mean wheel-turning escape latencies of the unrestrained-escape and restrained-escape groups on each of the first 12 trials of acquisition and on the remaining 24 trials in two blocks of 12 trials each.


Unrestrained Escape
Restrained Escape
Mean Escape Latency in Seconds
13
12
11
10
9
8
7
6
5
4
3
2
1
1 2 3 4 5 6 7 8 9 10 11 12 13-24 25-36
Trials

INTRODUCTION II

A principal finding of this experiment was that both the unrestrained-escape and unrestrained-classical groups developed conditioned accelerative heart-rate responses while the corresponding restrained groups displayed conditioned decelerative responses. In neither unrestrained or restrained conditions did the opportunity to make a wheel-turning escape response influence the direction or magnitude of the conditioned heart-rate changes. Furthermore, there was no evidence that the subjects receiving escape training responded somatically to the CS signalling shock as might have been expected had this type of training produced a general state of excitation. Thus, the amount and pattern of motor activity shown by the escape groups to CS+ was not different from that revealed by the classical groups.

The fact that all of the subjects in the restrained-escape group showed heart-rate decelerations to CS+ during the last 12 trials of conditioning appears to demonstrate that immobilization was the dominant factor controlling the direction of the heart-rate change and that the escape contingency played no role. On the other hand, it is conceivable that the wheel-turning response in this group was poorly learned or that it required too little effort to encourage the development of an anticipatory state of preparedness that would in turn produce cardioaccelerations. From an examination of the response latencies shown in Figure 10 it can be seen that there was very little improvement over the course of conditioning in the speed with which the restrained-escape subjects rotated the wheel to terminate the shock. Due to the proximity of the subjects to the wheel it seems likely that the wheel may have been rotated fortuitously on some trials by shock-elicited struggling. These

occasions would, of course, be counter productive to the learning of an integrated wheel-turning reaction.

An attempt was made in a second experiment to insure that a vigorous well organized escape response was learned by restrained rats during the conditioning session. To accomplish this objective the rats were required on each trial to rotate the wheel continuously throughout a 1-min. period in order to either terminate the shock or postpone its recurrence.

METHODS II

Subjects

Twenty female Long-Evans hooded rats served as subjects. The rats were purchased from Charles River Laboratories and housed with ad lib access to food and water in facilities provided by the Department of Animal Care at the University of Oregon Health Sciences Center.

Apparatus

With the exception of the restraining device, the basic apparatus including the wheel-turning chambers and ECG recording system were the same as those used in Experiment I. The restrainer was constructed of Plexiglas and meausured 20.5-cm long x 5.0-cm wide x 6.5-cm high at the front tapering to 5.0 cm at the back. The rear of the restrainer was drilled with a matrix of holes with 1.3-cm spacing through which rods were placed to adjust the size of the holder to the subject. This restrainer provided the subjects with slightly better access to the wheel than did the wire mesh cage used in Experiment I.

The CS was a 5-kHz tone presented through the speaker mounted in the ceiling of the chamber. The US was delivered to the rat's tail using the same electrodes and shock parameters as those employed in Experiment I. The total duration of shock received to the nearest 0.1 sec. and the number of wheel-turning responses made were automatically printed out on a Massey Dickinson cumulative printer.

Procedure

Electrodes for recording ECG were implanted using the same surgical procedure as that employed in Experiment I. The experiment was

comprised of one experimental group (n=14) and one pseudoconditioning control group (n=6). Subjects in the experimental group were given 40 training trials. Each trial began with the presentation of the CS followed 6 sec. later by the simultaneous raising of the shield covering the wheel and the delivery of the US. The US, which was programmed to last 60 sec., was terminated for 1.6 sec. by a 180^{o} rotation of the wheel. Each subsequent rotation during the US-off period served to prolong the recurrence of the shock for an additional 1.6 sec. Thus, after the initial presentation of shock it was then possible for the subjects to avoid further shock by rotating the wheel 180^{o} at least once every 1.6 sec. The CS remained on for the 60-sec. period that this contingency was in effect. The control subjects were given the same escape-avoidance training except that the CS was presented either 80, 90, or 110 sec. ($\bar{X}$=90 sec.) after the US. The duration of the CS for control subjects was 7 sec. The intertrial interval for both groups was either 200, 220, or 240 sec. ($\bar{X}$=220 sec.).

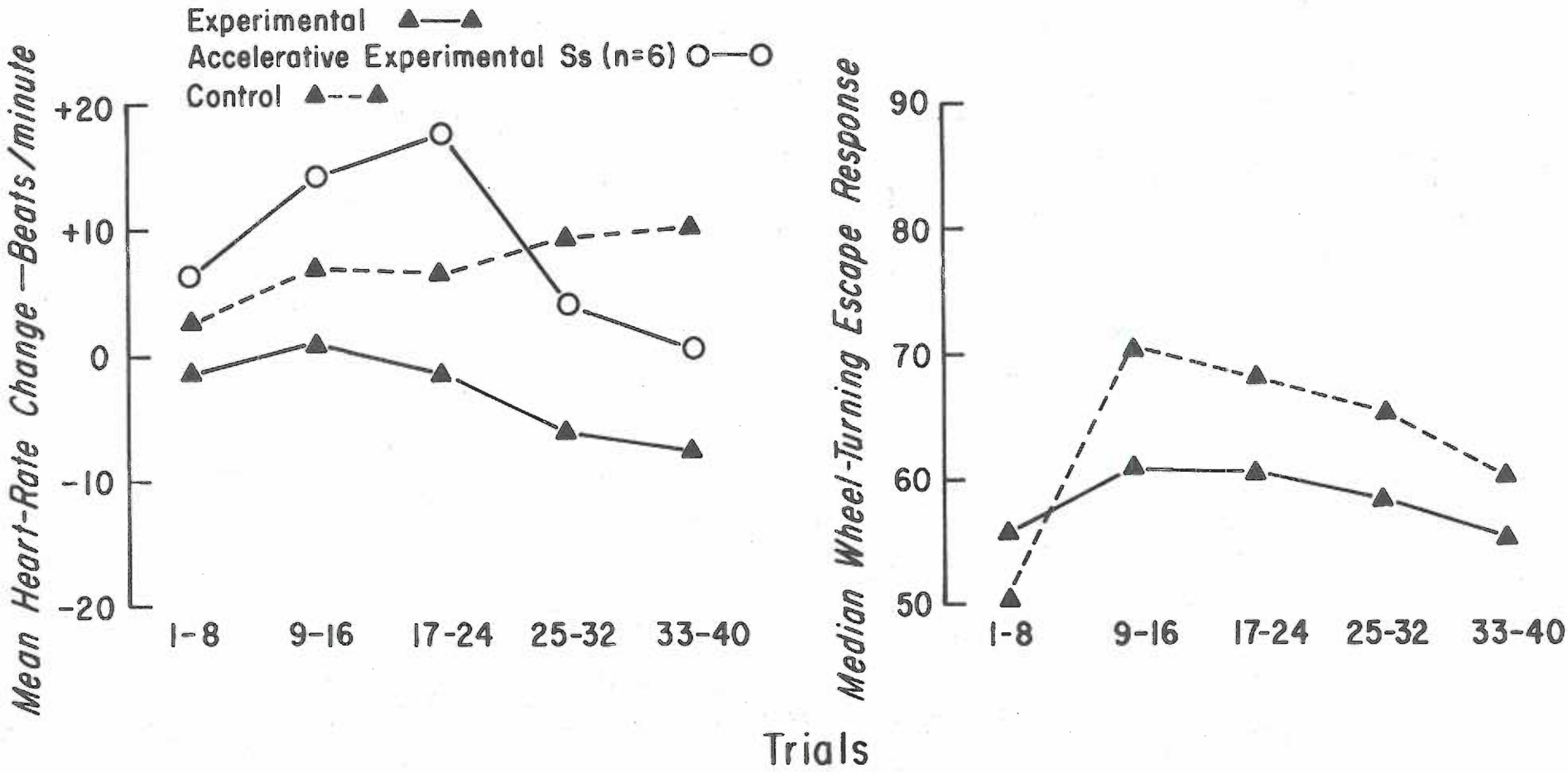
Heart beats were counted in a 6-sec. interval immediately preceding the CS, in three 2-sec. intervals during the CS and during a 6-sec. interval following the offset of the US. The number of heart beats in each 2-sec. period was converted to beats per minute and corrected for base level by subtracting the pre-CS beats-per-minute heart rate.

RESULTS II

Mean CS minus pre-CS heart-rate difference scores of the experimental and control groups are plotted in the left half of Figure 12 as a function of eight-trial blocks during acquisition. Examination of this part of the figure provides no evidence of a conditioned accelerative heart-rate response in the experimental group. The change in heart rate shown by this group was a slight deceleration which increased slightly in magnitude over the conditioning trials. By contrast, heart rate accelerated in the unpaired control group with the magnitude of the reaction gradually increasing over trials.

A groups x trials analysis of variance revealed that the overall responses of the experimental and control groups were not reliably different. However, a reliable conditioning effect was obtained in the form of a significant groups x trials interaction ($F=32.69$, $df=4/72$, $p < .001$). This interaction reflects the divergence of the heart-rate responses of the experimental and control groups over conditioning. The trials effect was also significant ($F=5.22$, $df=4/72$, $p < .01$). Inspection of the results of individual subjects within the experimental group indicated that six of the fourteen subjects showed an overall acceleration to the CS during conditioning. The open circles plotted in Figure 12 reveal that these subjects showed a rapid increase in the magnitude of heart-rate acceleration over the first 24 trials of conditioning but that heart rate then returned toward base line. Although not reliably different from the control group the responses of these six subjects represent one of the very few examples of relatively sustained cardioacceleration in restrained rats to a CS signalling shock.

Figure 12. Mean CS minus pre-CS heart-rate responses of the experimental and control groups in eight trial blocks during acquisition are shown in the left of the figure. The triangles represent group means whereas the open circles depict the mean heart-rate responses of the six experimental subjects showing accelerative heart-rate reactions. The median number of wheel-turning escape responses of the experimental and control group averaged over blocks of eight trials are plotted in the right side of the figure.


Experimental
Accelerative Experimental Ss (n=6)
Control
Mean Heart-Rate Change —Beats/minute
+20
+10
0
-10
-20
1-8
9-16
17-24
25-32
33-40
Median Wheel-Turning Escape Response
90
80
70
60
50
1-8
9-16
17-24
25-32
33-40
Trials

The median number of wheel-turning responses of the experimental and control groups during the 1-min. unconditioned stimulus are presented in the right-hand portion of Figure 12. It can be seen that the escape procedure led to vigorous responding with the number of responses of both groups increasing over the first eight trials, reaching a peak during the second block of eight trials, and then showing a very slight reduction over the remainder of the trials. In general, the number of wheel-turning responses shown by the control group appears higher than the experimental group throughout acquisition. However, a Mann-Whitney U test performed on the differences between the number of responses on the first and last blocks of trials for each group demonstrated that there was no significant difference between the groups.

DISCUSSION

Degree of Restraint and Escape Contingency

The principal outcomes of the present investigations with respect to the effects of the conditioning procedures employed on conditioned heart rate in the restrained and unrestrained conditions were that: (1) both unrestrained-escape and unrestrained-classical groups developed conditioned accelerative heart-rate reactions while the corresponding restrained groups displayed conditioned decelerative heart-rate responses; (2) regardless of the degree of restraint, the opportunity to control the duration of the shock US failed to influence the direction or magnitude of the conditioned heart-rate reaction; (3) the escape contingency did not alter the amount or pattern of general movement or of EMG activity in the escape as compared to the classical groups in either the unrestrained or restrained conditions; (4) sorting the conditioning trials of the combined unrestrained and restained groups on the basis of whether heart rate increased, showed no change, or decreased to CS+ revealed that the heart-rate reactions of the unrestrained groups were less consistent than those of the restrained groups. Careful examination of the results of the unrestrained group revealed that two types of subjects could be identified, those showing cardioacceleration and those showing cardiodeceleration; (5) pre-CS heart rate of the unrestrained groups declined over acquisition while for the restrained groups it remained relatively unchanged; (6) in Experiment II in which restrained rats learned a vigorous wheel-turning escape response, six experimental subjects showed initial accelerative reactions with the group as a whole showing a small decelerative response.

One of the main objectives of the present experiment was to examine the influence of being able to control the duration of an aversive US on

the direction of the classically conditioned heart-rate responses in both unrestrained and restrained rats. Previous studies have shown that unrestrained rats demonstrate cardioacceleration to a CS that signals a foot-shock US. It was hypothesized that such a shock could be partially escaped by jumping off the shock-grid and that this instrumental escape contingency may have led to an excitatory state that resulted in cardioacceleration to the CS.

In the present study the shock US was delivered to the tails of the unrestrained rats providing little if any opportunity to escape the shock. Nevertheless, the unrestrained-classical group developed conditioned heart-rate accelerations with the magnitudesof their responses being similar to those of the unrestrained-escape group. Finding conditioned cardioaccelerations in the unrestrained groups is consistent with what has been shown in previous studies (Black & Black, 1967; Borgealt et al., 1972; Duncan, 1972; Fehr & Stern, 1965; McDonald et al., 1963). However, the fact that acceleration also occurred in the unrestrained-classical group provides little support for the notion that instrumental-escape contingencies were the primary determinant of such reactions.

The cardioaccelerative reactions of the unrestrained groups are in contrast to the results reported by Teyler (1971) who found that freely-moving rats receiving an inescapable chest-shock developed reliable conditioned decelerations in heart rate. A possible explanation for this apparent conflict may have been the location of the electrodes delivering the shock in the two studies. In Teyler's experiment the electrodes were sutured through a fold of skin on either side of the rib cage. Wires connecting the rat to the shock source terminated in a commutator mounted in the ceiling of the test chamber. Such an arrangement may have encouraged biting at the electrodes and wires during the intertrial interval.

Cessation of this activity when the CS was presented could account for the heart-rate decelerations and the decreases in general activity that were observed.

An additional factor that could have conceivably contributed to the conflicting outcomes of the two studies was the difference in duration of the shock USs. Teyler employed a brief 1.0-sec. shock while in the current study the duration of the US was controlled by the wheel-turning latencies of the unrestrained-escape subjects. Consequently, the mean shock duration ranged from approximately 12.5 sec. on the first two trials to a mean of 3.5 sec. during the final two blocks of acquisition. It may be that these long shocks produced an intense state of excitement that enhanced the development of conditioned cardioaccelerations.

The decelerative heart-rate reactions that were found in the present experiment are consistent with the results of numerous other studies involving restrained rats (Fitzgerald & Martin, 1971; Fitzgerald & Teyler, 1970; Fitzgerald et al., 1972; Fitzgerald et al., 1966; Holdstock & Schwartzbaum, 1965; Vardaris, 1968). The wide variety of conditioning parameters employed in these studies attest to the powerful influence of restraint in the production of conditioned cardiodecelerations in rats. The failure of the wheel-turning escape response to alter the direction of the conditioned heart-rate responses in restrained rats again suggests that contrary to expectation instrumental contingencies may play a relatively insignificant role in determining the direction of classically conditioned heart rate.

On the assumption that the escape response in Experiment I was poorly learned or that it required too little skeletal-motor activity to produce an anticipatory state of excitement, a second study was carried out

in which the rats were forced to rotate the wheel throughout a 1-min. period to keep the shock off. This procedure also failed to produce an overall conditioned increase in heart rate. In fact, by the end of acquisition the heart-rate response of this group was slightly decelerative. However, that requiring a vigorous well-learned escape response can increase the incidence of cardioacceleration was revealed by the fact that six experimental subjects showed sizeable accelerative reactions during the first two-thirds of acquisition. These results are in contrast to what was found in the first experiment. In that study, none of the restrained-escape subjects showed heart-rate acceleration during conditioning. However, it is important to note that the heart-rate responses of the six experimental subjects in Experiment II were probably not conditioned as they were highly similar to those shown by the unpaired control group. One methodological implication of these findings is that failure to employ adequate control conditions to evaluate nonassociative factors could easily lead to an erroneous conclusion regarding the occurrence of classically conditioned heart rate.

In evaluating the results of Experiment II it may be pertinent to point out that the heart-rate reactions of the unpaired group were considerably larger than those shown by unpaired control groups in previous studies (Fitzgerald & Martin, 1971; Fitzgerald & Teyler, 1970; Fitzgerald et al., 1973; Teyler, 1971). One possible explanation of this difference has to do with the duration of the shock US and the time that was allowed to elapse between the presentation of the US and the CS. In prior studies a relatively brief 1-sec. shock was employed in conjunction with a US-CS interval that averaged approximately 1.5 min. This interval was selected to minimize the direct effects of the US on

the responses made to the CS. Although a comparable interval was used in the current study the average duration of the shocks was approximately 15 sec. It is conceivable that these shocks together with the vigorous wheel turning that occurred during the 1-min. shock period produced a general state of emotional arousal that persisted throughout the US-CS interval and increased the likelihood of heart-rate acceleration to the CS. Perhaps, the magnitude of this unlearned response would have been reduced had a longer US-CS interval been used.

In the present study, pre-CS+ heart rate of the unrestrained groups decreased from 445 beats per minute at the beginning of acquisition to 415 beats per minute by the end of acquisition. The rate shown by the restrained groups remained relatively constant at 440 beats per minute. However, there was no indication that pre-CS+ heart rate influenced the direction of the conditioned heart-rate reactions. For example, the unrestrained-classical group showed conditioned cardioacceleration throughout acquisition while pre-CS+ heart rate steadily declined. In fact, the conditioned heart-rate reactions were smallest at the end of acquisition when lower basal heart rate would presumably result in augmented cardioaccelerations. Although similar pre-CS+ heart-rate changes took place in the unrestrained-escape group, conditioned heart-rate accelerations occurred only during the last 12 acquisition trials. In addition, when the subjects in the unrestrained groups were separated on the basis of whether they displayed accelerative or decelerative responses there was no difference between the basal heart rate of the two types of subjects. In both restrained groups, equivalent conditioned heart-rate decelerations developed during the final block

of acquisition while pre-CS+ heart rate showed a slight decrease for the escape group and no change for the classical group. These findings demonstrate the relative independence of CS initiated heart-rate changes and base-level heart rate and are consistent with previous studies (Fitzgerald & Martin, 1971; Fitzgerald & Teyler, 1970; Teyler, 1971) in which large shifts in pre-CS heart rate occurred over the course of acquisition without any apparent influence on the magnitude or the direction of conditioned heart rate.

The only indication of a positive relation between base-level heart rate and conditioned heart rate appeared when all of the CS+ trials were sorted on the basis of whether heart rate increased, showed no change, or decreased. In this case, pre-CS+ heart rate was reliably higher when heart rate decelerated to CS+ than when heart rate accelerated. However, in neither instance were the magnitudes of the heart-rate changes correlated with base-level heart rate.

In the current investigation, the unconditioned heart-rate responses were uniformly accelerative with the magnitudes of the reactions being approximately 20 beats per minute larger in the unrestrained as compared to the restrained condition. This difference in response magnitude was apparently not related to shock duration or amount of skeletal activity since there were no reliable differences between these measures in the two conditions. A possible explanation might involve the fact that the tails of the unrestrained subjects were held in place through a slot in the floor of the conditioning chamber. It may be that this arrangement increased the painfulness of shock-elicited struggling leading to larger unconditioned heart-rate changes.

Finding unconditioned cardioaccelerations to shock is in general agreement with the results of previous studies involving both unrestrained and restrained rats (Borgealt et al., 1972; Duncan, 1972; Fitzgerald & Teyler, 1970; Fitzgerald et al., 1966; Holdstock & Schwartzbaum, 1965; McDonald et al., 1963; Teyler, 1971). The fact that the conditioned and unconditioned responses of the unrestrained groups were both accelerative is consistent with traditional theories of classical conditioning that maintain that the two responses should be similar. On the other hand, the conditioned cardiodecelerations of the restrained groups are not consistent with these formulations. However, temporary unconditioned heart-rate decelerations have been observed in rats (Fitzgerald & Teyler, 1970; Stainbrook, 1975; Teyler, 1971) leaving open the question of the true nature of this reaction.

A potentially important finding was the difference in the consistency of the heart-rate reactions of the unrestrained and restrained groups. The unrestrained groups showed heart-rate increases on 49% of the trials and decreases on 39% of the trials while the restrained group demonstrated heart-rate decreases on 70% of the trials. Also, approximately one-half of the subjects in the unrestrained groups showed reliable cardioaccelerations to CS+ while the remaining subjects showed nonsignificant cardiodecelerations. In the case of the restrained groups, only two subjects failed to show cardiodeceleration to CS+ during the latter part of conditioning. In general, these results are consistent with those reported by Teyler (1971) involving freely-moving rats receiving a foot-shock US. In that study approximately one-half of the subjects showed heart-rate accelerations that were reliably larger

than those shown by control subjects. The remaining half of the subjects showed heart-rate decelerations that were not reliably different from those of controls. One possible explanation for the apparent lack of consistency in the direction of the heart-rate responses of unrestrained rats is that the absence of restraint provides an opportunity for the animals to engage in behaviors that may compete with the formation of learned associations between the CS and US. It is also conceivable that cardioacceleration is the primary reaction that becomes conditioned in unrestrained rats and that decelerations in heart rate may represent instances in which shifts in base-line activity occur.

Two final observations can be made regarding the influence of escape contingency and degree of restraint. First, the results revealed that the movement and EMG reactions to CS+ in the escape groups were comparable to those shown by the classical groups. In both cases, more skeletal-motor responding occurred at the beginning than at the end of the CS+ period. This finding was rather surprising since it might be expected that rats would show an increase in gross-motor activity just prior to making an escape response. That this did not occur may have to do with the fact that the shield covering the response wheel prevented any responding during the CS-US interval with the rats receiving approximately 0.5 sec. of shock before an escape response could be made. Under these conditions, it is conceivable that the cue for wheel-turning was the occurrence of shock in combination with the raising of the shield rather than the programmed CS. This hypothesis gains support from the results of studies suggesting that in discriminated-avoidance conditioning situations shock may be a more potent signal for responding than the CS (D'Amato & Schiff, 1964; D'Amato, Keller, & DiCara, 1964).

Second, it was found that the unrestrained groups showed significantly more movement activity to both CSs than did the restrained groups. However, this difference was so small that it cannot account for the opposite direction of the heart-rate responses of the unrestrained and restrained groups.

In summary, the results of the present study provide little evidence that instrumental-escape responses influence the direction of the conditioned heart-rate reactions of rats. Similarly, base-level heart rate appears to have virtually no effect on the direction of heart-rate changes to the CS. Finally, the results suggest that the conditioned heart-rate reaction of unrestrained rats is primarily cardioacceleration while that of restrained rats is almost without exception cardiodeceleration with the major factor(s) determining these reactions being the degree of restraint imposed upon the subject.

Relation Between Heart Rate and Motor Activity

The principal findings of the present investigation with respect to the relation between heart rate and skeletal-motor activity were that: (1) conditioned changes in movement and in EMG activity rarely occurred in any of the conditions. In those instances in which they were found, they were, with one exception, not associated with conditioned heart-rate reactions. The amounts and patterns of movement and of EMG activity in the escape groups were not different from those observed in the classical groups. In both cases, there was a burst of responding at the onset of CS+ and CS- followed by a rapid decline in activity toward pre-stimulus levels; (2) an analysis in which all of the CS+ trials were sorted into three categories on the basis of whether heart

rate increased, showed no change or decreased revealed a correspondence between the direction of the mean heart-rate responses and the direction of the mean movement reactions. However, an examination of individual trials within each of the three heart-rate categories indicated that heart rate and movement were frequently independent and in fact changed in opposite directions on some trials.

An attempt was made in the current study to determine whether learned changes in skeletal-motor activity occurred in rats during classical conditioning and if so whether such changes were associated with classically conditioned heart rate. Although the relationship between motor activity and heart rate has been examined in many previous studies, appropriate statistical analyses were frequently not carried out to establish that the responses were in fact learned. In a majority of studies, such an analysis was precluded by the fact that the control conditions required to rule out nonassociative changes in responding were not incorporated into the design of the experiments. While the results of these investigations may bear on the general question of the extent to which heart rate and movement reactions occur together, the processes, i.e., learning, sensitization, pseudoconditioning, etc., underlying the reactions cannot be identified.

A major finding of the present study was that although conditioned movement and EMG reactions sometimes occurred they were not systematically associated with learned changes in heart rate. For example, the unrestrained-escape group showed a conditioned increase in movement to CS+ during the first 12 acquisition trials whereas conditioned cardioaccelerations did not occur until the last 12 acquisition trials. Learned EMG reactions occurred during the last two blocks of acquisition

for the unrestrained-classical group while conditioned heart-rate accelerations took place throughout acquisiton. In the restrained-classical group, learned EMG responses were present only during the middle of acquisition in the absence of conditioned heart rate. A positive relationship between conditioned heart rate and movement was shown by the subgroup of unrestrained subjects (N=16) demonstrating cardioacceleration to the CS on the last 12 acquisition trials. The fact that more evidence of conditioned somatic activity was not obtained agrees generally with the results of prior studies in which both restrained (Fitzgerald & Teyler, 1970; Holdstock & Schwartzbaum, 1965) and unrestrained (Teyler, 1971) rats receiving traditional classical conditioning training failed to demonstrate learned skeletal-motor responses.

There was a clear tendency in all groups for gross movement and EMG activity to increase sharply at the onset of both CSs. In the case of movement, this startle-like reaction showed evidence of conditioning in the unrestrained-escape and restrained-classical groups, whereas conditioning of the EMG component was demonstrated in the two classical groups. It may be important to note that Roberts and Young (1971) mentioned that small movement reactions to the onset of the CS+ regularly occurred in rats receiving CER training. However, for some unspecified reason these reactions were ignored in their analysis of the movement data.

In the present case, the conditioning effect of the onset responses was due primarily to the inhibition of movement to CS- with the magnitude of the response to CS+ being highly similar to that which occurred during the pre-test trials with the CS alone. In spite of the presence of this differential skeletal-motor responding there was no evidence of comparable differentiation in the heart-rate reactions to the onsets of the CSs.

Without exception, the magnitudes of the differences in the heart-rate responses were smaller during the first interval of the CSs than in any of the other intervals.

At least in terms of conditioned changes, the results outlined above offer almost no support for those hypotheses proposing that the somatic and cardiac systems are coupled together centrally. It would appear likely that the presence of conditioned activity in a center purportedly controlling both heart rate and movement would lead to synchronous changes in both responses.

Some of the most obvious demonstrations of the independence of heart rate and skeletal-motor activity were obtained when the topographies of the reactions were considered. All groups, regardless of the degree of restraint, showed a burst of movement and EMG activity to the onsets of both CSs. These responses were then followed by decreases in activity toward pre-stimulus levels. In spite of these highly similar somatic reactions, the unrestrained groups showed cardioacceleration whereas the restrained groups displayed cardiodeceleration. Furthermore, the heart rate and motor reactions were not temporally coincident for any of the groups in that somatic activity always peaked during the first counting interval while the largest heart-rate reaction consistently occurred just prior to US onset. These results are in apparent contrast to those obtained in the CER procedure with rats (Roberts & Young, 1971), in varied instrumental procedures with dogs (Sutterer & Obrist, 1972), and in classical conditioning with unrestrained cats (Howard et al., 1974). In these latter instances, both increases and decreases in heart rate during the duration of the CS seemed to be paralleled by comparable changes in movement.

To provide an overall analysis of the relation between heart rate and movement, CS+ trials in both restrained and unrestrained conditions were sorted into three categories (see Figure 5). These categories included trials in which heart rate either increased, showed no change, or decreased. The results of this procedure provided an apparent indication of overall dependence between heart rate and movement activity. It was found that increases in heart rate in the unrestrained group were accompanied by reliable increases in movement while in the restrained group decreases in heart rate were associated with significant decreases in movement. Those relatively few instances of cardioacceleration in the restrained condition were also paired with reliable increases in motor activity. These findings correspond closely to those reported by Roberts and Young (1971) for rats receiving CER training. In that study heart-rate changes during the CS were tabulated when increases, decreases, or no change in movement occurred. They found that decreased movement was associated with reliable cardiodecelerations while increased movement was accompanied by significant cardioaccelerations.

Although not emphasized by Roberts and Young, it is perhaps worthwhile to point out that in their study pre-CS heart rate paralleled pre-CS movement and lever pressing activity. In the presence of high rates of pre-CS lever pressing, heart rate and gross movement were elevated, whereas during low rates of pre-CS lever pressing, heart rate and gross movement were reduced. Comparable relationships were obtained in the present study in that base-level heart rate paralleled pre-CS movement activity. Other studies (Borgealt et al., 1972; Brady et al., 1969; Duncan, 1972; Sampson et al., 1974) have also reported elevated heart rate during lever pressing. Together, these findings suggest that at

times heart rate may be a rather sensitive indicator of ongoing somatic activity.

An additional point with respect to the Roberts and Young study is that decelerations in heart rate occurred to the CS on trials when pre-CS heart rate and movement were elevated while accelerations were elicited by the CS on those trials in which base-level heart rate and movement were depressed. In general, the present experiment furnished similar results. Thus, pre-CS movement and heart rate were elevated on trials in which movement and heart rate decreased to the CS (see Figure 5 and Table 1). To a lesser extent this also tended to be true when movement and heart rate increased to the CS. The presence of high pre-CS movement and heart rate on trials in which decreases in both responses occurred, suggests that these trials represent instances in which the presentation of the CS produced a cessation of ongoing struggling, leading to an augmentation of heart-rate deceleration. Similarly, elevated pre-CS movement and heart rate on trials in which increases in both responses were observed could indicate that on these trials ongoing motor activity became more vigorous in the presence of the CS producing an amplification of heart-rate acceleration. In those cases in which the change in movement was opposite to that of heart rate, the magnitudes of the heart-rate reactions whether accelerative or decelerative, were reduced. Comparable outcomes have also been obtained in other classical conditioning studies (Cohen, 1969; Duncan, 1972; Obrist & Webb, 1967; Teyler, 1971). In these studies, as in the present experiment, changes in heart rate occurred independently of movement, but when movement changed in the same direction as heart rate the cardiac reactions were augmented.

Evidence of independence between heart rate and gross-motor activity was obtained in the present experiment by resorting the CS+ trials in the three heart-rate categories (increase, no change, or decrease) on the basis of whether movement increased, showed no change, or decreased on these trials (see Table 1). First, regardless of whether the rats were unrestrained or restrained, large increases and decreases in heart rate occurred in the absence of measurable movement. It may be argued that on these trials small changes in movement occurred but that because of the insensitivity of the movement detector they were not recorded. However, the magnitudes of such changes in comparison to those observed for heart rate would offer little support for cardiac-somatic formulations (Obrist et al., 1972) which have suggested that adjustments in heart rate are made to meet the metabolic demands of somatic activity. Second, both increases and decreases in heart rate occurred even though movement changed in the opposite direction. These trials provide some of the most compelling evidence of independence of heart rate and movement activity since it cannot be argued that activity changes were present but not detected. Third, relatively large increases as well as decreases in movement occurred without any corresponding changes in heart rate.

In summary, the above results provide examples of the independence of heart rate and movement during classical conditioning and suggest that the responses elicited by the CS were mediated by processes other than those controlling skeletal-motor activity. Furthermore, it should be emphasized that these outcomes are in contrast to what was obtained when mean responses were calculated over a number of trials. Clearly, heart-rate reactions can occur quite unrelated to changes in movement but this independence may be masked by the averaging procedure.

General Theoretical Considerations

In a recent review of the central structures involved in cardiovascular control, Cohen and MacDonald (1974) provide a description of what they termed a "central-exercise pathway". This pathway apparently arises in the motor cortex, traverses the hypothalamus, and subsequently courses through the brain stem ventral and ventrolaterally to enter the spinal cord (Cohen & MacDonald, 1974; Eliasson, Lindgren, & Uvnas, 1952; Hilton, 1966; Rushmer et al., 1960; Schramm & Bignall, 1971). Electrical stimulation of these central structures has been shown (Rosen, 1961a, 1961b) to produce sympathetic vasodilation of skeletal muscle, vasoconstriction of high resistance vessels such as skin, intestines, and kidneys, and cardioacceleration combined with increased strength of contraction. However, systemic arterial blood pressure remained relatively unchanged. Presumably, cardioacceleration associated with the initiation of motor activity (Rushmer et al., 1960) or voluntary muscle contraction (Freyschuss, 1970; Petro et al., 1970) is mediated via this central system.

It may be possible that changes in activity in this "central exercise pathway" could account for the results of most CER and other complex instrumental procedures upon which cardiac-somatic formulations are primarily based. For example, skeletal-motor activity associated with ongoing lever-pressing tasks could have an excitatory effect on the pathway which in turn would elevate heart rate. Increases in skeletal responding would result in cardioacceleration while cessation of responding would lead to withdrawl of excitatory influences and to cardiodeceleration. The general finding that base-level heart rate was elevated during lever pressing (Borgealt et al., 1972; Brady et al., 1969; Duncan, 1972; Roberts

& Young, 1971; Sampson et al., 1974) and that cessation of lever pressing during the CS was associated with a decrease in heart rate are consistent with this hypothesis.

The findings in the present study along with those obtained in other investigations (Cohen, 1969; Duncan, 1972; Sampson et al., 1974; Teyler, 1971) that heart-rate changes are sometimes augmented or reduced by movement activity may also reflect cardiac-somatic coupling in which heart rate is adjusted for changes in skeletal-motor activity. Consistent with this notion is the fact that pre-CS heart rate and movement were elevated on trials in which decreases in movement during the CS seemed to augment cardiodecelerations. Similarly, when cardioaccelerations appeared to be augmented by increased movement during the CS, pre-CS heart rate and movement activity were relatively low. In other words, these trials most likely represent instances when struggling or cessation of struggling, which are exercise-type activities, exert an influence on the heart-rate reactions.

Although the exercise mechanism provides a means for cardiac-somatic coupling to occur, its operation depends largely on changes in skeletal activity. The present results demonstrated that changes in heart rate whether accelerative or decelerative could occur independently of gross-movement activity. Furthermore, heart-rate increases in response to exercise or increased motor activity are, at least in humans, mediated by a decrease in vagal activity with increasing sympathetic involvement during vigorous activity (Freyschuss, 1970; Robinson, Epstein, Beiser, & Braunwald, 1966). In restrained rats, in which background activity is held to a minimum, conditioned decelerative heart-rate responses are controlled primarily by increased vagal activity (Fitzgerald et al., 1973). Taken

as a whole, these findings suggest that classically conditioned heart rate in rats may be mediated by processes other than those related to the metabolic demands of exercise.

An alternative conceptual framework, in which the conditioned heart-rate responses of rats may be viewed, involves the notion of species-specific defense reactions. Bolles (1970) outlined three types of species-specific behaviors that can be identified in the rat. These characteristic response patterns included fleeing, freezing, or some form of attack which Bolles termed "pseudoaggressive behavior". Furthermore, he suggested that when a rat is shocked, normal exploratory and grooming behavior drop out and the response repertoire changes to one consisting primarily of species-specific defense reactions. It was also emphasized that once elicited these behaviors are not blindly reeled off in inappropriate circumstances. For example, training rats to make a jumping response to avoid shock is very difficult. However, if the rat is trained to jump out of a shock box, learning proceeds very readily (Maatsch, 1959). Similarly, if running is an effective response, in that it allows the rat to flee from a shock environment, it will be easily learned as an avoidance response. Thus, the type of defense reaction as well as the extent to which it appears may depend upon critical features of the learning procedure.

Recent evidence provided in a study by Blanchard and Blanchard (1971) supports the notion that defense reactions in rats may be, to a large extent, determined by the particular circumstances of the situation. These investigators described either flight or freezing reactions that occurred when a rat was faced with a natural predator. An approaching cat consistently elicited flight reactions and in fact, the rats would even cross an electrified grid in order to escape the cat. However, when

the shock intensity was sufficiently high to inhibit escape, the predominant reaction was changed to one of freezing.

It is possible that certain aspects of the classical conditioning situation, particularly the degree of restraint imposed upon the rats influence the pattern of natural defense reactions that occur. Thus, restrained rats may develop an inhibitory reaction related to natural freezing behavior because in this instance escape from the shock US is impossible. That freezing and cardiac deceleration frequently occur together in rodents was shown by Hofer (1970). He captured six different species of rodents and recorded heart rate during behavioral freezing. Periods of prolonged immobility ranging from 2 to more than 60 minutes were observed, accompanied by very low heart rates and a high incidence of cardiac arrhythmias. The type of arrhythmias described by Hofer have also been observed in the present laboratory in restrained rats receiving classical conditioning suggesting that similar inhibitory processes may be operating.

In unrestrained rats, the potential for dealing with the shock could lead to anticipatory aggressive behavior having as one of its components cardiac acceleration. It was recently shown that cardioacceleration was elicited by the first forelimb strike in fighting cats (Zanchetti, Baccelli, Mancia, & Ellison, 1972). Furthermore, rats readily attack or bite objects in the conditioning chamber during the presentation of shock and shock is also known to produce aggressive responses in rat pairs (Ulrich, 1966). Finally, Farris, Gideon, and Ulrich (1970) have reported that aggressive reactions in rats can be classically conditioned.

Two neural systems have been described that may make it reasonable to distinguish between aggressive and inhibitory defense reactions. The

first system, which includes the pathways mediating aggressive behavior, has been outlined in considerable detail. The course of this pathway originates from a restricted portion of the amygdala, continues to the medial hypothalamus via the amygdalofugal pathway, traverses the subcollicular tegmentum, and finally reaches the central gray area of the midbrain (Cohen & MacDonald, 1974; Gray, 1972). Upon stimulation of the amygdala in the cat, a characteristic defense reaction gradually emerged, beginning with increased alerting responses and pupillary dilitation, then vocalization, and finally, piloerection and increased agitation. However, when the hypothalamus was stimulated, an immediate and full-blown attack reaction occurred. The cardiovascular components of this reaction included increased heart rate and contractility, increased arterial blood pressure, augmented blood flow to muscle and a reduction in blood flow to the skin (Zbrozyna, 1972).

A second system has been described (Gray, 1972) that is primarily concerned with behavioral inhibition and involves a series of interconnected structures including hippocampus, medial septal area, orbitofrontal cortex, and caudate nucleus. Lesions of this system impair passive avoidance of shock and extinction of appetitive behavior (Gray, 1972). Unfortunately, there is little information on this system with respect to cardiovascular responses. Kaada (1960) described what he termed as an arrest reaction that resulted primarily from electrical stimulation of orbitofrontal cortex and cingulate gyrus in cats. At the onset of stimulation all spontaneous activity ceased, accompanied by a fall in blood pressure and a decrease in heart rate. Zanchetti et al. (1972) observed that during immobile confrontation in cats, cardiodeceleration frequently occurred and suggested that this may have been

mediated by activation of the inhibitory septo-hippocampal system.

Recent research by Blanchard and Blanchard (1972a, 1972b) has implicated both the amygdala-hypothalamus defense system and the inhibitory septo-hippocampal system in the control of species-specific defense reactions in the rat. Blanchard and Blanchard (1972a) found that rats with lesions restricted to the corticomedial nuclei of the amygdala, failed to avoid an immobile cat or an approaching shock prod. In fact, the rats approached both objects. In a second experiment (Blanchard & Blanchard, 1972b), hippocampal lesions enhanced the avoidance responding of rats to a cat stimulus and reduced freezing during the intertrial interval. In addition, rats were considerably more active in the presence of an inescapable cat which normally would have elicited freezing responses. They concluded that hippocampal lesions interfered with the specific unconditioned defensive reaction of freezing.

Classically conditioned heart-rate accelerations in pigeons are known to be mediated primarily by increased sympathetic activity (Cohen & Pitts, 1968). Furthermore, Cohen (1975) recently demonstrated that lesions of the amygdala homologue, a primary structure in the neural pathway mediating aggression, greatly reduced conditioned accelerations in pigeons. That direct electrical stimulation of structures in defense pathways can function as a US supporting classically conditioned heart rate in rabbits was recently shown by Elster et al. (1970). These results suggest that classically conditioned heart-rate reactions may be intimately related to systems controlling natural defense reactions.

A detailed investigation of the effects of lesioning the septo-hippocampal system on conditioned heart rate in restrained rats was carried out by Holdstock (1970). Although he found that septal lesions

did not impair the conditioning of heart rate deceleration, the possible effects of lesioning other parts of the system were not examined. The fact that the magnitude of the conditioned heart rate response failed to decrease during extinction was viewed by Holdstock as evidence of the involvement of the septal region in behavioral inhibition.

In the preceding discussion it was suggested that classically conditioned heart-rate responses in rats may be related to natural species-specific defense reactions. In addition, it may be that the direction of the classically conditioned heart-rate response depends largely on whether neural systems related to aggressive reactions become activated as opposed to the activation of systems related to inhibition or freezing behavior. The results of the present experiments demonstrated that the primary determinant of whether cardioaccelerations or cardiodecelerations develop was the degree of restraint imposed upon the subjects. Consequently, it may be that this factor determines the selection of either inhibitory or aggressive response systems.

To evaluate the usefulness of these hypotheses requires detailed information on the dynamics of the cardiovascular responses that occur during conditioning in both unrestrained and restrained rats. In addition, the precise specification of the neural structures involved in the development of the classically conditioned heart-rate response and its relation to other behaviors must be determined. Hopefully, with this type of information, a more thorough understanding of the heart-rate conditioning process can be obtained.

SUMMARY AND CONCLUSIONS

The present investigation was designed to examine the effects of instrumental reinforcement contingencies on the direction of classically conditioned heart-rate reactions of rats. In addition, both gross-movement activity and electromyographic (EMG) activity were recorded to determine the relation of skeletal responding to the development of the heart-rate changes. In Experiment I, a 2 x 2 factorial design was employed with the two dimensions being the conditioning procedure (signalled escape training vs. yoked classical conditioning) and the degree of restraint imposed upon the subjects (unrestrained vs. restrained). All subjects were given a differential conditioning procedure in which CS+ (either a 1-kHz or 5-kHz tone) was always paired with a tail shock unconditioned stimulus(US) and CS- was presented alone. All subjects received 15 min. of adaptation to the conditioning chamber, 24 CS-alone trials, and 80 acquisition trials consisting of 40 CS+ and 40 CS- presentations. Reinforced escape trials began with the presentation of CS+ followed 6 sec. later by the raising of a shield covering the response wheel and the onset of the US. The US remained on until a response occurred or if no response occurred within 60 sec. the US was automatically terminated. Reinforced classical conditioning trials were identical to escape trials except that an escape response was prevented by blocking the rotation of the response wheel. CS- trials in both escape and classical conditions consisted of a 7-sec. CS without the US or the raising of the shield. The index of conditioning for heart rate, movement, and EMG was a difference score computed by subtracting pre-CS activity from activity during the CS period.

Experiment II was carried out to determine if a vigorous wheel turning response would result in conditioned cardioaccelerations in

restrained rats. The basic apparatus and procedures were the same as those employed in Experiment I. However, in this experiment the subjects were required to rotate the wheel throughout a 1-min. period in order to terminate and postpone the recurrence of shock. Conditioned changes in heart rate were assessed by employing a control group receiving unpaired presentations of the CS and US.

The principal findings of these studies were:

1. Both unrestrained-escape and unrestrained-classical groups displayed conditioned cardioaccelerations whereas the corresponding restrained groups showed conditioned cardiodecelerations.
2. In the unrestrained and restrained conditions, a wheel-turning escape response failed to influence the direction or magnitude of the conditioned heart-rate reactions.
3. In Experiment II, six experimental subjects showed initial cardioaccelerative responses, however, the group as a whole displayed an overall small cardiodecelerative reaction.
4. Conditioned changes in gross movement and EMG activity were generally not associated with corresponding conditioned changes in heart rate.
5. Examination of heart rate and movement on a trial by trial basis demonstrated that the two responses were frequently independent and in fact changed in opposite directions on some trials.

In summary, the results of the current study provided little evidence that heart-rate accelerations seen in unrestrained rats were attributable to an excitatory state dependent on instrumental escape contingencies. Similarly, the movement results were not consistent with cardiac-somatic coupling hypotheses proposing that increases in movement lead to cardioaccelerations while decreased movement results in

cardiodecelerations. Rather, the major determinant of the direction of the heart-rate responses was the degree of restraint imposed upon the subjects. It was suggested that the degree of restraint may influence whether an inhibitory freezing type of reaction or an aggressive threat type of behavior develops each of which may be accompanied by characteristic cardiovascular responses.

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