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# THE EFFECTS OF CONTROL, FEEDBACK AND PREDICTABILITY ON PSYCHOPHYSIOLOGICAL INDICES OF STRESS

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# THE EFFECTS OF CONTROL, FEEDBACK AND PREDICTABILITY ON PSYCHOPHYSIOLOGICAL INDICES OF STRESS

by

SARAH RUTH BAKER

A thesis submitted to the University of Plymouth in  
partial fulfilment for the degree of

DOCTOR OF PHILOSOPHY

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## ABSTRACT

### THE EFFECTS OF CONTROL, FEEDBACK AND PREDICTABILITY ON PSYCHOPHYSIOLOGICAL INDICES OF STRESS

by Sarah Baker

The primary aim of this thesis was to examine the psychophysiological effects of predictability, personal control and feedback within the biopsychosocial framework of the stress-diathesis model proposed by Steptoe (1991).

Five studies were carried out. In the first, cardiovascular, electrodermal and behavioural effects of providing feedback were examined. The results indicated that the provision of feedback increased individual's performance on a complex, decision-making task and maintained that performance over time. In contrast, feedback appeared to have minimal effect on autonomic activity. In the second and third studies, the physiological effects of predictable and unpredictable noxious events were examined. In general, it was found that predictability was beneficial in terms of a reduction in cardiovascular reactivity. However, despite some suggestive effects, no evidence for the influence of predictability was obtained from the electrodermal system. The fourth study investigated the interactive effects of predictability with those of control. Again, the results yielded support for the beneficial effects of predictability, but only in situations during which individuals had no control over the duration of aversive stimulation. Where personal control was available, predictability augmented cardiovascular reactivity. As before, there was little differential effect in terms of electrodermal activity. In the fifth study, further support was obtained for these additive effects of predictability and control in the cardiovascular system. Moreover, the provision of feedback enhanced these additive effects.

These findings were discussed in terms of the stress-diathesis model which was extended to incorporate the effects of feedback. A framework for integrating the physiological effects of these three factors within the stress-diathesis model was formulated based upon the concepts of effort and distress.



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Finally, to my parents Val and Robin Baker who have supported me over the years and waited patiently for the fruition of this work.

## AUTHOR'S DECLARATION

At no time during the registration for the degree of Doctor of Philosophy has the author been registered for any other University award.

The work presented in this thesis was solely that of the author, with the exception of the first study which formed part of a collaborative project undertaken with Dr. J. A. Spinks, Ms. M. Wong and Dr. B. M. Jones at the University of Hong Kong. The author was an equal collaborator in regard to the design of the experiment. However, statistical analyses undertaken for the study was solely the work of the author. The project was funded by the University of Hong Kong. The remaining work presented in this thesis was financed with the aid of a studentship from the University of Plymouth.

A programme of advanced study was undertaken, which consisted of guided reading in the area, supervised by Dr. D. Stephenson, supervised instruction of psychophysiological techniques and attendance at relevant conferences.


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# CHAPTER 1

## INTRODUCTION

Stress is a notoriously vague term, has initiated a number of debates over its definition, and in the long-term may prove irrelevant as a single concept than as a general framework for a broad range of phenomena. The problem has been summed up in a statement by Steptoe in which he suggested, "stress has become an umbrella for discussions of emotional and behavioural influences on physical disorders, referring to almost every method by which personality, actions or environment affect illness" (Steptoe, 1980, p. 55). Alternatively, stress may be important in linking emotional and behavioural reactions to events and, in turn, their physiological correlates. These links, in turn, may have the potential to influence the aetiology, maintenance and/or recurrence of certain diseases. Indeed, a review of the vast literature reveals that the majority of research into ill-health uses the concept of stress with most employing some concept of demand to connect environmental events to pathophysiological ones.

Historically, systemic arousal has been studied in great detail, although the validation and examination of stress as an explanatory concept is relatively new. From this work two broad approaches have emerged. On the one hand, stress has been defined as a response or dependent variable which identifies patterns of physiological responding elicited in different situations (e.g. Cannon, 1935; Selye, 1956, 1976). The sole criterion of whether or not an organism has been stressed is whether or not a pattern of 'stressful' responses are present, regardless of the nature of the experience undergone. Thus, stress responses are non-specific. The alternative approach defines stress as a stimulus or an independent variable which may be observed or even manipulated with the aim of specifying its effects on the organism (e.g. Holmes & Rahe, 1967; Ursin, Baade & Levine, 1978). Thus, a variety of factors ranging from major life events to minor hassles, from sleep deprivation to overcrowding, from electric shock to aversive noise, may be examined for their supposed effects on behaviour.

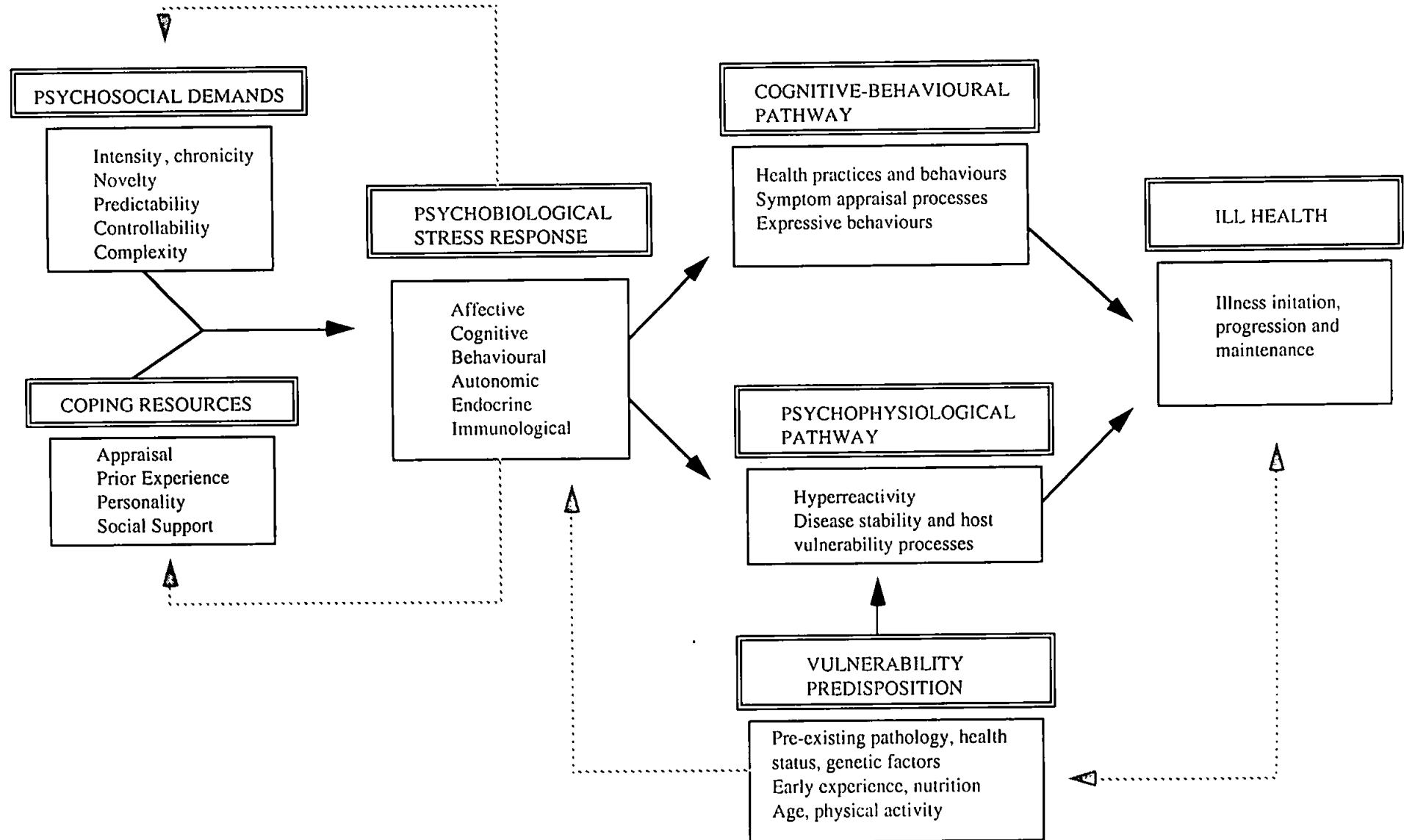
However, a contemporary approach to understanding stress is interactive incorporating both the "stress outside" and "stress inside" approaches (e.g. Leventhal & Tomarken, 1986; Levi, 1987; Steptoe, 1989, 1990, 1991). The development of these

interactive or 'stress-diathesis' models are indicative of the complexity of the stress concept. Indeed, with the acceptance of this type of model, the all encompassing stress concept is replaced by narrower and more precise hypotheses concerning the various possible interrelationships between psychological, social, environmental and biological components.

One exemplary stress-diathesis model is that espoused by Steptoe (1991) (see Figure 1). The aim of the model is to incorporate environmental influences and their interactions with biological and genetic factors in order to describe psychobiological stress responses that may be precursors to disease. According to this model, both physiological and psychological factors present demands upon an individual's resources and capacity for coping which, in turn, lead to responses involving both the psychophysiological and cognitive-behavioural systems. Coping resources included in the model are; the individual's appraisal of the situation, prior experience, the availability of social support and various personality variables, such as, the Type A behaviour pattern. Likewise, a variety of important environmental factors or 'psychosocial demands' are identified including not only the most obvious, such as, the intensity and duration of the situation or event, but also its novelty or complexity, and the amount of control or predictability available to an individual. Whether or not the interaction between psychological and environmental factors leads to changes in the psychophysiological or cognitive-behavioural systems depends upon an individual's predisposition or vulnerability. Various constitutional factors have been identified including; an individual's genetic background, sex, age, physical fitness, diet, and smoking and alcohol intake. These vulnerable or predisposed individuals have the capacity to develop chronic physiological reactions such as, heightened haemodynamic responses and these, in turn, have been implicated in the early stages of the aetiology but more commonly in the course, severity and prognosis of 'stress-related' disorders.

Implicit in this interactive approach to understanding the manifestations of stress is the assumption that stress may lead to physical, psychological and/or behavioural outcomes or symptoms. However, the role of stress in health remains a contentious issue, mainly because of difficulties in delineating the mechanisms that link stress with disease states. Thus, what is required is not only an examination of the physiological and constitutional

Figure 1. Elaborated stress-diathesis model (Stephens, 1991; p. 639)



systems involved, but also a detailed understanding of the potential moderators of the response to stress. Investigations into specific mediating variables in order to identify those properties of the situation that make them potentially stress-inducing and to understand their relationships with physiology and behaviour have to be undertaken whilst recognising that there is seldom, if ever, any simple, direct or univariate link between environmental stimuli and physiological or stress-related processes. However, by identifying formal properties of the situation, it is possible to provide a taxonomy of those properties that are especially relevant to how stress processes and health-related outcomes are determined. Indeed, various environmental or perceived environmental factors have been identified as potentially important in terms of stress-related outcomes. The influential work of Weiss (1968, 1970, 1971a, 1971b, 1971c) identified three particular variables, those of predictability, control and feedback.

Predictability has been a major theme in stress-related research since the 1950's (see Badia, Harsh & Abbott, 1979; Phillips, 1989; Steptoe, 1989), especially in experimental studies involving nonhuman subjects. A critical review of the literature on the role of predictability in promoting or reducing stress responses, detailed in Chapter 2, suggests the data are both complex and contradictory. The most popular view is that nonhuman subjects, if given a choice, prefer conditions which are predictable and, that these conditions lead to a reduction in physiological indices of stress. This view also prevails in the human literature, with individuals choosing predictable over unpredictable events. On a psychophysiological level, it is suggested that predictable aversive events lead to a lowering of autonomic and subjective responses compared with unpredictable events. However, on closer examination, there appears to be inconclusive support for this position. Moreover, there appears an equal amount of data which suggests that predictability leads to an increase in psychophysiological stress compared with unpredictable conditions, or, reports no difference.

These conflicting results can be partially explained by individual differences or experimental parameters. With regard to the latter, these have arisen partly because predictability is an ambiguous concept and the majority of researchers have failed to distinguish amongst its many different components. In the vast majority of studies, for example, researchers have confounded varying types of predictability in one experiment;



i.e. when the event will occur (temporal predictability); the probability of whether an event will occur or not; and the physical characteristics of the event such as, its quality and intensity. Other experimental factors have also been implicated including; the severity and chronicity of aversive stimulation, levels of shock attenuation, response interference, and the indices of 'stress' employed. However, even when these factors are taken into consideration there still remains a fair amount of controversy. What is clear, however, is that there are other psychological factors which are involved in the majority of the experiments concerning predictability which may help to explain these discrepancies. The most frequently occurring are those of control and feedback.

The concept of control has been widely accepted as having significance for stress-related behaviour (see Steptoe & Appels, 1989). Indeed, there has been a growing interest in the concept over recent years with the tendency to assume that control, or the perception of control, over aversive stimulation has a beneficial or stress-reducing effect and, that loss of control is debilitating. Indeed some researchers (e.g. Sells, 1970) have argued that lack of control is a necessary if not sufficient condition for stress whilst others have gone even further. For example, Lefcourt (1973) concludes; "... the sense of control, the illusion that one can exercise personal choice, has a definite and a positive role in sustaining life" (Lefcourt, p. 424). Despite these conclusions, and the formidable amount of literature documenting the deleterious effects of lack of control, this position has not gone unchallenged. Indeed, a critical review of the literature, detailed in Chapter 4, reveals the term 'control', as seen previously with predictability, to be both complex and ambiguous. Indeed, some researchers have suggested that it has developed into a concept of such vagueness, encompassing such a wide variety of heterogeneous ideas, that its usefulness is in question (Phillips, 1989). For example, the following includes some of the types of control which can be found in the current literature, a large number of which have been used interchangeably; illusion of control (Langer, 1975), locus of control (Folkman, 1984), preference for control (Overmier, Patterson & Wielkiewicz, 1980), loss of control (Baum & Valins, 1977), perception of control (Geer, Davison & Gatchel, 1970), control as a motivator (Rodin, Rennert & Solomon, 1980), control as choice (Corah & Boffa, 1970), control as a reinforcer (White, 1959), behavioural control (Miller, 1979), cognitive

and decisional control (Averill, 1973), retrospective control and control as information (Thompson, 1981) and, finally, self control (Bjorkstrand, 1973).

Despite these difficulties in definition, however, the concept has been instrumental in stimulating research into the possible relationships between psychological factors and stress-related disorders (see Steptoe, 1983, 1989). Indeed, when these problems have been taken into consideration, it is clear that, in certain situations, control can have both stress-reducing and stress-inducing effects depending on a number of environmental and individual factors. With regard to human subjects, these factors range from the type of physiological index being measured to the effort or difficulty experienced in exerting control. For example, it has been suggested that control may only lead to a reduction in stress responses in situations where the outcome of the behavioural response is certain. Thus, only when certainty is high will control be stress-reducing. In contrast, situations where control is difficult to achieve leads to heightened responses i.e. it is stress-inducing (see Obrist, 1981; Steptoe, 1983). However, even when control is reliable there are a number of other important contributing factors, such as, whether the control response is exerted over the initiation, termination or frequency of the event and the availability of incentives. There may also be differences between the immediate and longer-term physiological responses. Thus, several dimensions of control affect physiological response patterns during aversive stimulation and it is, therefore, reasonable to assume that the experience of control is determined, in part, by the qualitative nature and meaning of the control response to the individual, and not just upon its effectiveness in preventing or mitigating the impact of a potentially aversive event. On a subjective level, absence of control has been associated with alterations in cognition, affect and personality patterns. With regard to the latter, several patterns have been identified including; neuroticism, depression, Type A behaviour pattern, loneliness and anxiety (see Steptoe & Appels, 1989). Furthermore, lack of control has also been found to influence health-related behaviours. As with biological processes, however, the mechanisms underlying subjective and behavioural changes are far from being well defined.

Although control is itself a potentially important variable in ameliorating psychophysiological stress, it may be that this variable can also account for a large proportion of the evidence currently attributed to predictability in experiments where

aversive stimuli are employed. For example, evidence suggests that in situations where individuals have control, predictability may be less aversive (e.g. Weiss, 1971a). In situations where no control is available, predictability frequently appears to be more aversive (e.g. Pare, 1964). Whether control is the important variable, or, whether control and predictability interact to produce an effect, is difficult to determine based on the experimental data available. Indeed, in the majority of predictability studies the factors of predictability and control have been confounded and *vice versa*. This is not surprising as it is extremely difficult to manipulate the two independently of one another. For example, in most situations predictability affords a weak sense of control in the sense that it provides the individual with information as to when something is going to happen. Indeed, there are a number of researchers (e.g. Averill, 1973) who believe that it is the element of predictability in control that is critical in accounting for the importance of control. Consistent with this view is that predictability without control is just as effective in reducing stress responses as control without predictability (e.g. Burger & Arkin, 1980). Other authors have suggested that the effects of predictability can be reduced solely to those of control (e.g. Cantor 1981, Dinsmoor 1983). However, it may be that predictability and control are independent constructs whose effects are additive (e.g. Phillips, 1989).

It would appear, therefore, that not only are the two factors difficult to manipulate independently of one another but, in addition, various relationships between the two factors are possible many of which appear to make identical predictions regarding the outcome of any factorial experiment. For example, the additive model predicts that an individual will experience the greatest psychophysiological stress when neither control or predictability are present. Individuals who can either control or predict should show intermediate levels of psychophysiological stress whilst those with both control and predictability should show the least amount of psychophysiological stress. However, if predictability and control reduce to a single common factor, the *same* predictions would be made; namely, those individuals with neither control or predictability experience the least of this common factor; individuals who have one or the other experience intermediate levels; and individuals with both control and predictability experience the greatest amount of the underlying factor.

Feedback is a second factor which may be involved in studies investigating predictability. Indeed, feedback has been shown to have a significant effect in determining physiological responses in nonhuman subjects, as shown by the work of Weiss (1971a, 1971b, 1971c) and others (e.g. Tsuda & Hirai, 1975). However, in spite of the considerable interest concerning the effects of feedback in the applied, behavioural literature, there has been relatively little attempt to understand the relationship between feedback and psychophysiological stress in human subjects, to any great extent. The literature relating to feedback is reviewed in Chapter 3.

The general term used for a variety of forms of psychological feedback is knowledge of results (KR), with the most common form of KR being where the experimenter takes a performance measure which is not normally available to the subject and feeds this information to them. With regard to both the laboratory and applied literature involving human subjects, KR has been found to enhance performance; improve rates of learning; that it augments motivation; that the more specific it is, the greater the impact; and, that when feedback is decreased, performance sometimes decreases. These findings have been reported using a variety of aversive and non-aversive tasks and, in numerous applied situations. In terms of those studies employing nonhuman subjects, these have generally reported that conditions in which feedback is provided leads to lower levels of physiological stress compared with no feedback conditions.

However, as with both predictability and control, there are a number of factors associated with KR which may augment or attenuate these beneficial effects, such as, its timing, frequency, specificity and source as well as the availability of incentives and/or goals. Furthermore, there has been some controversy in the literature as to why KR produces any beneficial effects. One line of evidence suggests that it is due to its informational component. For example, KR - defined previously as information about an aspect of subject's performance - may be used to significantly alter the individual's ability to cope with the situation not only by virtue of the feedback provided but also because KR and improved performance may simultaneously increase stimulus predictability and control over the situation. Therefore, an increase in an individual's performance could equally be a result of any or all of these components, and to attribute any beneficial effects to feedback *per se* would be erroneous. However, whether enhanced coping is or is not a result of an

increase in predictability and control depends partly on the amount and extent of information given to a subject via KR. For example, it is reasonable to assume that as the amount of detailed information is increased so too is the amount of perceived predictability and control. Thus predictability and control may help explain some of the conflicting results found in many of the studies involving feedback. Conversely, the availability of KR in many of the studies examining the supposed effects of control and/or predictability, may help explain some of the inconsistencies found in this literature. For example, some researchers have hypothesised that predictability may only be effective in reducing psychophysiological stress if it provides feedback i.e. because of the information given by the warning signal about safety periods (e.g. Seligman, 1975). Thus, feedback may be an important variable in accounting for the effects of predictability, and to the extent that feedback is an important variable in accounting for predictability, it may also be related to the fact that control is present in most of these experiments. For example, many effects ascribed to control may be a result of the added feedback inherent in control, i.e. information about the efficiency of a response. Thus, the degree of observed psychophysiological stress may be the product of an interaction between the availability of control, the predictability of the event and the amount of feedback received (see Weinberg & Levine, 1980; Weiss, 1971a). Weinberg and Levine (1980), for example, hinted at possible interrelationships based on a review of studies carried out with predictability. They suggested that the positive effects of predictability are primarily attributable to control and the negative effects to a lack of information or feedback. Thus predictability may always be aversive unless the situation also offers a type of control, or, unless the individual can gain feedback concerning the situation.

It appears therefore that all three environmental factors, control, predictability and feedback, may be potentially important with regard to the pattern and extent of psychophysiological stress. The effects of these variables are investigated in the experimental chapters of this thesis. The effects of feedback are examined in Chapter 6, predictability in Chapters 7 and 8, the interaction of predictability and control in Chapter 9 and the interaction of all three variables in Chapter 10.

However, the mechanisms underlying the effects of these variables are far from clear. For example, the psychobiological stress response identified in Steptoe's (1991)

interactive model (see Figure 1) is a loosely coupled system incorporating affective, cognitive, behavioural, autonomic, endocrine and immunological changes. The precise details of each of these components and their relationship to the stress-illness link are still relatively vague. In terms of biological function, for example, most current models of psychophysiological mediation state that an organism responds to aversive stimulation with large autonomic or neuroendocrine reactions and a reduction in these responses after termination of that stimulation. The relationship between these responses and disease is therefore that repeated or sustained exposure to the event may be pathogenic (e.g. Selye, 1956; Ursin, 1980). However, more recent research has suggested that this view is oversimplified (see Phillips, 1989; Steptoe, 1991). Indeed, direct evidence for this type of process has been elusive, although there is some evidence from the hypertension literature (see Obrist, 1981). Furthermore, by taking such an over-simplified view of the psychophysiological process, it is possible to ignore other potentially important mediating mechanisms. It may be, for example, that over-reactivity is not the only possible mediating process between stress and ill-health for *all* individuals, important variations may exist. Greater emphasis should therefore be placed on understanding patterns of physiological responses to stress. One particular approach taken in this thesis was suggested by Seligman's Safety Signal hypothesis (1975), in which it is suggested that the most important period, in terms of reducing an organism's physiological stress response during conditions employing aversive stimulation, was the amount of time in which the organism "felt safe" or had the opportunity to "relax". Thus, the converse to the over-reactivity response process - an organism's ability to lower physiological function - may have important consequences in aversive situations. This proposition has rarely been examined with regard to the stress-illness link (e.g. Lewis, Meyers, Kagan & Grossberg, 1963) and, in particular, with reference to specific environmental factors, such as, predictability, control or feedback. However, such response measures might be of potential significance and environmental factors may differentially effect the physiological response, with lower levels of physiological activity having significance in particular situations. These possibilities would be clarified if several response parameters were monitored simultaneously during experimental conditions. In order to achieve this, various physiological parameters will be examined in relation to the data presented in this thesis.

The physiological, psychological and statistical basis underlying these measures are discussed in Chapter 5.

To summarise: Despite decades of research and theorising on the role of predictability and control and to a lesser extent, feedback, little consensus exists as to the benefits of these factors to the individual. Further, a large body of evidence has tended to yield a variety of conflicting findings and, it has been difficult to find a framework that can account for these inconsistencies. Thus, this thesis attempts to investigate the conditions which do and do not lead to a change in psychophysiological stress in aversive conditions. Firstly, the literature in the areas of predictability, feedback and control will be reviewed in the context of relevant theories. Some of the methodological issues involved in the measurement, quantification and analysis of psychophysiological stress will then be discussed. This is followed by five experimental chapters investigating the effects and interactions of predictability, control and feedback. Finally, an integrative framework for the understanding, delineation and analysis of the concepts of control, predictability and feedback will be discussed.

## CHAPTER 2

### PREDICTABILITY

#### 1. INTRODUCTION

Since the late 1950's there have been a great number of studies carried out suggesting that warning about aversive events is advantageous to both human and nonhuman subjects. However, an extensive review of the predictability literature reveals a number of inconsistencies. Indeed, several studies have indicated that predictable aversive events are perceived as neither more preferable nor less aversive than unpredictable stressors.

Various factors which may alter an organisms response to aversive stimulation are examined in this chapter. Namely, the benefit which may accrue from information about when an aversive event will occur (i.e. temporal predictability). Subjectively, benefit may be in the form of lower distress or perceived aversiveness. Psychophysiologically, it has commonly been thought of in terms of smaller autonomic changes to event impact and less autonomic activation in anticipation of that event. With regard to nonhuman subjects, physiological benefit has been examined in terms of lower levels of plasma steroids, body temperature, stomach ulceration and a greater body weight. Yet another measure of benefit, in both human and nonhuman subjects, has been in terms of behavioural choice. All three areas of benefit will be examined in this review.

Although a large amount of studies have investigated aspects of predictability in applied settings, many of which identify individual differences to account for differences in the benefit derived from either predictable or unpredictable aversive situations (e.g. Averill & Rosenn, 1972), only laboratory experiments will be reviewed in this chapter. The major reason for excluding applied studies is that of space. Moreover, for the purposes of replication, laboratory studies provide a more reliable source of experimental findings.

Several theories have been put forward in order to account for the supposed benefit of predictability of aversive events. These are the 'preparatory activity' theories which centre on events prior to or around the impact point of an aversive event. Two such theories will be examined in this thesis; namely, the Preparatory Response (Perkins, 1968)



and Preception (Lykken, 1962) Theories. A second approach involves the concept of safety signals (Seligman, 1968), and focuses its explanation on the period away from the impact point of an aversive event; namely, when the organisms perceives itself safe.

## 2. THEORIES OF PREDICTABILITY

### 2.1. Preparatory Response Theory

According to the Preparatory Response Theory (Perkins, 1968), stimuli that precede aversive events allow subjects to prepare for those events either peripherally or centrally. This, in turn, has the effect of reducing the aversive effects of that event. When the event is unsignalled, however, this preparatory response cannot be made at the appropriate time, leading to either no change or an increase in the impact of the aversive event.

A number of testable predictions are made by the preparatory hypothesis. First, that predictable aversive events should be perceived as less intense than unpredictable events. Second, that autonomic indices of stress should be lower during predictable compared with unpredictable aversive events, and lastly, that if given a choice, subjects should prefer predictable compared to unpredictable aversive events.

There are a number of advantages to the Preparatory Response Theory in that it accounts for the effects of factors, such as, warning signal duration and variability in determining preference for predictability and levels of physiological response to the situation. For example, it has been suggested that longer signals allow more adequate preparation than shorter signals, and data supporting this position has been found in various experimental studies. Signal variability may also be preparatorally important. For example, optimal preparation responses must be precisely timed, and conditions that allow for this should be preferred to conditions that do not. Indeed, it has been found that subjects prefer fixed over variable signal durations and immediate over delayed shock.

Thus, one of the strengths of the preparatory hypothesis would appear to be that it is explicitly testable. In testing its predictions, however, one must make specific assumptions about the nature of the preparatory response, and unfortunately, when the

various assumptions made across experiments are viewed as a whole, they often conflict. For instance, the finding of a preference for immediate over delayed shock and of fixed over variable signal durations, suggests that precisely timed responses are optimal and, that delayed or variable shock makes timing difficult. However, many studies have found greater preference for longer signal durations leading some researchers to suggest the opposite. That is, longer warning allows more effective preparation than shorter signals.

A similar conflict occurs when examining shock intensity. Some studies have reported that preference for predictable events increases as shock intensity increases, presumably because the need for preparation is greater at higher intensities. However, others have suggested the opposite, namely, that preparation should occur equally at all shock intensities. Furthermore, it has been reported that preference is maintained when the aversive event in the predictable condition is increased up to three times the intensity, four times the density, and nine times the duration of the same event in the unsignalled condition. It seems unlikely that a preparatory response of the degree of effectiveness required to explain the latter data could be found. The empirical data examining these, and related questions, will be examined in Sections 3.2.1. and 3.2.2 for nonhuman and human subjects respectively.

As mentioned previously, a basic requirement of the Preparatory Theory is that autonomic indices of stress should be lower during predictable compared with unpredictable aversive conditions. However, the experimental data is contradictory. In terms of those studies employing human subjects, many have reported a reduction in physiological stress as a function of predictability, others an increase, whilst the majority reveal no difference between predictable and unpredictable conditions. Moreover, this appears to be case for both anticipatory and event impact data. With regard to nonhuman subjects, however, the vast majority of studies have found a reduction in autonomic indices as a function of predictability. The literature relating to physiological indices of stress will be reviewed in Sections 3.3.1 and 3.3.2 for nonhuman and human subjects respectively.

## 2.2. Preception Theory

At the same time as Perkins and colleagues were developing the preparatory response theory, Lykken (1959) reported a study in which he found that electrodermal responses elicited by electric shock were lower in predictable compared to unpredictable trials. Lykken hypothesised that the aversiveness of the signalled shock was reduced by preparatory activity or a "phasic and selective inhibitory process initiated by the warning signal and centred upon the expected time of arrival of the noxious stimulus" (Lykken, 1962). This preparatory activity was termed preception.

The Preception Theory makes similar, although more specific, predictions as the Preparatory Response Theory regarding the outcome of predictability studies (see Section 2.1). For example, an organism's response to predictable events will be a function of the length of the inter-stimulus interval (i.e. the time between the signal and the event). That is, preception should be optimal at shorter compared to longer intervals. This is because in the former, event onset is more predictable. Furthermore, the theory predicts that the effect of preception is a function of the intensity of the noxious event; namely, stronger stimulation should augment the preception effect to a greater extent than weaker ones.

General predictions made by the Preception Theory include; firstly, that smaller physiological responses should occur with predictable aversive events than with unpredictable events. Secondly, that if subjects are given a choice, they should prefer predictable over unpredictable aversive events.

In terms of the empirical data, the Preception and Preparatory Response Theories can account for the same proportion due to the fact that they both make similar predictions concerning subject's behaviour in aversive situations, although for different reasons. According to Perkins' theory, preparatory responses are manifestations of classical conditioning, whereas, the preception hypothesis implies a more 'cognitive-perceptual' approach. However, there is a lack of specificity about how the preception effect may operate. Indeed, the primary weakness of the preception hypothesis is that it implies a neurophysiological mechanism which, based on the data available, cannot be objectively identified.

The empirical data relating to Lykken's Preception Theory in human subjects will be reviewed in Sections 3.2.2. and 3.3.2, although as mentioned previously, those studies employing nonhuman subjects and associated with the Preparation Response Theory (Sections 3.2.1 and 3.3.1) might also be explained in terms of preception.

### 2.3. Safety Signal Theory

An alternative explanation to these preparatory theories, is in terms of the Safety Signal Theory (Seligman, 1968; Seligman, Meier & Solomon, 1971). The safety hypothesis in its simplest form divides the aversive situation into discriminable shock and shock-free periods. For example, when a warning signal reliably predicts the occurrence of shock, then safety, the absence of shock, is also predicted by the absence of the signal for shock. Under the signalled condition, therefore, shock (danger) periods and shock-free (safe) periods are reliably predicted. However, in the unsignalled condition, neither safe or danger periods can be reliably predicted. According to the safety hypothesis, subjects choose the signalled shock condition over the unsignalled one because the safe periods are identifiable and longer, as they include the total inter-stimulus interval. This, in turn, generates a pattern of alternating arousal (during the signal) and relaxation (during signal absence). Whereas, if no safety signal is present, the subject will remain in chronic fear over a longer time period and will therefore maintain a high state of physiological arousal.

The Safety Signal Theory is relatively explicit about when and why arousal and/or distress will differ between predictable and unpredictable conditions. Consequently, it can account for data which suggests that organisms show greater anticipatory distress prior to predictable events compared with unpredictable aversive events. This is because, in the former condition, subjects are in the presence of danger signals (the warning signal) which lead to greater levels of arousal than situations where no danger signals are present i.e. the unpredictable condition. However, difficulties have arisen in that the theory does not make any specific predictions about the way in which an organism will respond to the impact of an aversive event, and it cannot reconcile the fact that a minority of people when given a choice will prefer the unpredictable condition.

Furthermore, in terms of experimental parameters such as signal duration, a number of conflictory predictions have been made in the literature. For example, the finding that organism's prefer longer over shorter signal durations is presumably incompatible with a safety analysis which suggests that shorter signal lengths should be preferred. This is because the latter are easily discriminable, and lead to both shock and shock-free periods being easily identifiable. Conversely, longer signal durations, it is suggested, lead to less easily discriminable shock-free periods. Thus, if safety is the only factor determining preference for the predictable condition, strong preferences should develop at the shorter durations. However, the opposite assumption has also been suggested from a safety view, namely, that longer signal durations lead to more easily identifiable safe and danger periods compared with shorter signal durations. Longer signal durations should therefore be preferred.

Other, slightly tenuous results have been pointed to in the literature in order to suggest that safety is not a necessary condition for preference. For example, a handful of studies examining the effects on preference of varying the probability of shock and shock-free periods reveal that preference for the predictable condition remains even when unpredictable shocks occur in the supposedly safe (shock-free) periods. Preference was only eliminated when the probability (dependability) of safety was reduced to a minimal level. It has been argued that in terms of the safety analysis it is difficult to reconcile these findings, because the shock-free period was not 100% safe in the latter condition. These and related findings involving both nonhuman and human subjects will be reviewed in Sections 3.2.1 and 3.2.2 respectively. The Safety Signal Theory as it applies to autonomic indices of stress will be reviewed in Sections 3.3.1 and 3.3.2, for nonhuman and human subjects respectively.

### 3. LITERATURE REVIEW OF PREDICTABILITY

#### 3.1. Methodological Issues In Predictability Research

The psychophysiological differences between predictable and unpredictable events can be examined in various ways. These include when the event will occur (temporal

predictability), the probability of whether an aversive event will occur or not and the physical characteristics of the aversive event. The following review of the literature will be largely concerned with temporal predictability, as this has been the most extensively studied in the laboratory.

There are a number of methodological problems associated with past studies in the area of temporal predictability. The most salient of these problems is that predictability has been confounded with other variables. For example, in a predictable condition, the warning signal itself elicits an autonomic response, and therefore any comparisons between predictable and unpredictable conditions are confounded by the response elicited by the physical properties of the warning stimulus. This has been termed response interference (see Kimble & Ost, 1961; Peeke & Grings, 1968).

Some researchers have tried to avoid the problem of response interference by omitting the warning signal and comparing temporally random versus non-random aversive events. Alternatively, other studies have investigated the differential effects of fixed versus variable signal-event intervals. However, both of these procedures introduce difficulties, for instance, if the inter-trial (or stimulus) interval is not short, subjects will not be able to reliably discriminate the length of fixed or random intervals. Thus, the aversive event becomes subjectively unpredictable for all subjects regardless of experimental condition.

Other procedures have been employed to minimise this problem, many of which have resulted in very complex procedures and as a result, their findings have often been difficult to interpret. For example, Furedy and Klajner (1972) employed a complex secondary signalling procedure within which they examined the physiological response pattern produced by the secondary signal rather than to the event itself. One problem with this type of design is that the response elicited by the secondary signal which signalled whether the subsequent aversive event was to be predictable or unpredictable, cannot be taken as a simple unambiguous index of anticipated aversiveness of subsequent stimuli, because a response produced by a signal can be qualitatively different from a response produced by the event itself.

These alternative measures have been employed to a greater extent in recent studies involving human subjects, rather than the older, animal experiments. All relevant literature

will be reviewed in the following sections, regardless of experimental design manipulations, although the methodological and conceptual differences should be noted.

### 3.2. Preference For Predictability

3.2.1. Nonhuman Studies. “Laboratory studies of preference for predictability of aversive events in choice situations have been consistent in the finding that where animals may choose behaviourally between either receipt of predictable electric shock (i.e. shock preceded by a warning signal or delivered at fixed temporal intervals) or unpredictable shock (i.e. non-signalled shock or variable interval presentation) then the predictable option is preferred” (Phillips, 1989; p. 241).

Some of the earliest research specifically directed toward the question of preference was performed by Perkins, Levis and Seymann (1963) and by Lockard (1963). In Lockard's study, rats were given a choice between a warning signal preceding unavoidable shock (experimental condition) and random presentation of the warning signal and shock (control) using a shuttle box procedure. Experimental parameters and results for Lockard's study and the remaining studies included in this section are summarised in Table 1. During the acquisition period, the control group spent an average of 50% of their time in the signalled side, compared with 90% for the experimental group. During the extinction phase, occupation of the warning signal side rapidly decreased to 50% of the time.

Perkins *et al's* (1963) results were similar to those of Lockard's. After ten sessions, 14 out of 16 of their subjects were spending about 70 to 80% of the time in the signalled shock side of the shuttlebox. When the conditions were reversed, however, a reversal in preference did not occur. This failure was due, according to Perkins *et al.*, to the short time allowed for the reversal phase i.e. only three test days.

A subsequent series of experiments by Perkins, Seymann, Levis and Spencer (1966) used identical shuttlebox apparatus and procedures as Perkins *et al.* (1963). In these studies, the effects of shock and warning signal duration and length of inter-shock intervals on preference were investigated.

Experiment 1 examined the differential effects of shock duration (0.05 or 5 sec) on preference. They found that for subjects receiving the 0.05 sec shock there was no clear

Table 1. Examples of nonhuman studies investigating choice between predictable and unpredictable shock  
(adapted from Badia, Harsh & Abbott, 1979, p. 1114-1116).

<u>Study</u>	<u>Choice Procedure</u>	<u>Parameters</u>	<u>Test Session</u>	<u>Results</u>
Abbott & Badia (1979)	Changeover	Scrambled, .5 sec, 1mA; Signal length varied	Multiple 6 hour sessions	Preference for P related to signal duration
Badia & Abbott (1980)	Changeover	Scrambled, .5 sec, 1mA; 5 sec signal	Multiple 6 hour sessions	All subjects preferred P
Badia, Coker & Harsh (1973)	Changeover	Scrambled, .5 sec, 1mA; 5 sec signal	Multiple 6 hour sessions	Preference for P when P shocks 2-8 times more frequent
Badia & Culbertson (1972)	Changeover	Scrambled, 1mA escapable (Expt 1) or inescapable (Expt 2), .5 sec; 5 sec signal	Multiple 6 hour sessions	All subjects changed to P schedule
Badia, Culbertson & Harsh (1973)	Changeover	Scrambled, duration and intensity varied; 5 sec signal	Multiple 6 hour sessions	Preference for P when P shocks up to 9 times longer & 6 times stronger
Badia, Culbertson & Lewis (1971)	Changeover	Scrambled, 1mA, .32 sec avoidable shock; 5 sec signal	Multiple 6 hour sessions	All subjects changed to a P schedule



Badia <i>et al.</i> (1975)	Changeover	Scrambled, .5 sec, .8mA; 5 sec signal	Multiple 6 hour sessions	Preference for P schedules
Badia, Harsh, Coker & Abbott (1976)	Changeover	Scrambled, .5 sec, 75mW; 5 sec signal	Multiple 6 hour sessions	Choice related to dependability of signal
Biederman & Furedy (1973)	Concurrent chain schedule	Unscrambled, scrambled or tail shock, 5 sec, 1mA; 5 sec signal	1 approx. 4 hour session	Preference for P with unscrambled shock only
Biederman & Furedy (1976)	Shuttlebox	Scrambled or unscrambled, 5 sec, .6mA; 3 sec signal	2, 3 hour sessions	Without pretraining, preference with unscrambled shock only
Frankel & Vom Saal (1976)	Shuttlebox	Scrambled, 1 sec, .5 mA; 12 sec signal	4, 10 hour sessions	More time in P schedule
French <i>et al.</i> (1972)	Shuttlebox	Unscrambled, 1mA, 1 sec; signal varied	Multiple 1 hour sessions	Preference for P shock
Gliner (1972)	Shuttlebox	Scrambled, 2 sec, intensity varied; 10 sec signal	5, 6 hour sessions	Preference for P in high & low intensity gps
Harsh & Badia (1975)	Changeover	Scrambled, .5 sec; 5 sec signal	Multiple 6 hour sessions	Preference related to intensity
Harsh & Badia (1976)	Changeover	Scrambled, .5 sec, 1mA; 30 sec signal	Multiple 6 hour sessions	Preference related to ITI

Knapp, Kause & Perkins (1959)	T maze	Unscrambled, .7 sec, 60V; 30 sec signal	50 sessions (8 trials a day)	80-90% of free-choice trials to P side
Lockard (1963)	Shuttlebox	Unscrambled, 2 sec, .28mA; 5 sec signal	12, 1 hour sessions	90% of trials on P side
Miller, Daniel & Berk (1974)	Shuttlebox	Direct, .5 sec, .4mA; 10 sec signal	30-50, 3 hour sessions	Clear preference for P, with reversals
Miller, Marlin & Berk (1977)	Shuttlebox	Direct or scrambled, .5 sec, intensity varied, ITI varied; 5 sec signal	Multiple 1.5, 2 or 4 hour sessions	Preference related to parameters most subjects preferred P
Perkins <i>et al.</i> (1963)	Shuttlebox	Unscrambled, .5 sec, 500V; 3 sec signal	6, 10 hour sessions	75-80% of time on P side
Perkins, Seymann, Levis & Spencer (1966)	Shuttlebox	Unscrambled or direct, 500V, duration, ITI varied; signal duration varied	6-9, 10 hour sessions	Up to 90% of time on P side, successful reversals
Safarjan & D'Amato (1978)	Changeover	Scrambled, .5 sec, .8mA; 5 sec signal	Multiple 4 hour sessions	Preference related to dependability of shock free period

Note: VT=variable time; FT=fixed time; P=predictable; ITI=intertrial interval. All subjects are rats unless otherwise indicated.

preference. However, subjects who received the 5 sec shock duration did reveal a clear preference. Although the difference between the two groups did not reach significance.

During Experiment 2, the effects of signal duration were examined, with three groups receiving either a 0.5, 3 or 18 sec signal. Shock duration was 0.5 sec. Preference for signalled shock increased with increasing duration of the warning signal. The 0.5 sec group produced no clear preference, the 3 sec group demonstrated some preference and the 18 sec group spent significantly longer in the signalled shock side.

Experiment 3 examined the differential effects of inter-shock interval. Subjects were assigned to one of four experimental conditions with shock every 1 min, every 5 min, every 30 min or six shocks every 30 min at irregular intervals. Shock duration was 0.5 sec and signal duration 18 sec. Those subjects who received the two shorter inter-shock intervals revealed a markedly greater preference than did the 30 min groups.

During Experiment 4, Perkins *et al.* (1966) examined whether behavioural preference was reversible, with all subjects receiving three consecutive days of training (acquisition) followed by six days of reversal in which the schedule on each side of the shuttlebox was reversed. Inter-shock interval remained at six shocks every 30 min, irregularly spaced. All other parameters were identical to Experiment 3. All subjects produced a marked and significant preference for the signalled side during acquisition and, unlike Perkins *et al.* (1963), also during reversal schedules. Perkins *et al.* concluded from these studies, that parameter values were important determiners of choice, and that optimal conditions for preference to occur were when subjects received a greater number of shocks, with a long warning signal duration and a longer shock duration.

Perkins (1968) favoured a preparatory response explanation to account for these and related findings (Knapp, Kaue & Perkins, 1959; Lockard, 1963, 1965; Perkins *et al.*, 1963). It was argued that the presence of a signal preceding shock onset allows the elicitation of a well timed response acting to mitigate event impact. The finding that animals preferred situations with longer warning signals (Perkins *et al.*, 1966) lent support to this view, in that longer signals allow more adequate preparation than shorter signals. These findings were later confirmed by studies employing operant changeover procedures (detailed later) (e.g. Abbott & Badia, 1979; French, Palestino & Leeb, 1972). Particularly salient are the findings of Abbott and Badia (1979) who found that animals did not show a

preference in situations employing short signal durations (i.e. 0.5 or 1.0 sec) but when durations were longer (i.e. 1.5 or 2.0 sec) a significant preference did emerge.

Experiments manipulating signal variability provided additional support for the Preparatory Response Theory. To be maximally effective, preparatory responses should be precisely timed, and conditions that allow for this should be preferred to conditions that do not. Indeed, in addition to the findings of Perkins *et al.* (1966), Safarjan and D'Amato (1977) reported a study in which subjects preferred fixed over variable warning signals. The authors employed a changeover procedure in which subjects could choose between one of two conditions - fixed or variable warning - by the press of a lever. In both conditions shocks (0.5 sec duration and 1.0 mA intensity) were preceded by a 5 sec white noise (82 dB) warning stimulus. However, in the variable condition the probability of the warning signal terminating in shock was less than one whereas in the fixed condition probability always equalled one. As mentioned previously, a significant preference for the reliable signal emerged. The assumption of precisely timed preparatory responses also provides the rationale for predicting preference for immediate over delayed shock (e.g. Knapp *et al.*, 1959), and greater preference in situations employing shorter compared with longer inter-shock intervals (e.g. Perkins *et al.*, 1966).

Nevertheless, several studies have reported findings that are supposedly difficult to reconcile with the preparatory hypothesis. For example, the results of an experiment by Harsh and Badia (1975) revealed that preference for predictability increased as shock intensity increased. Subjects were tested in repeated sessions with inescapable shock delivered on a variable-time schedule, using a changeover procedure. Shock intensity was varied within a range of 0.15 to 3.0 mA, with all subjects remaining at each intensity level for a minimum of three days. Signal and shock durations were 5 and 0.5 sec respectively. As mentioned previously, the findings suggested that strength of preference for signalled shock varied as a result of shock intensity. At the lowest intensity (i.e. 0.15, 0.20 and 0.30 mA), subjects revealed no clear preference. However, at higher shock intensities, subjects were spending significantly greater amount of time in the signalled condition. An explanation in terms of the original Preparatory Response Theory (Perkins, 1968), would suggest that the preparation effect should occur equally at all shock intensities. However, it may be (as suggested in Section 2.1) that preparation is a function of the strength of the

aversive event whereby stronger or more intense stimuli augment the preparation effect more than weaker ones. Indeed, this type of explanation would be favoured by the Preception Theory.

A series of experiments carried out by Badia and colleagues (e.g. Badia & Culbertson, 1972; Badia, Coker & Harsh, 1973; Badia, Culbertson & Harsh, 1973; Badia, Harsh & Coker, 1975; Badia, Harsh, Coker & Abbott, 1976; Harsh & Badia, 1975) are, however, the most difficult to reconcile with a preparatory explanation. These studies were carried out using an operant changeover procedure in which changeover responding was monitored during a fixed training phase.

Badia *et al.* (1976) carried out an experiment in order to investigate the differential effects of varying the dependability of the warning signal, in which the total number of signals was held constant at 180 whilst the number of shocks was varied parametrically between 180 and three. Thus, the probability of shock in the event of a signal varied, but the probability of shock in the absence of a signal always remained constant. The authors found that subjects changed to the predictable condition when a signal preceded shock 100% of the time, and when the probability of shock was reduced to less than 100%. Badia *et al.* suggest the dependability of a signal identifying a shock period was not as important as the signal indicating safety. In a follow-up experiment, Badia *et al.* (1976) reduced the dependability of the warning signal identifying the shock-free (safety) period. It appeared that preference for predictability varied as the dependability of the signal identifying safety varied. Findings similar to these have been reported by Safarjan and D'Amato (1978) and Arabian and Desiderato (1975). Arabian and Desiderato (1975), for example, found that signalled shock was preferred to unsignalled shock because, in the former condition, the absence of the pre-shock warning signal acted like a safety signal. Indeed, they concluded that " ... contrary to the preparatory response hypothesis, a warning stimulus is not a condition animals seek to maintain; rather, they seek to avoid it (other things being equal)" (Arabian & Desiderato, p. 195).

Although these conclusions are slightly tenuous, the work mentioned above did serve as justification for those researchers espousing a safety signal compared with preparatory explanation for the beneficial effects of predictability (e.g. Badia & Culbertson, 1972; Badia, Coker & Harsh, 1973; Badia, Culbertson & Harsh, 1973). For example,

Badia, Coker and Harsh (1973) and Badia, Culbertson and Harsh (1973) examined the strength of the supposed preparatory mechanism by testing whether preference for predictability could be maintained even at the cost of a more aversive stimulus.

Unsignalled, inescapable shocks were presented to rats. By pressing a lever, subjects could change to a signalled schedule for 3 min. After a minimum of three 6 hour sessions, higher values of signalled shock intensity or duration were introduced. During Experiment 1, the duration of signalled shock was increased in increments of 0.5 sec. In the second experiment, the intensity of signalled shock was increased in increments of either 0.2 or 0.4 mA. Subjects in Experiment 1 all chose signalled over unsignalled shock at a high level, ranging from 70 to 96% when shock duration was 0.5 sec. When unsignalled shock duration remained at 0.5 sec while signalled shock increased, four subjects continued changing over for the first 3 increments i.e. up to 2.0 sec. At this point, the rate of changeover responding for one subject dropped to 50% whereas, the remaining three subjects continued changing over, at the same levels, up until shock duration was 2.5 sec. Following this, additional 1 sec increments were added. One subject decreased responding at 3.5 sec to about 60% whereas, the other two subjects continued responding at high levels (61-95%) with a signalled shock duration of 4.5 sec. This duration of shock represents nine times the duration of unsignalled shock.

During Experiment 2, the shock intensity was systematically varied revealing similar results to those of Experiment 1. Subjects in the unsignalled condition received shock intensity of 1.0 mA throughout the experiment compared with signalled shock which increased in increments of 0.2 or 0.4 mA. Changing over to the signalled shock schedule continued at a high rate (58 to 99%) for all subjects until shock intensity reached a level of 1.8 or 2.0 mA i.e. approximately twice that of unsignalled shock. However, additional increments in shock intensity produced varied results for different subjects. For some subjects responding dramatically reduced whereas, for others responding continued at a high rate (90%) at levels of 3.0 mA. These results suggest that subjects preferred the signalled condition over the unsignalled one when shock intensity was two to three times more intense. Although these findings could merely be indicating how effective subject's preparatory responses were, it has been suggested (e.g. Badia, Harsh & Abbott, 1979) that

it is unlikely that a preparatory response of the degree of effectiveness required to explain the latter data could be found.

However, several findings also appear difficult to accommodate within the alternative safety analysis. One of these concerns warning signal duration. Numerous studies have reported that nonhuman subjects prefer longer over shorter signal durations (Abbott & Badia, 1979; French *et al.*, 1972; Harsh & Badia, 1976; Perkins *et al.*, 1966). In terms of a Safety Signal explanation, it has been suggested that short signals are easily discriminable allowing shock and shock-free periods to be identified, therefore, stronger preferences should develop with these signal durations rather than with the longer length signals. Thus, it has been concluded that discriminable shock-free periods cannot be a sufficient condition for preference (e.g. Harsh & Badia, 1976). However, it may equally be that longer length signals allow safe and danger periods to be more easily identified leading to greater preference for these types of signals. A conclusion substantiated by the empirical data. Nevertheless, further evidence against a safety analysis comes from those studies investigating the dependability of the stimuli identifying shock and shock-free periods, in which the authors suggest that preference for predictable conditions can be maintained even when a number of unpredictable shocks are given during the shock-free (safe) period. Indeed, only when the dependability of safety was reduced to a low level was preference eliminated (Badia *et al.*, 1976 detailed earlier). These results would appear to be difficult to reconcile with the safety signal view because the shock-free period was only partially safe, not totally safe.

Clearly, from a review of the literature, evidence for and against both the preparatory activity and safety signal analyses exists, most of which can be used interchangeably depending on which theoretical interpretation is preferred by any particular researcher. However, irrespective of whatever theory is applied to the literature, the data does suggest that subjects preferred predictable over unpredictable situations whether the shocks were avoidable, escapable or inescapable (Badia & Culbertson, 1972; Badia, Culbertson & Lewis, 1971) or even when they received either longer, stronger, or more frequent shocks under the predictable condition (Badia, Coker & Harsh, 1973; Badia, Culbertson & Harsh, 1973). In spite of these findings, however, one major criticism which has dominated the preference for predictability literature is that of shock

modification. It has been argued, for example, that behavioural preference only occurs because subjects are able to partially avoid or minimise the aversiveness of the shock by postural adjustments. This may especially be true for past experiments in which unscrambled shock was delivered through the grid floor (e.g. Lockard, 1963; Perkins, Levis & Seymann, 1963; Perkins, Seymann, Levis & Spencer, 1966). It is plausible, therefore, that these postural adjustments are more effectively executed when signals precede shock than when signals are absent. If this is so, then subjects receive less severe shocks in the predictable condition than they do in the unpredictable condition.

According to Biederman and Furedy (1973, 1976), differential modification of shock accounts entirely for the observed preference for the predictable condition. Biederman and Furedy (1973) investigated the effects of shock modifiability when shock was unscrambled (modifiable), scrambled or through fixed (tail) electrodes (unmodifiable). In Experiment 1, subjects were allocated to one of three groups in which shocks were delivered through unscrambled foot grids, scrambled foot grids or fixed tail electrodes. During the testing phase subjects received 50 trials. Each trial consisted of a shock the offset of which was contingent on pressing either of two experimental levers (i.e. escapable shock). This was followed by, a series of five shocks with fixed duration of 5 sec: i.e. unaffected by lever pressing. An attempted escape response produced a 5 sec noise signal before each of the inescapable shocks during that trial. If subjects used the other bar, no signal was provided for that trial. Thus, bar preference determined whether the inescapable shocks would be signalled or unsignalled. In order to maximise discriminability between the escapable and inescapable shocks, the escapable shocks were 0.8 mA while the latter were 1.0 mA.

The results revealed that only those subjects in the unscrambled group significantly preferred the signal producing bar. There were no significant differences between predictable and unpredictable conditions involving either the scrambled or fixed shock groups. Biederman and Furedy then went on to compute correlations between signalled modification and the preference for predictability data for each individual subject across the three groups. Shock modification was judged by examining each individual subject's milliammeter recordings and the percentage of departures from an average waveform. The results for the unscrambled group revealed a weak, but significant, positive correlation.



Scrambled and direct shock produced no significant correlation between shock modification and preference for predictability. Biederman and Furedy concluded from these findings - that preference for the signalled side only reached significance in the group where shocks were delivered through unscrambled shock grids - that behavioural preference could be accounted for in terms of a modification effect. It was argued that the observed correlation between modification and preference provided further support for this interpretation.

It would appear, therefore, that modifiability may play an important role in barpress studies involving a changeover choice procedure. Furedy and Biederman (1976, Experiment 3) also assessed the differential effects of modifiability using a shuttlebox procedure, which provided much of the past data (e.g. Lockard, 1963; Perkins *et al.*, 1963). The results revealed a marked preference for the signalled side (e.g. 73%) in the unscrambled condition, but not in the scrambled (e.g. 59%). A result consistent with a shock modification explanation. In addition, during a second experiment carried out by the same authors, an assessment of shock modification on an individual-subject, within-trials basis revealed no significant preference for the signal in those subjects who showed no modification whereas, subjects extensively modifying shock showed a significant preference for the warning signal. Furedy and Biederman concluded that behavioural preference emerges "... when animals develop the capacity for external and unauthorised shock modification by spending an appreciable time on grids of the same polarity during the delivery of supposedly inescapable shock" (Furedy & Biederman, p. 5).

Against this, there are many studies which have found a strong preference for the predictable condition when shock was both scrambled, delivered through surface electrodes (e.g. Badia & Culbertson, 1972; Harsh & Badia, 1975, 1976; Miller, Daniel & Berk, 1974; Miller, Marlin & Berk, 1977) or through implanted electrodes (Griffin, Honaker, Jones & Pynes, 1974). These studies suggest therefore, that preference for predictable shock does not depend on overt modification of shock.

Furthermore, these results have been replicated and extended by Badia and Abbott (1980, Experiment 1) who examined whether differential modification occurred between predictable and unpredictable conditions. The apparatus was identical to that used in previous experiments (e.g. Badia, Culbertson & Harsh, 1973). They employed a

changeover choice procedure in which the signalled schedule consisted of a 5 sec tone warning signal which terminated with shock onset. In the unsignalled condition, no signals were presented. All subjects received two sessions of signalled followed by, two of unsignalled shock. The duration of shock received under the signalled and unsignalled conditions was nearly identical across both training sessions.

During the first session, the signalled condition was preferred. However, no differential trends in the duration of grid contact time appeared even after as many as 60 hours of shock occurring 30 times per hour. Badia and Abbott concluded that no differential modification of shock occurred, although the figures did reveal some degree of reduction in absolute contact time. For example, while 100 msec shock duration was typical, two subjects reduced contact time to an average of 60 msec. This appeared to be due to subjects running and leaping during shock. However, similar behaviour occurred during both signalled and unsignalled shock.

It would appear, from the results of both Badia and Abbott and those of numerous other studies mentioned above, that preference for the predictable condition does occur despite the absence of differential shock modification. The unusual findings of Furedy and Biederman are examined by Badia and Harsh (1977), centering on certain experimental parameters chosen by the authors, including, signal duration (3 sec), amount of training (none), length of testing phase (two, 3 hour sessions) and inter-shock interval (45 sec). Many of these factors have been demonstrated in the past (Badia & Culbertson, 1972; Perkins *et al.*, 1966) to weaken preference behaviour.

Further methodological difficulties were also pointed to by Biederman and Furedy (1976). These involved the operant changeover procedures used during a majority of the studies mentioned above. Namely, subjects were given the opportunity to change from unsignalled to signalled shock schedules but were not allowed to change from signalled to unsignalled. According to Biederman and Furedy, this results in an "asymmetrical design" whereby the responses produced during the signalled and unsignalled conditions are significantly different. Although Harsh (1978), in a wide ranging critical review of Biederman and Furedy's work, states that they have simply described the experimental conditions of Badia and Culbertson's (1972) study during which subjects could only

change from unsignalled to signalled shock schedules, and not *vice versa*. However, use of the changeover procedure does not require that one schedule as opposed to the other be selected as the imposed schedule. Indeed, subjects have been given the opportunity to change in both directions (e.g. Badia *et al.*, 1975; Safarjan & D'Amato, 1977), which in turn leads to what Biederman and Furedy term a "symmetrical" design. Moreover, both of these studies confirmed and extended the findings of Badia and Culbertson (1972).

In summary, a review of the animal literature suggests that rats, pigeons (Griffin, Honaker, Jones & Pynes, 1974) and fish (Fisher & Badia, 1975) generally prefer predictable to unpredictable aversive events. However, there has been some inconsistency as to the precise experimental parameters which are most crucial in determining the *strength* of preference in aversive situations. Furthermore, the various methodological problems associated with shock modification have not always been recognised.

3.2.2. Human Studies. There have been a variety of studies with human subjects investigating the behavioural preference for predictability. However, despite the generally held view that people will most often choose information about when an aversive event will occur, a review of the literature points to only a handful of reports supporting the beneficial effects of predictability (e.g. Badia, Suter & Lewis, 1967; Katz, 1984; Lanzetta & Driscoll, 1966; Lykken & Telling, 1974).

One of the first studies to support this view was reported by Lanzetta and Driscoll (1966) in an experiment investigating subjects choice of information (i.e. predictable) or no information (i.e. unpredictable) where outcomes were uncertain (shock-no shock, reward-no reward, shock-reward) and unavoidable. During each trial, subjects were presented with an amber pre-warning light which indicated when they could press one of two buttons labelled "information" or "no information". By pressing the button labelled information, subjects received a warning signal indicating whether or not shock would be presented. Pressing the no information button, led to a 10 sec period (with no signal) before shock onset. All subjects received a shock duration of 0.5 sec and at an intensity level set by the subject beforehand as "annoying".

Subjects demonstrated a significant preference for information in all three outcome conditions. However, there were large and consistent individual differences in preference

behaviour. In addition, Lanzetta and Driscoll's experiment was not devised to test for a preference *per se*. Rather, subjects were instructed to indicate whether they would like information as to whether or not a 10 sec interval would be followed by an unavoidable shock and were forced to push a button marked "information" or "no information". Furedy and Doob (1972) argued that it is conceivable that given such an alternative, subjects tended to press the information button simply because they thought it was the most appropriate response in the situation. The experiment also confounded different types of predictability. For example, by choosing "information" subjects were gaining both *whether* and *when* information, whereas, by choosing "no information" subjects were receiving neither.

A similar study by Badia *et al.* (1967) in which subjects were asked, after a certain number of trials, whether they would prefer to receive additional trials with or without a warning signal revealed just over half (55%) of the subjects chose the signalled condition.

In a later study designed specifically to overcome methodologically problems such as, confounding of different types of predictability, Katz (1984) found that a substantial majority of people preferred predictable over unpredictable aversive events. Predictability was manipulated on a within-subject basis in which each subject received six predictable trials and six unpredictable trials in a random order. Shock intensity was set at a level determined as aversive by each individual subject. Two sets of ten lights were set in front of the subject, each numbered 1 to 10. One set of lights was blue, the other was amber. During the amber light trials, the amber lights lit up in a sequential order up until light number 8 when a 1 sec shock occurred (predictable condition). During the blue lights each light was lit up in a random order until light number 8 when shock occurred (unpredictable condition). Each light duration was 3 sec. After 12 trials, subjects were told that an additional six trials would follow and that they could choose whether they wanted blue or amber light trials.

Katz found that 64% of the subjects preferred additional trials of predictable shock compared with 36% preferring unpredictable trials, and this difference was significant. Nevertheless, there was a relatively substantial minority who preferred uncertainty. This conclusion is consistent with other studies which report that some subjects do prefer unpredictable events (e.g. Averill & Rosenn, 1972; Averill, O'Brien & deWitt, 1977).

The results of the studies mentioned above, all of which lend support to a behavioural preference for predictability, cannot distinguish between either preparatory or safety signal explanations to account for this preference. Furthermore, these explanations cannot be supported by the majority of experiments which have failed to demonstrate a clear preference for predictable over unpredictable aversive events, or, a reduction in the subjective impact of that event (Baltissen & Boucsein, 1986; Furedy, 1975; Furedy & Doob, 1972; Furedy & Klajner, 1972; Furedy, Fainstat, Kulin, Lasko & Nichols, 1972; Furedy & Ginsberg, 1973; Furedy, Katic, Klajner & Poulos, 1973; Kimmel, 1967; Klemp & Rodin, 1976; Lykken, Macindoe & Tellegen, 1972). In addition, some studies have found that signalling can sometimes even increase aversiveness (Bowers, 1971; Furedy & Doob, 1971). A comprehensive series of studies carried out by Furedy and co-workers are representative. In these, Furedy and colleagues varied the type of event (electric shock versus loud noise), its intensity (0.5-2.5 mA or 80-120 dB) and inter-stimulus intervals (0.5 to 5.0 sec). For example, Furedy and Doob (1971) carried out three experiments investigating the effects of shock intensity (0.5 to 2.5 mA), shock duration (0.3 to 2.0 sec) and inter-shock interval (up to 8 sec), and examined the differential effects on ratings of aversiveness (unpleasantness versus intensity) and preference behaviour (actual versus questionnaire). In terms of rated aversiveness, they found no difference between signalled and unsignalled conditions. During both of these experiments Furedy and Doob had used a single index of rated aversiveness; judged intensity. A second index, that of judged unpleasantness, was employed during Experiment 3 in order to replicate the previous findings. As before, signalling the shock failed to result in a reduction in rated aversiveness. On the contrary, there was a significant effect in the opposite direction; i.e. the signalled group rated the event as more unpleasant than the unsignalled group.

In terms of the preference data, Experiments 2 and 3 failed to find a significant difference between signalled and unsignalled conditions. However, the data collected during these experiments were based on questionnaires rather than actual behaviour. Experiment 4 assessed the actual preference of subjects and produced similar results.

In other studies carried out by Furedy and co-workers, it was demonstrated that a reliably significant preference for predictability did emerge (e.g. Furedy & Klajner, 1972; Furedy, Fainstat, Kulin, Lasko & Nichols, 1972), but that it was a function of stimulus

and timing parameters. That is, stronger intensities and shorter signal-shock intervals elicited preference to a greater degree. This is consistent with the nonhuman literature reviewed in the previous section, in which stronger preference emerged with high intensity shock and short inter-stimulus intervals (e.g. Perkins *et al.*, 1966).

Overall, neither the preparatory or safety signal explanations can account for the general finding that subjects show no clear preference for either predictable or unpredictable aversive events. Nor can they account for those studies which suggest that predictable aversive events are rated as more painful than unpredictable ones (e.g. Bowers, 1971). Bowers assessed pain ratings in a within-subject design, in which subjects received three conditions: Condition 1 involved subjects being told that they would be shocked 20 sec after white light offset (predictable); Condition 2, in which they were told that shock might or might not occur at any time during the 20 sec orange light duration (unpredictable) and Condition 3, in which the blue light signalled a 20 sec period in which light was never administered (non-shock). The mean pain ratings for Condition 1 were significantly higher than for Condition 2. A finding similar to that of Furedy and Doob (1971, Experiment 3) who found a slight but statistically reliable increase in perceived shock aversiveness (rated intensity) in those conditions preceded by a warning signal. It must be noted, however, that they had failed to find a statistical difference in rated intensity during two previous experiments. In addition, when subjects were asked to choose which condition they preferred, there was little difference between the signalled and unsignalled conditions when averaged across all three experiments. Indeed, the majority of subjects expressed no preference for either condition.

According to Katz (1984), methodological differences can offer an explanation for these discrepancies. He set out three possible difficulties. First, some past studies have investigated differing information (e.g. shock intensity and duration, warning signal duration, length of inter-stimulus intervals) in one experiment, thereby making it difficult to determine whether their results were attributable to predictability *per se* or even what factors were most relevant in determining subject's preference behaviour. That is, whether it was knowing exactly when the event would occur or knowing the exact nature of the aversive event. For example, experimenters have threatened electric shock but have never administered it (e.g. Miller, 1979; Petry & Desiderato, 1978). Temporal predictability has

also been confounded with predictability about what the event would be. For example, Furedy and Doob (1972) varied shock intensity unsystematically within subjects and within temporal predictability, making it impossible for subjects to predict shock intensity on each trial. Indeed, this is true for all of the studies reported in Table 2, with the exception of Katz's (1984) study in which subjects were provided with complete information about the intensity, duration and probability of the shock occurring. The only factor that was varied, therefore, was the actual timing of the shock. The results of which showed a clear preference for predictability.

Second, in other studies (e.g. Furedy, 1970, 1975; Kimmel, 1967; Klemp & Rodin, 1976), mild stimulus intensities have been employed. Thus, subjects may not have cared whether they did or did not have temporal information about an event. In addition, few researchers have considered the effects of individual differences in stimulus threshold levels. Indeed, there is abundant evidence suggesting that subjects have widely differing thresholds at which they find an event aversive (e.g. Bowers, 1971). Moreover, even Furedy (1975) has reported that preference only emerges at high intensities and that in other situations subjects may feel that the effort to discriminate between predictable and unpredictable conditions is "not worth it" (Furedy, p. 72). From the data reported by Furedy and other researchers it is impossible to determine if individual subjects found the level of shock to be aversive. One way of avoiding this problem is by using a work-up procedure whereby subjects choose intensities which are, for example, "highly annoying" (Peeke & Grings, 1968). Two studies have been conducted in which subjects have been motivated to accept their highest tolerance levels. The results of these two studies (Bowers, 1971; Katz, 1984), however, complicate the issue even further with one study (Katz, 1984) producing a clear preference for the signalled condition whereas, the second study (Bowers, 1971) pointed to signalled shock being rated as more painful than unsignalled shock.

Third, there are vast differences between studies in the way preference information was gathered. For example, most studies either employed a post-experimental questionnaire in which subjects had to indicate which condition they preferred (e.g. Furedy, Fainstat, Kulin, Lasko & Nichols, 1972; Furedy & Doob, 1972, Experiments 1 to 3), or, subjects had to rate the pre-shock portions of the trial on a scale ranging from "not

at all distressing" to "very distressing" (e.g. Bowers, 1971; Klemp & Rodin, 1976). Alternatively, two studies employed a situation where subjects were led to believe that they would experience additional trials in their preferred condition (Furedy & Doob, 1972, Experiment 4; Katz, 1984). The rationale behind the latter design is that if people knew that they were going to actually receive additional trials and had to choose one type or another, then their choices would be more representative of their actual behaviour. However, the findings of the two studies employing actual rather than expressed behaviour revealed opposite results, with Furedy and Doob (1972) finding no significant difference between signalled and unsignalled conditions compared with Katz (1984), who found that the majority of subjects preferred the signalled condition.

The variables influencing people's preference for predictable or unpredictable noxious events appear to be a function of both the experimental paradigm, and individual differences. No specific conclusions can be made based on the experimental data presented here. Nor can any theoretical perspective offer a coherent explanation of the inconsistent pattern of results reviewed in this section. The studies outlined in this section are summarised in Table 2, along with experimental parameters and rating procedures.

### 3.3. Physiological Consequences of Predictability

3.3.1. Nonhuman Studies. Given the reliability of preference for predictability, it would be expected that these conditions are also less physiologically stressful than unpredictable conditions. Indeed, there have been a variety of studies reporting that unpredictability may have debilitating physiological consequences; such as, greater stomach ulceration, loss of body weight, higher levels of plasma corticosterone and body temperature (e.g. Gliner, 1972; Mezinkis, Gliner & Shemberg, 1971; Price, 1972; Seligman, 1968; Seligman & Meyer, 1970; Simpson, Wilson, DiCara, Jarrett & Carroll, 1975; Weiss, 1970, 1971a).

Weiss (1970) carried out an extensive series of investigations into the effects of stressor predictability on a variety of physiological responses; including, stomach ulceration, plasma corticosterone concentration, body temperature, food and water intake and body weight changes. Each of the four experiments in the series employed essentially



Table 2. Examples of human studies investigating choice and subjective ratings between predictable and unpredictable aversive stimuli.

<u>Study</u>	<u>Rating Procedure</u>	<u>Parameters</u>	<u>Results</u>
Lanzetta & Driscoll (1966)	Preference: pressing a button	Signal 5 sec; Shock .5 sec, intensity varied; ISI 10 sec	Subjects preferred the information condition
Kimmel (1967)	Intensity: rotating lever after shock offset	Signal 1, .3 or 5 sec; Shock 1 sec, 2.5mA; ISI 5 sec	Short signal; greater with UP Long signal; greater with P
Furedy (1970)	Intensity: rotating lever after shock offset	Signal 3 sec; Shock .3 sec, 0.5-2.5mA; ISI 3 or 5 sec	0.5mA; greater with P Higher intensity; no difference
Bowers (1971)	Pain: rating from 1 to 5 after shock offset	Signal varied; Shock 2-3 msec; 1.5-7.8mA; ISI 4 to 19 sec	7 out of 8 subjects rated P shock as more painful
Furedy & Doob (1971)	Intensity: rotating lever after shock offset Preference: questionnaire	Signal 3 sec; Shock .3 sec, 1.0 or 2.5mA; ISI 5 sec	Intensity: no diff (Expt 1&2), P greater (Expt 3) Preference: P greater (Expt 1), no difference (Expt 2 & 3)
Furedy & Doob (1972)	Intensity: rotating lever after shock offset Preference: expressed (Expt 1-3), actual (Expt 4)	Signal 3 sec; Shock .3 sec, 0.5-2.5mA; ISI 5 sec	Intensity: as above Preference: no diff (Expt 1-4)

Furedy & Klajner (1972)	Intensity: rotating lever after shock offset Preference: questionnaire	Signal .5 sec; Shock .3 sec, 1.0 or 2.5mA; ISI 0 sec	Intensity: no difference Preference: no difference
Lykken, Macindoe & Tellegen (1972)	Intensity: rating from 0 to 20 after shock offset	Signal 1 sec; Shock intensity & duration varied; ISI 5 sec	No difference between groups
Furedy & Ginsberg (1973)	Intensity: verbal after a series of 5 shocks Preference: questionnaire	Signal .5 sec; Shock .3 sec, 1.0 or 2.5mA; VI 20 sec	Intensity: no difference Preference: no difference
Klemp & Rodin (1976)	Distress: rating 1 to 8 after shock offset Intensity: rating 1 to 8 after shock offset	Shock 3.0mA; VT 5 or 20 sec	Distress: greater in 20 sec gp Intensity: no difference
Katz (1984)	Distress: rating 1 to 9 after shock offset Aversion: rating 1 to 9 after shock offset Preference: actual	Signal varied; shock 1 sec, 3.5 to 8mA; ISI 0 sec.	Distress/Aversion: greater for UP Preference: 64% chose P

Note: VT=variable time; VI=variable interval; P=predictable; UP=unpredictable; ISI=interstimulus interval.

the same apparatus which included three compartments in which a rat was lightly restrained. Shock was delivered through fixed tail electrodes, so that no differential modification of shock could occur (see Section 3.2.1). Each subject was randomly assigned to a triplet, all matched for body weight. In each of these triplets, one subject was randomly selected as the non-shock control, and never received shock. The other two subjects received shock (3.5 mA and 2 sec duration) on a variable schedule with an average inter-shock interval of 60 sec. The tail electrodes for the two shock subjects were wired in series, so that shock received by these subjects was of exactly the same intensity and duration. One shock subject was assigned to the predictable group, receiving a tone signal that began 10 sec before shock onset. The other shock subject received the same signal, but this occurred randomly with respect to shock (unpredictable group). Shock for both predictable and unpredictable groups was inescapable and unavoidable.

The first experiment in the series examined the effects of shock predictability on the development of stomach ulceration. Experimental session duration was 19 hours. Data relating to three measures of stomach ulceration was collected; the percentage of subjects showing gastric lesions; the mean number of lesions and, the mean total length of lesions. There were significantly more subjects with gastric lesions in the unpredictable compared with non-shock group whereas, there was no significant difference between the non-shock and predictable groups. The unpredictable group had more animals with lesions compared with the predictable group, but this did not reach significance. The mean number of lesions was significantly greater in the unpredictable than either the predictable or non-shock groups and there was a significant difference between the latter two groups. Finally, the unpredictable group showed significantly longer lesions than the predictable and non-shock groups, which again, significantly differed from one another. The stomachs of each triplet were also compared for the extent of ulceration, ranging from a rank of 1 (least ulceration) to 3 (most ulceration). The results revealed that in every case the unpredictable groups differed significantly from both the predictable group and the non-shock group, while the difference between the predictable and non-shock groups did not reach significance. Weiss concluded, that animals which received unpredictable shock developed markedly more ulceration than animals which received either the same shock preceded by a signal or no shock. However, animals for whom shock was predictable did not differ significantly from

the non-shock control subjects, with the exception of results relating to the mean length of lesions, in which the predictable group produced longer lesions than the non-shock subjects.

During Experiment 2, two other somatic responses were examined; mean change in body temperature and concentration of plasma corticosterone. The apparatus and procedure were otherwise identical to Experiment 1. Body temperature was recorded at one hourly intervals for the first 5 hours of the session. Corticosterone concentration was determined at the end of the session which lasted either five or 24 hours. After one hour of the session, both the predictable and unpredictable groups produced a marked increase in body temperature compared with the non-shock group. The unpredictable group showed a greater increase in temperature compared with the predictable group, although this only approached significance. However, this difference increased during the second hour, as the temperature of the predictable group decreased while that of the unpredictable group continued to rise. By the end of the second hour, the difference was significant and remained so at the end of the third, fourth and fifth hours. However, from the end of the second hour the body temperature of the unpredictable group started to decrease at a similar rate to that of the predictable group. Those ten triplets who remained in the experimental session for 24 hours revealed no significant difference between the predictable and unpredictable groups by the end of the session, although both shock groups showed a greater level of body temperature than the non-shock controls.

Concentration of plasma corticosterone revealed a significant difference between the three groups after five hours, with both predictable and unpredictable groups showing a significantly greater concentration of corticosterone than the non-shock group. The unpredictable group also had significantly higher levels than the predictable group. At 24 hours, the unpredictable group still showed significantly higher steroid levels than the predictable group whereas, there was no significant difference between the two shocked groups and the non-shock controls. Although the difference between the unpredictable and non-shock groups approached significance.

During Experiment 3, Weiss examined differential effects on changes in body weight. A dependent variable often used in past studies, producing differential effects as a function of predictability. Apparatus and procedures were the same as Experiment 1 with

the exception that prior to the experimental session, all animals were housed in individual cages where water and food intake was measured daily. Again, the three subjects in each triplet were matched for body weight and also for weight gain across a period of at least one week's duration. The experimental session was then conducted as in Experiment 1. Body temperature was recorded as in Experiment 2. The session lasted five hours, after which the animals were weighed and returned to their individual cages. Body weight, food and water measures were obtained every 24 hours thereafter. Body weight was expressed as a mean change from pre-experimental body weight, and was measured before and after five hours and at five, 24 hour intervals thereafter. During the experimental session, unpredictable subjects lost significantly more body weight than both the predictable and non-shock groups. The weight loss of predictable subjects was only slightly greater than that of non-shock subjects. During the first 24 hours following the session, both shocked groups lost weight, although the unpredictable groups lost more than the predictable group. This difference did not reach significance. At the end of the first 24 hours after the session, and at every 24 hours thereafter, the weight loss remained significantly greater for the unpredictable group than for the predictable group. However, weight loss did steadily decrease for both groups during the five, 24 hour periods.

Weiss also measured food and water intake changes during Experiment 3 across the five, 24 hour periods, with the three days prior to the experimental session used as a baseline. After the first 24 hour period, the unpredictable group showed significantly greater depression of food intake than both the predictable and non-shock groups. Intake for the predictable subjects was also significantly more depressed than that of the non-shock group. These differences declined over subsequent 24 hour measurement periods. The results of differential water intake revealed relatively similar results, with all subjects showing a depression in water intake after the first 24 hour session. The intake of the unpredictable subjects was significantly more depressed than that of the non-shock group whereas, there was little difference between the two shock groups. After the second 24 hour period, the unpredictable group revealed a slight depression in water intake compared with both the predictable and non-shock groups for whom water intake was similar to baseline levels. The difference between unpredictable and predictable groups, and between the unpredictable and non-shock subjects reached significance whereas, the predictable and

non-shock groups had similar intake levels. It would appear then, that the non-shock control group ate and drank considerably more than the unpredictable subjects during the two days after experimental stress, but in comparison to predictable subjects, differences were found in relation to food intake only.

Body temperature was also examined during Experiment 3, and in general, the results of Experiment 2 were replicated.

The results of Experiment 3 were not in accordance with those of earlier studies using body weight as a dependent variable, all of which found decreased body weight in the signalled condition (e.g. Brady, Thornton & deFisher, 1962; Friedman & Ader, 1965; Pare, 1964). One notable difference between the methodology of Experiment 3 and that of past studies was that animals were mildly restrained in Experiment 3 whereas, they were free-moving in all previous studies. Experiment 4, therefore, assessed the differential effects of predictability on body weight in free-moving animals, with fixed tail electrodes. The procedure was the same as in Experiment 3, except that animals were placed in eight by eight by eight inch boxes. The warning signal was a blinking light instead of a tone as used in Experiments 1 to 3. Signal duration was still 10 sec. Two experimental sessions were given each three hours in duration and 48 hours apart. Body weight loss for each of the groups was assessed after each session. In general, the results of Weiss' first three experiments were replicated.

The findings of Weiss' series of experiments indicate that a situation in which shock is not preceded by a warning signal is more physiologically debilitating than when a warning signal precedes shock. This finding was found to varying degrees across the various dependent measures used. Unfortunately, Weiss' experiments and the vast majority of other studies showing support for the beneficial effects of predictability, do not permit a choice between theoretical explanations in terms of preparatory activity or safety signals. For example, does predictable shock reduce shock impact because the warning signal allows the animal to know just when the aversive event will occur, or, is it reduced because the animal knows when the shock will not occur i.e. when it is safe? Sawrey (1961), for example, found that animals receiving light followed by shock 100% of the time, and buzzer never followed by shock, formed fewer gastric ulcers than did animals that received half of the lights and half of the buzzers followed by shock. The safety signal

analysis predicts that for the 100% group, the buzzer indicates a shock-free (safety) period because it is never followed by shock. For the 50% group, neither the buzzer nor the light are safety signals. The 50% group therefore spends more total time in a state of fear and thus forms a greater amount of stomach ulcers.

Similarly, the safety analysis argues that the situation in which delayed shock occurs causes subjects to be in fear for a longer period of time than does the immediate shock situation because the timing of the aversive event is not accurate. For example, if a brief shock is delivered immediately on entering the experimental chamber, and the subject is released 45 sec later (Knapp, Kause & Perkins, 1959), it receives a shock followed by 45 sec shock-free (safe) period. However, if the shock is delayed for 45 sec, subjects receive 45 sec of shock (danger) period, followed by shock. So the rats chooses the chamber in which it is fearful less of the time.

However, the preparatory activity theories can also explain these results, because in Sawrey's experiment it is assumed that the 100% condition allows an animal to accurately prepare for the impact of the aversive event whereas, the 50% condition is unpredictable so preparation cannot be made at the appropriate time. In terms of the results found by Knapp *et al.*, the preparatory theories assume that a precisely timed preparatory response (i.e. the immediate condition) is more effective than in a delayed condition.

One study carried out by Weiss (1970) attempted to separate these two alternatives by exposing three groups of rats to intermittent shock for 48 hours. In the first condition, a high pitched tone preceded shock by 20 sec with three minutes of silence between each tone-shock trial (signalled). In the second condition, shock was also preceded by the beeping tone, but the three minute period was divided into six, 30 sec periods. Silence occurred for the first 30 sec after which, a series of five steady tones were introduced, each lasting for 30 sec and each increasing in pitch and intensity with respect to the previous tone (progressive-signalled). In the third condition, no stimulus of any kind preceded shock (unsignalled). Therefore, in the progressive-signal group the steady tones provide an external clock, so it was assumed that this group had better information than those animals in the first group as to when the stressor would occur. This condition should therefore be less pathogenic if predictability is beneficial because it allows the organism to know exactly when the stressor will occur and therefore improves the animal's ability to prepare for it.

Alternatively, the external-clock condition provides less 'safe-time', as it presents an aversive stimulus (the warning signal) for over three quarters of the inter-stimulus interval. Therefore, the single tone should be more beneficial if the benefits of predictability are due to the presence of signals indicating safety.

The effect of these conditions was examined upon development of stomach ulceration, corticosterone concentration levels and body weight loss. In terms of the mean number of gastric ulcers, animals in the no-signal condition developed more ulceration than they did in either the signal or progressive-signal conditions. However, when the two signalled conditions were compared separately, it appeared that the progressive-signal condition developed greater stomach ulceration than the signal group, suggesting that safety is the more important factor in reducing stress. Findings later confirmed by Tsuda, Tanaka, Hirai and Pare (1983). Furthermore, Caul, Buchanan and Hays (1972), found that extent of stomach pathology was directly proportional to the amount of time animals were in the presence of the stimuli associated with the stressor. It must be noted, however, that Weiss (1970) found no differences between the signal and progressive-signal conditions in terms of the amount of body weight loss and levels of plasma corticosterone. Although both measures were greatest in the no-signal condition compared with the signal and progressive signal conditions.

Unfortunately, there are a number of studies which found increased physiological activation, such as, greater ulcer development (Tsuda & Hirai, 1976) and a decrease in body weight and higher mortality rates (Brady, Thornton & deFisher, 1962; Friedman & Ader, 1965; Pare, 1964) in predictable compared with unpredictable conditions. All of which cannot be explained by either preparatory or safety signal theories.

Pare (1964), for example, ran subjects in seven by eight by seven inch boxes in which a 3 mA shock was preceded by a 5 sec tone in the predictable condition. Inter-trial intervals were 5 min, and the experimental session duration was 23 days, 22 hours daily. One methodological discrepancy must be noted however, that is during Pare's study subjects were allowed food and water throughout the experimental sessions. During Weiss' experiments animals had access to food and water only during one hour, daily feeding periods.



In terms of gastric ulceration, Pare found no significant differences between the groups. However, data relating to body weight, adrenal weight/body weight ratio and adrenal ascorbic acid concentrations did reveal differential effects as a function of predictability. Those subjects in the non-shock control group gained weight steadily over the experimental session, always showing a significant difference compared with the predictable and unpredictable conditions. All subjects in the two experimental groups initially lost weight during the one to three days after the start of the session, but increased in body weight up to five days after. During the five to 23 days after the onset of the experiment, the unpredictable subjects gained significantly more weight compared with the predictable group. However, these results should be considered in the light of differential consummatory rates, in which the predictable and unpredictable groups showed significantly less food consumption compared with the non-shock control. There was no significant difference between the experimental groups. It has been noted, that food deprivation and shock produce greater development of ulceration than either condition alone (Farren, 1961) (discussed in detail later).

In terms of the adrenal weight/body weight ratio and adrenal ascorbic acid concentrations, those subjects in the predictable condition had both a significantly greater ratio and greater ascorbic acid concentrations compared with the unpredictable and non-shock control subjects. Those subjects in the unpredictable group did not differ significantly from the control condition. The type of adrenal hypertrophy seen in the predictable subjects has repeatedly been associated with a stress response (e.g. Pare, 1962; Selye, 1949). In addition, increases in concentration of ascorbic acid in the predictable condition is in agreement with others (e.g. Brodish & Long, 1960; Katsh, Katsh & Osher, 1954) who observed increases in ascorbic acid concentration in subjects exposed to prolonged stress.

Weiss (1970), suggested that one possible explanation for the discrepancy between his experiments and those carried out by Pare and others, could be in terms of differences in methodology. For example, during all three of the contradictory studies mentioned above, shock was delivered through the grid floor whereas, during Weiss' experiments shock was delivered through fixed tail electrodes. Weiss suggested that the differential effects of predictability found across these experiments are due to attempted modification of

shock through postural adjustments when shock is delivered through the grid floor. Indeed, he suggests that these changes in posture are in fact coping attempts which, necessarily, are inefficient since the shock is inescapable. He points to previous results (Weiss, 1968) in which coping responses, if inefficient, produce a greater physiological response than if no coping responses are attempted. If the shock is preceded by a warning signal then it may permit the animal to initiate the responses mentioned above, to a greater extent than animals in the unsignalled group. Therefore, in those studies delivering shock through the grid floor, the predictable-unpredictable manipulation may be confounded by inefficient coping responses which, in turn, may increase physiological responding. In contrast, with the use of fixed tail electrodes the shock is consistent in duration and intensity across trials, therefore, the animal learns that any behaviour is ineffective in changing shock and all coping responses stop. Thus, the experimental manipulation is not confounded by any modification of shock.

A second explanation to account for the discrepancies between studies was suggested by Abbott, Schoen and Badia (1984) in a wide ranging review of the literature. In general, they suggested that there were several reasons for these discrepancies involving differences between types of design and procedures. In particular, differences in the intensity, duration and frequency of shock and in shock schedules, method of shock delivery and the number and duration of experimental sessions. However, when studies were grouped in relation to the physiological measures they employed, four salient features emerged: (i) length of the experiment (short or long term), (ii) the severity of shock parameters, (iii) the method of shock delivery and (iv) the availability of food. Abbott *et al.* examined each of these factors in relation to several physiological indices; namely, weight loss, gastrointestinal pathology (gastric acid secretion and stomach lesions) and pituitary-adrenal functioning (adrenal weight and plasma corticosterone concentration).

In terms of experimental length, studies were divided into two categories. First, there were long-term experiments in which subjects spent 15 to 24 hours in the apparatus during a session, with repeated sessions over a number of weeks. Conversely, there were short-term experiments of one long (i.e. 24 hour) or a few short (i.e. 5 hours or less) sessions. In terms of body weight, Abbott *et al.* assessed five long-term studies (Brady, Thornton & DeFisher, 1962; Friedman & Ader, 1965; Friedman, Ader &

Glasgow, 1965; Pare, 1964, 1965), all of which delivered shock to the grid floor and three short-term experiments (Gliner, 1972; Price, 1972; Weiss, 1970, Experiment 3) in which shock was delivered through tail electrodes.

Abbott *et al.* concluded that the contradictory findings of weight loss studies were in part related to the length of the experiment. In terms of short-term studies, weight loss either did not differentiate between predictable or unpredictable shock schedules (Gliner, 1972; Price, 1972) or was greater under the unpredictable condition (Weiss, 1970). Among the long-term studies weight loss was consistently greater under the predictable shock schedule. Nevertheless, as Abbott *et al.* repeatedly point out, these results do not mean that the length of the experiment *per se* was the most important factor, as there were also differences in the locus of shock delivery with the long-term studies delivering shock through the grid floor compared with tail shock in the short-term studies. There were also differences in the degree of restraint used and the availability of food. For example, the long-term studies used unrestrained subjects where food was available whereas, subjects in the short-term studies were restrained and food was unavailable. Food availability may play an important role in the differing outcomes between long and short term studies, but cannot offer a complete explanation. For example, where subjects have no access to food, weight loss differences can arise only through differential respiration, urination and defecation. Where food is available, differences can also arise from differential eating. Weight loss may therefore, arise from different behavioural responses to stress under the predictable and unpredictable conditions. However, in short-term studies where suppression of food responding has been measured (e.g. Seligman, 1968), more suppression has been found under unpredictable conditions. Weight loss findings from long-term studies indicate more suppression of eating under predictable conditions (e.g. Pare, 1964).

In terms of stomach ulceration the long-term studies are inconclusive due to methodological problems (Pare, 1964; Sawrey, 1961). In both, unscrambled shock was delivered through the grid floor, subjects were free-moving and food was available. Sawrey (1961) assigned subjects to either of two groups: predictable, in which a 5 sec light or buzzer occurred on a FT 150 sec schedule. Shock followed the light but never the buzzer. In the unpredictable group, the light and buzzer received identical presentations to

the predictable group. However, during half of the presentations shock followed the light and in the other half shock followed the buzzer. Sawrey found that those subjects who received the unpredictable schedule developed more ulcers than those subjects in the predictable group. However, during the unpredictable schedule shock occurred following signals and, was more probable than after no signal. Hence the condition was not truly unpredictable. Pare (1964), using truly predictable and unpredictable conditions, found that there was only limited ulcer development in both predictable and unpredictable subjects i.e. two out of eight subjects in the unpredictable condition developed one ulcer each, essentially the same as in the predictable group. These data are consistent with those of Pare (1972) in which rats became resistant to stomach ulceration development over the course of the experimental session.

Abbott *et al.* reviewed ten short-term studies, three of which used repeated short sessions and grid shock (Gliner, 1972; Seligman, 1968; Seligman & Meyer, 1970). All remaining studies employed one single session with tail shock (Caul, Buchanan & Hays, 1972; Guile & McCutcheon, 1984; Mezinkis, Gliner & Shemberg, 1971; Price, 1972; Weiss, 1970, Experiment 1) or both tail and grid shock in separate groups (Tsuda & Hirai, 1976). The majority of these studies found that ulcer development was greatest in the unpredictable condition compared with the predictable condition, with the exception of two studies (Price, 1972, Experiment 1; Tsuda & Hirai, 1976). Price (1972, Experiment 1) used a similar design to that of Weiss (1970), but with unrestrained subjects. In the predictable group subjects received a 8 sec light prior to shock compared with the unpredictable subjects who received the warning signal after shock, and the non-shock controls who received no shock. There were no significant differences in ulceration between those subjects receiving either a predictable or unpredictable schedule. However, when Price (1972, Experiment 2) tested additional subjects using an identical procedure but with subjects which were restrained, he found that five out of ten subjects in the unpredictable group developed ulcers. This was compared to one out of ten subjects in the predictable group and three out of ten non-shock controls. Price concluded that when shock is preceded by a warning signal it leads to less physiological stress than when the warning signal follows termination of the shock, but only in situations where subjects are restrained.

Tsuda and Hirai (1976) found similar results using tail and grid shock with restrained rats in three conditions: in Group 1 the probability of signals preceding shock was 1.0; in Group 2 the probability was 0.5 and in Group 3 the probability was 0. The results indicated that for tail shock conditions, those subjects in Groups 2 and 3 (the unpredictable subjects) developed more stomach ulceration than did subjects in Group 1 (predictable condition). However, subjects receiving unpredictable grid shock developed significantly fewer ulcers than those in the predictable grid shock group. There were no significant differences between the unpredictable tail and unpredictable grid shock groups, or, between the predictable tail and predictable grid shock groups. These findings are in line with the coping hypothesis suggested by Weiss (1970) (see Page 45), in which he suggests that shock delivered through the grid floor leads to the predictable group developing a greater number of ulcers compared with the unpredictable group. This is because in the former condition subjects are producing more physiologically harmful coping responses than subjects in the unpredictable condition. However, when shock is delivered through fixed tail electrodes the predictable-unpredictable manipulation is not confounded by levels of coping responses, and the supposedly beneficial effects of predictability, in terms of lower physiological responses, becomes apparent.

Abbott *et al.* concluded that these results taken as a whole point to the beneficial effects of predictability, with 12 out of the 17 studies they reviewed revealing a greater degree of stomach ulceration in the unpredictable condition. Among the five remaining studies, only the grid shock group in Tsuda and Hirai's (1976) experiment produced greater ulceration in the predictable group, the other studies found no differential ulceration between conditions. For studies that found no differential effects, Abbott *et al.* attributed this to a failure to provide experimental conditions that were severe enough. As with weight loss, the possibility that other factors such as shock parameters, locus of shock delivery or food availability playing an important role cannot be ruled out. For example, Mikhail (1980) has argued that ulceration is due to food deprivation *per se*, and that any difference in ulceration can be attributed to differences in eating, and not as a result of the action of psychological variables such as predictability. In support of this, other studies have found that stomach ulcers develop with food deprivation in the absence of shock stress (e.g. Mikhail, 1973) and are not always increased by the addition of shock (e.g.

Friedman & Ader, 1965). However, differential eating cannot account for differences in ulceration produced by predictable versus unpredictable shock where all subjects were deprived of food (e.g. Gliner, 1972; Guile & McCutcheon, 1984; Weiss, 1970).

A review of the literature involving pituitary-adrenal functioning revealed similar conflicting results to those of weight loss. For example, studies assessing the differential effects on adrenal weight found either no difference between predictable and unpredictable shock in the short-term studies (Friedman & Ader, 1965; Price, 1972) or greater adrenal hypertrophy in subjects receiving predictable shock in the long-term studies (Pare, 1964). Studies involving plasma corticosterone measures also revealed conflicting results, although all were classed as short-term (Bassett, Cairncross & King, 1973; Davis & Levine, 1982; Davis, Porter, Livingstone, Herrmann, MacFadden & Levine, 1977; Hennessy, King, McClure & Levine, 1977; Weiss, 1970, Experiment 2). In the first three studies, the investigators found higher levels of plasma corticosterone in those subjects receiving unpredictable schedules compared with the predictable subjects, whereas, the last two experiments revealed no significant differences between groups.

In summary, it would appear that the literature relating to predictability and physiological stress is relatively inconsistent as a whole. However, when studies are classed according to whether they are long (chronic) or short (acute) term the following conclusions can be drawn. First, during chronic exposure, predictable conditions produce a greater weight loss than unpredictable conditions. Among acute studies the opposite is true. In terms of gastric ulceration, the unpredictable conditions produce more severe ulceration than predictable conditions in short-term studies. Whereas, the only valid long-term study found no differential effects. No firm conclusions can be drawn from the data relating to steroid measures due to inconsistent findings. Thus, the data *generally* support the view that unpredictable aversive events are more debilitating than predictable ones in acute studies and less debilitating or even beneficial in chronic studies.

Abbott *et al.* suggest that this can be understood from a safety signal view. In predictable conditions, subjects can easily discriminate between shock (danger) and shock-free (safe) periods whereas, during unpredictable conditions these periods are not easily discriminable due to the absence of a warning signal. Abbott *et al.* suggest that predictable conditions generate periods of alternating arousal (during the signal) and relaxation (during

signal absence), which initially leads to reduced physiological arousal compared with unpredictable conditions. However, the authors go on to suggest that with long term exposure, " ... chronic physiological arousal may promote an adaptation, perhaps via opponent processes (Soloman & Corbitt, 1974). Such adaptation would be hampered under the phasic conditions of predictable stress because opponent processes would be aroused only during brief periods of signal presence" (Abbott *et al.*, p. 65). This explanation would account for the differential responding under short and long term conditions. During short term studies, chronic arousal is induced by unpredictable conditions whereas, the safe periods associated with predictable schedules would be expected to provide relief from physiological arousal, and thus render the predictable condition less physiological debilitating than the unpredictable condition. This explanation is consistent with those short term studies reporting greater weight loss, more severe ulceration and higher steroid levels in subjects experiencing unpredictable shock schedules. However, in long term studies, the phasic nature of predictable shock and the resultant inability to adapt would lead to an increase in the debilitating effects of the predictable condition. Again, this is consistent with findings from long term studies showing greater stomach ulceration and levels of gastric acid, and suppression of eating and resultant loss of body weight.

Unfortunately, Abbott *et al.*'s explanation relies on a hypothetical "adaptation" of physiological arousal via "opponent processes". To date, no study has been devised to investigate the interaction of such processes with predictability. Perhaps a more plausible explanation would be in terms of conditioned aversiveness (e.g. Mowrer, 1947), which suggests that the pairing of neutral stimuli (i.e. the warning stimuli) with an aversive stimuli, will, through classical conditioning, acquire unpleasant properties of the aversive event. Thus, organisms subjected to repeated or chronic exposure to predictable and unpredictable aversive stimuli, will show less distress and lowers levels of physiological arousal in the unpredictable condition than in the predictable one. However, this may not necessarily be the case with acute exposure, in which stimuli preceding the aversive event initially acquire positively reinforcing properties (see Schoenfeld, 1948).

Nevertheless, irrespective of interpretation, there are further discrepancies in the studies reviewed by Abbott *et al.*. In particular, those chronic studies which involved

'weaker' experimental manipulations (i.e. unrestrained animals), reported a reduction or elimination of the effects of predictability. This suggests therefore that the difference between acute and chronic exposure may not have been only due to the length of the experiment *per se*. Instead, other factors such as availability of food, method of shock delivery and restraint also have to be taken into consideration. Unfortunately, these variables have been confounded across experiments. It would appear from the literature, therefore, that several plausible classification schemes are possible as an explanation of the conflicting findings. A summary of study outcomes is shown in Table 3, including findings involving weight loss, gastric acid, stomach ulceration, adrenal weight and corticosterone concentration levels.

In summary, it appears that the nonhuman literature is inconsistent in relation to reduced physiological stress as a function of predictability. In part, this relates to the complexities of physiological response systems, in which no one physiological measure can be used as an index of the 'stress' response, and to other methodological and design difficulties.

**3.3.2. Human Studies.** Psychophysiological studies investigating predictability have invariably measured some physiological variable such as; Heart Rate (HR), Skin Conductance (SC) or plethysmographic digital Volume-Pulse Change (VPC) as an index of states such as 'arousal'. In the majority of cases, there is a lack of concordance between physiological and subjective (see Section 3.2.2) measures due to either, researchers assessing subjective states by employing no more than one verbal report (e.g. intensity or unpleasantness), or, assessing physiological stress as specific activity within one autonomic system only. As a result, the psychophysiological literature concerning human subjects appears as confused as the material dealt with in the preceding section in relation to nonhuman subject's physiological response patterns.

A large number of studies indicate that signalling an impending event leads to an increase in anticipatory arousal in terms of electrodermal levels (e.g. Miller, 1979; Monat, Averill & Lazarus, 1972), a higher frequency of non-specific electrodermal responses (e.g. Miller, 1979; Monat *et al.*, 1972) and increases in HR (e.g. Bowers, 1971 (a & b); Monat *et al.*, 1972). In contrast, a handful of studies have reported the opposite. That is,



Table 3. Summary of nonhuman study outcomes across long and short term experimental sessions (adapted from Abbott, Schoen & Badia, 1984, p. 62).

<u>Study</u>	<u>Physiological Measure</u>				
	<u>WL</u>	<u>GA</u>	<u>UL</u>	<u>AW</u>	<u>CL</u>
<u>Short-Term</u>					
Bassett, Cairncross & King (1973)					P
Caul, Buchanan & Hays (1972)			U		
Davis & Levine (1982)					P
Gliner (1972)	N		U		
Guile & McCutcheon (1984)			U		
Hennessy <i>et al.</i> (1977)					P/U
Mezinskis, Gliner & Shemberg (1971)			U		
Price (1972)					
Expt. 1 (unrestrained)	N		N		
Expt. 2 (restrained)	U		U	N	
Seligman (1968)			U		
Seligman & Meyer (1970)			U		
Simpson <i>et al.</i> (1975)				U	N
Tsuda & Hirai (1976)					
Tail Shock			U		
Grid Shock			P		
Weiss (1970)					
Expt. 1			U		
Expt. 2					U
Expt. 3	U				
<u>Long-Term</u>					
Brady, Thornton & DeFisher (1962)	P				
Friedman & Ader (1965)	P			N	
Friedman <i>et al.</i> (1965, Expt.3)		P			
Glavin & Mikhail (1976)					
Solid Diet			N		
Liquid Diet			U		
Mikhail (1971, Expt. 2)		P	N		
Pare (1964)	P		N	P	
Pare (1965)	P				
Pare & Isom (1975)		P			
Sawrey (1961)			U		

Note: P=positive effect of predictability; U=positive effect of unpredictability;  
N=nonsignificant; WL=weight loss; GA=gastric acid; UL=ulceration;  
AW=adrenal weight; CL=corticosterone level.

unpredictability produces an increase in autonomic arousal as measured by HR, electrodermal levels (e.g. Averill & Rosenn, 1972; Lykken, Macindoe & Tellegen, 1972; Price & Geer, 1972) and a higher frequency of non-specific electrodermal responses (e.g. Bowers, 1971 (b); Price & Geer, 1972). Yet other studies have reported no difference in electrodermal levels, non-specific electrodermal responses or VPC (e.g. Bowers, 1971 (a); Glass, Singer & Friedman, 1969; Katz, 1984).

The validity of some of this data (especially those studies which have reported decreased physiological activity in predictable conditions) has been questioned by many researchers, such as Furedy (1970), since the predictable and unpredictable conditions involved essentially different experimental stimuli i.e. smaller autonomic responses produced in the predictable condition could be due to response suppression. In some of the studies mentioned above (e.g. Lykken *et al.*, 1972), the warning signal in the predictable condition elicited a response, which did not fully recover, and may have acted to reduce the magnitude of the response produced by the aversive event.

Furedy and Klajner (1972) have reported a study in which they employed a complex secondary signalling procedure in order to minimise the effects of response interference (see also Furedy and Doob (1971) (see Section 3.2.2)). This included presenting subjects with a series of five, 3 sec shocks delivered on a variable interval schedule of 20 sec (range 10 to 30 sec). Shock was varied within subjects for two factors; intensity (1.0 or 2.5 mA) and signalling (presence or absence of a 0.5 sec tone). Each subject received the four types of shocks series presented randomly, and the nature of each imminent shock was indicated by one of four secondary-signal warning lights. The offset of the secondary warning light was followed within 10 to 20 sec by the first in the series of five shocks. Interest focussed on the physiological response produced by the secondary signal preceding the aversive event.

The difference between signalled and unsignalled conditions did not approach significance for any of the electrodermal measures. Nor was there any interaction between signalling and shock intensity. The study therefore confirmed the subject's verbal intensity ratings (see Section 3.2.2), that signalling unmodifiable shocks does not reduce the aversiveness of shock. This finding was replicated by Furedy and Ginsberg (1973) using similar apparatus and experimental parameters.

However, there are certain difficulties with Furedy and co-workers studies. Primarily, that the electrodermal response produced by the secondary signal may be significantly different from that elicited by the aversive event itself. Furthermore, in Furedy and Klajner's (1972) experiment they employed signal-shock intervals of 0.5 sec. It has been suggested that this is too short an interval to allow for a reduction in impact. In terms of an animal's preference for predictability, preference does not develop at such short inter-stimulus intervals (e.g. Perkins *et al.*, 1966). Furedy and Klajner do suggest that differential responding may have been achieved with a slightly longer inter-stimulus interval. Moreover, higher ratings of aversiveness have been reported with unpredictable shock at both 1 and 3 sec inter-stimulus interval durations (Kimmel, 1967). Although, it must be noted, that a reduction in the rated aversiveness of shock as a function of predictability failed to emerge with longer inter-stimulus intervals of 5 sec (Furedy, 1970) and 8 sec (Furedy & Doob, 1971).

The results concerning event impact are as inconsistent as those mentioned above. For example, some studies report smaller electrodermal response amplitudes at impact with predictable events (e.g. Baltissen & Boucsein, 1986; Katz, 1984; Lykken, Macindoe & Tellegen, 1972; Lykken & Tellegen, 1974; Peeke & Grings, 1968; Price & Geer, 1972), while others found no differential responding in terms of electrodermal activity or VPC (e.g. Bowers, 1971 (a); Furedy & Chan, 1971; Furedy & Klajner, 1972; Furedy & Ginsberg, 1973). One study reporting cardiovascular activity found lower HR levels at event impact when shock was preceded by a warning signal (Lykken *et al.*, 1972). However, as with studies using anticipatory measures of activation, these impact indices are subject to similar methodological criticisms concerning differing experimental stimuli between predictable and unpredictable conditions.

Of those studies employing unconfounded experimental techniques which have also found a reduction in autonomic arousal as a function of predictability, Katz's (1984) study is an excellent example (a detailed description of the experimental design is given in Section 3.2.2). During each trial, 40 sec segments of data were recorded, beginning 10 sec before trial onset. Katz took three response measures: first, the physiological response to shock onset (the largest SCR during the 1.5 to 4 sec after shock); second, the largest SCR during the 1.5 to 4 sec after trial onset and third, the response occurring after trial onset with a

latency longer than 4 sec, but prior to shock onset. The latter two measures were included in order to give an indication of subject's anticipatory activity. Although anticipatory responses were larger in the unpredictable condition, the difference was not significant. Katz did find a highly significant difference between conditions at shock impact, with larger SCRs in the unpredictable group. Furthermore, as reported in an earlier section (Section 3.2.2), subjects also reported feeling less distress during the interval before predictable shocks compared to when they were waiting for unpredictable shocks. Differences in rated aversiveness of the shock at its impact revealed that subjects reported higher ratings when the shock was unpredictable.

Katz explains these findings in terms of preception, where the warning signal allowed subjects to engage in preparatory activity in order to reduce the aversiveness of the event. For example, on a subjective level, predictable aversive stimuli were rated as less unpleasant than unpredictable events of the same intensity and duration. The majority of subjects, when given a choice, preferred predictable over unpredictable conditions and, finally, when the aversive event was preceded by a warning signal, subjects had lower levels of electrodermal activity at event impact than when there was no warning signal present. Katz conclusion that, "... the findings are reminiscent of Lykken's preception hypothesis" appear misplaced, however, as it is impossible to differentiate between an explanation in terms of either preparatory response or preception based on the data available.

Nevertheless, an earlier study by Lykken *et al.* (1972) has been cited as providing support for a preception explanation, although response interference effects may also be partially responsible. In a within-subject design, subjects received 24 predictable and 24 unpredictable shocks. In the two predictable conditions, a 1 sec tone preceded shock by 5 sec followed by, a 1 sec tone immediately before shock onset. Lykken *et al.* assessed both individual response curves (averaged across trials) for each condition and, mean response levels for each of the conditions in terms of range-corrected HR and SC.

When predictable and unpredictable conditions were compared in terms of mean changes in HR to event impact, the results revealed a highly significant difference, with the unpredictable conditions producing marked HR acceleration to shock onset compared with predictable conditions. Electrodermal activity followed a similar pattern to cardiovascular

responding, with the predictable group producing smaller SCRs to shock onset than the unpredictable group.

However, magnitude ratings of shock intensity were unaffected by the presence of a warning signal in the predictable condition, a finding inconsistent with an explanation in terms of preception. The authors explain this inconsistency by referring to the finding that magnitude estimates were positively correlated with both SCRs and HR elicited by shock onset. They suggest that an autonomic response to an aversive event is a function of the subjective intensity of that stimulus and that "... subjective intensity in turn depends upon objective stimulus magnitude as modulated by afferent control mechanisms such as preception, habituation, and the like. Thus, subjective intensity and the resulting SCR will vary directly with shock intensity when the shocks are unpredictable" (Lykken *et al.*, p. 323). Whilst magnitude estimates are believed to be a function of shock intensity *per se*, autonomic responses, on the other hand, are believed to be functions of both intensity and predictability.

Even if this tenuous argument is accepted, the Preception Theory cannot easily incorporate the results of a study by Price and Geer (1972) designed to examine predictions based on the safety signal hypothesis. They compared electrodermal activity in two groups exposed to either predictable or unpredictable aversive stimuli. The stimuli consisted of a series of pictures of dead bodies, picture duration was 5 sec. For the predictable group, an 8 sec tone was given before each picture whereas, the unpredictable group received no warning signal.

Predictions based on a safety signal analysis would be in terms of differential responding to predictable and unpredictable conditions during the inter-trial interval compared with similar reactions to the aversive event itself.

Three measures of electrodermal activity were assessed: first, the number of non-specific fluctuations (NSFs) during the inter-trial interval: second, the skin conductance response (SCR) during the 0 to 4 sec after onset of the aversive stimulus and third, the change in skin conductance (SC) occurring 4 to 8 sec after aversive stimulus onset. When subjects were compared on the number of NSFs during the inter-trial interval, the unpredictable group showed significantly more NSFs than the predictable group. In addition, the unpredictable group produced significantly greater SCR's to event impact than

the predictable group. There were no differences between the two groups in terms of the change in SC during the 4 to 8 sec after stimulus onset.

Price and Geer suggest that differential responding to event onset was confounded due to response suppression caused by the warning signal in the predictable condition (see Section 3.1). Thus, the results should not be used to support the beneficial effects of predictability. However, the significantly greater number of NSFs during the inter-trial interval in unpredictable compared with predictable conditions is in line with the safety analysis which suggests that signalling an aversive event reduces the aversiveness of the *total situation*, rather than at event impact *per se*. Against this, those few studies which have included a measure of autonomic activity during the inter-trial interval and have found no differential effects of predictability (e.g. Bowers, 1971 (a); Katz, 1984). Indeed, Bower's results (see Section 3.2.2. for a detailed description) revealed no difference between predictable and unpredictable conditions in terms of the mean frequency of SCRs during the course of the 20 sec trial, or, in SCR magnitude to event onset. Both measures did clearly distinguish between non-shock and experimental groups. Furthermore, cardiovascular measures revealed an increase in anticipatory HR levels for those subjects receiving a signal before shock onset compared with those for whom shock was unpredictable. There was little difference between the unpredictable and non-shock control groups.

The findings of Bower's study, along with many others (e.g. Furedy & Chan, 1972; Furedy & Klajner, 1972), suggest that there are no differences between predictable and unpredictable aversive events, or that predictable events lead to greater levels of autonomic activity. None of these results can be explained either in terms of preparatory or safety signal theories.

There are, however, a variety of methodological differences between the studies mentioned above and a number of those reporting decreased autonomic activity as a function of predictability (e.g. Price & Geer, 1972), many of which may account for some of the reported discrepancies. For example, in the study carried out by Bowers (1971) baseline HR measures were utilised at random intervals throughout the course of the experiment. Not only does this method not take into consideration any individual pre-experimental differences in cardiac activity, but it also confounds baseline measures with

the predictability manipulation. His rationale for using this procedure was to incorporate into the baseline measure any heart rate effects due to the threatening nature of participating in a shock-based experiment. From this running baseline, specific effects of shock threat on cardiac activity could be determined unconfounded by a more generalised threat reaction. In contrast, the majority of past studies typically utilised a baseline monitored before experimental manipulation began, and more importantly before an announcement that shock would be employed during the experiment. Bowers suggested that this procedure created a baseline that was artificially low and/or insensitive to specific differential effects of shock as a function of predictability.

In addition, there are numerous other methodological and procedural differences, some of which were outlined in detail in Section 3.2.2. First, a large majority of the studies confounded temporal predictability by investigating differing information (e.g. shock intensity and duration, warning signal duration and length of inter-shock intervals) in one experiment. Second, experimenters have not taken into account that different subjects have differing thresholds at which they find an event aversive. Thus, if the event is not aversive to a subject then he or she may not find it necessary to discriminate between having information or not having information about a noxious stimulus.

In summary, the results of both anticipatory and impact measures are inconsistent and often confusing. Some studies report greater impact and anticipatory arousal with unpredictability while others report the opposite effect or no difference. The variables that influence whether prediction will be advantageous or debilitating have yet to be determined, but may include the type of event used as the aversive stimulus, the signal-shock interval and even the intensity of the noxious event. A summary of study outcomes, including anticipatory and impact measures is shown in Table 4. Due to the methodological problems mentioned above, theoretical explanations to account for the available data have, in the majority of cases, not been discussed and there has been little attempt to differentiate between preparatory and safety analyses. However, it may be that factors associated with individual differences in people's preference for predictability can account for some of these differences. Indeed, the majority of studies supporting the beneficial effects of predictability have also found that a minority of subjects preferred, and showed lower

Table 4. Summary of human study outcomes investigating the effects of predictable and unpredictable aversive stimuli on physiological activity.

<u>Study</u>	<u>Stimulus</u>	<u>Physiological Measure</u>			
		<u>Anticipatory</u>		<u>Impact</u>	
		<u>EDA</u>	<u>HR</u>	<u>EDA</u>	<u>HR</u>
Averill & Rosenn (1972)	S		U		
Baltissen & Boucsein (1984)	N			P	
Bowers (1971)	S	N	P	N	
Furedy & Chan (1972)	S			N	N
Furedy & Doob (1971)					
Experiment 1	S		U		
Experiment 2	S		N		
Experiment 3	S		U		
Furedy & Klajner (1972)	S			N	
Furedy & Ginsberg (1973)	S			N	
Glass, Singer & Friedman (1969)	N	N			
Katz (1984)	S	N		U	
Lanzetta & Driscoll (1966)	S			U	
Lykken & Tellegen (1974)	S			U	
Lykken <i>et al.</i> (1972)	S	U	U	U	U
Miller (1979)	S	P			
Monat, Averill & Lazarus (1972)	S	P	P		
Peeke & Grings (1968)	S			U	
Price & Geer (1972)	V	U		U	

Note: S=shock; N=noise; V=slides; P=positive effect of predictability; U=positive effect of unpredictability; N=nonsignificant; EDA=electrodermal activity; HR=heart rate.



physiological arousal, in situations of unpredictability. Individual difference factors have yet to be clearly investigated.

4.

## CONCLUSION

In conclusion, preference for predictability studies in the laboratory have consistently shown that nonhuman subjects choose predictable over unpredictable shock. However, it is not necessarily the case that behavioural choice is accompanied by less severe physiological stress. It has been suggested that some of the inconsistencies apparent in the predictability literature, may be explained by the differing physiological measures used and by certain experimental variables including, the chronicity of the experimental session, shock intensity, method of shock delivery and the availability of food and water. Similarly, studies with human volunteers have obtained inconsistent and conflicting findings especially with regard to behavioural indices of rated aversiveness and/or preference for predictability. While physiological indices, especially electrodermal reactions, generally show an increase in anticipatory and response magnitude as a function of predictability, or no difference between predictable and unpredictable conditions. However, the validity of this data has been questioned on both methodological and conceptual grounds, especially with reference to the effects of response interference.

In terms of theoretical explanations, it is clear that the situation is more complex than either the preparatory or safety signal analyses suggest. It is possible that both preparatory responding and safety play an important part, they are certainly not mutually exclusive and it may be that both preparatory and safety interpretations can make a valid contribution under specific experimental conditions.

## CHAPTER 3

### FEEDBACK

#### 1. INTRODUCTION

There has been an extensive amount of research concerned with the consequences of providing feedback in both laboratory and applied settings. The purpose of this chapter is to provide an overview of this work in order to clarify what is meant by feedback in specific situations and, to highlight a number of important issues which have been raised as a consequence of this research. In particular, the various dimensions of feedback ranging from its timing, frequency, source and interpretation to the specific function feedback may have, such as, incentive or informational.

In general, the majority of laboratory and applied studies involving feedback have looked at its effects in improving performance. Although these studies have contributed widely to our knowledge of how feedback enhances, for example, learning and motivation, they are of only secondary interest to the subject of this thesis. However, improved performance may have an indirect effect on the consequences of aversive situations in that it improves the ability to produce adequate coping responses and, therefore, perception of control. Consequently, the literature will be briefly summarised rather than reviewed in detail. The remainder of the chapter will concentrate on a detailed examination of the literature concerned with the physiological consequences of providing feedback, in both human and nonhuman subjects.

#### 2. LITERATURE REVIEW OF FEEDBACK

##### 2.1. Performance Consequences of Feedback

2.1.1. Laboratory Studies. There is an extensive amount of literature involving feedback in the laboratory (for a detailed discussion of the issues discussed in this section, see Annett, 1969). In general, the data indicates that the provision of some kind of feedback generally results in improved performance on a variety of motor, perceptual and verbal

tasks. In particular, feedback appears to enhance rates of learning and augment motivation. It also seems that the more specific it is, the greater the impact, the longer the delay between performance and feedback, the greater the effect and, that when feedback is decreased, performance sometimes decreases.

However, there is considerable inconsistency within the literature as to why feedback produces these beneficial effects. One line of evidence suggests that it is due to the informational component of feedback, in which subjects are provided with information about some aspect of their performance which may be used to control their subsequent performance (i.e. it makes possible a better orientation to the task when given continuously across trials) whilst the second approach suggests an incentive or motivational component (i.e. it enhances some measure of performance such as speed, accuracy or effort). In terms of the latter, the incentive effect is not believed to be caused by feedback but rather is mediated by it in that feedback provides the subject with the means of achieving their aim. For example, in simple reaction time experiments, subjects are told to respond "as fast as possible" and yet to avoid pressing the button when no signal is present (i.e. a false positive). Thus, as Annett (1966) states, a higher reaction time can be traded-off against the risk of false positives but only if subjects are given feedback so that they can adjust their reaction time on the basis of the feedback they receive. This is especially true if a large penalty (i.e. an electric shock) is involved. The incentive properties of feedback have been investigated in some detail (e.g. Annett, 1966; Chapanis, 1964; Hauty & Payne, 1955; Mace, 1935).

The informational function of feedback concerns the distinction between knowledge of results (KR) and response feedback (RF). The former provides an individual with information about some aspect of their performance which can be used to control subsequent responding. In contrast, the second type of feedback, RF, provides information only to the level of 'success-failure' or 'right-wrong'. A number of researchers have elaborated on this distinction (e.g. Nuttin, 1953). Nuttin distinguishes between these two types of feedback with reference to two types of tasks; one in which the subject believes they are continuing to learn the same thing across trials (an 'open' task) and the other in which the subject believes they have to achieve a new goal on each trial (a 'closed' task). Thus in an open task (i.e. one in which KR is provided), Nuttin believes that each

response gives the subject two types of information: first, there is the aspect of 'sanction', success or failure concerning the given response and second, an 'informative' aspect which relates to the part of the task which has still to be completed. During closed tasks (i.e. where only RF is provided), once the response concerning each trial has been given, that particular part of the task is closed. Therefore, a 'correct' or 'good' response has no further implications or it has nothing more than the 'reduction of the need to make a good response' or a 'reward' in the Thorndikian sense.

Obviously within this latter type of task it is possible for the subject to ask '*how* good' or '*how* bad' was the response (i.e. KR). However, by adding additional information or KR, the question then becomes one of how much information is actually being given to subjects. Conversely, by providing simply 'good-bad' RF the absolute definition of what is 'good' or 'bad' is left to the subject. Intuitively, it would seem that RF is more clear-cut than KR, which, in providing more detailed information may simply increase the potential for ambiguity or confusion, and subsequently decrease an individual's performance level. Thus, the results of those studies employing KR will depend not only on the presence or otherwise of KR, but also upon the amount and complexity of feedback given as a result of the individual's response. For example, a number of studies have found that when subjects are given precise KR, they show improved performance compared with subjects who receive less precise KR (e.g. Annett, 1959; Trowbridge & Cason, 1932). Conversely, other researchers have reported findings which suggest that detailed KR is not always beneficial, and that performance varies with the different amounts and kinds of information received (e.g. Crafts & Gilbert, 1935; Green, Zimilies & Spragg, 1955; Swets & Sewell, 1963).

In general, there appears to be an optimum amount of KR that will result in beneficial effects, and that additional information will not improve performance and may even lead to its deterioration. Moreover, the differential effects of KR depend not only on the nature of the information but also what is done with it. For example, the hypotheses which subjects use in an attempt to find a solution to the problem. These hypotheses, in turn, interact with KR, for example, if a subject's hypotheses are incorrect the interaction will normally lead to below-optimal performance. Furthermore, KR changes a subjects ability to cope with the situation not only by virtue of the feedback provided but also

because KR and improved performance may simultaneously increase stimulus *predictability* and *control* over the situation. Therefore, an increase in subject's performance could equally be a result of any or all of these components and to attribute any beneficial effects solely to feedback *per se* could be erroneous. In contrast, simple right-wrong (or RF) feedback provides little detailed information as to the nature of the response which, in turn, provides the subject with minimal additional stimulus predictability or control over the situation.

However, a number of studies have reported difficulties associated with even the simplest form of RF. In particular, that RF type feedback does not help individuals acquire knowledge about the properties of complex tasks and, that RF has a limited usefulness in the acquisition of knowledge (e.g. Hammond, Summers & Deane, 1973; Schmitt, Coyle & King, 1976). Instead, they suggest that feedback which contributes to performance improvement should consist of "cognitive material" (i.e. KR) instead of response-orientated material, which enables subjects to perceive not only that their response was incorrect, but *why* it was incorrect.

In summary, those studies employing laboratory tasks have generally found that the presence of feedback improves performance and rates of learning across a variety of situations. However, the beneficial effects of feedback can be mediated by other variables such as the timing of feedback and the availability of incentives. Nevertheless, the most important function of feedback appears to be in terms of its information component. Indeed, a number of studies have differentiated between simple 'right-wrong' feedback and feedback involving 'how and why' information. The latter type of feedback appears to be more beneficial in complex, cognitive tasks although this, in turn, depends on the amount and nature of the information involved.

2.1.2. Applied Studies. A review of the applied literature reveals only a handful of studies which have manipulated some aspect of performance feedback, the majority of which have found that feedback can have a beneficial effect in complex, decision making situations (e.g. Braunstein, Klein & Pachla, 1973; Carver & Scheier, 1982; Catano, 1976; Greller, 1980; Ivancevich & McMahon, 1982). These beneficial effects are dependent on various factors, most of which have been discussed in relation to laboratory studies

involving feedback. For example, simple right-wrong (RF) feedback has limited usefulness in complex cognitive tasks compared with more detailed cognitive (KR) feedback (e.g. Jacoby, Troutman, Mazursky & Kuss, 1984). Additional factors are those concerning the timing and frequency of feedback (e.g. Bloom & Bourdon, 1980; Chhokar & Wallin, 1984; Joseph & Maguire, 1982), and the differential effects of goal-setting. Indeed, numerous studies have reported that the setting of goals or targets is necessary before feedback can improve or sustain performance. Furthermore, some report that when there is goal-setting in feedback and no feedback situations, feedback has no additional effect on performance (e.g. Bandura & Simon, 1977; Locke, 1967). However, other studies suggest that it is the presence of feedback which is the mediating factor in the beneficial effects of goal-setting; i.e. that goals without feedback are not sufficient to improve performance (e.g. Erez, 1977; Strang, Lawrence & Fowler, 1978).

In short, it would appear that the differential effects of having a goal or target and having feedback are complex in nature. For example, feedback can be the means by which a goal is specified and a means by which a subject can relate their performance to the goal, but feedback *per se* may not necessarily have any incentive effect. The incentive function of feedback seems to involve providing individuals with a performance target to aim for and information necessary for corrective action. Thus, although goal-setting and feedback appear to be different and independent factors, the applied data suggest that goal-setting is an intermediate step whereby feedback leads to a goal and then to an increase in performance. However, it should be borne in mind that there are relatively few studies which have examined goal-setting and provision of feedback independently.

To summarise: the basic findings involving feedback in applied settings suggests that information supplied to the subject can enhance performance in a variety of tasks and/or situations. However, these beneficial effects are mediated by the type of feedback presented to the subject. A conclusion partially supported by work carried out in the laboratory. In addition, a number of studies have found that the timing and frequency of feedback may play an important role. One additional component of feedback, not discussed in relation to laboratory studies, was that of goal-setting. Indeed, some studies have reported that the provision of feedback and goal-setting are optimal to enhance performance, and that without goals the presence of feedback is not sufficient.

## 2.2. Physiological Consequences of Feedback

2.2.1. Nonhuman Studies. A review of the animal literature reveals only a handful of studies that have investigated the physiological consequences of feedback in nonhuman subjects. The majority of these have included only an indirect manipulation of feedback, with the exception of two studies carried out by Weiss (1971b, 1971c).

Weiss (1971b) randomly allocated six rats to two types of triplets, each matched for body weight. For one triplet type, animals were assigned to either avoidance-escape, yoked or non-shock conditions. The avoidance-escape rat could turn a wheel at the front of its cage to postpone shock for 200 sec, or if the shock had begun, it was immediately terminated and the next shock did not occur for 200 sec. The yoked rat received identical duration and intensity of shocks as the avoidance-escape animals, but had no control over the shock. Those animals acting as the non-shock controls, never received shock. For the second triplet type, each response made by the avoidance-escape animal produced a tone (feedback stimulus) of 5 sec duration. Yoked and no-shock control animals in this feedback condition also received the tone stimulus whenever the avoidance-escape animals produced it. A total of 32 triplets were used. The experimental session lasted for 48 hr after which, two measures of stomach pathology were taken; first, the total length of lesions and second, the mean number of gastric lesions.

Weiss found that those avoidance-escape animals which received no feedback showed significantly greater number and length of gastric lesions than those avoidance-escape animals which did receive a feedback stimulus. Indeed, animals in the latter condition produced similar levels of stomach ulceration as the non-shock control subjects. In contrast, the extent of stomach ulceration in the avoidance-escape animals without a feedback stimulus was significantly greater compared with the no-shock control subjects. There were no significant differences between feedback and no feedback conditions for either the yoked or, non-shock control groups. Furthermore, for those avoidance-escape animals which did not receive a feedback stimulus, ulceration increased as the number of coping responses increased, whereas, those subjects which received feedback produced no increase in ulceration as a function of an increase in responding. These results also point to the fact that having control over the shock is not the most significant factor; rather, the

extent of ulceration is determined by having control which, in turn, means relevant feedback from coping responses is high.

These and other related findings (Weiss 1968, 1970, 1971a), led Weiss to suggest that it was not control *per se* that was responsible for the reduction in pathology, but rather a combination of the number of *coping responses* an animal makes (the greater amount being more ulcerogenic) and the proportion of response-produced *relevant feedback* (less feedback being more ulcerogenic). Relevant feedback is said to occur when a response produces a stimulus unassociated with the original stressor; for example, a lever press terminating a warning tone in a signalled avoidance schedule. The amount of relevant feedback produced depends on how different the stimulus situation becomes and how far removed these new stimuli are from any new association with the stressor. Thus, feedback of low positive, zero or negative values coupled with large amounts of coping responses produces debilitating symptoms, such as, weight loss, gastric ulceration and greater plasma corticosterone concentrations, whereas, feedback with high positive values and few coping responses is associated with little or no debilitating symptoms. Consequently, the level of ulceration can be accurately predicted if the extent of these two factors is known, and the ulcerogenic nature of a situation can be increased or decreased by withholding or giving feedback. In general, the theory makes a number of testable predictions. If an animal makes no coping responses, it will not ulcerate whether or not it receives feedback. Similarly, if relevant feedback is at a maximum, an animal will not ulcerate whether or not it makes any coping responses. However, as the amount of feedback decreases and the number of coping responses increases, the extent of ulceration will become more severe. These predictions were indeed substantiated by the findings of Weiss (1971b).

This conclusion can also be supported to a certain extent by a number of other studies, which have suggested that even in the absence of control, feedback information does have the capacity to reduce physiological responding to aversive stimuli. For example, Hennessy, King, McClure and Levine (1977) reported that the presence of a signal following shock onset resulted in a reduced adrenal response to the stimuli in the absence of control. Similarly, Tsuda and Hirai (1975) have shown that by manipulating response rates and the amount of relevant feedback produced, it is possible to reverse the beneficial effects of having control over an aversive stimulus. They employed matched



triplets of animals, in which the avoidance-escape subjects had to make either one, two, five or eight responses in order to avoid shock. Thus as the response requirement increased, the response rate increased and the amount of relevant feedback produced by each response decreased. According to the Relevant Feedback Theory, therefore, gastric pathology amongst the avoidance-escape animals should increase as the response requirement increased whereas, yoked animals should show similar amounts of ulceration in each condition as the amount of relevant feedback remained the same. These results were obtained, with the avoidance-escape animals producing more lesions in each successive response rate condition. In addition, the avoidance-escape animals showed significantly less severe stomach ulceration than the yoked animals when required to make only one response. However, when five or eight responses were required to control shock the avoidance-escape animals produced more severe ulceration.

Weiss (1971c) went on to further test the Relevant Feedback Theory, in particular to examine the effects of negative feedback on the development of gastric ulceration. He predicted that by making response-produced feedback negative, ulceration should increase for the avoidance-escape animals but not for the yoked rats. The avoidance-escape, yoked and no-shock conditions were identical to the previous experiment (Weiss, 1971b). These conditions were maintained during a 48 hr session, however, during the second half (last 24 hr) a shock was delivered each time the avoidance-escape subject turned the wheel (i.e. made a coping response), thereby punishing the correct response. In terms of the Relevant Feedback Theory it would be hypothesised that a situation in which animals received negative feedback (i.e. responses produced stimuli associated with the stressor), should be more ulcerogenic than a situation in which relevant feedback was zero (i.e. yoked condition).

These results were indeed found during the second half of the experimental session, with animals in the avoidance-escape condition developing significantly more severe gastric ulceration than their yoked counterparts. A second measure, the mean number of lesions, also showed that the avoidance-escape animals had more severe ulceration than their yoked counterparts, although this did not reach significance. In addition, the avoidance-escape animals had significantly greater plasma corticosterone

concentrations than the yoked animals. For all measures, the two shock groups had significantly greater levels of pathology than the non-shock control subjects.

It is unlikely that the additional shocks increased the ulceration or plasma corticosterone levels in the avoidance group, since both avoidance and yoked subjects received extra shocks, yet only those animals which made avoidance-escape responses showed increased ulceration. Indeed, the correlation between the number of punishment shocks and ulceration was very close to zero ( $-.07$ ) for the yoked animals whereas, for the avoidance-escape animals the correlation reached  $0.66$ . Instead, it would appear that altering the feedback information increased the amount of ulceration. For the yoked animals, the punishment shocks had no effect on the relevant feedback received i.e. feedback remained at zero. However, for avoidance-escape animals the punishment shocks altered the relevant feedback significantly. For example, in the first session coping responses produced stimuli unassociated with the stressor whereas, when punishment shocks were added coping responses produced stimuli associated with the stressor, indeed coping responses produced the stressor itself. This, in turn, led to a  $500\%$  increase in ulceration involving the avoidance-escape animals.

Weiss concluded that the Relevant Feedback Theory could incorporate the effects not only of positive feedback, but also those associated with punishment and conflict, with the debilitating effects of punishment being derived from coping responses which produce low amounts of relevant feedback. Indeed, conflict situations produce a large amount of coping behaviour because in order to terminate an aversive stimulus animals must make a response even though that response is subsequently punished.

However, there are a number of difficulties associated with the Relevant Feedback Theory. First, it does not in itself propose a mechanism of ulcerogenesis, although the data does indicate one possibility. Animals developing the greatest ulceration also produced the greatest increases in plasma corticosterone levels (Weiss 1971b). Plasma corticosterone levels have been shown to have a number of deleterious biological effects, such as, contributing to the development of gastric ulcers (e.g. Miller, 1980). This type of relationship suggests, therefore, a role for corticosteroids in the production of stress-induced stomach ulceration. However, it is possible that increased corticosterone and stomach lesions are linked together as parallel effects of aversive stimulation rather than in

a causal sequence. Second, there are general methodological problems with Weiss' work. For example, Weiss studied the effects of coping responses, relevant feedback and predictability in one situation therefore limiting the generalisability of his findings to a specific intensity of shock, for a specific type of stressor and during a specific task with a specific degree of task difficulty. Similarly, any extrapolation of Weiss' findings to the effects of feedback in humans must be treated with caution, because of the complexity of higher cognitive processes such as verbal communication and self-evaluation which may markedly alter the way an aversive stimulus or situation is perceived. Moreover, there are likely to be large differences in the effects of the stimulus used for nonhuman (electric shock) and human (motor, perceptual or verbal tasks) subjects, which make extrapolation from Weiss' findings to humans problematic.

2.2.2. Human Studies. Compared to the extensive literature involving performance consequences of providing feedback, there have been very few studies which manipulate feedback as an independent variable in an attempt to examine the effects on physiological indices of stress in human volunteers (Grilly, 1978; Light & Obrist, 1980). Unfortunately, these studies employed entirely different methodologies. For example, they differ in the type and consequences of feedback, the task and level of difficulty employed, the aversive stimulus used and the physiological indices studied.

Light and Obrist (1980) examined the physiological effects (systolic and diastolic blood pressure (SBP and DBP)), heart rate (HR) and pulse transit time (PTT) of coping and performance feedback in a reaction time task involving electric shock as the aversive stimulus. The main factor under investigation was the opportunity to avoid shock but additional factors such as, prior experience with the aversive stimulus and performance feedback were also examined. Light and Obrist hypothesised on the interrelationship between feedback, control and cardiovascular activity, stating that "... subjects with performance feedback would have greater opportunities for control over the task than those without feedback, and so should show enhanced cardiovascular changes" (Light & Obrist, p. 244). They suggested that by providing feedback subjects can use it to enhance active coping mechanisms. For example, if subjects are provided with a display of their reaction

times as they do the task, they attempt to control the task not just by attempting to avoid the shocks but also by trying to improve on their best reaction time score.

All 72 subjects took part in an unsignalled shock avoidance RT task in which they had to press a button "as fast as possible" whenever a tone was heard. Subjects were randomly allocated to one of eight conditions. The two opportunity to avoid groups were achieved by yoking avoidance and no-avoidance subjects in pairs so that they received identical shocks (3 mA, 1 sec duration). Both avoidance and no-avoidance subjects were instructed to try to react faster on each trial. However, the avoidance subjects were also told that each time they did not react faster, they might receive an electric shock. Whereas, no-avoidance subjects were not told of any contingency between RT and shock. Subjects in the feedback groups had their RT's displayed on a screen after each trial whereas, subjects in the no-feedback groups received no RT display. Subjects in the prior shock experience groups received one shock before the beginning of the task whereas, no experience subjects received the first shock during Trial 1 of the task. All subjects received 34 trials, with inter-trial intervals ranging from 10 to 30 sec.

In terms of subject's RT performance, those subjects who received feedback reacted significantly faster than those without feedback. There were no significant interactions with either avoidance or prior shock experience. With regard to the cardiovascular data, feedback produced a greater increase in SBP and a greater decrease in PTT from pre-experimental baselines, compared with the no feedback, but only among those subjects who had no control over the shock. Feedback-no avoidance groups also produced greater increases in HR compared with the no feedback-no avoidance groups, although this did not reach significance. In addition, the HR data produced a feedback by time interaction, due to subjects showing a significantly greater increase in HR at the beginning of the trial when feedback was available compared to when feedback was not available. There were no differences in the later periods of the task. There were no interactions between prior experience and feedback across any of the cardiovascular measures.

In general, it would appear that when subjects could not control shock, providing trial-by-trial feedback led to greater changes in cardiovascular activity, especially SBP and PTT compared with a no-feedback situation. However, when subjects could actively

control whether or not they received an aversive stimulus, the presence or absence of feedback made little differences in terms of cardiovascular activity. Light and Obrist examined their findings with reference to two key points. That is, operationalisation of control and task difficulty. For example, Light and Obrist's study differs considerable from those carried out by other researchers (e.g. Averill & Rosenn, 1972; Gatchel & Proctor, 1976) in the way in which control was manipulated. For example, in the latter studies subjects were required to perform a simple motor response (i.e. finger tapping, pushing a lever or button pressing), whereas, in Light and Obrist's experiment the task was both effortful and difficult. Thus, in the former studies, having control was never in doubt (i.e. it was certain), whilst in the Light and Obrist study, exercising control was difficult, and each subject could not be sure in advance whether they had indeed been successful (i.e. it was uncertain). It may be that this uncertainty is the crucial mediating factor. Indeed, this interpretation would be supported by recent studies examining the effects of effortful coping and/or task difficulty (e.g. Contrada, Glass, Krakoff, Kehoe, Isecke, Collins & Elting, 1982; Light & Obrist, 1983; Light, Obrist & Godaert, 1981; Manuck, Harvey, Lechleiter & Neal, 1978; Smith, Houston & Stucky, 1985; Solomon, Holmes & McCaul, 1980). These studies suggest that difficulty in achieving control, and the effort required to exercise control, is a contributing factor to differential cardiovascular activation (see Chapter 4, Section 3.1.1 for details of these studies). Unfortunately, there have been no studies investigating the differential effects of when control is difficult and/or uncertain compared with situations in which control is both effortless and predictable in relation to performance feedback. Furthermore, the avoidance and no-avoidance conditions differ not only with regard to active-passive coping but also in the incentives and encouragement those subjects in the control condition receive as a result of the nature of the task. As has been seen previously in relation to performance effects (Section 2.1), incentives are an important dimension of feedback in that they mediate its motivational properties.

Indeed, Grilly (1978) examined the effects of 'right-wrong' feedback (or RF as termed throughout this chapter) on electrodermal activity, in order to differentiate between the informational and motivational (incentive) function of RF type feedback. Those laboratory and applied studies manipulating RF in relation to performance effects (see Sections 2.1.1 and 2.1.2), reported that simple 'right-wrong' feedback had little effect

upon performance especially in relation to difficult tasks such as, learning multiple-cue probabilities (Hammond, Summers & Deane, 1973) or decision-making in applied organisational settings (Jacoby, Mazursky, Troutman & Kuss, 1984). There have been numerous studies which have attempted to clarify the informational value of certain stimuli and, in turn, the effect on physiological systems. However, none of these have examined the effects of feedback *per se*. For example, electrodermal activity has been shown to vary with the amount of information inherent within the stimulus or event (e.g. Spinks & Siddle, 1976; Velden, 1974), the behavioural significance of the stimuli (Ray & Piroch, 1976) and the amount of information processing in the presence of feedback stimuli (deSwart & Das-Smaal, 1976).

During Grilly's study the informational value of RF was indexed by the rate of habituation of the SRR to the feedback stimulus. In addition, he examined whether the informational value was affected by the presence of a contingency between subject's choice on a task and the feedback received. This was achieved by employing a matching task where subjects were provided with 'right-wrong' feedback which was or was not contingent upon their actual performance. This resulted in a situation in which subject's correct and incorrect responses were sometimes followed by a 'right' feedback stimulus and sometimes a 'wrong' feedback stimulus. It was predicted that if RF feedback had an informational function then SRRs would habituate faster to non-contingent than to contingent RF. In contrast, the opposite results would be predicted by Weiss' Relevant Feedback Theory, which states that conflict situations involving negative feedback (i.e. the non-contingent condition in Grilly's study) should result in greater physiological pathology. In addition, Grilly specifically hypothesised that there would be higher magnitude SRRs on incorrect choice 'right' trials than on correct choice 'right' trials and, a higher magnitude SRRs on correct choice 'wrong' trials than on incorrect 'wrong' trials. If either of these predictions were supported it would indicate that RF does indeed have a informational function whereas, if only 'right-wrong' RF produced differential effects then an incentive function would be indicated.

Subjects were allocated to one of two groups differing in the kind of RF received and the degree to which the RF corresponded to the subject's actual choices. Group 1 were provided with 'right' RF following all correct choices and 'wrong' RF after all incorrect

choices (i.e. feedback was contingent on their responses). Group 2 was provided with non-contingent RF however, all of these subjects were 'yoked' to subjects in Group 1 so that they received identical frequencies and sequence of 'right' and 'wrongs'. All subjects received 24 trials.

In terms of matching performance both contingent and non-contingent subjects produced a significant increase across trials, although there were no significant differences between the two groups. These findings suggest; firstly, that RF type feedback does not serve any informational function and secondly, that predictions made by the Relevant Feedback Theory, concerning greater physiological activity in negative feedback situations, were not substantiated in terms of performance on this type of task. Similar results were found for electrodermal activity i.e. no significant differences between contingent and non-contingent conditions in terms of the magnitude of SRRs. Thus, Grilly's findings substantiated those of past studies which suggested that RF type feedback does not facilitate performance in complex tasks (e.g. Hammond, Summers & Deane, 1973). However, the finding that subjects produced greater mean SRRs to the 'right' feedback stimulus than to the 'wrong' feedback stimulus regardless of RF contingency, led Grilly to suggest that RF does have motivational or incentive properties.

The findings of Grilly's study demonstrated, therefore, that it was possible to assess the differential effects of the informational and incentive functions of feedback independently of a subject's actual performance. Nevertheless, his slightly tenuous results need to be substantiated in further studies employing different tasks with various levels of difficulty, and where aversive consequences are involved. In particular, studies in which feedback affects performance on the task. Indeed, it may be that feedback has beneficial effects in terms of performance only when performance can be differentially affected by the subject's own actions. A notion supported by the findings of Light and Obrist (1980). It would then be possible to further investigate the effects of feedback in terms of its informational and/or incentive properties.

In summary, literature reviewed in this section suggests; firstly, that feedback has differential physiological effects in specific situations and secondly, that feedback may serve two types of function, either informational or as an incentive. With regard to the former, the most important mediating variable would appear to be an individual's ability to

control the aversive consequences of their performance, such that, subjects who cannot control shock by their task performance produced a greater increase in cardiac activity but only when trial-by-trial feedback is present. When subjects can actively control whether they will or will not receive an aversive stimulus, the presence of feedback makes little difference in terms of cardiovascular activation. Moreover, it was suggested that task difficulty may play an important role in mediating the effects of feedback, although this has yet to be confirmed since no feedback studies have manipulated task difficulty independently. In terms of the function that feedback has, the preliminary data suggest that the informational properties of RF type feedback are weak, and that any beneficial effects are a result of the motivational or incentive properties of feedback. However, it would be premature to draw any firm conclusions based on the data available at present.



## CHAPTER 4

### CONTROL

#### 1. INTRODUCTION

##### 1.1. Problems Of Methodology

The multidimensional construct of control is one of the most widely used in psychological research, with an extensive literature involving studies with both human and nonhuman subjects. A large number of which have investigated the relationship between controllability and stress. In general, control has been defined as the individual's perception that he or she can execute (or has the potential to execute) some action that changes an aversive stimulus or event. This definition, therefore, includes all types of control and recognises that a control response does not have to be executed for it to be effective. Indeed, it merely has to be perceived as 'real'.

This lack of definitional specificity has meant that the majority of studies have not taken into consideration the complex set of relationships between the different types of control employed in different situations. Obviously this is of critical importance in order to understand the relationship between stress and control, as each type of control is related to stress in a complex way; sometimes increasing it, sometimes reducing it, and sometimes having no effect at all. Moreover, the research literature to date suggests that the stress-inducing or stress-reducing properties of control depends upon a variety of factors such as the meaning of the control response for the individual and the context or situation in which it is employed and not merely upon its effectiveness in ameliorating or mitigating the impact of an aversive stimulus or event. Thus, as Phillips (1989) points out, "... there appears a real danger therefore, that just as the concept of 'stress' has proven difficult to justify (Stephoe, 1983), 'control' may also disguise the complexities and problems that it is supposed to explain" (Phillips, p. 239).

Recent reviews (Averill, 1973; Miller, 1979; Thompson, 1981) have sought to provide descriptive typologies of control based on an analysis of experiments manipulating controllability with very different results. These differences give an indication of the

difficulty in providing an adequate conceptualisation of control.

Averill's (1973) threefold typology is the one employed most extensively. He distinguishes between behavioural control, defined as the availability of a response that can modify objective characteristics of a noxious event; cognitive control, which involves the processing of information; i.e. its interpretation or appraisal and decisional control, he defines as choice. Averill further distinguishes between two types of behavioural control (regulated administration and stimulus modification) and two types of cognitive control (information gain and appraisal). However, Averill's typology is argued to be unsatisfactory (Fisher, 1986; Thompson, 1981) " ... in that cognitive control does not refer to control at all and use of the term is tautological because cognitive control is "that which reduces long-term distress". Therefore, by definition, it is stress-reducing. There is no reason to assume that gaining information *per se* is a form of cognitive control" (Fisher, p. 28). Indeed, in many situations information can lead to a subject believing that the event is uncontrollable. In addition, Averill's use of decisional control is argued by Fisher and Thompson to be unsatisfactory because few studies have examined this variable in relation to stress, because subjects should be given decisional control for ethical reasons and decisional control involves choice and is, therefore, usually considered to be a form of behavioural control or certainty of behavioural control (see Corah & Boffa, 1970).

Thompson (1981) went on to propose a four category typology. First, behavioural control which was defined as a "belief that one has a behavioural response available that can affect the aversiveness of an event " (Thompson, p. 90). Second, cognitive control which is " ... the belief that one has a cognitive strategy available that can affect the aversiveness of an event" (Thompson, p. 90). Thompson further distinguishes between two types of cognitive control; avoidant strategies and non-avoidant or sensitising strategies (Lipowski, 1970). The third type of control defined by Thompson is that of information which is delivered to the subject. However, as Thompson states, " ... it is probably not useful to think of information as one conceptually homogenous variable, since there is no reason to expect that these different forms of information will have the same effects on stress " (Thompson, p. 91). Information can, therefore, be a warning

signal which gives temporal information concerning a future event, or it can be information about some characteristic of the event, such as, the procedures involved in a situation. The fourth type of control is termed retrospective control and deals with the "beliefs about the causes of a past event" (Thompson, p. 91). Thus, retrospective control is not about the feelings of control when experiencing an event but attributions about the causes of a past event.

There are a number of difficulties associated with Thompson's typology: first, the concept of information as a form of control is very similar to what Averill (1973) termed cognitive control and, is therefore subject to the same criticisms i.e. information may at times engender feelings of control in an individual but information gain *per se* is not necessarily a *form* of control. Second, retrospective control is a form of attributional analysis about the cause of an event and, consequently, is of no great importance in the majority of studies investigating the basic issues in controllability research, that is, whether the availability of control does indeed ameliorate the effects of an aversive stimulus or situation. Although evaluation of past situations may partly determine response and strategies in this situation.

Although the difficulties mentioned above, involving both Averill and Thompson's typologies are important, perhaps the most salient problem involves the type of control which has been employed most widely; namely, behavioural control. This is because, in the typical behavioural control experiment, controllability and predictability are confounded. That is, the group with instrumental control or self-administration has more control *and* more predictability than the group without control. For example, when a subject administers shock to themselves, they not only control it, but they also know exactly when it will start. On the other hand, if the shock is experimenter-delivered, the subject can neither control it, nor do they know when it will start. Similarly, when a subject can escape an aversive event they know exactly when it will terminate, and when they can avoid an event they know precisely when it will not occur. Thus, predictability facilitates controllability by providing crucial information concerning the timing of coping

responses. As a result any beneficial effects produced during the experimental procedure cannot easily be attributed to either control or predictability independently.

This methodological confound also poses difficulties for Miller's (1979) proposed typology of behavioural control. The first two types of behavioural control she identifies are instrumental control and self-administration. Instrumental control is where "a person is able to make a response that modifies the aversive event" (Miller, p. 287). A variant of instrumental control suggested by Miller is that of perceived instrumental control in which subjects believe that they have control, but objectively, they have not. From the subject's point of view, instrumental and perceived instrumental control do not differ i.e. they are psychologically identical. However, methodologically Miller believes the difference to be important. For example, perceived instrumental control allows subjects in all experimental conditions to receive identical events, without the disadvantages of the yoked control design (see Levis, 1976).

The second type of behavioural control is self-administration where a subject delivers the aversive event to themselves. As mentioned previously, both instrumental control and self-administration paradigms are confounded with predictability. For example, in the instrumental control paradigm, when a subject escapes a noxious stimulus, they not only control it but they also know exactly when it will be turned off. Conversely, if a subject cannot escape an aversive event, they neither control it or know exactly when it will terminate. In terms of the perceived instrumental control paradigm, perceived control may be varied but differences in predictability are minimised because the aversive event occurs independently of responding. However, if the subject believes that they have control, then events may appear subjectively predictable. Thus, although actual control may be unconfounded by actual predictability, perceived controllability may still be confounded with perceived predictability.

Therefore, in all of the experimental paradigms discussed so far, any differences that emerge between control and no control groups can be attributed to differences in controllability *or* predictability with no way of differentiating between the two. Because of this confounding, Miller (1979) proposed two further types of behavioural control. First, actual control equated for predictability, in which predictability and controllability are kept

methodologically distinct by providing the no controlling subject with an external time cue to signal when the aversive event will occur. Such a cue can then be used to signal when the event will arrive in a self-administration experiment, terminate in the case of instrumental escape or, occur or not occur in the case of instrumental avoidance. This type of paradigm therefore eliminates any differences in timing between the control and no control groups.

The second type of behavioural control Miller proposed was potential control, in which a person believes that they have control available but does not employ the response. This type of study therefore differentiates between future and present control; i.e. the controlling subject does not employ the control response now but knows that at some time in the future it will be available. Thus, subjects with and without control are exposed to exactly the same experimental procedure, equally predictable for both individuals. However, although this paradigm is very effective in eliminating any differential effects as a result of the control-predictability confound, it does involve additional methodological problems. That is, the effect of controllability depends upon the actual and perceived differences between potential control and no-control conditions. Subjects are asked, or even instructed with varying degrees of emphasis, to refrain from using the controlling response. Thus, controllability experienced by the subject can be limited by strong experimenter demands. Furthermore, in some experiments, those subjects who use the controlling response are replaced by new subjects (e.g. Corah & Boffa, 1970).

This introduction has highlighted the complexities surrounding the concept of control. Each of the various conceptions of control currently in use have merit and substantial research evidence can be arranged to support any one of these approaches. However, during this review the term control will be reserved for instances of behavioural or *objective control* over environmental contingencies or events which either terminates those events, or makes them less probable, less intense or of shorter duration. Therefore, objective control will consist of Averill (1973) and Thompson's (1981) concepts of behavioural control and, Miller's (1979) categories of actual and perceived instrumental control and self-administration. The actual control equated for predictability and potential control paradigms will also be included, as theoretically, these designs are very important (see Section 2).

The exclusion of *subjective control* (i.e. Averill (1973) and Thompson's (1981) cognitive control), that is control over emotional reactions, covering such areas as individual differences in the need, preference or desire for control or the belief in control (i.e. locus of control) and strategies designed to regulate the impact of aversive events, is not meant to imply that it is unimportant. However, it is peripheral to the issue of instrumental control, in which an individual's actions can affect the occurrence or non-occurrence of aversive stimulation physically. Furthermore, the definition of control as a belief indicates that it is an individual difference construct; something which, when assessed, varies among individuals and within an individual over time. This leads to the results being both confusing and often contradictory.

Given the conceptual complexities and large amounts of literature involved, it is necessary to rely on laboratory studies in order to examine the effects of controllability. The reliance on laboratory studies obviously threatens the external validity of the findings. Ethical concerns dictate the level of noxious stimulation, and subjects are given the right to terminate the experiment at any time. Thus, subjects always have the final control. Moreover, laboratory situations necessarily involve a limited range of aversive stimuli or events such as, shock, loud noise and intelligence tests, whereas, applied settings include various situations with different demands. Thus, the difficulties and limited range of laboratory studies should be noted when considering the experimental evidence. This review will also not deal with certain closely related areas of research such as, learned helplessness.

As well as conceptional differences, many researchers have differed in terms of the exact part of the process of receiving an aversive event they examine; the anticipatory period *before* the aversive event occurs; the impact period *while* the aversive event occurs; the post-event period *after* the aversive event has occurred or the long-term post-event period. These distinctions are useful in organising the vast amount of studies concerning stress and control and two of them - the anticipatory (including the warning signal and inter-stimulus interval) and impact periods will be employed during this review as they involve the large majority of laboratory studies with human volunteers. Physiological and subjective (self-report ratings) indices of stress will be examined across each of these periods. In addition, this chapter will include a review of those nonhuman studies

manipulating objective control, the majority of which have assessed differential physiological effects during the short and long-term post-event periods. Behavioural (preference) measures will also be discussed with regard to human and nonhuman subjects.

## 2. THEORIES OF CONTROL

In addition to examining the empirical data concerning control, this chapter will review some of the theoretical explanations which have been put forward to account for these findings. In general, there have been three main approaches. The first is what Miller (1979) referred to as "circular theories" all of which state that control reduces 'stress' but do not propose a mechanism by which 'stress reduction' occurs. There are several examples of this, Glass and Singer (1972) proposed that individuals choose control in order to avoid feelings of helplessness. So "feelings of helplessness" explain why individuals who have no control are aroused. This explanation seems tautological because it is not made clear how feelings provide a stress mechanism, or how they can be assessed independently of stress *per se*. Similarly, Sells (1970) proposed that uncontrollability is more arousing than having control (also Lazarus, 1966 and Epstein, 1972 for similar explanations), but presents no mechanism to explain why. Learned helplessness (Seligman, 1975) may be useful in accounting for why uncontrollable noxious events produce motivational and cognitive deficits, but does not provide a mechanism for why lack of control may be more arousing and not preferred. Finally, there are those explanations which claim that control is beneficial from an evolutionary perspective (e.g. Averill, 1973), or, that control provides "mastery of the environment" (Fernichel, 1945; Hendrick, 1942). In psychoanalytic terms, Hendrick proposed that the instinct to master is a primary pleasure, which forms an inborn drive "to do" and "learn how to do". Fernichel produced a variant on this theme in which the reason behind a person seeking mastery of the environment was that it was anxiety-reducing. Thus, when an aversive event is mastered, the following anxiety reduction is pleasurable. Although these two proposed

arguments are not strictly tautological, they are extremely difficult, if not impossible, to assess. Furthermore, in terms of an evolutionary perspective, it would be slightly impractical to test them, particularly as there are not many grant-making bodies which give awards lasting millennia. These theories will not be discussed further.

The second group of theories have in common the proposition that the presence of control provides additional predictability, and therefore, the effects of controllability are reducible to those of predictability. Included here are Seligman's (1968) Safety Signal Theory, Weiss' (1971a) Relevant Feedback Theory and Perkin's (1968) Preparatory Response Theory. A detailed examination of these theories is given in Chapters 2 and 3. Briefly, the safety signal hypothesis states that in situations where a reliable warning signal predicts shock, the absence of the signal is a reliable predictor of shock absence (i.e. safety). Predictable shock generates a pattern of alternating arousal (during the signal) and relaxation (during signal absence) which is not possible with unpredictable shock, so subjects in the latter group remain in a high state of physiological arousal over virtually the entire test session. During instrumental and self-administration studies, those subjects who have a controlling response available have more predictability and a greater number of safety signals than those subjects without control. These safety signals provide information about when shock will not occur (avoidance), when it will go off (escape) or, that it will not come on (self-administration). In contrast, during no control conditions, subjects have fewer safety signals available and therefore less information.

Thus, the safety hypothesis predicts; (i) instrumental control should be preferred as it provides more safety signals and therefore less anticipatory arousal; (ii) self-administration should also be preferred and, lead to lower levels of anticipatory arousal because the subject knows precisely when the aversive event will occur, and consequently, when it is safe. These effects, and those mentioned in (i), should be particularly clear in avoidance paradigms but less clear with escape, since shock offset itself provides a safety signal for the no control condition; (iii) fewer or no effects of control (instrumental or self-administration) at event impact.

The Relevant Feedback Theory (Weiss, 1971a) is similar to the safety signal hypothesis. It proposes that the availability of a control response is less physiological stressful because these responses are followed by relevant feedback. Relevant feedback is



said to occur when a response produces a stimulus unassociated with the original stressor. For example, an escape response is followed by the relevant feedback of warning signal offset and the non-occurrence of the aversive event. Thus, one of the functions of feedback is to aid prediction of the outcome of an event. Predictions made by the relevant feedback analysis include; (i) preference for instrumental control; (ii) less anticipatory arousal with control if the mechanism by which relevant feedback reduces stress is because it provides a safety signal, as defined in the preceding theory; (iii) no predictions concerning event impact; (iv) no differential effects in self-administration studies because the event that follows a controlling response is not a stimulus unassociated with the stressor (relevant feedback) but the stressor itself (i.e. onset of shock). Thus, subjects should not prefer, nor show less anticipatory or impact arousal with self-administered events.

The Preparatory Response Theory (Perkins, 1968) suggests that when an event is predictable, it is less arousing since the warning signal allows subjects to prepare for that event either peripherally or centrally. In addition, subjects should choose predictable events because the signal elicited preparatory response reduces the perceived aversion of the event. When a subject can control an event by avoidance, they can predict its occurrence or non-occurrence and by escape, they can predict its duration. So the Preparatory Response Theory predicts; (i) preference for instrumental control and less arousal at impact because the subject is relatively certain about the non-occurrence, or the duration, of the aversive event. Thus, they can make well timed preparatory responses in order to mitigate the effects of that event; (ii) similar predictions for self-administration, since the self-administering group has better timing about exactly when the aversive event will occur; (iii) no specific predictions regarding anticipatory arousal for either instrumental control or self-administration. However, the theory may be extended in order to incorporate anticipatory periods. For example, it might be suggested that lower autonomic activity should occur with controllability because in instrumental control, or self-administered conditions the aversive event is more predictable compared to no control or experimenter-administered conditions. In turn, additional predictability should lead to a reduction in arousal because the individual is anticipating a less painful event since they are able to prepare for it.

However, these predictability theories do not provide a satisfactory explanation of why potential control could be sufficient for mitigating the effects of aversive stimuli. Nor

would they suggest that the effect of controllability could be greater than those of predictability alone. For example, if a group could not control but were given warning signals in order to predict the occurrence of an aversive event, the above theories would predict the same outcome as for the group which could control.

These problems, led Miller (1979) to suggest the minimax hypothesis. This suggests that controllability provides an organism with more than just extra predictability. Specifically, it states that controllability in an aversive situation acts as a signal about the expected outcome of that situation. That is, a subject with control expects a less aversive outcome than a subject without control. According to the hypothesis, this is achieved by providing a guaranteed upper limit on maximum future danger. Therefore, the minimax analysis implicitly proposes that organisms want to minimise the maximum danger to themselves and, that controllability provides a mechanism by which to do so. As Miller states, "A person who has control over an aversive event ensures having a lower maximum danger than a person without control. This is because a person with control attributes the cause of relief to a stable internal source - their own response - whereas a person without control attributes relief to a less stable, more external source" (Miller, p. 294).

Specific predictions made by the minimax hypothesis are that subjects should prefer instrumental control because it minimises maximum intensity, duration and/or frequency of a noxious stimulus. Anticipatory arousal should be lower when subjects have control over an event because the maximum danger presented by that event is lower than when the event is uncontrollable. However, the hypothesis makes no predictions concerning event impact, as it proposes no mechanism whereby expectations concerning the maximum danger presented by the event affect responses to that event once it occurs. Similar predictions are made for self-administration studies, this is because when a subject delivers the aversive event to themselves they know that when it becomes intolerable, they can simply stop delivering it. In contrast, when shock is experimenter-administered, subjects have no guarantee that when it reaches an intolerable level they will not receive another one.

In situations where control is equated for predictability, subjects will prefer controllability and, produce less anticipatory arousal. This is because subjects believe control is caused by an internal, stable factor - their own responding. Therefore, subjects know that in the future they only have to produce the same response to minimise danger.

This *stability* is crucial to the minimax hypothesis because, "If a subject attributes the cause of relief to a stable, internal factor - such as their own response - they have a reliable predictor that in the future, danger will only occur up to some relatively low, maximum amount. In contrast, if the attribution is to some unstable, external factor - such as the experimenter's whims - future danger is not guaranteed to be restricted to any relatively low maximum" (Miller, p. 295). Thus, in terms of instrumental control equated for predictability, a subject's own response is a more stable guarantee of future danger levels compared with external signals, even though these same signals have given identical predictability of shock onset and offset up until the present point. The self-administration equated for predictability paradigm is logically similar. If a subject can turn the shock off themselves then they are dependent on their own responses this, in turn, means the subject can predict the minimum amount of time it takes to perform a controlling response. Subjects in the self-administration condition, therefore, show reduced arousal compared with an individual who is reliant on an experimenter's actions, even if this experimenter has been reliable and predictable in the past. The minimax hypothesis makes no predictions concerning event impact in actual control equated for predictability studies.

The predictions concerning potential control are similar to those listed above. Since subject's with the ability to control show a level of arousal proportional to the maximum level of shock they believe to be possible and this, in turn, is related to the latency of their reaction time to press the button (or the time it takes to perform the appropriate controlling response). Subjects with no control have no guaranteed maximum level and, therefore, will reveal greater levels of anticipatory arousal.

As mentioned previously, the minimax hypothesis can successfully account for a large amount of the empirical findings. Moreover, another advantage of the minimax hypothesis is that it makes specific predictions about when subjects will relinquish control. Obviously this is of importance in 'real life' where people often voluntarily yield control into the hands of a third party i.e. an 'expert'. Furthermore, in laboratory settings, there is always a minority of subjects who, when given a choice, prefer uncontrollable aversive events (e.g. Averill & Rosenn, 1972). The Minimax Theory predicts that subjects will relinquish control when they *believe* that some factor other than their responding provides a more stable guarantee of the maximum limit of danger. There are various situations in

which this may occur, for example, where an individual is uncertain as to whether or not they can reliably perform the necessary controlling response (i.e. on a difficult task). A second situation would be where an individual is uncertain that performing a controlling response would reliably lead to the desired outcome (i.e. where the probability of avoiding shock varies). For example, Averill et al. (1977) carried out a study in which they varied the probability (1.00, 0.66 or 0.33) of avoiding shock by pressing a button. The authors found that preference for control decreased as the probability of shock avoidance decreased from 1.0. Finally, a third situation would include where individuals have to learn the contingency between the response that reliably leads to the desired outcome.

In all of the above situations, having an internal controlling response does not guarantee stability. Thus, uncontrollability may be preferred and lead to lower levels of anticipatory arousal. In addition, the minimax analysis would predict that this effect should decrease as the judged stability of internal controlling factor increases. Unfortunately, a review of the literature reveals no study that bears directly on this question.

Although the Minimax Theory provides an adequate explanation of some of the effects of behavioural control, it is not clear how the hypothesis can account for any beneficial effects of controllability during the impact period. One possible way of incorporating the impact data may be to argue that when subjects have a controlling response available they experience less anticipatory arousal and, against this background of 'relaxation', the noxious stimulus leads to less arousal at event impact. Furthermore, habituation to noxious events may be facilitated when the individual is in a 'relaxed' state (see Lader & Matthews, 1968). However, the majority of human studies which have employed impact measures would not support this extended version of the minimax hypothesis, since they found no differential effects with controllability. Further clarification of this issue would mean that the minimax hypothesis must make more precise predictions about the timing of the supposedly beneficial effects of controllability. For example, a differentiation between short and long-term effects would be advantageous, since it may be that control is stress-reducing in the long-term even though in the short-term it may be stress-inducing. Furthermore, there are a number of other issues left unresolved through the theory's lack of specificity. For example, it makes no predictions about how control differentially affects responding as a function of the quality, frequency, intensity, duration

or other characteristics of the stressor. Nor can it explain the observed desynchrony between anticipatory physiological and self-report data, since it makes no predictions about the various dependent variables which are components of the process 'stress'.

### 3. LITERATURE REVIEW OF CONTROL

#### 3.1. Anticipatory Period

##### 3.1.1. Instrumental Control

3.1.1.1. Subjective Measures. The majority of studies manipulating instrumental control have found that it decreases subjective ratings of discomfort and/or anxiety (Bowers, 1968; Gatchel, McKinney & Koebernick, 1977; Glass, Reim & Singer, 1971; Houston, 1972; Stotland & Blumenthal, 1964; Szpiller & Epstein, 1976). However, one study (Averill & Rosenn, 1972) found no difference in anxiety ratings between control and no-control groups. The results of these studies and those mentioned in the following sections are summarised in Table 5.

A typical example of one of these studies is that by Szpiller and Epstein (1976). Subjects were allocated to one of three groups: Group 1, were informed that if they tapped rapidly during a specified period at the end of an anticipatory phase, they could avoid receiving shocks; Group 2, were told to tap as rapidly as possible but were not informed of any relationship between tapping and shocks and Group 3, who neither tapped nor avoided shock but were included to assess the effects of motor activity on physiological measures. All three groups were yoked so that they received identical shocks (5 mA and 0.5 sec duration). The task consisted of six trials of a 12 point count-up displayed visually on a counter in front of the subjects, with shocks presented on the count of 10. The interval between each number was 15 sec. Thus, subjects in the avoidance group could avoid receiving a shock by tapping as rapidly as possible on a telegraph key, between the numbers eight and 10. Additionally, the non-avoidance subjects were told to tap as rapidly as possible between the numbers eight and 10 to determine the effects of motor activity on reactivity. Subjects in the no-tapping group were given no task to perform. Subjects had to

Table 5. Examples of studies investigating the effects of control upon physiological, subjective and behavioural indices of stress in human volunteers (adapted from Arntz and Schmidt, 1989, p. 137).

<u>Study</u>	<u>Type of Control</u>	<u>Stimulus</u>	<u>Preference</u>	<u>Anticipation Subjective</u>	<u>EDA</u>	<u>HR</u>	<u>Impact Subjective</u>	<u>EDA</u>	<u>HR</u>
Averill & Rosenn (1972)	I	S		NS	C	NS		NS	
Averill, O'Brien & deWitt (1977)	I	S	C	C					
Bowers (1968)	I	S		C				C	NS
Champion (1950)	I	S							C
Contrada <i>et al.</i> (1982)	I	N/S				NS			
Corah & Boffa (1970)	I	N						C	C
DeGood (1975)	I	S				C			
Elliot (1969)	I	S	C	NS					NC
Gatchel <i>et al.</i> (1977)	I	N		C	NC				NC
Gatchel & Proctor (1976)	I	N			NC	NS			NC
Houston (1972)	I	S		C		NC			
Light & Obrist (1980)	I	S				NC			
Manuck <i>et al.</i> (1978)	I	N/S							
Obrist <i>et al.</i> (1978)	I	S							
Sandman (1975)	I	V	C			NS			
Smith, Houston & Stucky (1985)	I	S				NC			
Staub <i>et al.</i> (1971, Expt.2)	I	S				NS		C	C
Szpiller & Epstein (1976)	I	S		C	NS	NS			

Ball & Vogler (1971)	SA	S	C						
Haggard (1949)	SA	S				C			C
Pervin (1963)	SA	S	C	C				NS	
Staub <i>et al.</i> (1971, Expt.1)	SA	S						NS	
Arntz & De Jong (1989)	CP	S					C		
Bjorkstrand (1973)	CP	S			NS			C	
Carr & Wilde (1988, Expt.1)	CP	N		NS	C	C	NS	C	C
Geer, Davison & Gatchel (1970)	CP	S		C			NS	C	NS
Glass <i>et al.</i> (1973)	CP	S		NS				C	NS
Geer & Maisel (1972)	CP	V			C/NS				C
Carr & Wilde (1988, Expt.2)	P	N		NS	C	C			
Corah & Boffa (1970)	P	N						C	C
Glass, Singer & Friedman (1969)	P	N						C	NS
Glass, Reim & Singer (1971)	P	N				C		NS	NS

Note: I=instrumental; SA=self-administration; CP=control equated for predictability; P=potential control; S=shock; N=noise; V=slides; C=positive effect of control; NC=positive effect of no control; NS=non-significant; EDA=electrodermal activity; HR=heart rate

better their previous performance on the immediately preceding trial (i.e. perform more taps), in order not to receive a shock on the next trial.

Szpiller and Epstein found that those subjects who had an avoidance response available rated their anxiety levels as lower than those subjects who had no available avoidance response. Both avoidance and non-avoidance groups had greater levels of anxiety than the no-tapping control group. These findings have been substantiated in other studies employing a variety of aversive stimuli; including, shock (Bowers, 1968), high intensity (95 and 110 dB) noise (Gatchel *et al.*, 1977; Glass *et al.*, 1971 respectively), an intelligence test (Stotland & Blumenthal, 1964) and a cognitive task with the threat of electric shock (Houston, 1972).

The study that reported no difference between control and no control groups (Averill & Rosenn, 1972) assessed the differences between vigilant and non-vigilant coping strategies on subject's reactions to the anticipation of electric shock. Thus, methodologically, it differed considerably from the majority of control studies mentioned above. The vigilance condition consisted of subjects listening for a warning signal during an anticipatory period before onset of electric shock whereas the non-vigilant condition consisted of a subject listening to music and thus ignoring the warning signal. Each subject could switch between two channels on a tape recorder. On the first channel, a warning signal occurred 5 sec before shock onset, and on the second, there was background music but no warning signal. Additionally, one group of subjects could avoid shock by pressing a button within the 5 sec interval following the warning signal, whereas, the second group had no controlling response. The experimental session consisted of eight trials which terminated in a shock of either zero, low (1.5 mA), medium (2.5 mA) or high (3.5 mA) intensity, all shocks were of 1 sec duration. Two subjective measures were examined; the painfulness of the shock and how relaxed or tense a subject felt.

The results of the relaxation-tension ratings revealed no significant differences between avoidance and no avoidance groups at any of the shock intensity settings. In terms of pain ratings, a similar pattern emerged. Thus, providing subjects with an avoidance response did not lead to reduced stress, as measured by subjective indices. One reason behind the discrepancies between Averill and Rosenn's findings and those mentioned previously may be that, in the former study, subjects in the avoidance condition could



choose whether or not to exercise control by monitoring the warning signal. By examining Averill and Rosenn's findings it is clear that a number of subjects (23%) in the avoidance group did not actually have control; i.e. they chose not to monitor the warning signal at any of the shock intensities. In contrast, a smaller percentage (20%) of those subjects in the avoidance group chose to control during all shock intensities. The remaining subjects in the avoidance group chose control or no control depending on whether the shock was of zero, low, medium or high intensity. Therefore, it may be that having the ability to choose whether or not to employ an avoidance response, when it is readily available, has differential effects.

3.1.1.2. Physiological Measures. In terms of electrodermal indices of stress, a number of studies have reported a decrease in non-specific SCRs (Gatchel & Proctor, 1976; Sandman, 1975; Szpiller & Epstein, 1976) and tonic SCL (Averill & Rosenn, 1972; Sandman, 1975) as a function of control. Conversely, one study produced no differential effects of controllability as measured by SCL (Szpiller & Epstein, 1976) and a further two an increase in SCL with control (Gatchel & Proctor, 1976; Gatchel *et al.*, 1977).

In terms of non-specific SCRs lower levels of activity have been found in within (Sandman, 1975) and between-subject designs, employing both escape (Gatchel & Proctor, 1976) and avoidance (Szpiller & Epstein, 1976) tasks. However, the data concerning tonic electrodermal levels is contradictory. For example, contrary to their findings for non-specific SCRs, Szpiller and Epstein (1976) found no significant difference between avoidance and no avoidance groups in SCL. Thus, it would appear that the non-specific skin conductance responses may measure something different to other electrodermal measures. A number of studies have noted a marked degree of independence between these measures in the past (e.g. Kilpatrick, 1972; Wilson & Dykman, 1960), although in other situations these measures have been positively correlated (e.g. Silverman, Cohen & Shmavonian, 1959).

Two studies by Gatchel and co-workers (Gatchel & Proctor, 1976; Gatchel *et al.*, 1977), found that those subjects who had a control response available produced increased SCL compared with subjects who were allocated to the no control condition. These differences may be partially explained in terms of methodological discrepancies; specifically the type of controlling response available to subjects. For example, in the

Gatchel studies, subjects had to learn what the controlling response was, whereas, in the remaining studies, subjects were told beforehand what controlling response they had to produce. In the Gatchel studies, both avoidance and non-avoidance subjects were told that a loud noise (1000 HZ) would come on and that if they watched the two lights in front of them, this would tell them how the noise could be controlled. If they succeeded, then a light marked "S-out" would come on; i.e. the subject had stopped the tone. However, if they did not succeed, a light marked "time-out" would come on when the tone finished; i.e. the tone had been stopped automatically. The correct response was actually four presses on the response button, and subjects could learn this after several trials.

Miller (1979) suggests that differences in controlling response do not lead to differences in effort, because the control subjects in the remaining studies (e.g. Szpiller & Epstein, 1976) were also exerting effort. Instead they result in increased alertness and vigilance in those subjects trying to learn a response. This, in turn, leads to increases in tonic electrodermal levels in the control condition. However, this explanation cannot account for why controlling subjects in Gatchel and Proctor's (1976) study produced decreases in non-specific SCR activity compared with no control subjects, unless it is argued that increased vigilance affects SCL and non-specific SCRs differentially.

In addition to differences in available controlling responses, there are a number of other discrepancies between Gatchel's studies and those reporting opposite results. For example, Gatchel *et al.* employed a learned helplessness paradigm in which subjects were pre-treated with escapable or inescapable shock. Following this, subjects returned for a second experimental session in which physiological and/or subjective deficits were investigated. In all previously mentioned studies the primary variable of interest was control versus no control, and subjects were tested in one experimental session only. Furthermore, during Gatchel *et al.*'s (1977) study subjects were clinically depressed volunteers. The effects of clinical depression upon physiological response patterns are unclear (e.g. McCarron, 1973), and nor is it known how this may, in turn, affect the differential effects of having control.

The empirical data concerning cardiovascular activity is also contradictory. Four studies produced no differential effects of control (Averill & Rosenn, 1972; Gatchel & Proctor, 1976; Staub, Tursky & Schwartz, 1971; Szpiller & Epstein, 1976), whereas, the

majority have reported an increase in cardiovascular activation (Contrada, Glass, Krakoff, Krantz, Kehoe, Isecke, Collins & Elting, 1982; Hodapp, Heiligrag & Stormer, 1990; Houston, 1972; Light & Obrist, 1980; Lovallo, Wilson, Pincomb, Edwards, Tompkins & Brackett, 1985; Manuck, Harvey, Lechleiter & Neal, 1978; Sandman, 1975; Smith, Houston & Stucky, 1985). As with the electrodermal data, Miller (1979) explains these contradictory results in terms of inconsistencies in the type of controlling response available to subjects. For example, in those four studies which found no differential effects of control, subjects were required to perform a simple motor response (i.e. finger-tapping, pushing a lever or button-pressing), whereas, in the remaining studies, the task was effortful and/or difficult. In Houston's study, for example, subjects performed a cognitive task; the Digits Backward Test from the Wechsler Adult Intelligence Scale (Wechsler, 1955). In the avoidance condition, subjects were told that they would be given a set of digits and that they might receive a shock if they made a mistake. In contrast, subjects in the non-avoidance condition were told that they would receive a set of digits and that sometime during this period they would receive one or more shocks. In fact, none of the avoidance or non-avoidance subjects were ever shocked. Contrary to expectation, HR increased more in the avoidance group than in the non-avoidance, although this difference did not reach significance.

Houston attributed these findings to differences in effort between the groups, with subjects in the avoidable group expending more effort in order to get the digits right and avoid the threat of shock compared with the non-avoidance subjects. Houston argued that this interpretation was supported by the fact that the task was difficult, requiring performance near to a subject's maximum ability. Thus, considerable effort would be required to perform well enough not to make mistakes.

It would appear that an individual's physiological response to an aversive situation or event in which they may or may not have a controlling response available, is more complex than has been proposed by past studies in the area. Indeed, differential effects may result from both the *availability* of control and the response demands; that is, according to the *difficulty* of control. This interpretation is supported by a number of more recent studies examining the effects of effortful coping (e.g. Contrada *et al.*, 1982; Light & Obrist, 1980; Lovallo *et al.*, 1985; Smith, Houston & Stucky, 1985). For example, Light

and Obrist (1980) employed a reaction time task with electric shock as the aversive stimulus, and found elevated HR, Systolic Blood Pressure (SBP) and Pulse Transit Time (PTT) in avoiding compared with non-avoiding subjects. Similar results were demonstrated by Contrada *et al.* (1982), using a complex visual choice reaction time task with white noise and/or shock as the aversive stimulus. Control subjects produced larger increases in SBP and Diastolic Blood Pressure (DBP) from pre-experimental baselines compared with no control subjects. However, there were no differences in HR levels between the two groups. Differences in HR activity were found in a study by Smith *et al.* (1985), with those subjects who had a controlling response (as in Houston, 1972) available to them producing higher SBP and HR than no control subjects. There were no differential effects with regard to finger pulse volume responses.

All of these studies would suggest that active control is associated with increased cardiovascular activation. Difficulty in achieving control and the effort required to exercise control would seem to be a contributing factor. Indeed, the assumption that performance on a effortful task leads to a different pattern of physiological reactivity would be predicted by a number of studies investigating the effects of manipulating task difficulty (e.g. Carroll, Turner & Hellawell, 1986; Light & Obrist, 1983; Light, Obrist & Godaert, 1981; Obrist, Gaebelein, Teller, Langer, Grignolo, Light & McCubbin, 1978; Wright, Contrada & Patane, 1986).

Studies by Obrist and his associates typically employed an active reaction time coping task. In this task, subjects were asked to press a key whenever they heard a certain tone. This tone occurred without warning after intervals ranging from 10 to 45 sec throughout a 14 to 16 min task. These studies provided a powerful aversive incentive for active coping - the threat of shock when reaction times were not fast enough. In addition, the study by Light, Obrist and Godaert (1981) offered a further incentive for effortful coping. There was a small monetary reward whenever their reaction time met a specified criterion. This criterion was varied between subjects so that for some individuals the task was easy and they won most of the time, for others it was difficult so winning occurring approximately 50% of the time, and finally, for the remaining subjects, it was impossible and they never won. In addition, the hard condition involved loss of money each time the

subjects reaction time was slower than on the previous trial, and subjects were instructed beforehand that they might receive shock for slower reaction times.

Light *et al.* hypothesised that the difficult condition would elicit more sustained effortful coping than either the easy or impossible conditions, and this, in turn, should produce more sustained cardiovascular activity. Initially, substantial cardiovascular responses were observed in all conditions irrespective of task difficulty, but over time, distinctions did emerge between groups. Subjects in the impossible condition showed a greater decline over time in HR, SBP and PTT than subjects in the difficult condition. However, contrary to expectations, although the more reactive subjects in the easy condition showed a greater decline in HR than those in the difficult condition, their blood pressure levels were similar and sometimes even higher than subjects in both of the other groups. Average reaction times did not differ between groups, so cardiovascular reactivity was not simply a function of differential motor activity levels.

Obrist, Gaebelein, Teller, Langer, Grignolo, Light & McCubbin (1978) found that subjects in the hard condition showed elevated HR, SBP and PTT compared with subjects in either the impossible or easy conditions. There were no differences between the latter two conditions. Furthermore, in a post-experimental interview, subjects in the hard condition stated that they were more involved, tried harder and gave up less frequently compared with both the easy and impossible conditions. The authors attributed these differential effects to levels of perceived control in each of the three conditions i.e. the impossible and easy conditions were equivalent to no control and control situations respectively whereas, the hard condition involved a situation where control was possible but not inevitable. Thus, the authors conclude that " ... when the probability of control was manipulated, the initial elevations of SBP, HR, and PTT were more sustained under conditions of some control than conditions of either almost perfect control or little control" (Obrist *et al.*, p. 109). However, cardiovascular responses would not appear to be dependent solely on successful control, since sustained alterations were not produced by subjects in the easy condition.

This conclusion would only be partially supported by more recent studies employing appetitive reaction time tasks (Light & Obrist, 1983), cognitive tasks, mental arithmetic and Raven's matrices manipulated to offer easy, difficult and impossible levels

of difficulty (Carroll, Turner & Hellawell, 1986; Manuck, Harvey, Lechleiter & Neal, 1978; Wright, Contrada & Patane, 1986). For example, Carroll *et al.* found increases in HR from easy to difficult and virtually impossible versions of mental arithmetic and Raven's matrices tasks, but no differences between the two more difficult conditions. In addition, the finding that there were no significant differences in oxygen consumption between conditions, implies that differential HR patterns were a function of task difficulty, and not a result of differences in energy expenditure (see Blix, Stromme & Ursin, 1974). Furthermore, self-report ratings revealed a significant difference in reported active engagement as a function of task difficulty, with both mental arithmetic and Raven's matrices producing reports of less engagement in the easy condition compared with both the hard and impossible conditions. There were no significant differences in reported engagement between hard and impossible tasks. A similar pattern emerged for self-reported arousal.

Carroll and his colleagues concluded that the effects of task difficulty on cardiac activity are more complex than initially predicted by Obrist *et al.* (1978). For example, Obrist *et al.* predicted no sustained cardiac activity when tasks were easy, and a reduction in activation with impossible tasks. However, these predictions have not been substantiated, with sustained HR increases in both impossible and easy conditions (e.g. Carroll *et al.*, 1986; Light & Obrist, 1983). Furthermore, subject's self-report ratings in Carroll *et al.*'s study did not differentiate between hard and impossible conditions, leading the authors to suggest that " ... difficulty level affects cardiac activity only in as much as it fosters variations in the extent to which subjects feel engaged in and aroused by a task" (Carroll *et al.*, p. 180).

The findings reported above appear to suggest that factors such as task difficulty and effortful coping are important in experimental situations which measure cardiovascular activation patterns. However, it would be rather premature to attribute the differential effects of task difficulty to differences in the availability of control. Since an impossible task is not necessarily uncontrollable nor, *vice versa*. That is, although a difficult task in general may involve less perceived control, control may still have independent effects i.e. they are not interchangeable. A review of the literature would suggest that this distinction has often been neglected.

However, those studies which have independently manipulated control of an aversive stimulus and levels of task difficulty, have found conflicting results. For example, Manuck *et al.* (1978) manipulated both factors in subjects performing a cognitive task under the threat of aversive loud noise. Those subjects in the control condition were told that they could avoid the noise (115 dB) if they solved a test item successfully whereas, subjects in the no control condition were told that the noise would occur randomly during the experimental session. No subject actually received the aversive stimulus. Within each of these conditions subjects were allocated to either a difficult or easy version of a concept formation task (from Feldman & Drasgow's (1959) Visual-Verbal Test). Subjects in both difficulty conditions received the same test items, however, the conditions varied on other task-related variables, for example, the number of solutions subjects had to report per item.

Manuck *et al.*'s findings showed a clear interaction between task difficulty and perceived control in SBP, with higher levels produced in those subjects who had control and were given the difficult version of the task. During the easy task, there was a non-significant trend for the control subjects to have lower levels of SBP than the no control group. There were no differential effects involving DBP, either in terms of control or task difficulty. These findings have been supported by other studies. Solomon, Holmes and McCaul (1980) used finger pulse volume responses and confirmed the results of studies reported earlier in which control over noxious events led to elevated cardiac activity only in situations where there was some degree of difficulty or effortful coping involved (e.g. Contrada *et al.*, 1982; Lovallo *et al.*, 1985). Thus, control *per se* may not be crucial. Rather, it is the behaviours demanded by different experimental situations that determine cardiovascular reactivity.

However, based on the empirical data available at the present time, it is not possible to offer a convincing explanation for why task difficulty and control should have these effects. For example, Manuck and colleagues argued that " ... the availability of an avoidance response acts primarily as an incentive for effortful responding, such that the performance aspects of a control task induce greater arousal in coping subjects" (Manuck *et al.*, p. 549). The mechanism by which this is achieved is "sensory rejection" (Lacey & Lacey, 1970), with those subjects in the control condition focusing more on "task-related demands for cognitive processing, shutting out extraneous or irrelevant stimuli" than their

no control counterparts. This, in turn, is associated with elevations of both HR and blood pressure (Lacey & Lacey, 1970). However, Manuck *et al's* empirical data offers no direct support for this explanation particularly as they report no measure of an individual subject's response "effort".

To summarise: the stress-reducing or stress-inducing effects of instrumental control in aversive situations would appear to be a function of the experimental situation; specifically, the nature of the controlling response available to individuals and the difficulty of that response. These factors are of particular importance in terms of cardiovascular responding, where a large number of studies have reported increased cardiovascular activation with effortful coping and/or difficult tasks. On the other hand, situations involving passive coping and/or impossible tasks have generally produced less sustained cardiovascular activation. It is therefore important to take these contributing factors into account when assessing the sometimes inconsistent and confusing research literature. The results of studies investigating subjective ratings of anxiety and/or distress and electrodermal activity, have been relatively consistent in the finding that when subjects have control, subjective arousal and phasic non-specific SCRs are reduced compared with a no control condition. The findings concerning electrodermal levels are contradictory. For example, some studies have reported no difference in SCL as a function of controllability whereas, others have produced increases in SCL. Unfortunately, as with the cardiovascular data, these inconsistent results can be partially attributed to methodological problems such as, differences in the nature of the controlling response.

3.1.2. Self-Administration. Only two studies have been reported that have investigated the effects of self-administration on anticipatory arousal (Haggard, 1949; Pervin, 1963). During Haggard's experiment subjects were presented with a list of words, one of which was always followed by shock. In the self-administration condition, a signal came on at a specified time after which subjects administered shock to themselves, whereas, the remaining subjects had shock administered by the experimenter. Haggard found that self-administered shock resulted in smaller changes in skin conductance to the stimulus indicating shock onset compared with experimenter-administered shock. However, these results do not necessarily indicate that control over the delivery of shock reduces



physiological arousal. Those subjects in the self-administered condition received a warning signal before shock onset whereas, shocks in the experimenter-administered condition were not preceded by a signal. Thus, control and predictability were confounded (see Chapter 2, Section 3.1). Moreover, Haggard did not measure non-specific SCRs or subjective indices of psychophysiological stress during the anticipatory period. It is, therefore, difficult to draw any firm conclusions from these findings.

The study carried out by Pervin (1963) did include indices of subjective anticipatory arousal and attempted to investigate the effects on control independently from those of predictability. He manipulated predictability (signal, no signal, inconsistent signal) and control (self or experimenter-administered) in a within-subject design and assessed the differential effects on anxiety and pain ratings. The results indicated a non-significant trend for subjects to rate self-administered shock as less anxiety-inducing and less painful compared with experimenter-administered shock. The relationship between self and experimenter-administered shock varied according to the type of signal received; i.e. as the signal was lost (became more inconsistent), the shock was rated as increasingly more painful and anxiety-inducing. However, this interaction did not reach statistical significance.

Pervin suggested that the lack of any significant effects may have been due to low levels of anxiety. In support of this, he suggested that the differences which were observed were all at the beginning of the experiment when anxiety was at its highest, and that the greatest difference between self and experimenter-administered shock was in the no signal condition. Again, when anxiety levels were at their highest. However, a more plausible reason behind the non-significant results may be that the subjective estimates of pain and anxiety were given only after subjects had received two examples of each condition; i.e. after the 24th, 48th and 72th trials. It may be that these self-report ratings were not representative of an individual's level of subjective stress in this type of situation. Obviously, more research needs to be undertaken into control via self-administration before any firm conclusions can be drawn.

The finding that self-administration leads to lower levels of anticipatory arousal, both on a subjective and physiological level, is consistent with the safety signal and minimax hypotheses. For example, in terms of the Safety Signal Theory, self-

administration of an aversive stimulus means that the individual knows just when the event will occur (danger) and when it will not (safety), and therefore, subjects know when they can relax. Conversely, subjects in the experimenter-administered condition cannot differentiate between safe and unsafe periods which, in turn, means subjects relax less and have higher levels of anticipatory arousal compared with self-administered subjects. On the other hand, the minimax analysis suggests that self-administered shocks are more tolerable because the subject knows that when shock reaches a maximum danger level, they can simply stop administering it.

3.1.3. Actual Control Equated For Predictability. A review of the literature reveals four instrumental control studies which manipulated equated control (Carr & Wilde, 1988; Geer & Maisel, 1972; Geer, Davison & Gatchel, 1970; Glass, Singer, Leonard, Krantz, Cohen & Cummings, 1973), and one self-administration experiment (Bjorkstrand, 1973). All of these studies claimed to examine whether the effects of control were primarily due to the ability of the subject to predict the occurrence of an aversive event, or whether the effect of control was independent of predictability effects. Thus, the results of these studies are crucial in differentiating between the predictability and minimax theories.

In terms of the instrumental control studies, three out of four revealed a lower number of non-specific SCRs during the anticipatory period, or, smaller electrodermal responses to the warning signal with controllability (Carr & Wilde, 1988; Geer, Davison & Gatchel, 1970; Geer & Maisel, 1972). The fourth (Glass *et al.*, 1973), revealed no significant differences between control and no control conditions in terms of electrodermal activity.

Carr and Wilde (1988) also reported lower HR levels in control subjects compared with a no control group, although there were no differences with regard to subjective indices of stress. In this study (Experiment 1), all subjects were presented with six trials, the beginning of each trial was signalled by a tone (74 dB, 5 sec duration) followed by, 3 min and 10 sec of silence. Depending on the experimental condition, the trial ended with a 10 or 3 sec burst of white noise (100 dB). Subjects allocated to the control group could change the aversive stimulus from a 10 to 3 sec white noise by pressing an illuminated button in front of them. Conversely, those subjects in the no control condition were not

told of any contingency between button pressing and reduction of the noise. They were asked, however, to turn off the light inside the illuminated button by pressing it. Thus, any differential effects of button pressing between groups was minimised.

The findings revealed a significantly lower number of non-specific SCRs and, lower HR variability during the 3 min anticipatory period in control subjects compared with those in the no control group. However, subjective measures taken during the anticipatory period, revealed no differences between the two groups in self-reported tension, stress or arousal. Indeed, the data showed a trend in the opposite direction; i.e. the control subjects reported greater feelings of tension, stress and arousal than the no control group.

The finding that control led to a reduction of physiological responding supports the minimax hypothesis and fails to support the predictability theories, since the latter suggest that control reduces stress because it provides an individual with extra predictability and that control has no stress-reducing effects *above and beyond* more finely tuned predictability. Thus, when subjects in both control and no control conditions have equal amounts of predictability, the controlling response provides neither more safety signals, relevant feedback, or the ability to make more finely tuned preparatory responses.

However, none of these theories can account for the desynchrony between physiological and self-report measures of anticipatory arousal. An analysis in terms of minimax would predict a reduction on subjective levels as well as physiological. The findings of the other two positive studies (Geer *et al.*, 1970; Geer & Maisel, 1972) cannot elaborate any further, as neither included measures of anticipatory subjective arousal. A number of possible hypotheses may be offered to explain this observed desynchrony (see Lang, 1968; Hodgson & Rachman, 1974). It may be that having control over an aversive stimulus differentially modifies physiological and subjective responses as a function of certain characteristics of the stimulus, such as, its frequency, duration, intensity or, other characteristics of the stimulus or may be due to individual differences in the subjects. Indeed, perhaps one of the most damaging notions concerning control, in the present literature, is its over-simplicity with regards to behavioural, subjective and physiological responses. This has led to unwarranted generalisations about the impact of controllability in different systems in which responses are presumed to be interchangeable. Thus, the beneficial effects of control in one system is assumed to be equally beneficial for others.

However, the present research literature would suggest that such generalised responses are rare, and that the pattern of responding varies according to different psychological coping strategies available to each individual (see Steptoe, 1991).

It is not clear why Glass *et al's* (1973) study should yield results different to the other three, especially since their experimental procedure was similar to that employed by Geer *et al.* (1970). All subjects were told to react to the onset of shock (intensity varied, 6 sec duration) by pressing a reaction time button. A warning light (10 sec duration) preceded shock by 10 sec. After ten trials, half of the subjects were told that in the following ten trials they could reduce the duration of shock from 6 to 3 sec with faster reaction times (perceived control condition). The other half were not informed of a contingency between reaction time and shock (no perceived control). The data revealed that non-specific SCRs did not differentiate between perceived and no perceived control subjects.

One highly speculative reason suggested by Glass *et al.* to explain differences between their and Geer *et al's* (1970) findings is that of differences in the level of autonomic arousal during the experiment. For example, the average number of non-specific fluctuations in the perceived control condition in Glass *et al's* study was higher (165.5) than in Geer *et al's* (152.0). Thus, Glass and colleagues assume that their subjects were more "anxious" and that, " ... anxiety arousal somehow inhibited the effects of perceived control on phasic SC responses to the shocks" (Glass *et al.*, p. 592). Indeed, they point to past research that indicates that the ameliorating effects of cognitive factors surrounding the aversive event are impaired by high levels of arousal (e.g. Lazarus, 1968). However, Glass *et al.* do not suggest how this explanation accounts for related findings of their study in which controllability resulted in lower pain ratings and fewer behavioural after-effects.

The study that equated control for predictability in a self-administration paradigm revealed no significant differences between those subjects who had control and those who did not (Bjorkstrand, 1973). Bjorkstrand's experiment was a between-subject design with all subjects receiving shock (average 345 v, 0.05 sec duration) preceded by a warning light of 5 sec duration and a second warning light of 1 sec duration. Subjects were allocated to one of four groups: Group 1 were instructed to press a switch when the second warning light came on (press-shock); Group 2 received shock during the second warning light (no

press-shock); Group 3 pressed the switch at the second warning light onset but never received shock (press-no shock); Group 4 were instructed to attend to the lights only (no press-no shock).

The findings revealed a significant difference between press and no press groups during the period between the first warning signal onset and second warning signal offset. Those subjects in the press groups had higher electrodermal activity than the no press conditions. However, this was primarily due to the press-no shock subjects having higher levels than the three remaining groups, whereas, the press and no press shocked groups revealed similar levels of activity. Thus, it would appear that in the presence of shock control via self-administration produced no beneficial effects compared to a situation where no control was present.

To summarise: when control is equated for predictability, three out of four studies produced lower levels of anticipatory physiological arousal with instrumental control. However, the one self-administration study found no differential effects of controllability. It would appear, therefore, that equated control *possibly* reduces anticipatory arousal. Obviously, more detailed research is needed in this area.

3.1.4. Potential Control. In a potential control paradigm, the Safety Signal, Relevant Feedback and Preparatory Response Theories predict no difference in anticipatory arousal between control and no control conditions. For example, subjects in the control condition do not actually perform a controlling response, so they cannot receive relevant feedback. Levels of relevant feedback are, therefore, identical in both the control and no control conditions. Likewise, the safety signal analysis suggests that since control subjects have not yet responded, they have received no more safety signals than have those subjects in the no control group. Thus, all subjects are in the presence of exactly the same signals predicting shock and shock-free periods. In contrast, the Minimax Theory would predict differential responding, since the subject with a controlling response knows that in future trials if the maximum danger presented by the aversive event becomes intolerable, they have a response available to them. Conversely, the no control subjects have no guarantee that they will receive shock which is within their maximum danger level. Therefore, the latter group should produce greater levels of anticipatory arousal than control subjects.

A review of the literature reveals three studies which have investigated the effects of potential control on physiological responding. One employed a self-administration paradigm with a cognitive task as the aversive stimulus (Stotland & Blumenthal, 1964), the remaining studies employed instrumental control and noise (Carr & Wilde, 1988; Glass, Reim & Singer, 1971).

Two of these studies provided evidence that potential control significantly reduces anticipatory electrodermal activity (Carr & Wilde, 1988; Stotland & Blumenthal, 1964). The third revealed findings in the expected direction, but differences between groups did not reach significance (Glass, Reim & Singer, 1971). Only one of these studies assessed subjective ratings during the anticipatory period (Carr & Wilde, 1988), and these revealed no significant differences between potential control and no control conditions in self-reported tension, stress or arousal.

During Stotland and Blumenthal's experiment, subjects were told before the study began that they would receive a series of IQ tests. However, those subjects in the self-administration condition were also told that they could take component parts of the test in any order that they wished. After these instructions, but before the tests began or the order chosen, subjects in the control condition showed lower anticipatory electrodermal levels than their no control counterparts. Although this study is of a different design than most of the studies mentioned previously, the results do indicate that control via self-administration can mitigate the effects of an aversive situation provided it is complex enough to involve some degree of ambiguity.

The experimental design employed by Carr and Wilde (1988, Experiment 2) was similar to the previous study carried out by the same authors but investigating control equated for predictability (Carr & Wilde, 1988; Experiment 1), with the exception that the no control subjects were not required to press a button and, white noise duration was 3 sec. Those subjects in the potential control condition were instructed that if the noise became intolerable, they could press the button in front of them and the noise would stop. However, they were also asked to endure as much of the noise as possible. No subject ever used the button during the experimental trials.

The data revealed that the potential control subjects produced significantly lower levels of non-specific SCRs and HR variability compared with the no control group. In

addition, self-reported feelings of tension, stress and arousal during the anticipatory period produced a non-significant trend towards higher subjective ratings in the no control group.

The findings of Glass *et al.* (1971) also revealed a non-significant trend in the expected direction; i.e. lower SCL in the potential control condition. One possible reason for why the data was non-significant may be in terms of methodological differences between Glass' studies and those mentioned previously. For example, in Glass' study, subjects in the potential control condition believed they had control over noise through a third party seated in another room who would press a button to terminate the aversive stimulus only if they were signalled by the subject, to do so. Thus, potential control subjects were told, " ... that while he did not have the control button, he did have the option of communicating *once* (author's italics) to the "other subject". He was told that this communication would mean that he wanted the noise stopped. The subject was then given a toggle switch and was told that he could contact the confederate *only* by pressing the switch" (Glass *et al.*, p. 247). Therefore, not only was the subject incapable of producing a controlling response *themselves*, but in addition, they could only have control *once* during the whole experiment. Even though they received 24 bursts of white noise.

It might be suggested, therefore, that subjects in the potential control condition may have felt that they had less control over the white noise. However, post-experimental ratings scales assessing the success of inducing feelings of control over the noise, did significantly differentiate between control and no control subjects.

Obviously, subjects in the control condition did feel that they had some response available to them in the future, albeit it through a third party that could reduce the aversiveness of the white noise. However, the experimental design of the study does make direct comparisons with the remaining potential studies difficult. Moreover, it is difficult to assess Glass and colleagues' findings in terms of Miller's minimax hypothesis since the potential control manipulation is not fully in accordance with the literal definition of potential control stated by Miller (1979). Furthermore, subjects in Glass *et al.*'s study only had one controlling response available to them throughout the entire experiment whereas, in all other studies subjects could terminate *each* aversive event, if so desired. It may be that this type of manipulation alters a subject's perception of control which may, in turn, weaken any observed differential physiological effects as a function of controllability.

In summary, although potential control would generally appear to reduce anticipatory physiological arousal, the one study measuring subjective indices of stress produced no differential effects as a function of control. Methodological discrepancies, such as, differences in the manipulation of potential control, may partially explain some of these differences. As stated earlier, the positive findings of having potential control support the Minimax Theory only.

### 3.2. Impact Period

#### 3.2.1. Instrumental Control

3.2.1.1. Subjective Measures. In those studies measuring subjective indices of stress, the majority reveal that control reduces the impact of an aversive event (Bowers, 1968; Corah & Boffa, 1970; Staub, Tursky & Schwartz, 1971), whilst one study reported no difference between control and no-control groups (Averill & Rosenn, 1972). In addition, one of the positive studies even showed that subjects tolerated twice the level of pain when they had control over the aversive event compared with a situation where no control was available (Bowers, 1968).

In Bowers study, subjects were told that the experiment they were about to participate in involved electric shock (1 to 2 msec duration) randomly set at one of three levels; just noticeable, painful but tolerable or at a maximal level. Each of these levels were individually set by the experimenter before the beginning of the experiment. Half of the subjects were told that electric shock was dependent on their performance on a maze and, that they *should* (author's italics) learn to avoid the shocked responses. The remaining subjects were not told of any contingency between shocks and performance. Subjects were then instructed that they would receive 20 trials although no subject ever did. Indeed, they were all stopped after only two trials.

Bowers found that subjects in the avoidance condition selected, on average, more than twice the shock intensity (for the painful but tolerable level) than the non-avoidance subjects. Similar results were found for the level of shock judged to be maximally painful. In addition, when subjects were given an opportunity to receive additional trials on the maze (with electric shock) after completing the experiment, only six of the 32 subjects



withdrew, and of those six subjects, five were in the non-avoidance condition. Bowers therefore suggests that subjects found the non-avoidance condition more aversive than the avoidance condition.

These findings - that the availability of control leads to a reduction in the impact of aversive stimuli - have been replicated by other studies employing shock in which subjects had to rate the shock on four subjective indices; sensation, uncomfortableness, painfulness and endurance (Staub *et al.*, 1971; Experiment 2) and, in studies employing white noise as the aversive stimulus with ratings of discomfort (Corah & Boffa, 1970).

The one study (Averill & Rosenn, 1972) which found no significant difference between conditions has been discussed in detail elsewhere (see Section 3.1.1). In particular, the methodological discrepancies between Averill and Rosenn's study and those reporting positive effects of control, were noted and discussed. For example, in the former, the main issue under investigation was the differential effects of vigilant and non-vigilant coping strategies, and not the differential effects of control *per se*. As reported in Section 3.1.1, there were no differences in self-reported painfulness of shock, and nor in how relaxed or tense subjects felt as a function of control. Indeed, a large number of subjects in the avoidance condition chose to remain non-vigilant throughout the experimental session; i.e. they chose not to have control over whether or not they received shock. It was suggested, therefore, that having the ability to choose whether or not to employ an avoidance response when it is readily available has differential effects compared with situations in which subjects have control regardless of whether or not they want it.

Another tentative explanation was suggested by Miller (1979) which involved differences in the certainty of exerting control. In Averill and Rosenn's study, subjects had to be vigilant for a warning signal in order to avoid shock (Averill & Rosenn, 1972). Miller suggests that this would have made control over shock more uncertain. In contrast, of those studies which obtained positive findings, one found that a reduction in impact only occurred when the availability of the controlling response was made salient to subjects (Bowers, 1968). In the remaining two studies (Corah & Boffa, 1970; Staub *et al.*, 1971), the experimenter emphasised that subjects *should* control the noxious stimulus, and subjects were provided with an *easy* controlling response. Thus, in these studies, subjects were certain that they could control the stimulus. Moreover, in one of the studies (Staub *et*

*al.*, 1971), subjects in the control condition also had certainty about the intensity of shock; i.e. subjects had a switch which allowed them to control the increase in intensity of the shocks by one, two or three increments (each increment was 0.2 mA).

Overall, these studies indicate that if a subject is certain that they can exert control over a noxious stimulus or event, then they may show a reduction in pain impact ratings compared to a subject without control. In contrast, if the ability to control is uncertain, then control may have no differential effects upon subjective indices of stress. Obviously, this explanation is only tentative. However, the inconsistencies in the empirical data cannot easily be explained by other systematic methodological differences.

3.2.1.2. Physiological Measures. The inconsistent findings involving electrodermal measures might also fit such an explanation. For example, two studies produced lower SCR magnitudes as a function of control (Champion, 1950; Corah & Boffa, 1970), two studies produced no differential effects (Bowers, 1968; Sandman, 1975), whilst another two studies showed an increase in SCR with controllability (Gatchel & Proctor, 1976; Gatchel, McKinney & Koebernick, 1977). Those studies which reported a positive effect of having instrumental control emphasised that subjects should control shock, and they provided subjects with an easy controlling response (Champion, 1950; Corah & Boffa, 1970). Miller (1979) suggests, therefore, that control subjects in the above studies were certain that they could exert control, and that this certainty is an important mediating variable in the response to the availability of control. Where certainty of having control leads to smaller impact SCRs in subjects with control than a subject without control. Conversely, if the exertion of control is uncertain, a subject may produce greater SCRs to impact than a subject without control, or there may be no difference between control and no control subjects. For example, those studies which showed no difference or increased impact with instrumental control either did not emphasise that subjects should exert control (Sandman, 1975) and/or provided subjects with a difficult controlling response (Bowers, 1968; Gatchel & Proctor, 1976; Gatchel *et al.*, 1977). Indeed, those studies which reported an increase in SCR to impact required subjects to learn the control response during the experimental session (Gatchel & Proctor, 1976; Gatchel *et al.*, 1977) (see Section 3.1.1 for more details). Thus, as Miller (1979) suggests, subjects in both of the Gatchel studies would have been maximally uncertain about exerting control.

Therefore, in the absence of any other obvious, consistent methodological differences between the above studies, the certainty of exerting control may offer a partial explanation for the discrepancies in the electrodermal data. As Miller (1979) concludes, "The most parsimonious interpretation of these findings is that certainty of control reduces, and uncertainty of control augments, the autonomic impact of an aversive event" (Miller, p. 299).

In terms of the cardiovascular data, no consistent pattern emerges. One study reported a decrease in HR as a function of control (Staub, Tursky & Schwartz, 1971), one study found no differential effects (Gatchel & Proctor, 1976), and two studies found an increase in HR impact (Elliot, 1969; Sandman, 1975). Those studies which included a blood pressure measure all found decreased impact with control (DeGood, 1975; Hokanson, DeGood, Forrest & Brittain, 1971).

DeGood (1975), for example, employed a matching task in which half of the subjects were allowed to escape the task whenever they wished by requesting a 1 min rest period (escape condition). The remaining subjects were yoked and therefore received rest periods only when requested by an escape subject (no escape condition). In order to maintain a high response rate, the task was paced with electric shock as a punisher for failure to maintain the required pace. Each subject received approximately 30 shocks per experimental session, and each session lasted half an hour.

Those subjects in the escape condition showed smaller increases in SBP compared with the no-escape subjects. However, these results were not supported by the DBP data which produced no significant difference between groups. The systolic data replicates that found by Hokanson *et al.* (1971) in a study employing a similar experimental procedure. Unfortunately, the latter experiment did not include a measure of diastolic arousal.

The only study reporting a decrease in HR impact levels as a function of control (Staub *et al.*, 1971; Experiment 2) employed an entirely different procedure to that described above. Control subjects were given one switch that enabled them to administer shock to themselves and a second which allowed them to control the increase in intensity of the shocks by one, two or three increments (each increment was 0.2 mA). No control subjects were given neither of these switches. However, subjects were yoked to control subjects on the magnitude of shock increments. The results indicated that cardiac responses

to shock were greater in the no control condition compared with the condition in which control was available.

It is difficult to understand why the aforementioned studies produced positive effects as a function of controllability whilst the remaining studies either reported no difference or an increase in HR impact with control. Differences in the difficulty of the controlling response cannot be a significant factor, as all of the studies employed easy control responses with the exception of one (Gatchel & Proctor, 1976). The one possible unifying factor that Miller (1979) suggests is that studies reporting a decrease in cardiovascular activation with instrumental control were in some sense avoidance paradigms. Subjects could limit shock intensity before they actually received it (Staub *et al.*, 1971), or they could avoid some shocks altogether by requesting a rest period (DeGood, 1975; Hokanson *et al.*, 1971). Conversely, subjects in the remaining studies could only execute escape responses (Elliott, 1969; Gatchel & Proctor, 1976; Sandman, 1975). Thus, differences in results can be predicted by the type of controlling responses available to subjects. However, as Miller (1979) points out, there is no apparent explanation for *why* the type of response should differentially affect cardiovascular responses at impact.

In summary, it would appear that the findings relating to cardiovascular activation are generally more inconsistent than both the subjective and electrodermal data. Where positive findings of instrumental control are reported, these are predicted by the Preparatory Response Theory and an extended version of the minimax hypothesis only. The Relevant Feedback Theory makes no specific predictions regarding effects at event impact, whereas, an analysis in terms of safety signals would predict no significant difference between control and no control conditions.

**3.2.2. Self-Administration.** There have been three self-administration studies which have reported impact measures. However, only one of these included a measure of physiological responding the results of which showed decreased electrodermal arousal with self compared with experimenter-administrated shock (Haggard, 1949). However, as mentioned in a previous section (Section 3.1.2), Haggard's experiment has been criticised

due to a number of methodological problems which make it difficult to draw any firm conclusions from his findings.

Those studies which included a measure of subjective stress both found no differential effects (Pervin, 1963; Staub *et al.*, 1971, Experiment 1). Pervin observed the effects of self versus experimenter administration upon ratings of pain and anxiety and found a non-significant trend for the self-administered shock to be rated as less painful and anxiety-inducing (see Section 3.1.2). In contrast, Staub *et al.* examined the effects of shock intensity upon four subjective indexes of shock intensity; sensation threshold, uncomfortableness, painfulness and limit of endurance. The experimental design was similar to that employed by Staub and colleagues in a later experiment (Staub *et al.*, 1971, Experiment 2) (detailed in Section 3.2.1). Briefly, self-control subjects were given a switch which enabled them to give themselves shock. The experimenter increased the intensity of shocks (in 0.2 mA increments) from a imperceptible shock to the subject's limit of endurance. The experimenter turned on a light when a new level of shock was set and, self-control subjects were told that they could administer shock at any time after this light had come on. This signal and the controlling switch were not available to the no-control subjects.

The findings revealed no significant differences in average shock intensity at any of the four levels of subjective judgement. Moreover, there were no trends that even approached significance. One of the explanations put forward by the authors involves predictability. They reported that self-control subjects tended to administer shocks to themselves as quickly as possible, thus, the timing of the shocks for the no control groups became relatively predictable. Therefore, only the identity of the person who delivered the shocks differed; i.e. the subject or the experimenter. Under these conditions, differences in control over the onset of the stimulus did not differentiate between groups in terms of subjective arousal. However, control in this experiment did not mean the availability of response by which to terminate or avoid a noxious event, but rather the ability to influence the manner of experiencing them. In this type of situation, the importance of control may be to increase predictability and, when the ability to terminate or avoid an aversive stimulus or event is not available, predictability may reduce stimulus impact. This, in turn, should mean that control and no control subjects show similar levels of subjective arousal.

However, given the paucity of evidence available, it would seem premature to draw any firm conclusions concerning the effects of self-administration upon impact arousal.

3.2.3. Actual Control Equated For Predictability. In experiments where control has been equated for predictability, two studies have found reduced SCR to shock impact as a function of control (Geer & Maisel, 1972; Geer *et al.*, 1970), and two studies found no differential effects (Carr & Wilde, 1988; Glass *et al.*, 1973). In a self-administration study by Bjorkstrand (1973) in which control was equated for predictability, the author found reduced SCRs at impact with self-administration. In terms of subjective indices of stress, two studies found no differential effects of control when measured by pain ratings (Geer *et al.*, 1970) or reported tension, stress and arousal (Carr & Wilde, 1988), whereas, two studies found reduced pain ratings (Arntz & De Jong, 1989; Glass *et al.*, 1973).

Most of these studies have been discussed in some detail previously (see Section 3.1.3) with the exception of Geer and Maisel (1972). In this study, subjects were allocated to one of three groups: Group 1 had control over the termination of an aversive slide and complete prediction concerning timing of that slide; Group 2 had complete prediction of the occurrence of the slide, but they could not control it; Group 3 had neither control or prediction. Subjects in the former two groups were shown a series of ten slides of dead bodies and each slide was preceded by a warning tone (60 dB, 10 sec duration). Subjects in the no prediction-no control group received identical stimuli but were not told of any relationship between the warning signal and slides. By pressing a button, Group 1 subjects could terminate the slide at any time. Subjects in Groups 2 and 3 were not given a controlling button. However, they were yoked to control subjects so that duration of each slide was the same for all subjects.

Geer and Maisel found a significant difference between experimental groups with the prediction-no control and no prediction-no control groups showing greater responses to slide impact than subjects in the control-prediction condition. The authors suggest that the effects of control cannot be satisfactorily accounted for in terms of predictability *per se*, as subjects who had control over termination of the slide produced lower levels of electrodermal arousal than those subjects who had prediction alone.

These findings replicated those of an earlier study employing an escape paradigm with shock as the aversive stimulus (Geer *et al.*, 1970) and, a self-administration experiment with shock (Bjorkstrand, 1973). However, it is not clear why some studies failed to find positive effects of controllability either on a physiological (Carr & Wilde, 1988; Glass *et al.*, 1973), or subjective (Carr & Wilde, 1988; Geer *et al.*, 1970) level. There were no systematic methodological discrepancies between studies. Indeed, the experiment carried out by Glass and colleagues was a replication of that carried out by Geer *et al.* Similar problems arise when the subjective-physiological dissociation is considered. For example, why did control subjects in Geer *et al.*'s study exhibit a reduction in electrodermal arousal but not in subjective pain ratings, whereas the opposite was true for those subjects in the experiment carried out by Glass and colleagues. No plausible explanation for this disassociation or its contradictory nature has been offered. There are no reported studies which have specifically addressed this question.

In summary, no firm conclusions can be drawn from the present control equated for predictability literature. Where positive findings of control are reported, these are predicted by an extended version of the Minimax Theory only.

3.2.4. Potential Control. At the physiological level, one study (Corah & Boffa, 1970) found lower SCRs to noise with potential control compared with a situation in which control was not available. The remaining studies found no differential effects (Carr & Wilde, 1988; Glass, Singer & Friedman, 1969; Glass, Reim & Singer, 1971). Of those studies which included a measure of subjective arousal, two found a reduction in reported pain with potential control (Corah & Boffa, 1970; Glass, Singer & Friedman, 1969). The third found no differential effects (Carr & Wilde, 1988), although there was a trend in the expected direction; i.e. a reduction in ratings of tension, stress and arousal with potential control. As with the previous section, these studies have been discussed in detail earlier (see Section 3.1.4), and as with the equated control paradigm, there are no explanations which easily account for the inconsistent data.

### 3.3. Post-Impact Period

In this section I will focus on the post-event effects of controllability with nonhuman subjects, as the majority of human studies have not included post-event measures. Those that have are mostly concerned with behavioural effects; i.e. performance deficits and not physiological and/or subjective indices of arousal. This chapter is primarily concerned with the latter, although realizes that behavioural effects are important. Indeed, a number of studies have reported performance deficits on cognitive tasks after uncontrollable noise (see Cohen, 1980 for an excellent review).

Nonhuman studies assessing the differential physiological effects of controllability have all employed instrumental control. As has been mentioned previously (Section 1.1), the majority of studies manipulating instrumental control have not distinguished between the effects of control and those of predictability. However, there are a handful of nonhuman studies which have independently manipulated predictability of stimulus onset and controllability of stimulus offset, notably those by Weiss (1968, 1970, 1971a, 1971b, 1971c). All relevant nonhuman studies will be reviewed below, whether they have or have not equated control for predictability.

One of the earliest experiments devised to examine whether the availability of a controlling response in an aversive situation would alter its physiological consequences, was carried out by Brady, Porter, Conrad and Mason (1958) on monkeys. Eight monkeys were assigned to one of two groups: avoidance or non-avoidance. The avoidance animal could delay shock (5 mA, 0.5 sec duration) for 20 sec if it pressed a lever. In addition, each avoidance subject was yoked to a monkey in the non-avoidance condition so that all animals received shock of identical intensity and duration. Thus, there were four pairs of monkeys each of which received six hour experimental sessions, alternating with six hour no shock periods for 24 hrs each day. The experimental period lasted approximately six to seven weeks.

Stable avoidance lever-pressing was established at 15 to 20 responses per minute, which prevented only the occasional shock during the six hour avoidance periods. Indeed, shock rates never exceeded two per hour during this period. However, the authors found that death of the avoidance monkey occurred after only nine, 23, 25 or 48 days of the



experimental procedure. In all cases, the presence of extensive gastrointestinal ulcers were revealed. In contrast, none of the non-avoidance subjects showed any gastric lesions.

This reported incidence of gastric ulcers in monkeys exposed to avoidable shock has proven difficult to replicate (e.g. Foltz & Millett, 1964) even in those laboratory conditions under which the study originated (Brady, 1964). Furthermore, there were faults in the experimental design which will be discussed later. However, a more recent study investigating cardiovascular responding under avoidance conditions, did find similar results (Corley, Shiel, Mauck, Clark & Barber, 1977). Twenty-two monkeys were randomly assigned to either avoidance or yoked conditions. A Sidman avoidance paradigm was employed in which the avoidance subject had to press a lever which postponed or avoided shock (4 mA, 1.0 sec duration) for a 40 sec interval. If the avoidance animal made no response, inter-shock interval was 5 sec. The experimental period lasted for 24 hours. Animals were then sacrificed at intervals ranging from 24 to 146 hours in order to assess the chronicity of myocardium degeneration.

The authors reported that significant myocardial degeneration was apparent in both avoidance and yoked animals, whether the monkey died during the experimental period or, was sacrificed up to five days after the experiment had terminated. However, eight avoidance animals had significantly higher degeneration ratings than their yoked counterparts. The remaining three pairs had similar ratings. In addition, an examination of shock avoidance performance shows that it was not related to the extent of myocardial degeneration.

Corley and colleagues attribute the myocardial degeneration found in avoidance monkeys to " ... the added psychological stress of response contingencies in the avoidance situation" (Corley *et al.*, p. 328). This "added psychological stress" is related to either sympathetic inhibition or parasympathetic activation which can precipitate myocardial degeneration and even death. Indeed, four monkeys died during the experimental period or up to 48 hours afterwards. These findings were later replicated in a study employing a similar experimental design (Corley, Shiel, Mauck, Clark & Barber, 1979).

Unfortunately, the findings reported by Corley and colleagues (1977, 1979) and those of Brady (1958), are contradicted not only by those studies mentioned earlier (Brady, 1964; Foltz & Millett, 1964), but also by a number of other experiments employing rats

(Gliner, 1972; Moot, Cebulla & Crabtree, 1970; Weiss, 1968, 1970, 1971a, 1971b, 1971c), monkeys (Hanson, Larson & Snowden, 1976) and dogs (Dess, Linwick, Patterson, Overmier & Levine, 1983). The two studies by Weiss (1968) are excellent examples in which rats able to perform an avoidance-escape response developed less severe gastric ulceration and lost less body weight than yoked rats who received identical experimental procedures but had no control response available. In these studies matched triplets of rats were exposed to the experimental conditions simultaneously. Each triplet consisted of one animal that could either avoid or escape shock (0.5-3.0 mA, 0.5 sec duration) by jumping onto a platform, a second animal with no control over shock but shocked whenever the first animal received shock and a third control animal that never received shock. In all conditions, a warning signal preceded shock by 10 sec.

During Experiment One, physiological response to stress was measured by differential weight loss which, in previous studies, had been found to be a sensitive index of stress (e.g. Pare, 1964; 1965). The experimental session lasted for two and a half to three hours, with inter-trial intervals of 30 sec duration. Body weight and food and water intake were measured at 16 hours after the end of the experimental session and, for five 24 hour periods thereafter.

During the experimental session, there were no significant differences between avoidance and yoked animals, although both of these groups lost significantly more weight than the non-shock controls. However, comparisons at 16 and 24 hours showed that both the non-shock and avoidance subjects gained significantly more weight than the yoked group. The non-shock controls also gained more weight than the avoidance animals, although this failed to reach significance. Food and water intake measures revealed a similar pattern, although it was not possible to determine whether differential intake was responsible for the differences seen in body weight between conditions, because accurate defecation measures were not taken.

The second experiment carried out by Weiss (1968) involved a similar procedure, with three notable exceptions. First, gastrointestinal ulcers were employed as the stress index. Second, the experimental session was 21 hours in duration. Third, the avoidance response consisted of animals touching a metal plate situated in front of them. Following termination of the experimental session, subjects were rehoused in individual cages, and 12

hours later each animal was sacrificed. Two measures of stomach ulceration were included; the total number of lesions and the length of each individual lesion.

Both the avoidance and yoked animals showed a significantly greater percentage of lesions than the non-shock controls. Subjects in the yoked condition had a higher percentage of lesions than their avoidance counterparts. However, this did not reach statistical significance. A similar pattern emerged with the mean number of lesions, although the difference between yoked and avoidance subjects was significant. In addition, in those animals developing lesions, the mean total length was significantly greater for the yoked subjects than either the avoidance or non-shock control animals. The difference between the latter two conditions also reached significance.

The findings of Weiss's studies - that animals who could avoid or terminate shock developed a lower number and severity of gastric lesions and lost less body weight than their yoked counterparts - confirms the results reported by a large number of studies. However, they are opposite to those of Brady (1958) and Corley and co-workers (1977, 1979) who employed monkeys as their experimental subjects. The most obvious discrepancy between these studies is one of species difference i.e. Weiss (and the majority of other positive studies) employed rats whereas, Brady, Corley and colleagues used monkeys. However, as Weiss (1968) points out, there are studies which have reported the most effective schedule in producing ulceration in rats (six hours on, six hours off) was the only schedule to produce gastric lesions in monkeys (see Rice, 1963). Thus, different species do show some similarities in relation to experimentally-induced gastrointestinal pathology. Furthermore, there are studies which have produced positive effects of controllability with monkeys as the experimental subjects (e.g. Hanson *et al.*, 1976).

There are other methodological discrepancies between, for example, Weiss and Brady *et al.*'s experiments. Firstly, animals in Brady's study were not randomly assigned to the avoidance or yoked condition, rather the subject in each pair which learnt the avoidance response more quickly during a pre-test was selected as the avoidance subject. This selection factor may have contributed to the high rates of ulceration seen in the avoidance subjects. Indeed, this would be predicted by a study carried out by Sines, Cleeland and Adkins (1963), in which rats susceptible to gastric ulcers learnt an avoidance response more quickly than 'normal' control animals. The same could be true of monkeys.

Secondly, the chronicity of the experimental session may have confounded the differential effects of controllability. The second experiment by Weiss, for example, was of 21 hours duration whereas, the monkeys in Brady's study were in the experimental apparatus for between six and seven weeks. It may be that physiological responding differs when the experimental session is chronic rather than acute, a prediction supported by findings of studies investigating the differential effects of predictability (discussed in detail in Chapter 2, Section 3.3.1). However, this explanation cannot explain the findings of Corley and co-workers (1977, 1979), who employed experimental sessions of 24 hour durations and, found greater myocardial degeneration in monkeys with an avoidance response available. Nor can it explain the results of Pare (1971) who assessed the effects of instrumental control in experimental sessions lasting either five or 21 days. He found no differential effects in either the number of gastric ulcers, body weight, food and water intake or adrenal/body weight ratios with regard to the chronicity of the experiment.

A third explanation which takes into account those studies by Brady, Corley and co-workers is in terms of the type of schedule employed. For example, both of the above studies involved a Sidman avoidance schedule. In this type of paradigm no warning signal precedes shock, and therefore, animals have to maintain relatively high rates of responding in order to avoid shock. Subjects in Brady *et al's* study, for example, had to maintain extremely high rates of responding (15 to 20 responses per minute). Since the yoked subjects do not need to make these responses, there is a significant physiological difference between the animals. In contrast, both studies carried out by Weiss (1968), included a warning signal which preceded shock. Thus, this response appears to be easier than the unsignalled responses mentioned above. It may be that performing an easy controlling response leads to a reduction of physiological stress whereas, difficult responses lead to an increase in physiological responding. Certainly this explanation would be supported by findings from studies involving human volunteers, in which effortful and/or difficult controlling responses resulted in increased cardiac activity whereas, effortless and/or easy responses led to either a decrease in activity, or, no difference between control and no control conditions (see Section 3.1). Furthermore, there are a number of studies employing nonhuman subjects which have reported increased gastrointestinal pathology as a result of high rates of responding (e.g. Barbaree & Harding, 1973; Goesling, Buccholz & Carreira,

1974; Natelson, 1976; Tsuda & Hirai, 1975 ). For example, Tsuda and Hirai (1975) specifically manipulated both rates of responding and availability of control, such that, avoidance-escape animals in different triplets had to make either 1, 2, 5 or 8 responses in order to avoid/escape shock. Thus, from the explanation given above, it would be predicted that as the response requirement and amount of responding increased the amount of ulceration in the avoidance-escape animals should also increase. These results were indeed obtained. The avoidance-escape animals produced progressively more lesions in each successive response condition whereas, their yoked counterparts revealed similar pathology in all conditions.

In Weiss' (1971a) view, this type of finding indicates that physiological pathology may be a function of an interaction between an organism's performance - the frequency of "coping" responses - and the probability of response-dependent signals concerning safety - "relevant feedback". Primarily, the advantages of having control are related to the safety produced by performance of a successful avoidance response. Weiss confirmed this view in subsequent studies employing both negative (Weiss, 1971c) and positive (Weiss, 1971b) feedback. It has also been supported by other researchers (Murison, Isaken & Ursin, 1981) (see Chapter 3 for a detailed discussion of studies involving feedback).

In summary, it would appear that having a controlling response available, whether it is one of escape or avoidance, generally leads to a reduction in physiological pathology. However, as was reported earlier in relation to studies involving human volunteers, the pattern of physiological responding depends upon a number of situational factors such as the difficulty of achieving and/or sustaining the necessary controlling response and not merely on the availability of a controlling response. In addition, there are a number of factors such as, experimental design parameters, which have not been fully investigated in relation to controllability. For example, it may be that differential responding to control alters as a function of the duration, number, frequency and intensity of the aversive stimulus, the type of experimental schedule employed (sidman versus signalled) or the chronicity of the experimental session. Although these variables have been investigated and discussed in great detail in relation to the predictability data (see Chapter 2), they have mostly been ignored with regard to the control literature. The question, therefore, is not whether control reduces responding to 'stress', but under what conditions it has stress-

inducing effects, and in what conditions it has stress-reducing effects. At present, nonhuman (and human) studies do not provide an answer to this question.

With regard to theoretical interpretations of the data, the studies discussed above cannot differentiate between those explanations which propose that the presence of control merely provides additional predictability (e.g. the relevant feedback, safety signal and preparatory response hypotheses), and those that suggest that controllability provides an organism with more than just extra predictability (e.g. the minimax hypothesis). All four explanations suggest that instrumental control should lead to a reduction in physiological indices of stress compared with a no control condition. Since a situation in which instrumental control is available provides either more safety signals, relevant feedback, more finely tuned preparatory responses, or reduces the maximum intensity of the noxious stimulus or event.

### 3.4. Preference For Control

3.4.1. Instrumental Control. Preference for instrumental control is predicted by all four theories. The relevant feedback hypothesis proposes that instrumental control should be preferred because an organism's avoidance or escape response is always followed by relevant feedback. For example, an escape response is always followed by warning stimulus offset and the non-occurrence of shock (i.e. excellent relevant feedback). Conversely, subjects who have no instrumental control do not perform a controlling response which, in turn, means that they receive no relevant feedback. With regard to the safety signal hypothesis, subjects in the control condition have more safety signals than those subjects who have no control. For example, in an avoidance paradigm, feedback from a subject's response and the intention to respond reliably predict no aversive stimulus, and therefore, subjects can reliably predict both stimulus (safe) and stimulus-free (danger) periods. Thus, subjects with a controlling response available can relax more than a subject with no response available. In contrast, the Minimax Theory predicts that subjects will prefer instrumental control over no control because in the former condition they have a response available which enables them to minimise the maximum duration, intensity and/or

frequency of the aversive event or situation. This therefore reduces anticipatory arousal compared with a subject who has no controlling response available to them.

A review of the literature reveals only one human study which has measured preference for instrumental control, and six out of nine subjects chose the control condition (Elliott, 1969). Unfortunately, Elliott's findings can only be used as a mere indicator of behavioural preference due to a number of methodological problems. For example, he decided only half way through running the experiment to ask subjects to state preferences. This he did after he had seen the trend of the data he had collected from subjects so far. In addition, only nine subjects gave "clear answers". Therefore, he only collected preference data from nine out of 32 subjects.

With regard to the nonhuman studies assessing behavioural preference for instrumental control, the data are limited and generally indirect. Moreover, the data which are available are mostly concerned with appetitive situations (see Osborne, 1977 for a review). Indeed, a review of the literature reveals only one study which has measured preference in an aversive situation (Abbott & Badia, 1979). Abbott and Badia (1979, Experiment 2) employed a changeover choice procedure (detailed in Chapter 2, Section 3.2.1) in which subjects could choose, after initial baseline training, whether to receive escapable or inescapable shock. In the escapable condition, shock (scrambled 75 mW, 1.5 sec duration) could be terminated by the production of a single press of the escape lever during the first 1.5 sec of shock. If the animal failed to respond, shock terminated after 10 sec. In the inescapable condition, subjects received 1.5 sec shock independent of responding.

Abbott and Badia found that there was no significant difference in the amount of time spent in either the escapable or inescapable conditions. These results could not be explained by differences in shock duration between the two conditions, as there were virtually identical durations of escapable and inescapable shock. Furthermore, these findings were replicated in another experiment carried out by the same authors (1979, Experiment 3) employing avoidable-non avoidable shock. The authors believed that avoidance would be seen by subjects as a more "potent" response than escape, as they eliminate all contact with shock. The other experimental parameters were similar to those employed in Experiment 2. Again, the authors found that when subjects were given the

opportunity to change from avoidable to unavoidable shock or, *vice versa*, none of the 12 rats did so consistently.

Abbott and Badia concluded that subjects found controllable and uncontrollable aversive stimuli equally aversive whether the controlling response was one of escape, or, of avoidance. However, a number of factors may have contributed to the lack of positive effects. For example, shock duration was relatively short in both Experiment 1 (1.5 sec) and 2 (0.5 sec) and, therefore, termination of shock was equally predictable under both controllable and uncontrollable conditions. This increased predictability may have confounded the differential effects of control. Additionally, it may be that control is only preferred in certain situations; for example, when the aversive stimulus or event is more uncertain. Such situations would include longer or more variable shock durations. Indeed, a number of researchers have found that behavioural preference for signalled shock changes as a function of experimental parameters such as; shock and signal duration, shock intensity and the type of schedule employed (i.e. fixed or variable) (see Chapter 2, Section 3.2).

A second explanation may be in terms of the within-subject design employed by Abbott and Badia. Other studies assessing the behavioural and physiological effects of controllability have used between-subjects designs. It may be that these differing designs lead to different outcomes (see Grice, 1966). Indeed, Abbott and Badia suggest that " ... allowing comparisons between both conditions teaches the animal in a rather direct way that having control over shock does not result in any change in shock parameters relative to the uncontrollable situation" (Abbott & Badia, p. 155). However, it is impossible to examine this explanation empirically especially since there have been no between-subject preference studies involving nonhuman subjects.

To conclude; the one study assessing preference in humans did find a positive effect with controllability, and this would be predicted by all four theories. In contrast, the only behavioural preference study with nonhumans found no differential effects. However, this conclusion is based on minimal and far from convincing evidence.

3.4.2. Self-Administration. Studies employing a self-administration paradigm have rarely included a measure of subject's preference for self or experimenter-administered aversive



stimuli. Indeed, only two studies have reported such a measure, one of which employed expressed preference (Pervin, 1963) whilst the other used actual preference (Ball & Vogler, 1971). Both studies found a preference for those conditions which allowed self-administration of shock. For example, Ball and Vogler (1971) employed a within-subject design in which subjects administered shock to themselves after warning signal onset or had shocks administered to them at random intervals by an experimenter. After experiencing 18 trials, subjects were allowed to decide which condition they would like to be allocated to in future trials. However, they were also told that they would receive double the amount of shocks in their preferred condition versus one for the non-preferred. After 30 trials, the experimental session ended.

The authors found that out of 39 subjects, 25 of them showed a clear preference for a single self-administered shock over a single experimenter-administered one. A further 11 showed a preference for experimenter-administered shock whereas, three subjects showed no clear preference for either condition. In a post-experimental interview, 21 of the 25 subjects who chose self-administered shock said they had done so to avoid uncertainty whereas, the reasons given for choosing experimenter-administered shock were idiosyncratic i.e. the shock was "enjoyable", or, they were "curious" about it and preferred the excitement of random shock delivery. However, the majority of subjects did not stick with their preference when it meant doubling the amount of shock in the last 12 trials.

These positive findings of self-administration are similar to those reported by Pervin (1963) (detailed in Section 3.1.2), and are predicted by the minimax, safety signal and preparatory response hypotheses. For example, the Minimax Theory suggests that if a subject agrees to deliver an aversive stimulus to themselves, they know that if the situation becomes intolerable (i.e. it surpasses their maximum danger level) they can stop delivering it. Self-administration subjects also know that if they are required to give themselves a certain number of stimuli, they will not give themselves any more than that. Nor, for example, will they hold down the shock button too long. In contrast, in the experimenter-administered condition, subjects have no guaranteed upper limit of danger. Nor do they know how many noxious events they will receive, or, that the stimuli will not last longer than expected. The Safety Signal Theory is logically similar, for example, those subjects in the self-administered condition know exactly when the aversive event will occur and

therefore exactly when it will not occur i.e. they know when to relax. During experimenter-administered conditions, subjects have none of these safety signals and therefore relax less. In terms of the safety analysis this differential relaxation is crucial, as it predicts that individuals will choose those situations where they can relax more.

The only theoretical explanation which cannot account for these findings is the relevant feedback as it predicts no effects of self-administration. This is because in a self-administration paradigm the subject delivers the aversive event to themselves and therefore receives no positive relevant feedback; i.e. subjects are producing "stimuli associated with the stressor" - the aversive stimulus itself. Thus, in terms of relevant feedback, there are no differences between self and experimenter-administered conditions.

4.

## CONCLUSION

With reference to the empirical findings, there are some areas where more research is needed before definitive conclusions can be drawn, for example, studies measuring event impact effects in self-administration paradigms, and both anticipatory and impact measures in equated and potential control studies. There are also some areas where the relevant evidence is completely absent. In contrast, other areas have been extensively researched but many of the studies are methodological unsound limiting the conclusions which might be drawn. For example, the effects of control and predictability and/or, different types of control have been confounded in one experiment; e.g. control over stimulus intensity and control over avoiding, escaping or shortening the duration of the stimulus. Other potential confounding factors are the type of controlling response available (avoidance-escape or active-passive), difficulty of exercising the response or in learning the contingency between behaviour and response, task difficulty levels and, finally, differences in individual's perception of control. It is not surprising, therefore, that the results concerning behavioural control (instrumental and self-administration) are equivocal.

With regard to preference for control, it is relatively clear that self-administered events are preferred to experimenter-administered ones. Similarly, the findings of the only human study in the area suggests that instrumental control is preferred to a no control condition. In contrast, the one study manipulating preference for instrumental control in

nonhuman subjects found no differential effects. In terms of the large number of studies investigating anticipatory arousal, the majority have found that control leads to a reduction in subjective and electrodermal stress compared with no control conditions. In contrast, differential cardiovascular responses largely depend on situational factors, such as, the difficulty of the task and/or controlling response. No firm conclusions can be drawn from the impact data, with instrumental control having either stress-reducing, stress-inducing, or little effect compared with no control conditions. Moreover, the data relating to both anticipatory and impact measures in self-administration paradigms are just as inconsistent. Those studies employing nonhuman subjects generally report that the availability of a controlling response leads to a reduction in physiological indices of stress, such as, body weight, gastrointestinal lesions and plasma corticosterone levels during the short and long-term post-impact period.

The predictability theories, which suggest that control adds nothing over and above more finely tuned predictability, differ in their ability to account for these findings. Both the safety signal view and an extended version of the preparatory response hypothesis can account for the choice and anticipatory data involving instrumental control and self-administration. With regard to impact arousal, the positive findings in which control leads to a reduction in psychophysiological stress is consistent with a preparatory response but inconsistent with a safety signal view. The relevant feedback hypothesis can only account for the choice data and those findings reporting a reduction in anticipatory responding with instrumental control.

However, all three predictability theories are inadequate to account for the stress-reducing effects associated with the control equated for predictability and potential control paradigms. Since the safety signal, relevant feedback and preparatory response views would suggest no beneficial effects in these types of situations. Yet, such effects do emerge. For example, control has been shown to have beneficial effects in terms of anticipatory (equated and potential) and impact (equated) arousal, particularly in the electrodermal system. These positive findings would only be predicted by a minimax analysis. However, any conclusions drawn from either the equated or potential control studies should be treated with caution, since the data available is minimal and in some cases far from convincing.

## CHAPTER 5

### METHODOLOGY

#### 1. INTRODUCTION

This chapter will describe the methodology employed in all or most of the studies which follow. The first section will briefly describe the parameters of electrodermal activity employed and any statistical transformations which have been included in this thesis. Most of these, and related issues, have been discussed in great detail elsewhere (see Fowles, Christie, Edelberg, Grings, Lykken & Venables, 1981; Venables & Christie, 1973, 1980). The second section presents details of the measurement, quantification and analysis of cardiovascular activity. In particular, the various response parameters chosen to represent cardiac activity throughout this thesis. This is followed by a brief discussion of the difficulties associated with the operationalisation of tonic and phasic HR activity. Again, most of these issues have been dealt with elsewhere (see Jennings, Berg, Hutcheson, Obrist, Porges & Turpin, 1981; Siddle & Turpin, 1980; Stemmler & Fahrenberg, 1989).

#### 2. METHODOLOGICAL ISSUES

##### 2.1. Electrodermal Activity

Three parameters of exosomatic electrodermal activity were examined in this thesis. Tonic activity was measured in terms of skin conductance level (SCL) and the frequency of non-specific fluctuations (NSFs) which was defined as any artefact-free fluctuation greater than 0.02 microSiemens ( $\mu\text{S}$ ). The phasic measure employed was amplitude of the skin conductance response (SCR amplitude). This was defined as any artefact free fluctuation greater than 0.02  $\mu\text{S}$  which occurred 1-5 sec after stimulus onset. SCR amplitude was obtained by measuring the difference between the peak response level and the pre-stimulus level for each response. Unless otherwise stated, a standard within-subject range-correction procedure (see Lykken & Venables, 1971) was employed in order to reduce the amount of variance due to individual differences in responsivity. Change in conductance

was measured as a proportion of each subject's response to a standard stimulus presented before the pre-task baseline period.

The distributions of all three electrodermal parameters were examined for any deviations from normality (Winer, 1971). Where the results indicated any significant skew or extremes of kurtosis, the data were transformed prior to statistical analyses. Two transformations have been most commonly proposed in the literature; namely, a square root (Schlossberg & Stanley, 1953) and logarithmic (see Venables & Christie, 1980) transformation. Both transformations were examined. For range-corrected SCR amplitude, square root transformations were found to produce the best normally-distributed scores. No transformation of tonic SCL and frequency of NSF's was necessary.

## 2.2. Cardiovascular Activity

2.2.1. Measurement. Due to the large amount of data collected in the studies reported in this thesis, a major methodological problem was the appropriate summary statistic to employ in order to best represent cardiac activity. The most common approach in the literature has been to take the mean HR activity during specified time periods. However, this would appear wasteful of a large amount of data which could be of equal interest to the experimental question under consideration.

The strategy employed in this thesis was to divide the measurements into three basic response elements; namely, peak acceleration (maximum HR), peak deceleration (minimum HR) and average HR<sup>1</sup>. It was felt that including measures of minimum and maximum HR would provide additional relevant information in that experimental manipulation may effect the pattern of fluctuations (from a mean value) but not consistently change average HR. At the same time, however, both of these measures produce substantial correspondence with mean HR. Thus, by taking multiple dependent measures

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<sup>1</sup> A fourth parameter of cardiovascular activity was considered for inclusion in this thesis; namely, measures of HR variability. However, preliminary analyses of data from Experiments 1 and 2, in terms of variance/standard deviations, suggested that no additional information could be derived by employing such a measure. Further measures of HR variability were not available to the author. Thus, HR variability will not be discussed further.

(with comparable content) it was hoped that both the reliability and generalisability of the results would be increased.

There were a number of additional reasons for taking this approach. From a psychological perspective, some researchers have argued that physiological reactivity to psychological stress provides a meaningful reflection of emotional response (e.g. Light, 1981). In particular, a handful of studies which have employed measures of peak HR (e.g. Lipton, Steinschneider & Richmond, 1961; Opton, Rankin & Lazarus, 1966; Seraganian, Hanley, Hollander, Roskies, Smilga, Martin, Collu & Oseasohn, 1985), have suggested that it yields HR curves which are markedly elevated at the most psychological stressful points; i.e. the greater the degree of involvement or effort the subject is exerting, the greater the increase in peak HR. Although measures of minimum HR have been employed less frequently as parameters of the cardiac response profile (e.g. Carriero, 1975; Lewis, Meyers, Kagan & Grossberg, 1963), they too may be of psychological interest. For example, it may be that when perceptual-motor responding is minimal, this may be reflected in greater peak deceleration or lower levels of minimum HR. Indeed, this pattern of cardiac change would be expected on the basis of work carried out by Lacey and Lacey (1970) who suggested that various heart rate changes were associated with changes in attentional and task-related information processing. Thus, although it remains unclear from past work what precise psychological meaning the various response elements may possess, it would appear prudent at the present time to continue to examine response profiles rather than to reduce activity during tasks solely to an estimate of central tendency, such as, mean level.

From a physiological stance, the rationale behind this approach stemmed mainly from the concern with cardiovascular reactivity, and in particular exaggerated activity. Recent research has focused upon momentary surges in catecholamine activity during psychosocial stress (e.g. Dimsdale & Moss, 1980) suggesting a more detailed analysis of momentary surges in other indices. However, brief surges in cardiac activity could remain undetected if averaged over time. Thus by systematically exploring individual peaks rather than average activity, a more representative picture might emerge of cardiovascular response profiles. In addition, it was hoped to draw upon the seminal work of Obrist and colleagues (see Obrist, 1981 for a review), in which they challenge the implicit assumption

that neural-humoral influences modify HR in a direct synergistic manner; i.e. as HR increases there is a loss of parasympathetic restraint and an increase in sympathetic excitation and as HR decreases, the reverse process occurs. They suggest that under conditions requiring maximal attention to environmental events (e.g. challenging tasks that necessitate active coping), cardiac effects are sympathetic in origin and independent of somatic activities. That is, cardiovascular activity is in excess of metabolic requirements. In contrast, during less demanding tasks (e.g. where no opportunity to avoid exists), vagal or parasympathetic control is dominant and, in turn, these vagal influences are directionally related to somatic activity.

Unfortunately, due to technical restraints, measures inferring neural innervations, such as impedance cardiography (Kessley & Guethlein, 1990), were not available. Therefore, further non-invasive cardiovascular indices of autonomic divisions had to be developed. One approach suggested by the work of Mizejeski (1978), was to employ measures of average, maximum and minimum HR as tools for inferring the role of parasympathetic versus sympathetic mechanisms in autonomic arousal. The study carried out by Mizejeski, in which he employed a non-aversive motor (paced finger tapping) task, was to assess the effects of behavioural arousal upon cardiovascular activity. As would be expected, all three cardiac measures - average, minimum and maximum HR - increased with increased tapping. However, their patterns of change differed. Minimum HR showed a progressive increase with each increase in the rate of finger tapping. Conversely, both maximum and mean HR remained unchanged during moderate levels of responding, and only increased above resting levels when response rate was high. The author draws on previous studies to explain why maximum and minimum HR differ in their response to levels of behavioural arousal. For example, work involving sympathetic and parasympathetic blocking agents (e.g. Robinson, Epstein, Beiser & Braunwald, 1966) which suggested that mild exercise produces parasympathetically mediated increases in HR, whilst increases in HR during intense activity results in part from sympathetic activation.

Mizejeski suggests, therefore, that the different patterns of change involving average, minimum and maximum HR may mean that these measures index different neural mechanisms. For example, the monotonical relationship between minimum HR and

behavioural arousal suggests the incremental removal of parasympathetic inhibition, whereas the increase in maximum HR during high behavioural arousal suggests that the condition was intense enough to elicit a sympathetic response. However, Mizejeski's interpretation obviously requires caution mainly because neither parasympathetic or sympathetic blockers were administered. Therefore, any links between the three response measures and neural innervations remain at a purely hypothetical level.

It was therefore decided to build on the work of Mizejeski but using aversive, non-motor tasks. However, two limitations with this approach should be mentioned. First, as with Mizejeski's study, no sympathetic or parasympathetic blockers were administered to subjects. Thus, any differences in the pattern of activity between the three cardiac measures are purely speculative in terms of specific innervations. In addition, even when suggestions are made as to the patterns of neural control, it is still not possible to assess individual differences in the autonomic origins of this reactivity. For example, it may be that increases in average and maximum HR originate in elevated sympathetic reactivity, vagal withdrawal or the reciprocal activation of both autonomic divisions. Conversely, minimum HR could stem from low sympathetic and/or high vagal innervation. Second, metabolic activity (i.e. changes in O<sub>2</sub> consumption) was not assessed. It is therefore not possible to reliably attribute differential patterns of activity with changes in behavioural arousal as the response may simply be a result of the physical effort exerted in response generation. Although the motor components of the tasks, both reaction time and verbal, were not ignored in the studies which follow, it may be for example, that articulatory responding and the resulting interference with normal breathing patterns may have affected cardiac function. Nevertheless, use of the three measures remains potentially interesting if only indicative.

Although to a large extent based upon psychological and physiological principles, the decision to employ the three response measures also involved statistical examination of the data. This was principally to see whether average, maximum and minimum HR defined unique aspects of responsivity. The data from the 56 subjects in Experiment 1 were used for this purpose. Table 6 presents the raw data during each of the 17 time periods (see Chapter 6, Section 2 for a full explanation of the design) of Trial 1 for the three response measures. Visual inspection of the means reveals that the three measures, although



Table 6. Means and standard deviations for average, minimum and maximum HR across the time periods of Trial 1, Experiment 1.

Time Period	Means (SDs in parentheses)		
	Average HR	Minimum HR	Maximum HR
1	86.2 (12.5)	64.3 (13.9)	107.2 (11.1)
2	90.2 (12.6)	82.7 (14.8)	97.4 (12.6)
3	90.5 (13.8)	82.6 (15.5)	98.3 (13.4)
4	95.6 (13.5)	86.0 (16.5)	103.7 (11.9)
5	97.2 (12.8)	84.8 (18.7)	106.7 (11.8)
6	90.8 (13.3)	81.4 (16.4)	100.2 (12.5)
7	87.4 (13.6)	79.8 (14.3)	96.0 (12.5)
8	87.4 (13.6)	77.4 (14.2)	93.8 (11.4)
9	84.5 (12.5)	77.4 (13.6)	92.3 (11.7)
10	87.2 (12.8)	79.1 (13.7)	95.7 (12.8)
11	86.1 (13.6)	77.6 (14.9)	94.8 (13.9)
12	83.4 (11.8)	76.0 (12.5)	92.1 (12.4)
13	82.6 (11.7)	75.2 (12.3)	90.9 (11.9)
14	80.8 (11.6)	71.4 (13.0)	90.8 (12.2)
15	82.1 (12.8)	75.3 (13.0)	89.5 (13.1)
16	81.0 (10.5)	73.7 (12.6)	89.9 (13.0)
17	82.7 (10.5)	74.8 (11.4)	91.2 (11.9)

showing a large amount of communality, do have different patterns of change across phases of the experiment. Indeed, the data suggests that peaks and troughs in the response profile for each of the measures emerge at different times during the task i.e. the measures respond differently to the challenges exerted upon an individual and to the degree of effort expended. This can be seen particularly in the first half of the trial (i.e. Time Periods 1 to 8), during which time task uncertainty and involvement is at a greater level compared with later phases of the trial (i.e. Time Periods 9 to 17).

In order to further examine the data, the interrelationships between average, maximum and minimum HR were determined by correlational analyses. Inter-individual Pearson correlation coefficients were employed. As can be seen from Table 7, the use of correlational procedures appears to provide partial support for the inclusion of the three response parameters. As would be expected, on the basis of the raw scores, all three measures show a strong positive correlation with one another. In particular, the relationship between average HR and both minimum and maximum is highly significant across the time periods of the task, although the correlation between average and maximum HR is slightly higher. A finding which is predicted by the work of Mizejeski (1978). Furthermore, the relationship between average and maximum HR varies only slightly across time periods; i.e. they respond in a similar manner to the task, whereas average and minimum HR produce slight variations during 'low' (i.e. Time Period 1) (all trials with the exception of Trial 1) and 'high' (i.e. Time Period 5) behavioural arousal. The relationship between minimum and maximum HR shows a greater degree of variability across time periods. Nevertheless, all correlation coefficients are still statistically significant. Similar results were found for Trials 2 and 6.

To further complement this analysis, the data were examined using exploratory factor analysis. An orthogonal principle factor analysis followed by a Varimax rotation was employed on the average, minimum and maximum HR data. The analysis produced five factors all of which had eigen values greater than 1.0. Factors 4 and 5 were uninterpretable and loaded only on single or at most pairs of variables. However, three factors appeared which were interpretable and accounted for 80.4% of the variance. The loadings of these are shown in Table 8. Factor 1 clearly loads on Time Periods 11 to 17. Factor 2 loads on Time Periods 2 to 7 whilst the third factor loads on Time Periods 7 to 9. Therefore, three

Table 7. Pearson correlation coefficients for average, minimum and maximum HR across the time periods of Trial 1, Experiment 1.

Time Period	Correlation Coefficients		
	Average-Minimum HR	Average-Maximum HR	Minimum-Maximum HR
1	.68	.66	.48
2	.91	.94	.77
3	.92	.89	.74
4	.90	.93	.74
5	.76	.90	.54
6	.87	.93	.73
7	.95	.90	.83
8	.84	.87	.77
9	.97	.93	.85
10	.97	.89	.77
11	.89	.91	.68
12	.91	.91	.74
13	.91	.86	.66
14	.88	.88	.64
15	.91	.94	.79
16	.92	.89	.72
17	.90	.90	.67

Note: All correlations significant at the  $p < .01$  level

Table 8. Factor loadings of average, minimum and maximum HR on three factors from orthogonal factor analysis after varimax rotation.

Variables: *		Factor Loadings		
Time Period		1	2	3
1	AvHR	-.**	-	.50
	MnHR	-	.52	-
	MxHR	-	-	-
2	AvHR	-	.68	-
	MnHR	-	.56	-
	MxHR	-	.71	-
3	AvHR	-	.67	-
	MnHR	-	.55	-
	MxHR	-	.76	-
4	AvHR	-	.86	-
	MnHR	-	.77	-
	MxHR	-	.83	-
5	AvHR	-	.85	-
	MnHR	-	.62	-
	MxHR	-	.81	-
6	AvHR	-	.69	.56
	MnHR	-	.51	.64
	MxHR	-	.74	-
7	AvHR	-	.60	.71
	MnHR	-	.57	.74
	MxHR	-	.57	.63
8	AvHR	-	.60	.71
	MnHR	-	-	.68
	MxHR	-	-	.70

9	AvHR	-	-	.79
	MnHR	-	-	.80
	MxHR	-	-	.73
10	AvHR	.71	-	-
	MnHR	.76	-	-
	MxHR	-	.60	-
11	AvHR	.77	-	-
	MnHR	.66	.54	-
	MxHR	.77	-	-
12	AvHR	.85	-	-
	MnHR	.78	-	-
	MxHR	.87	-	-
13	AvHR	.83	-	-
	MnHR	.78	-	-
	MxHR	.79	-	-
14	AvHR	.81	-	-
	MnHR	.72	-	-
	MxHR	.79	-	-
15	AvHR	.84	-	-
	MnHR	.77	-	-
	MxHR	.85	-	-
16	AvHR	.86	-	-
	MnHR	.78	-	-
	MxHR	.84	-	-
17	AvHR	.89	-	-
	MnHR	.81	-	-
	MxHR	.82	-	-

Note: \* Only those variables having a loading on Factors 1, 2 and 3 > .50 shown in this table

\*\* Values < .50 indicated by a dash (-) for reasons of clarity

clearly defined responses do exist in the data. However, there are also slight variations in these response patterns across the three cardiac measures. For example, two time periods (1 and 10) show different loadings for each of the three measures. Time periods 1 and 10 correspond to phases of 'low' (i.e. the inter-trial interval) and 'high' task involvement (i.e. when subjects are asked for their second response) respectively. In addition, there are a minority of time periods in which the three measures load on more than one factor, such as Time Period 6, in which average and maximum HR load to a greater extent on Factor 2 whereas, minimum HR is accounted for more by Factor 3. Time Period 6 involves a phase of 'high' attention and effort (i.e. the 8 sec after subjects have been asked for their first response). Again, similar results were found for Trials 2 and 6.

The outcome of this analysis is to suggest that, on the whole, all three measures respond in a similar manner to this type of behavioural challenge. However, there are specific instances in which the patterns of change do differ and, although the statistical evidence was weaker than expected, it was decided that there was sufficient reason to make the use of all three cardiac measures potentially interesting.

**2.2.2. Quantification and Analysis.** The scaling of cardiovascular activity has at least three important aspects: first, the selection of a unit of measurement; second, the determination of a reliable baseline as a reference value; and third, the selection of a change score. The first of these points has been covered in Section 2.2.1. The second, baseline references, is concerned with the differentiation of tonic and phasic components of cardiac activity. Both of these components cause considerable difficulties in operationalisation. Tonic activity is usually conceived as a steady state without noticeable trends. However, the definition of an adequate baseline and the introduction of an initial rest period of reduced activity has been a controversial issue in cardiovascular psychophysiology (e.g. Gale & Baker, 1981; Fahrenberg, 1983; Jennings, Berg, Hutcheson, Obrist, Porges & Turpin, 1981) due to the fact that there is no such thing as a 'true resting heart rate' or a stationary phase. Instead, what is observed is a heart rate that has trends and periodicities. Measures of tonic activity employed in this thesis have, therefore, been taken over a long enough time scale to incorporate recurrent phases such as, respiratory sinus arrhythmia (RSA). Thus, what is meant in this thesis by tonic activity is a *relative* steady state

indicating resting conditions without marked trends during the observation interval before onset of the task.

In all of the studies which follow, these initial baseline values have been employed as a reference for computing change scores to represent phasic activity in the subsequent experimental period and, to eliminate any intra-individual variance due to differences in end-organ activity. These particular individual differences are mostly unrelated to task induced changes and, therefore, are not of direct interest to the experimental question under consideration. There have been serious criticisms expressed in the literature concerning change scores that refer to a specific 'initial value' due to biases such as the "psychophysiology of initial effects". These so-called biases occur because the initial baseline period is in itself a potentially stress-inducing condition and the magnitude of this effect may be confounded with various lasting (traits) or temporary (states) subject characteristics.

The identification of an adequate change score is therefore a fundamental problem in psychophysiology and, as such, cannot be discussed in detail here (see Levey, 1980; Fahrenberg, 1983 for reviews). Various measures have been proposed and several comparative analyses carried out (Fahrenberg, Foerster, Muller, Myrtek & Schneider, 1985; Llabre, Spitzer, Saab, Ironson & Schneiderman, 1991; Myrtek, 1984, 1985; Seraganian *et al.*, 1985; Stemmler & Fahrenberg, 1989). These have compared several kinds of change scores ranging from simple difference scores to those which take into account the Law of Initial Values (LIV) (Wilder, 1967). The most notable of these is covariance analysis, especially Lacey's (1956) Autonomic Liability Score (ALS). Based on several extensive data sets, Myrtek and co-workers concluded that simple difference scores were generally sufficient for use as change scores under a linear growth model, as they made fewer statistical assumptions, were sample independent, relatively stable, concrete and computable for single subjects. With reference to comparisons made with other more complex change scores, such as the ALS, they suggest the use of simple difference scores in the majority of situations with the ALS and other transformations being reserved for specific purposes. A position supported by Cattell (1982). Thus, in the studies reported here, an individual's reactivity will be assessed using raw scores and simple differences.

Similar problems arise when quantifying event-related phasic responses or the cardiac response curve (CRC). The CRC is usually expressed as the difference between a mean pre-stimulus level minus individual post-stimulus seconds. However, this approach presupposes that pre-stimulus activity is stable. The presence of large cyclic fluctuations such as, RSA limits such an assumption. Two particular problems arise. First, the mean pre-stimulus level may be influenced by the frequency, amplitude and phase of RSA, and second, the CRC is superimposed upon RSA bound cardiac activity and so the amplitude of these phasic responses will depend upon the direction of RSA at the time of event presentation. Therefore, the CRC will be composed of the response to the event and ongoing stimulus-irrelevant cardiac activity.

One possible way to reduce the amount of error associated with pre-stimulus variability is by extending the pre-stimulus sampling interval to obtain a more reliable measure of pre-stimulus level. Although this measure has been included in the studies which follow, it does not take into account any cyclic fluctuation in the post-stimulus period. Other procedures which attempt to overcome this problem such as, the RSA-corrected method (Turpin & Siddle, 1978) and time series analysis (Jones, Crowell & Kapuniai, 1969) were also considered. Criticisms of both of these techniques have been discussed in detail elsewhere (e.g. Cort, Edholm, Lobstein & Webb, 1977; Siddle & Turpin, 1980). After consideration, neither of these techniques were employed in this thesis, mainly because it was felt that the effects of RSA upon the CRC would be averaged out when stimulus presentations were random and when data was averaged across trials and subjects.



## CHAPTER 6

### EXPERIMENT 1

#### 1. INTRODUCTION

One of the psychosocial variables said to mediate responses to aversive stimulation is feedback. That is, information which is given to subjects concerning their performance on a task. As has been discussed earlier (see Chapter 3), two types of feedback have been identified. The first is termed KR, and involves providing subjects with some performance measure not usually available to them in order to facilitate subsequent performance. The effects of KR have been reviewed in Chapter 3, Section 2. In general, studies have found that KR can improve performance relative to no feedback conditions. In addition, those studies which included measures of physiological function have generally found that the availability of this form of feedback leads to an increase in responding compared to no feedback conditions. However, there are a number of conceptual and methodological problems associated with KR which make these findings difficult to interpret. The most important in terms of this experiment is that in the majority of KR studies it is not possible to attribute any changes in performance or physiological activity to feedback *per se*. By providing detailed 'directive' information, KR changes an individual's ability to cope with the situation not only by the feedback provided but also because KR and improved performance simultaneously increase perceived predictability and control over the situation. Thus, any changes in performance or autonomic function could be a result of any or all of these factors.

The second type of feedback, that is RF, consists of information only to the level of 'right-wrong' or 'success-failure'. RF therefore provides little or no additional information which individual's can use to control subsequent responding, or, to enhance perceived predictability and control over the situation. Manipulation of this type of feedback would therefore seem to be the most straightforward. Nevertheless, there has been relatively little work in the area. Those studies which have employed RF have mainly been concerned with performance effects, and in general have found that feedback can be of benefit in terms of increasing task performance compared with no feedback conditions. However, in

certain situations (i.e. complex, decision-making tasks), RF may lead to performance decrements over time because it provides no 'how and why' information which can be used to facilitate future responding through sustaining individual's involvement/engagement on the task. Few studies have examined the effects of RF on physiological function. Of these, the provision of feedback has not significantly changed electrodermal or cardiovascular responding in human volunteers during complex tasks compared to no feedback conditions. Those studies involving nonhuman subjects have reported greater levels of gastrointestinal pathology in no feedback compared to feedback conditions (see Chapter 3).

The aim of this first experiment was to separate out the effects of feedback from those of predictability and control via employment of a stress-inducing game. The game itself was of an information-handling, goal directed nature devised to manipulate RF whilst keeping task control and predictability relatively constant. It was also devised to produce strong autonomic reactivity patterns. The emphasis was therefore on psychophysiological stress evoked by the task and any changes of this stress produced by feedback. However, from the sparse information available in the literature, only tentative hypotheses could be made. Firstly, that the receipt of feedback will augment autonomic activity as seen by an increase in electrodermal and cardiovascular levels, compared to a no feedback condition. Secondly, that the provision of feedback will initially increase levels of task performance compared to a no feedback condition but that feedback, over the course of the task, will lead to performance decrements compared with a no feedback group who will produce little or no change in performance over time.

## 2. METHOD

### 2.1. Subjects

The subjects were 52 undergraduate volunteers (37 females and 15 males), with an age range of 18 - 40 years. All subjects were randomly allocated to one of the two experimental conditions; feedback (Fb) or no feedback (NFb). The only restriction to the

random allocation was that there were equal numbers of the smaller, male population in each of the two groups.

## 2.2. Apparatus

An IBM compatible computer was used to run the measurement software supplied by Contact Precision Instruments. This software controlled recording of all physiological and subjective responses via a micro couple interface, A-D converter (Type MC8) linked to the measurement hardware via a CL04 daisy chain connector system.

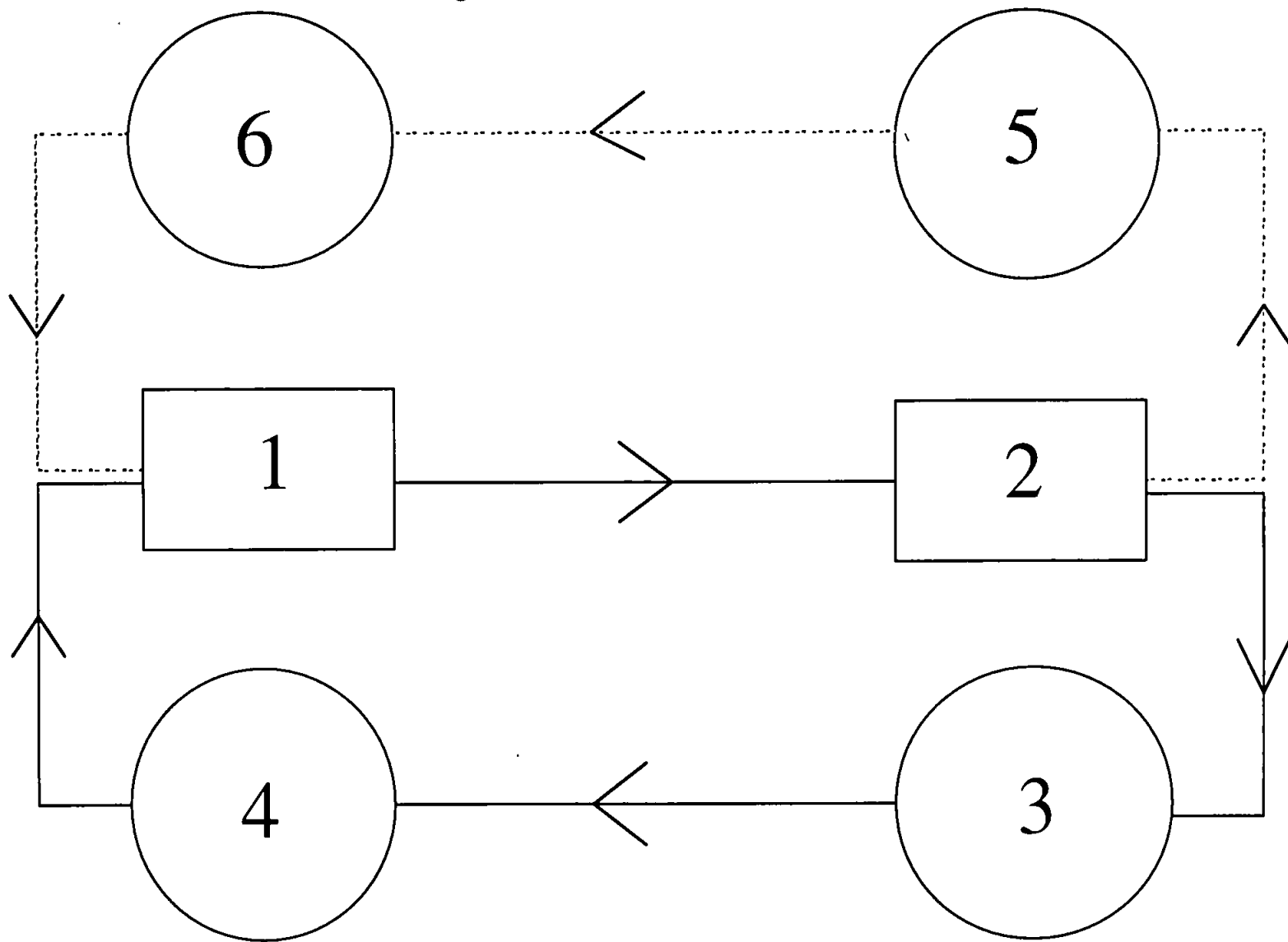
The measurement hardware was also supplied by Contact Precision Instruments and consisted of a Skin Conductance (SC) coupler (Type SC4) connected to two Ag-AgCl electrodes via a remote terminal block. The electrodes were filled with 0.05m NaCl electrolyte and attached to masked areas of the distal phalanx of the index and second fingers of the left hand. The total area from which conductance was recorded was 0.35 cm<sup>2</sup>. A constant voltage of 0.6V was applied across the electrodes. A second coupler recorded Finger Pulse Volume (FPV) from a plethysmograph transducer (Type PT1) attached to the fourth finger of the left hand. The output from the coupler was linked to an interval timer to provide a measure of Inter Beat Intervals (IBI) obtained on a beat by beat basis. The output from both couplers was recorded by the IBM compatible computer.

The air traffic control game was delivered using a BBC, model B microcomputer which was situated in the experimenter's room. The game was displayed on a colour monitor of 15 by 13.5 inches situated in the subject's room. This was positioned 18 inches from the subject and slightly to their right. The BBC also generated the noise used to signal the start of different parts of the trial, and this was presented via a loudspeaker of 12 by 7.5 inches situated in the subject's room. The loudspeaker was positioned 24 inches away from the subject and 5 inches above the colour monitor. Noise intensity was 68 dB.

The BBC was connected to the IBM compatible computer, so that all events being controlled by the BBC were also recorded alongside the subject's physiological and subjective responses.

The air traffic control game consisted of two juxtaposed oval flight paths which flowed in opposite directions (see Figure 2) and four 'planes' lettered A, B, C and D. The

Figure 2. Air Traffic Control Game



two paths coincided at points 1 and 2 after which, the dotted (upper) path continued in an anticlockwise direction, and the filled (lower) path followed a clockwise route. Therefore, all four planes could use positions 1 and 2. The lower points 3 and 4 were unique to planes B and D, and the upper positions 5 and 6 were unique to planes A and C. Planes A and C moved two places per move and Planes B and D one place per move.

The object of the game was to co-ordinate the flight of all four planes around their respective circuits without 'crashing'. This could be done by changing the speed of a plane; i.e. planes on the upper path (A and C) could be slowed down to one place per move, and planes on the lower path (B and D) could be speeded up to two places per move. However, the speed of only one plane could be changed for each move, and on the second move in a trial, any plane which had changed speed on the first move automatically reverted to its normal speed and could not change speed again for the second move. Each trial consisted of two moves for all planes given a start position which was unique to each trial. Correct responses over both halves of a trial constituted a successful move. Failure occurred where a 'crash' or illegal move was made during either or both halves of a trial.

An example of a successful attempt would, therefore, be as follows: When the starting positions are A2, B3, C5 and D1, correct and legal first positions would be A6, B4, C1 and D2, and correct second positions would be A2, B1, C5 and D3. As no two planes occupy the same position simultaneously (i.e. 'crash'), the trial would be successful.

The above example is considered to be an easy trial, as it was not necessary to employ the change of speed rules to avoid crashing. There were four types of trials: easy, as described above; intermediate, in which one change of speed was needed in order to prevent crashing; difficult, in which a change of speed was needed on both moves, and very difficult, in which subjects had to change the speed of a plane on the first half of a trial in order to avoid crashing in the second half. There were three easy, intermediate and difficult trials and two very difficult trials. These were presented in a randomised order (see Table 9).

A set of 5 keys, numbered 1, 2, 3, 4 and 5 were situated in the experimenter's room and were used to record the subject's reported feeling of control after each trial. The keys corresponded to a five point rating scale positioned at the subject's eye level. The

Table 9. Task difficulty as a function of trials and starting positions during Experiment 1.

Task Difficulty	Trial Number	Starting Positions
Easy	1	A2 B3 C6 D4
	7	A2 B4 C1 D3
	11	A5 B2 C1 D4
Intermediate	4	A5 B2 C6 D4
	9	A2 B3 C5 D1
	10	A6 B3 C5 D4
Difficult	3	A1 B4 C2 D3
	6	A5 B1 C2 D3
	8	A5 B4 C6 D3
Very Difficult	2	A2 B1 C5 D3
	5	A6 B4 C2 D3

rating scale had numbers one through to five from left to right, with 1 being labelled "Feel in complete control" and 5 labelled "Feel I have no control".

A two-way intercom was also in operation throughout. It was situated 8 inches from the subject and directly in front of them.

### 2.3. Procedure

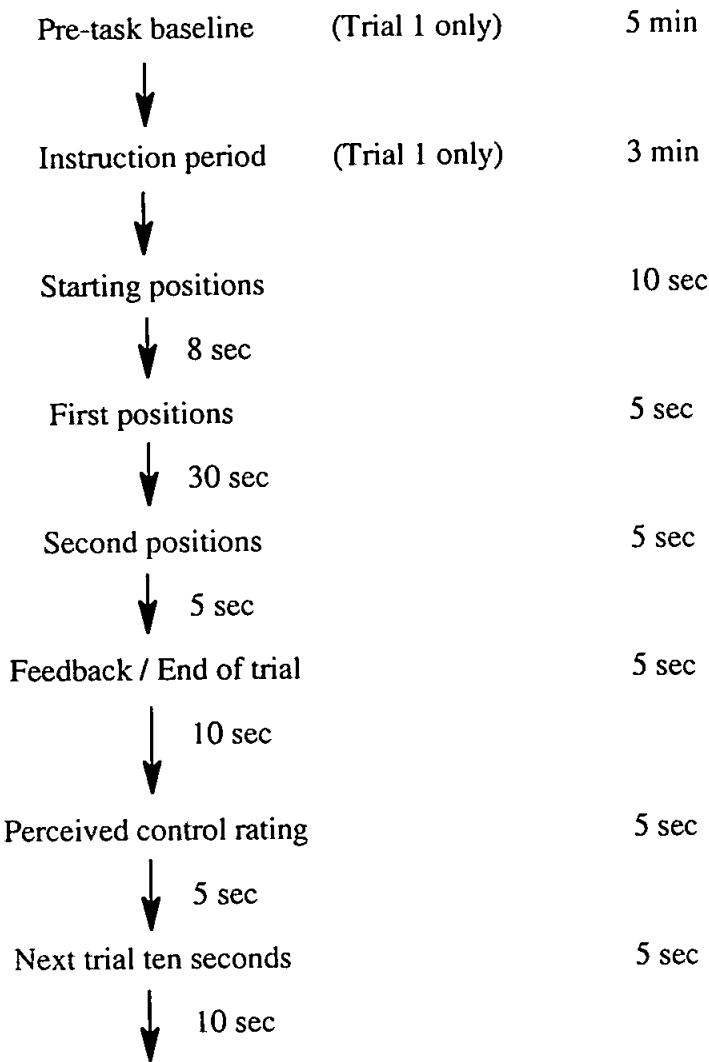
Subjects were seated in an upright padded chair in a soundproof room with a temperature range of 22-27°C. The stimulus equipment and recording apparatus were situated in an adjoining room. After the attachment of electrodes and transducer, subjects were informed of the procedure which was to follow.

The experimental procedure commenced with a five minute resting period. Following this, the subject was given the instructions necessary to play the game (see appendix). There were two sets of instructions, depending on the group to which subjects had been allocated; i.e. Fb or NFb. The only difference between these was that subjects in the Fb group were informed that, after every trial, they would be told on the screen whether they had been successful or not. Subjects in the NFb group were told that they would be informed on the screen that the trial was over. The subject was then given 3 min in which to study the instructions. During this period, the flightpath map was displayed on the subject's monitor.

Following these 3 min, the experimenter went back into the subject's room in order to confirm that subjects understood the instructions. The instruction sheet was removed, and subjects were informed that the experimental trials would begin immediately.

Each trial had a different starting position for all four planes, but following this, all eleven trials followed exactly the same format (see Figure 3). The start of each trial was signalled by a noise of 0.5 sec duration. The same noise was used to signal the presentation of other events on the screen; i.e. onset of feedback, end of the trial, the cue to give perceived control and information that the next trial started in ten seconds. The map of the flightpath was displayed from the beginning of each trial until the subject had been asked for the second position of the planes for that trial. The map was then removed. The starting positions of the planes were also displayed from the start of the trial and removed

Figure 3. Experimental procedure during Experiment 1.





after 10 sec. "What is your next move?" was then displayed for 5 sec during which there was a noise consisting of five single 'beeps' of 0.5 sec duration followed by one continuous 'beep' of 3.5 sec. The program then automatically stopped until subjects gave their next positions of the planes over the intercom. The experimenter then entered these into the BBC and re-started the program. There was a 30 sec wait before subjects were again asked "What is your next move". This remained on the screen for 5 sec and coincided with the same noise as described above. The program stopped and waited for the experimenter to enter the last positions. After entering the numbers and restarting the program, there was a 5 sec wait before subjects in the Fb group were given feedback on the screen consisting of "Your planes have crashed/are safe", and subjects in the NFb group were told on the screen "End of trial". After this, 10 sec elapsed before "Please rate your perceived control" was displayed on the screen. The subject then voiced a number from 1 through to 5 corresponding to the rating scale positioned in front of them. Following a 5 sec wait, subjects were informed that "The next trial would begin in 10 seconds". After these 10 sec had elapsed, the next trial began. After the subject had completed 11 trials, the experimenter unwired and debriefed the subject.

## 2.4. Scoring

2.4.1. Cardiovascular activity. IBI's were converted to HR and expressed as beats per minute (bpm). Three cardiovascular measures were assessed; average HR (avHR), peak acceleration (maximum HR (mxHR)) and peak deceleration (minimum HR (mnHR)). For a more detailed discussion of these measures, see Chapter 5, Section 2.1.1.

All three cardiovascular measures were assessed during the last 60 sec of the 5 min pre-task baseline in order to examine any pre-manipulation, between-group differences.

In order to demonstrate task influences on cardiovascular activity, each trial was divided into 17 separate time periods (see Table 10). Within-group comparisons were then made both across time periods and across trials. These within-group comparisons were carried out for both uncorrected and corrected HR for all three cardiovascular measures. Comparisons involving corrected HR were included in order to minimise any individual differences in cardiovascular activity. Corrected HR scores were calculated as the

Table 10. Time periods and stimuli during Experiment 1.

Time Period No.	Stimulus
1	Inter Trial Interval onset - offset
2	"Starting position" onset - offset
3	"Starting position" offset - "Next position" onset
4	"Next position" onset - offset
5	"Next position" offset - Subject's first response
6	0 - 8 sec after subject's first response
7	9 - 16 sec after subject's first response
8	17 - 24 sec after subject's first response
9	30 sec after first response - "Next position" onset
10	"Next position" onset - offset
11	"Next position" offset - Subject's second response
12	Subject's second response - "Fb/End of trial" onset
13	"Fb/End of trial" onset - offset
14	"Fb/End of trial" offset - "Perceived control" onset
15	"Perceived control" onset - offset
16	"Perceived control" offset - "Next trial 10 sec" onset
17	"Next trial 10 sec" onset - offset

difference between the last 60 sec of the pre-task baseline and raw HR and were expressed as changes in bpm.

In order to demonstrate between-group differences in cardiovascular activity, avHR, mxHR and mnHR were assessed across time periods and across pre-selected trials. The rationale behind choosing selected trials is given in Section 3.3. Both uncorrected and corrected HR were examined for all three cardiovascular measures.

2.4.2. Electrodermal activity. Tonic SCL and frequency of NSF's were assessed during the last 60 sec of the 5 min pre-task baseline.

Within-group comparisons were assessed for both tonic SCL and NSF's across time periods and across trials.

In order to demonstrate between-group differences in electrodermal activity, SCL and frequency of NSF's were assessed across time periods and across pre-selected trials. The trial numbers used in the analysis were the same as for cardiovascular measures.

2.4.3. Task Performance and Perceived Control. Between-group differences in both task performance and perceived control were assessed across trials.

### 3. RESULTS

In general, the results reported in this thesis have been derived using multivariate analysis of variance (MANOVA) models. Where these models have included one or several repeated measures factors, Greenhouse-Geisser epsilon was employed to calculate degrees of freedom (Winer, 1971). Unless otherwise stated, a significance level of 0.05 has been employed throughout this thesis. Likewise, unless otherwise stated, post-hoc comparison of means were examined using Fisher's Least Significant Difference (LSD) test.

### 3.1. Baseline Activity

With all measures, differences between groups were assessed during the last 60 sec of the baseline in a 1-way analysis of variance (ANOVA) with feedback (feedback/no feedback) as a between subject factor.

3.1.1. Cardiovascular activity. There was no significant main effect of feedback for any of the three measures (all  $F$ s < 1). Group means for avHR were 79.3 and 79.0 bpm for Fb and NFb groups respectively, mnHR means were 62.3 and 61.4 bpm and for mxHR, 100.2 and 100.1 bpm.

3.1.2. Electrodermal activity. There was no main effect of feedback involving tonic SCL ( $F < 1$ ) (1.9 and 1.7  $\mu$ S for Fb and NFb groups respectively). There was no significant effect of feedback for frequency of NSF's ( $F(1, 50) = 2.05$ ) (9.85 and 6.92 for Fb and NFb groups respectively).

### 3.2. Within-Groups Analysis

Within-group comparisons were carried out across all time periods and trials in order to assess the effects of experimental manipulation as a whole. All measures were therefore analysed in a 2-way MANOVA with repeated measures on both factors; trials (11 levels) and time periods (17 levels).

#### 3.2.1. Cardiovascular activity

3.2.1.1. Uncorrected Heart Rate. There was a highly significant difference across time periods for avHR ( $F(3, 153) = 62.9, p < .001$ ), mnHR ( $F(4, 192) = 37.4, p < .001$ ) and mxHR ( $F(5, 269) = 59.8, p < .001$ ). There was also a highly significant difference across trials for avHR ( $F(3, 151) = 29.3, p < .001$ ), mnHR ( $F(4, 187) = 17.1, p < .001$ ) and mxHR ( $F(3, 171) = 31.6, p < .001$ ). A trials by time periods interaction was revealed for all three cardiovascular measures: avHR ( $F(22, 1102) = 3.6, p < .01$ ), mnHR ( $F(23, 1162) = 2.79, p < .01$ ) and mxHR ( $F(26, 1306) = 3.4, p < .01$ ).

As the interaction involved multiple time periods and trials, visual presentation was used in order to facilitate its interpretation. First, for avHR (Figure 4), there was a similar pattern of responding across time periods for all trials. This consisted of an increase in levels of activity across Periods 1 to 5 with a steady deceleration from this peak until Period 9. At this point, there was a second increase in HR, but of a smaller magnitude than the first peak. Following this, there was a steady deceleration until Period 14 after which, there was a stabilisation in HR until the end of the trial. An inspection of Figure 4 suggests that the trials by time periods interaction is mainly due to differences across Periods 1 to 7. Trials 1 and 2 produced higher HR during these periods compared with the remaining trials whereas, during Time Periods 8 to 17 the difference between Trials 1 and 2 compared with the remaining trials was not as marked. There was also a more marked difference between the peak of activity (Time Periods 4 and 5) and the other time periods in the first two trials than was the case for subsequent trials. However, the overall pattern of responding appears to take a similar form for all trials, along with a general decrease in activity across trials.

MxHR (Figure 5) reveals a pattern of responding across trials which is very similar to avHR. There is the same increase, peaking at Time Period 5 and the secondary, smaller peak at Time Period 11. Again, this pattern of responding is more pronounced for the first two trials, particularly during the first peak. The most marked difference between avHR and mxHR is the elevated mxHR for the first trial during Time Period 1.

MnHR (Figure 6) reveals a less distinct pattern of responding across trials. All trials produced an overall increase in HR across Periods 1 to 4, followed by an inconsistent pattern of responding up until Period 9. Following this, all trials, with the exception of Trial 4, produced a second increase in levels of activity, but of a smaller magnitude than the first peak. Then, Trials 2 and 3 and Trials 5 through to 11 produced a sharp decrease in HR compared with Trials 1 and 4 which remained relatively elevated (Periods 10 to 12). After this point, all trials produced similarly inconsistent patterns of responding until the end of the trial.

This pattern of cardiovascular responding across time periods is revealed more clearly when data is averaged across trials. Multiple comparison of means reveal that for avHR (Figure 7), there was higher HR during Periods 2, 3 ( $p < .01$ ), 4, 5 ( $p < .001$ ), 6 ( $p$

Figure 4. Average HR as a function of time periods across Trials 1-11.

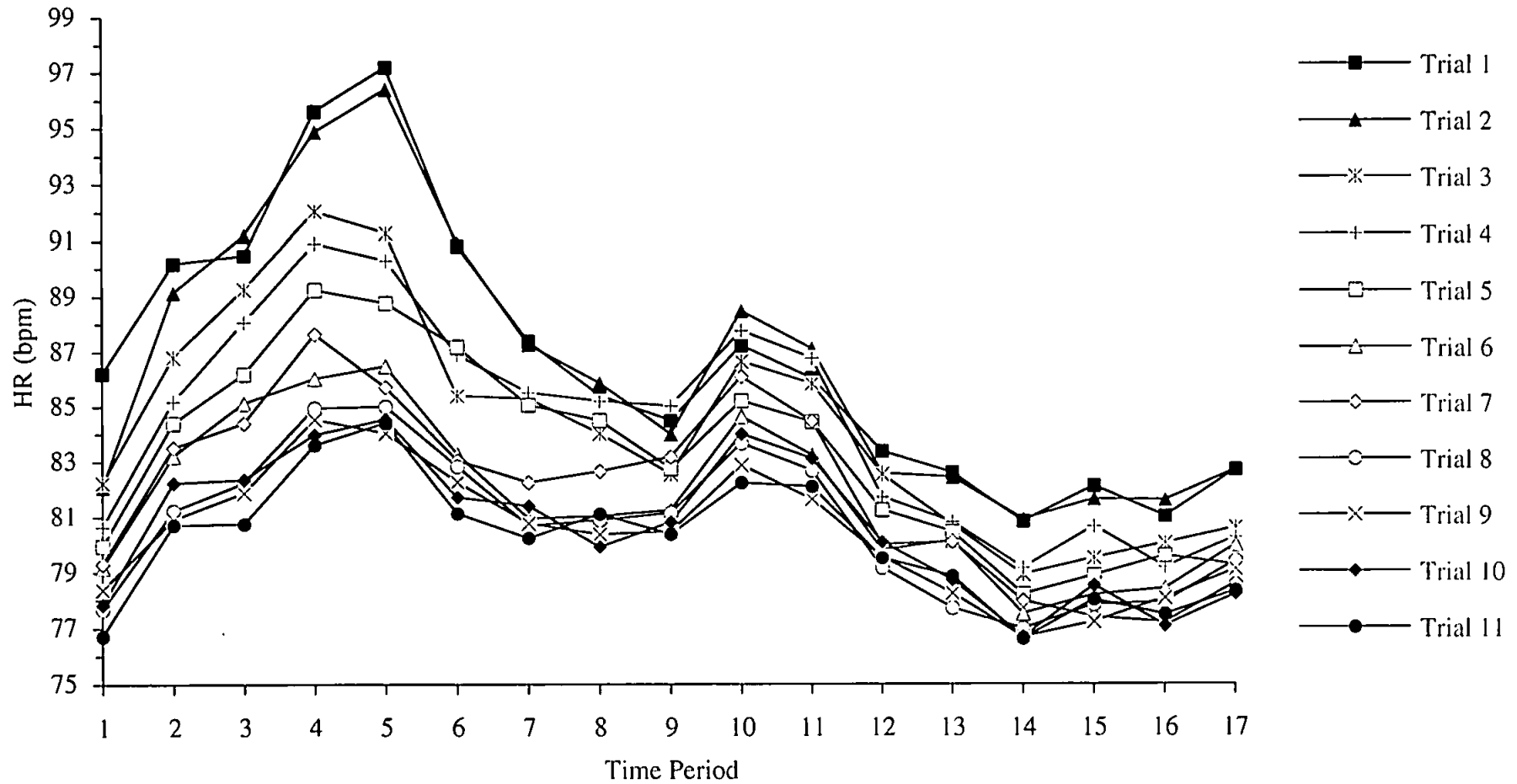


Figure 5. Mean maximum HR as a function of time periods across Trials 1-11.

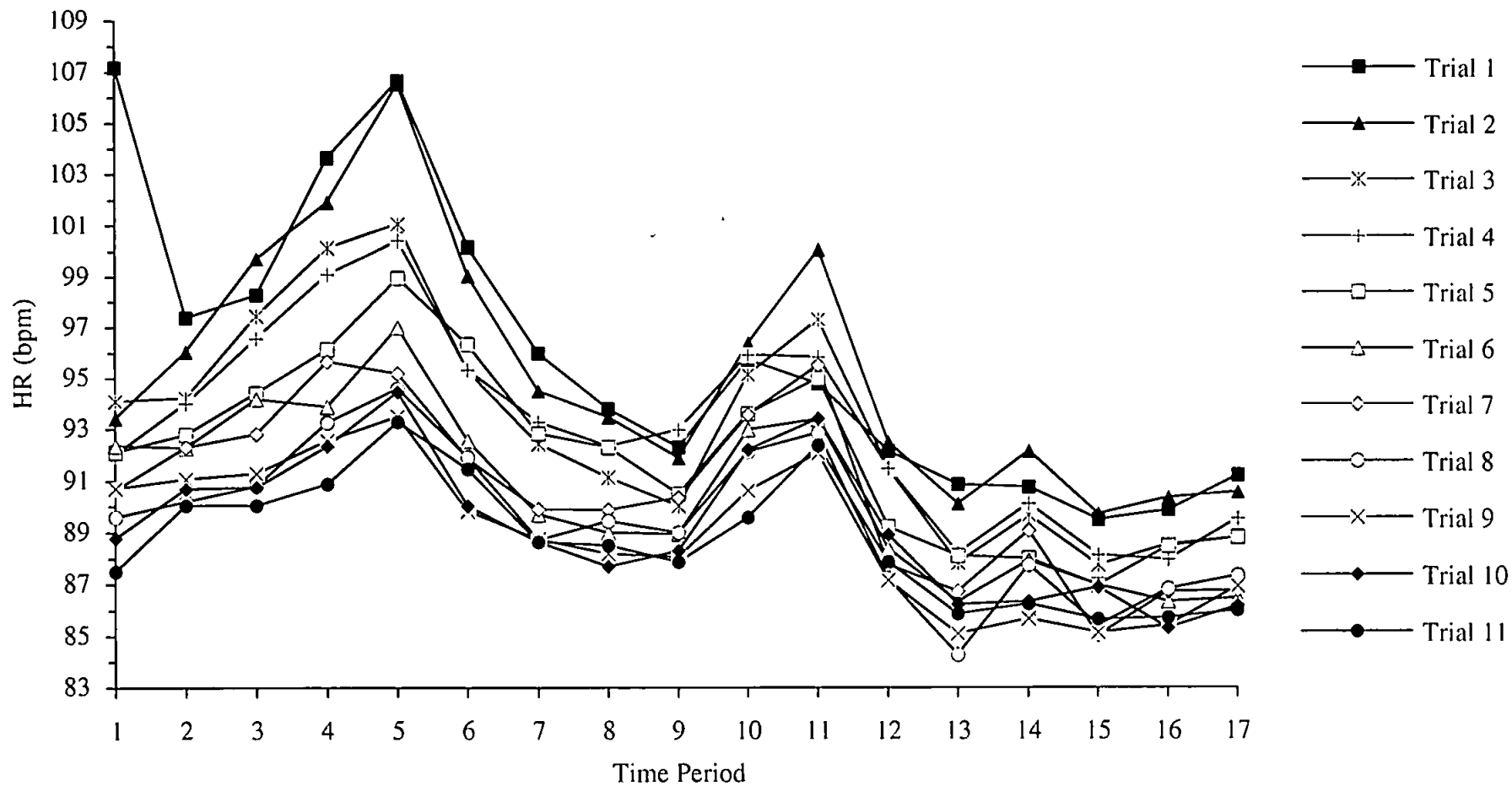


Figure 6. Mean minimum HR as a function of time periods across Trials 1-11.

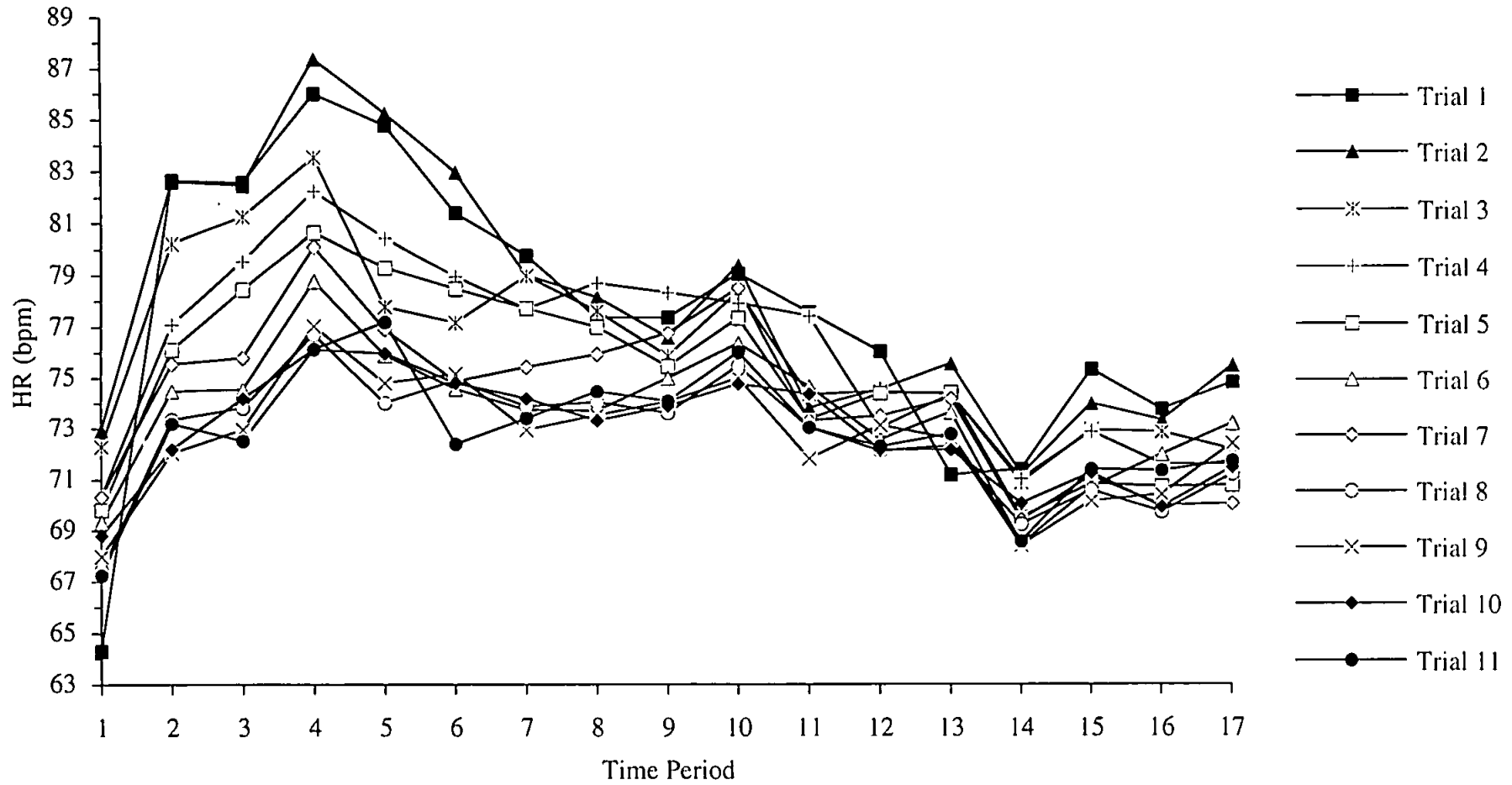
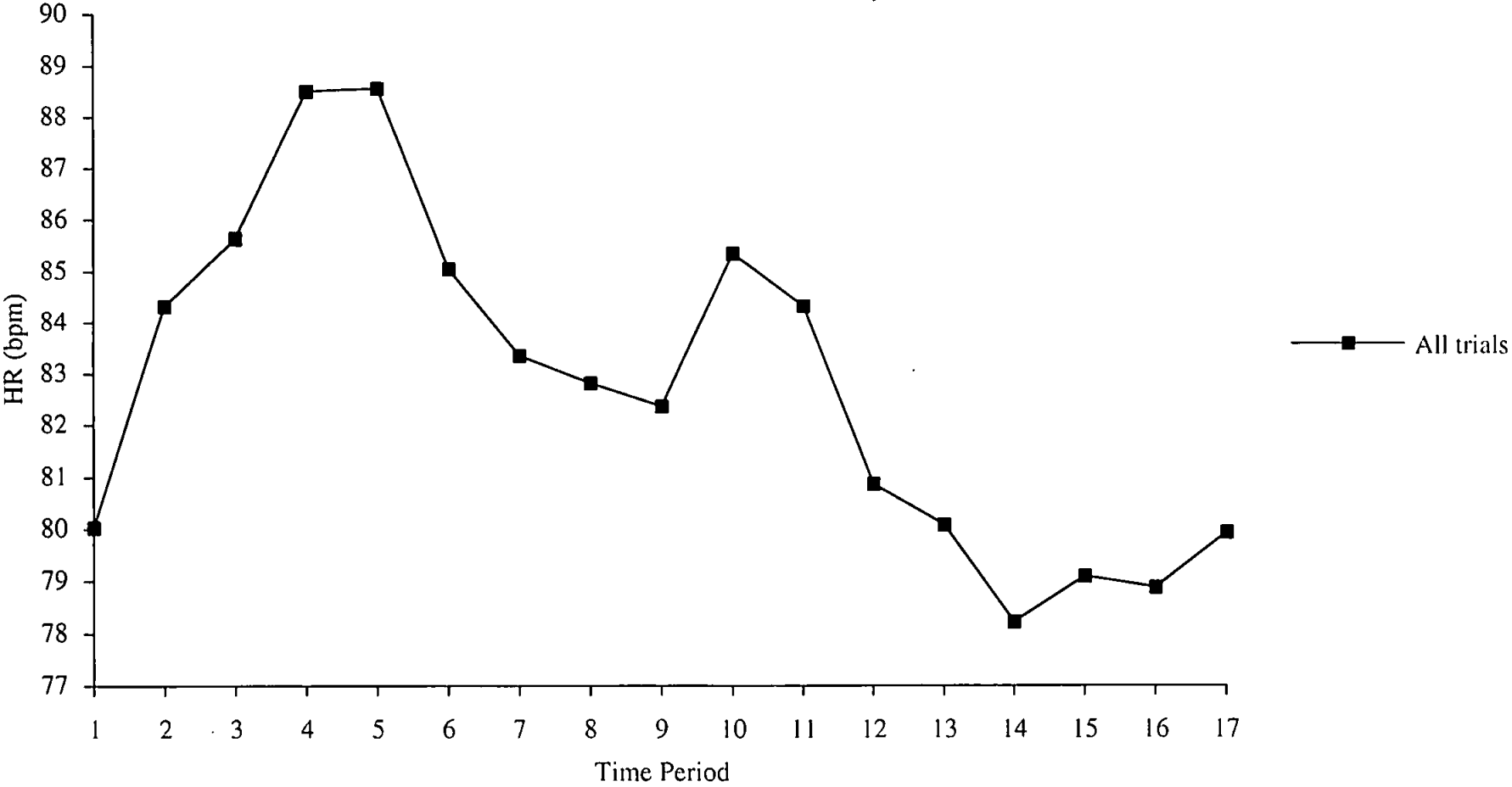




Figure 7. Average HR as a function of time periods averaged across Trials 1-11.



< .01), 7, 10 and 11 ( $p < .05$ ) compared with Time Period 1. There were no significant differences between Period 1 and Periods 12 through to 17. Time Periods 2 and 3 produced greater levels of activity compared with Periods 12 through to 17 ( $p < .01$ ). Time Periods 4 and 5 produced higher HR than Period 2 ( $p < .05$ ), Periods 6 to 9, Period 11 ( $p < .01$ ) and Periods 12 through to 17 ( $p < .001$ ). Time Periods 6, 7, 10 and 11 produced greater levels of activity than Time Periods 12 to 17 ( $p < .05$ ). Time Periods 8 and 9 produced greater levels of average HR compared with Periods 14 to 16 ( $p < .05$ ). There were no significant differences across Time Periods 12 to 17.

MxHR (Figure 8) revealed Time Period 1 to have higher HR than Periods 13 through to 17 ( $p < .01$ ). Time Periods 2 and 3 produced greater levels of activity than Periods 12 to 17 ( $p < .01$ ) compared with Time Period 4 which produced higher HR than Time Period 1 ( $p < .05$ ) and Periods 12 through to 17 ( $p < .01$ ). Time Period 5 produced greater levels of responding than Periods 1 to 3 ( $p < .05$ ), Periods 6, 7 and 8 ( $p < .01$ ) and Periods 9 through to 17 ( $p < .001$ ). Time Period 6 produced greater levels of activity than Period 9 and Periods 12 through to 17 ( $p < .01$ ), whereas, Time Periods 7, 8 and 9 produced higher HR than Periods 13, 15 and 16 ( $p < .05$ ). Time Period 10 produced higher HR levels than Periods 7, 8 and 9 ( $p < .05$ ) and Periods 12 to 17 ( $p < .01$ ), compared with Time Period 11 which produced greater levels of activity than Periods 1, 7, 8 and 9 ( $p < .05$ ) and Periods 12 through to 17 ( $p < .01$ ). There were no significant differences across Time Periods 12 to 17.

Post-hoc comparisons involving mnHR (Figure 9) revealed Time Periods 2 to 10 ( $p < .01$ ), Periods 11 to 13 and Period 17 ( $p < .05$ ) to have higher HR than Time Period 1. Time Periods 2 and 3 produced higher HR levels than Time Periods 14 to 16 ( $p < .05$ ) compared with Period 4 which produced greater levels of activity than Period 2, Periods 7 to 9 ( $p < .05$ ) and Periods 11 through to 17 ( $p < .01$ ). Time Period 5 produced greater levels of activity than Periods 11 to 17 ( $p < .01$ ), whereas, Periods 6 to 10 produced higher HR than Periods 14 to 17 ( $p < .05$ ). There were no significant differences across Time Periods 11 to 17.

In summary, avHR revealed a clear-cut pattern of responding across trials. At the beginning of the trial when subjects visually received the initial position of the planes (Period 2), and were asked for their first response (Period 4) there was a steady cardiac

Figure 8. Mean maximum HR as a function of time periods averaged across Trials 1-11.

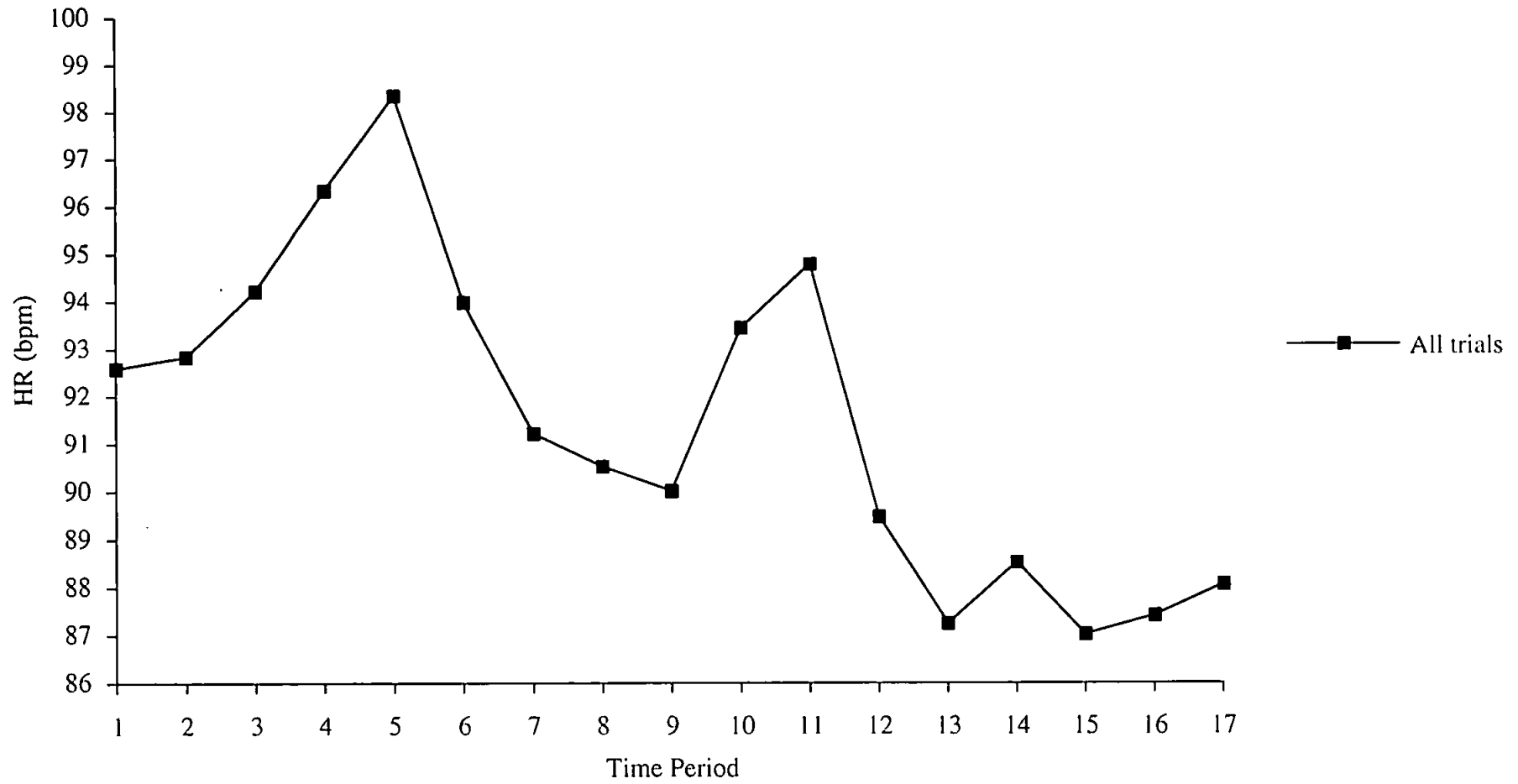
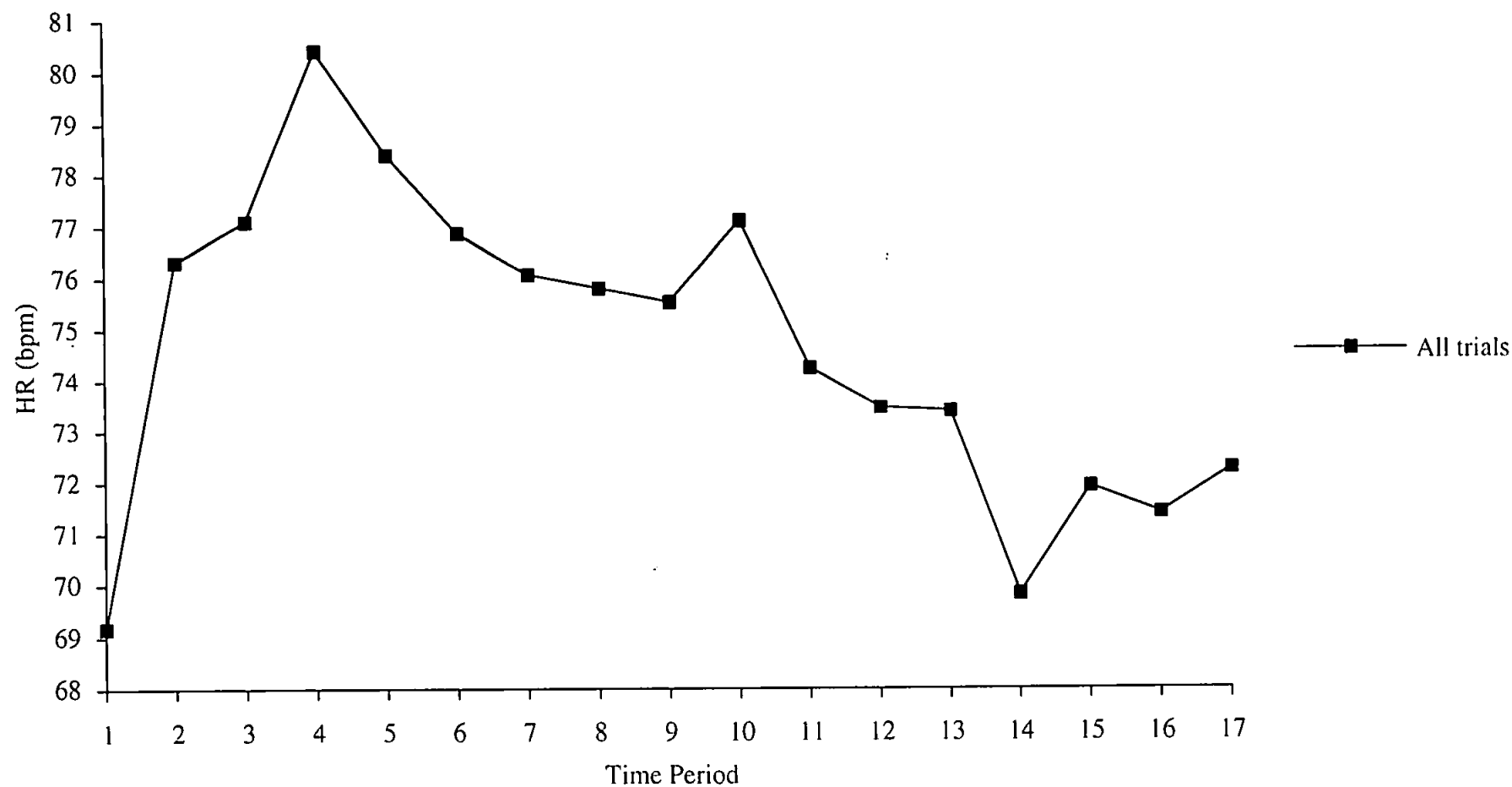


Figure 9. Mean minimum HR as a function of time periods averaged across Trials 1-11.



acceleration, reaching a peak when subjects were required to give their first response (Period 5). From this accelerative peak there followed an interval characterised by an anticipatory deceleration, during the thirty seconds after subject's first response (Periods 5 to 9). Until the time when subjects were asked for their second response (Period 9), at which point accelerative processes took over, but were of a smaller magnitude than the first response. Following the cue for subjects to give their second response (Period 10), there was a steady deceleration whilst subjects in the feedback condition received information concerning success or failure. There was then a slight acceleration until the end of the trial (Periods 14 to 17). Inspection of mxHR revealed a similar response patterning. MnHR produced a slightly different pattern, with the first HR peak occurring when subjects were asked for their first response (Time Period 4) compared with both avHR and mxHR which revealed an accelerative peak when subjects voiced their first response (Time Period 5). From this peak, mnHR produced an anticipatory deceleration, during the thirty seconds after subject's first response. Following this, there was a small increase in levels of mnHR when subjects were asked for their second response (Time Period 10) compared with a second accelerative peak for mxHR (Time Period 11) and avHR (Time Period 10). MnHR then revealed a steady deceleration until the point when subjects in the feedback group received information concerning success or failure (Period 13). After this, there was a sharp deceleration, followed by a steady acceleration until the end of the trial.

3.2.1.2. Corrected Heart Rate. The results for all three cardiovascular measures were very similar to those of uncorrected HR. They are therefore not reported in detail here.

### 3.2.2. Electrodermal activity

3.2.2.1. Skin Conductance Level. There was a significant difference across time periods ( $F(3, 135) = 10.67, p < .001$ ). Post-hoc comparison of means revealed that Time Periods 2, 4 and 5 had higher SCL than Periods 8 and 9 and Periods 12 through to 16 ( $p < .05$ ). There were no significant differences between the remaining time periods (Figure 10). There were no significant differences across trials ( $F < 1$ ) or any interaction between trials and time periods ( $F < 1$ ). Means across trials and time periods are plotted in Figure 11.

Figure 10. Mean skin conductance level as a function of time periods averaged across Trials 1-11.

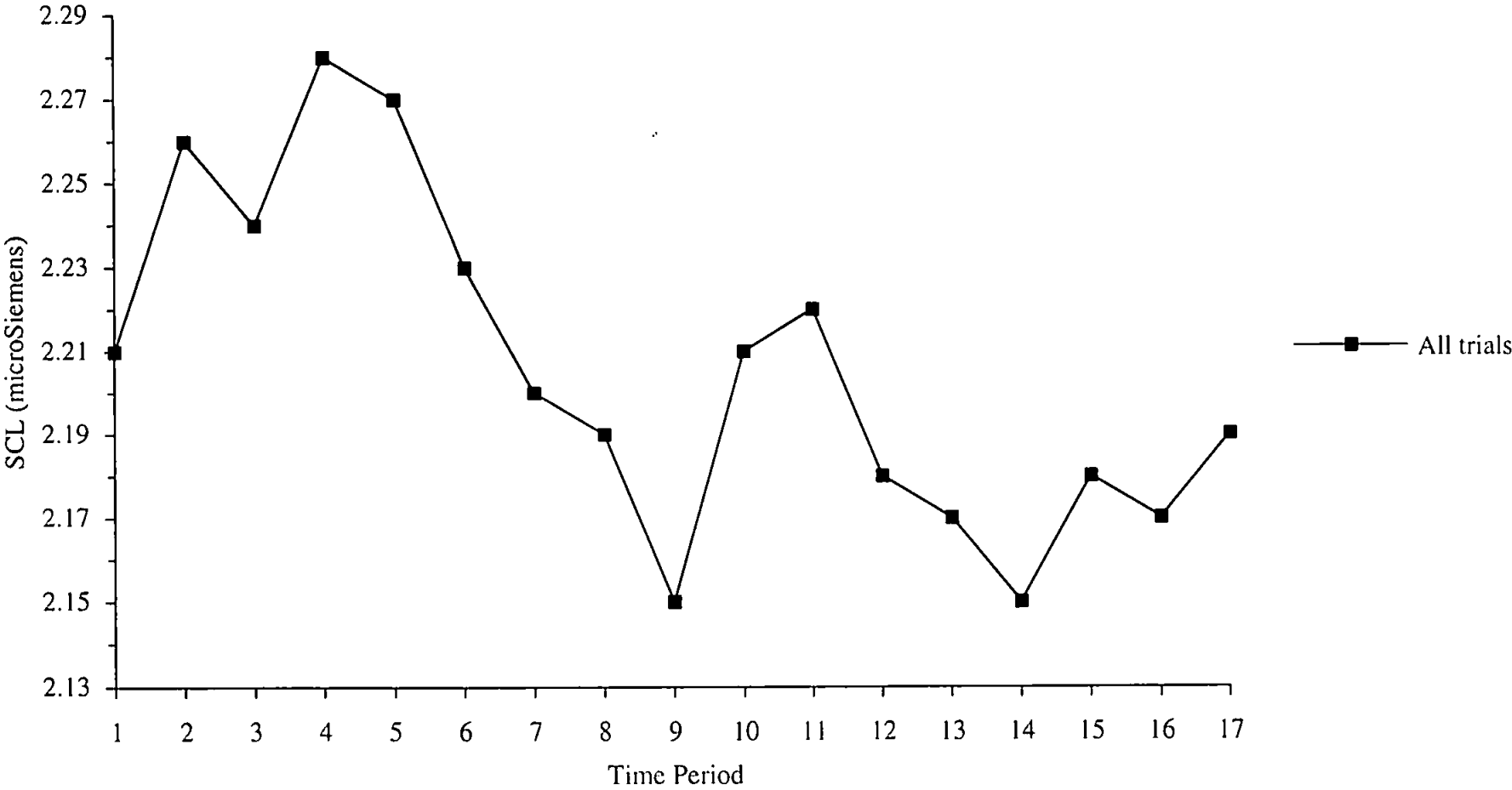
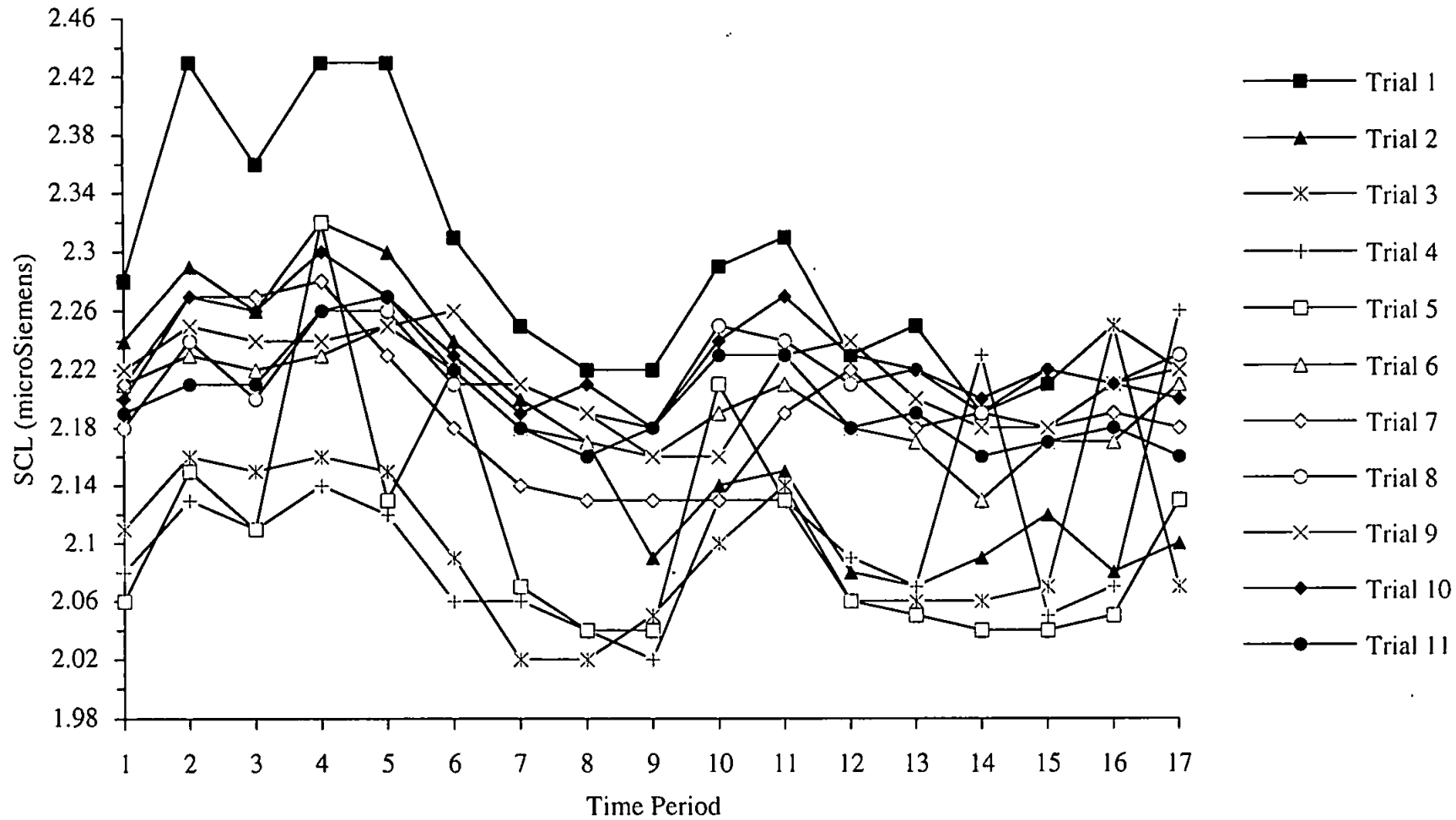


Figure 11. Mean skin conductance level as a function of time periods across Trials 1-11.



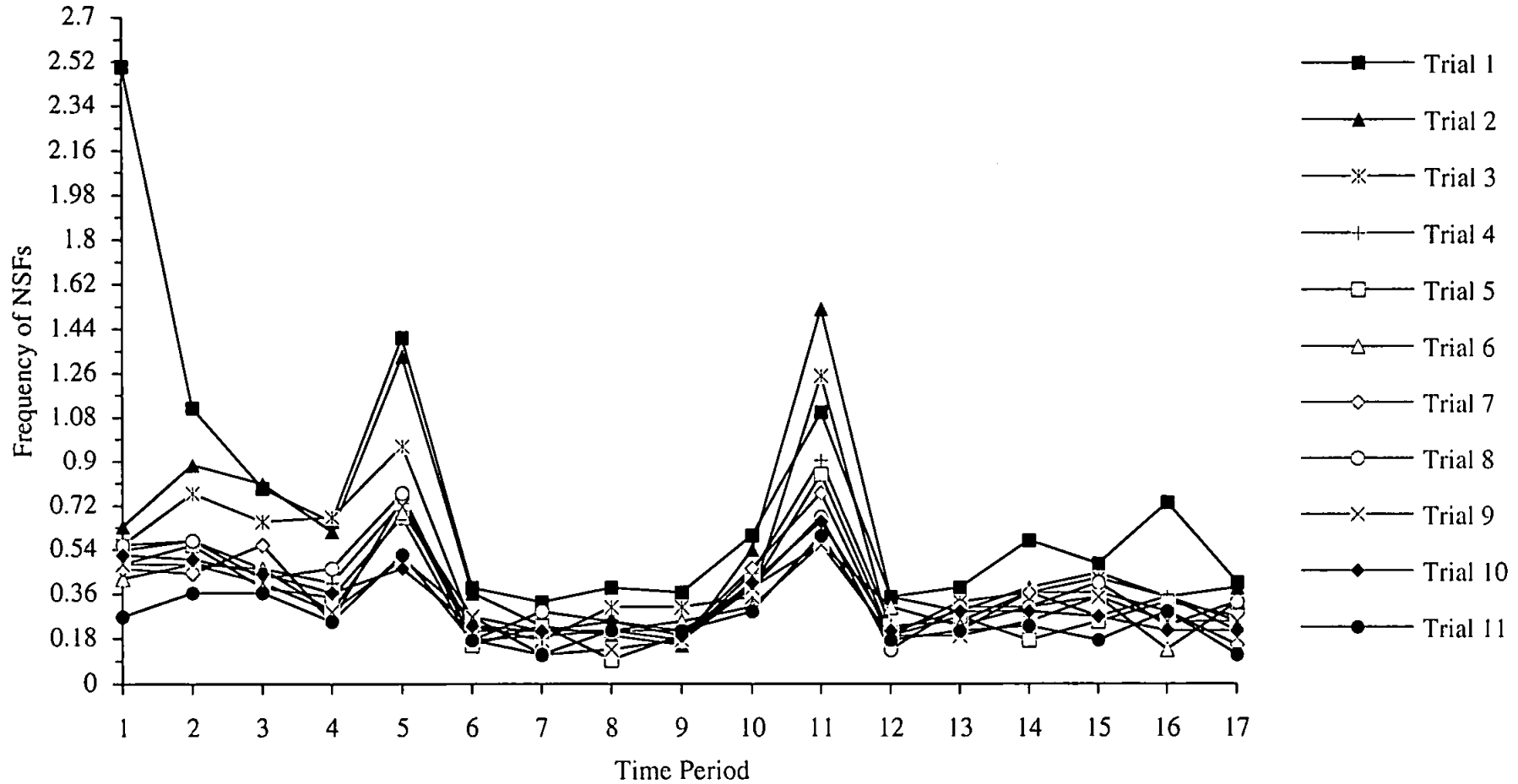
In summary, the pattern of responding involving tonic SCL is similar to that seen previously for cardiovascular activity. There was a steady increase in levels of activity when the initial positions of the planes were presented on the screen (Period 2) and when subjects were asked for their first response (Period 4). From this peak, there followed a decrement in SCL, during the thirty seconds after subject's first response until they were asked for their second response (Period 9). At this point, there was an increase in levels of activity, but of a smaller magnitude than was the case for the first peak. Following the cue for subjects to give their second response (Period 11), there was a steady decrease in tonic SCL whilst subjects in the feedback group received information concerning success or failure, and subjects in the no feedback group were told that it was the end of the trial. Following this, there was a slight increase in levels of activity while all subjects were asked for their ratings of perceived control up until the end of the trial (Periods 14 to 17).

3.2.2.2. Non-Specific Fluctuations. There was a highly significant difference across time periods ( $F(4, 185) = 41.1, p < .001$ ) and across trials ( $F(2, 116) = 65.9, p < .001$ ). There was also a trials by time periods interaction ( $F(3, 175) = 45.3, p < .001$ ). Visual presentation of the data (Figure 12) revealed a similar pattern of responding across time periods for all trials, with a steady number of NSFs across Periods 1 to 4. After this, there was a sharp increase in the number of NSFs during Period 5. From this peak, there was a sharp decrease in the frequency of NSFs followed by, a stabilisation in the number of NSFs until Period 9. Following this, there was an increase in the frequency of NSFs, of a similar magnitude as the first peak. From this peak, there was a sharp decrease in the number of NSFs followed by a stabilisation in the number of NSFs until the end of the trial (Periods 11 to 17). Inspection of Figure 12 suggests that the trials by time periods interaction may be due to Trial 1 producing a markedly greater frequency of NSFs during Period 1 compared with the remaining trials. Trial 1 also produced greater levels of activity during Periods 14 and 16 compared with the remaining trials. There was also a pronounced difference between the peak of activity (Time Periods 5 and 11) and the other time periods in the first three trials than was the case for subsequent trials.

This pattern of responding across time periods is revealed more clearly when data is averaged across trials. Multiple comparison of means then reveals that there was a greater frequency of NSFs during Time Periods 1, 2 and 3 compared with Periods 6 to 9



Figure 12. Frequency of non-specific fluctuations as a function of time periods across Trials 1-11.



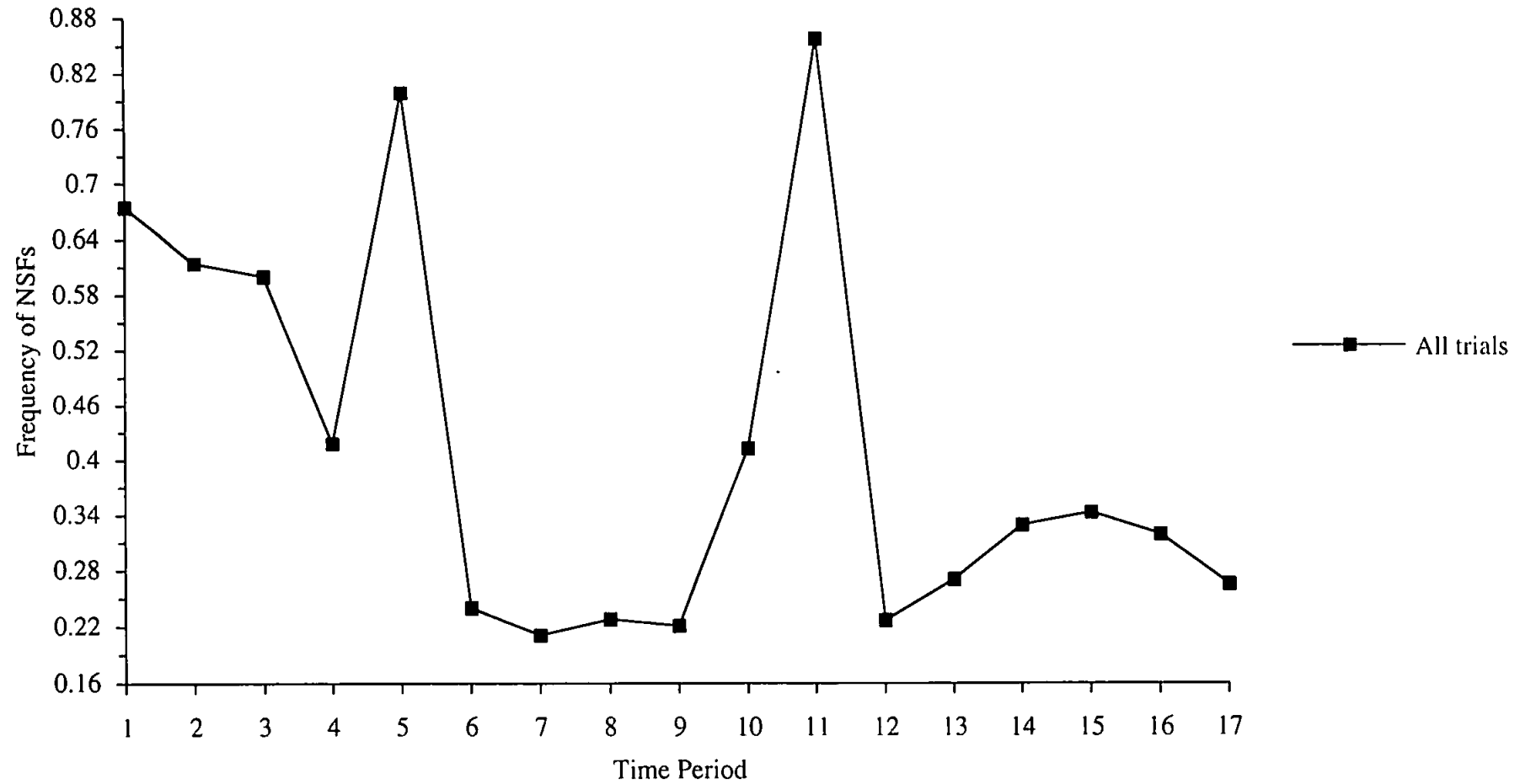
and Periods 12 through to 17 ( $p < .01$ ). Time Periods 5 and 11 produced a greater frequency of NSFs compared with Periods 1 to 4 ( $p < .01$ ), Periods 6 to 9 ( $p < .001$ ), Period 10 ( $p < .01$ ) and Periods 12 through to 17 ( $p < .001$ ). There were no significant differences across Time Periods 6 to 9 or Periods 12 to 17 (Figure 13).

In summary, the pattern of responding involving NSFs is somewhat dissimilar to that associated with tonic SCL and cardiovascular measures averaged across trials. There was a steady number of NSFs up until the point when subject were asked for their first response following by, a decrease in the number of NSFs while subjects were preparing to voice their response. After this, there was a sharp increase in the frequency of NSFs (Period 5). From this peak, there was a sharp decrease in the number of NSFs during the thirty seconds after subjects had given their response until they were asked for their second response (Periods 5 to 9). At this point, there was a second increase in the frequency of NSFs, of a similar magnitude to the first peak. Following the cue for subjects to give their second response (Period 11), there was a sharp decrease in the number of NSFs. After subjects gave their second response there was a slight increase in the frequency of NSFs until they were asked for their ratings of perceived control (Period 15). Following this, there was a slight decrease in levels of activity until the end of the trial.

### 3.3. Between-Groups Analysis

The between-group analysis was partially a repeat of previous analyses but included a between-group factor (feedback/no feedback). The number of trials used in the analysis was reduced due to problems with the size of the data set. Three trials were chosen which represented the early, middle and late stages of the experiment; namely, Trials 2, 6 and 9. All 17 time periods remained in the between-group analysis in order to assess the effects of experimental manipulation across the trial as a whole. All measures were therefore analysed in a 2 (feedback/no feedback) x 3 (Trials 2, 6 and 9) x 17 (time periods) model MANOVA. With repeated measures on the last two factors.

Figure 13. Frequency of non-specific fluctuations as a function of time periods averaged across Trials 1-11.



### 3.3.1. Cardiovascular Activity

3.3.1.1. Uncorrected Heart Rate. There was a highly significant difference across time periods for avHR ( $F(4, 198) = 35.7, p < .001$ ), mnHR ( $F(6, 274) = 19.6, p < .001$ ) and mxHR ( $F(7, 352) = 36.2, p < .001$ ). There was also a significant difference across trials for avHR ( $F(1, 68) = 43.1, p < .001$ ), mnHR ( $F(2, 73) = 27.8, p < .001$ ) and mxHR ( $F(1, 72) = 44.9, p < .001$ ). A trials by time periods interaction was revealed for all three cardiovascular measures: avHR ( $F(9, 463) = 4.4, p < .01$ ), mnHR ( $F(12, 597) = 3.7, p < .01$ ) and mxHR ( $F(12, 576) = 2.8, p < .05$ ). As expected, further analysis of this interaction revealed similar results as the within-group analysis (Section 3.2.1.1). The results are therefore not reported in detail here. There was no main effect of condition or any interaction involving condition across any of the three measures (all  $F_s < 1$ ). Group means across time periods and trials are plotted in Figures 14 to 16 for avHR, mnHR and mxHR respectively.

Visual inspection for all three measures reveals little difference between Fb and NFb groups during Trial 2. In later trials, however, the Fb group produced greater levels of activity during the early time periods of the trial (Periods 1 to 5) compared with the NFb group. Both groups had similar levels of activity in later time periods, with the exception of avHR and mxHR which revealed the Fb group to have higher HR than the NFb group during Periods 10 and 11 of Trials 6 and 9.

3.3.1.2. Corrected Heart Rate. The results for all three cardiovascular measures were very similar to those of uncorrected HR. They are therefore not reported in detail here.

### 3.3.2. Electrodermal Activity

3.3.2.1. Skin Conductance Level. There was a significant difference across time periods ( $F(2, 75) = 3.5, p < .01$ ). As expected, post-hoc comparison of means revealed similar results to the within-group analysis (Section 3.2.2.1). The results are therefore not reported in detail here. There were no significant differences across trials ( $F < 1$ ) or any interaction between trials and time periods ( $F < 1$ ). There was no main effect of condition or any interaction involving condition (all  $F_s < 1$ ). Group means across time periods and trials are shown in Figure 17. Visual inspection suggest that the Fb group may have had

Figure 14. Average HR as a function of groups, time periods and trials.

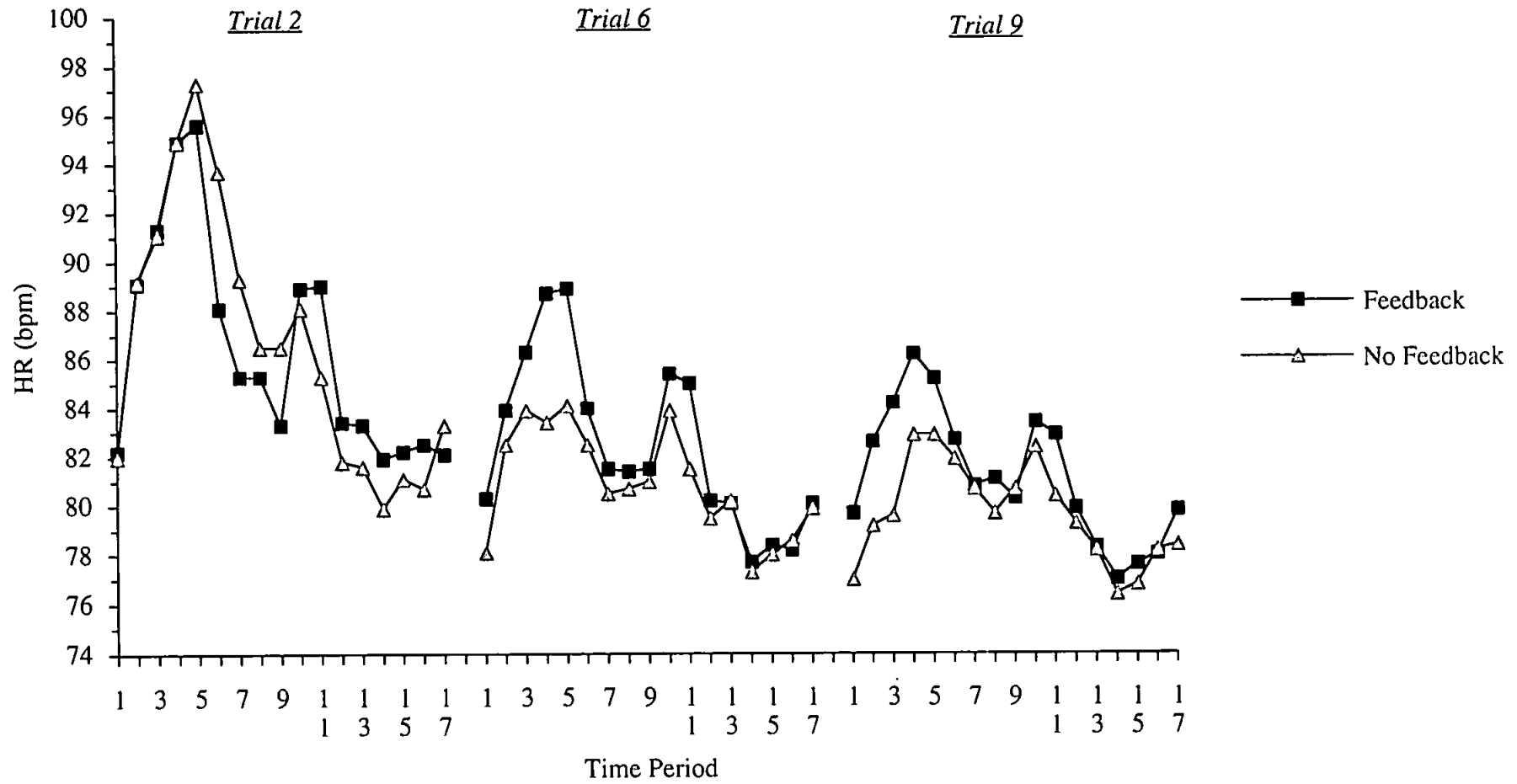


Figure 15. Mean minimum HR as a function of groups, time periods and trials.

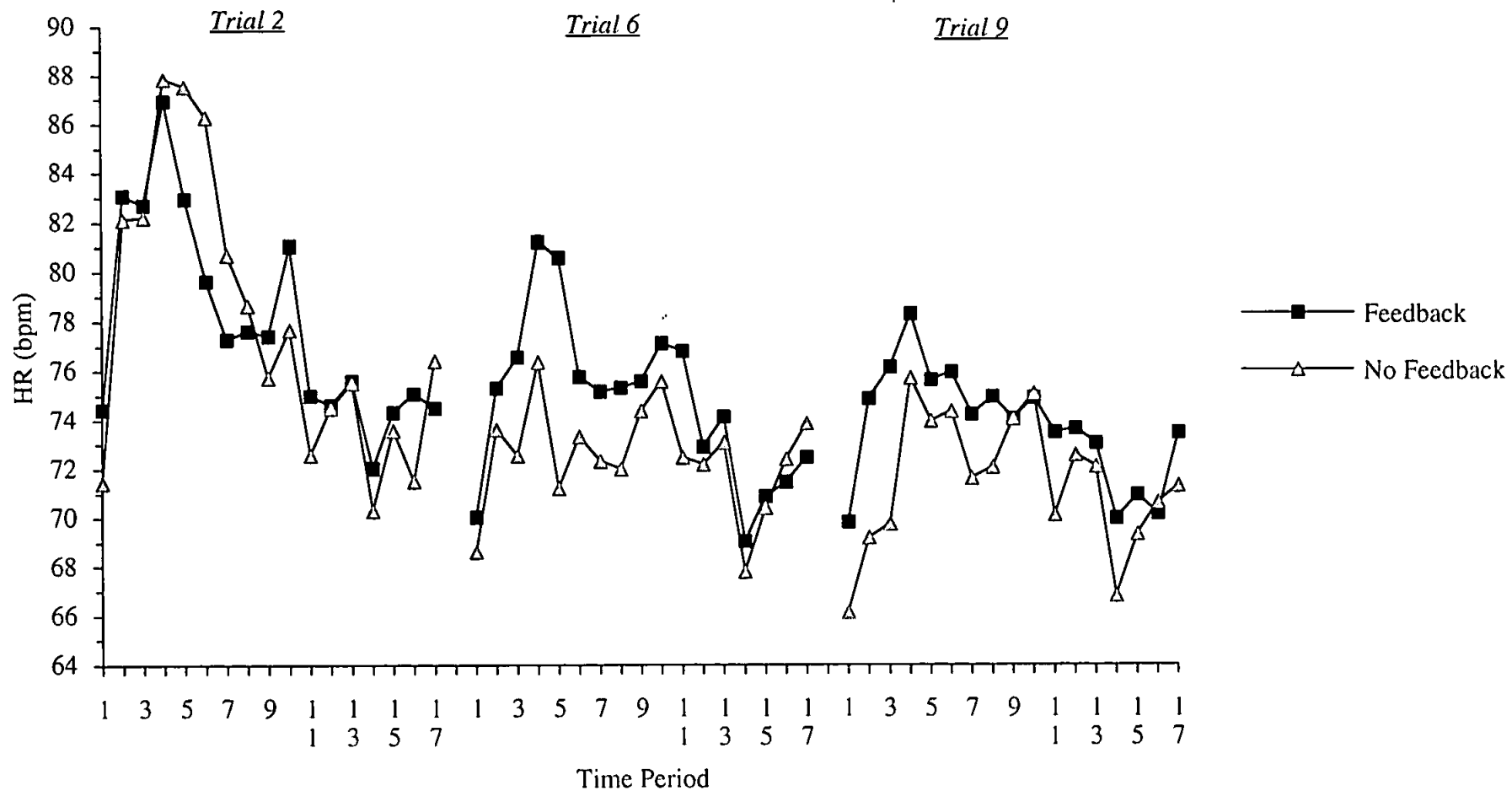


Figure 16. Mean maximum HR as a function of groups, time periods and trials.

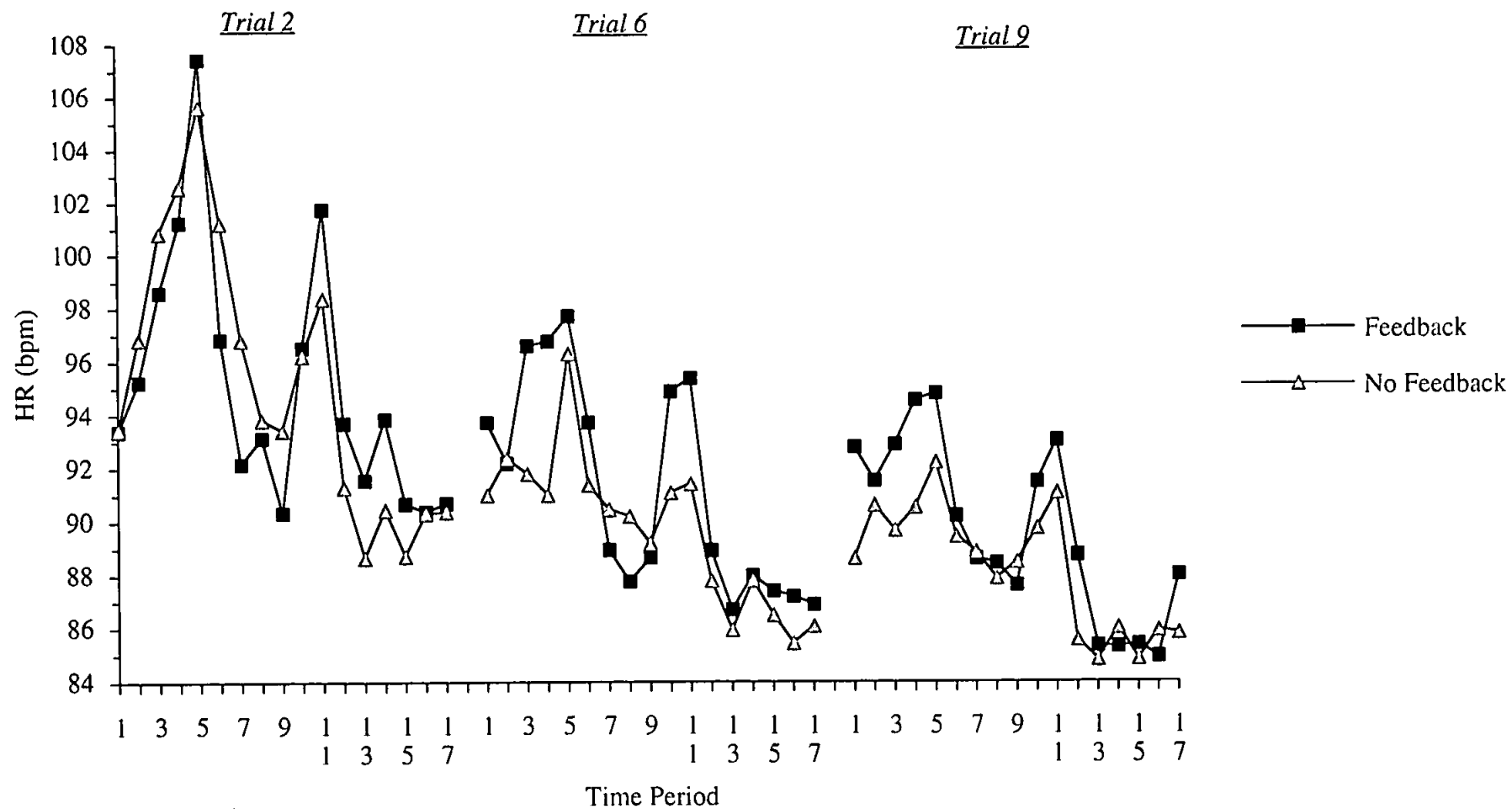
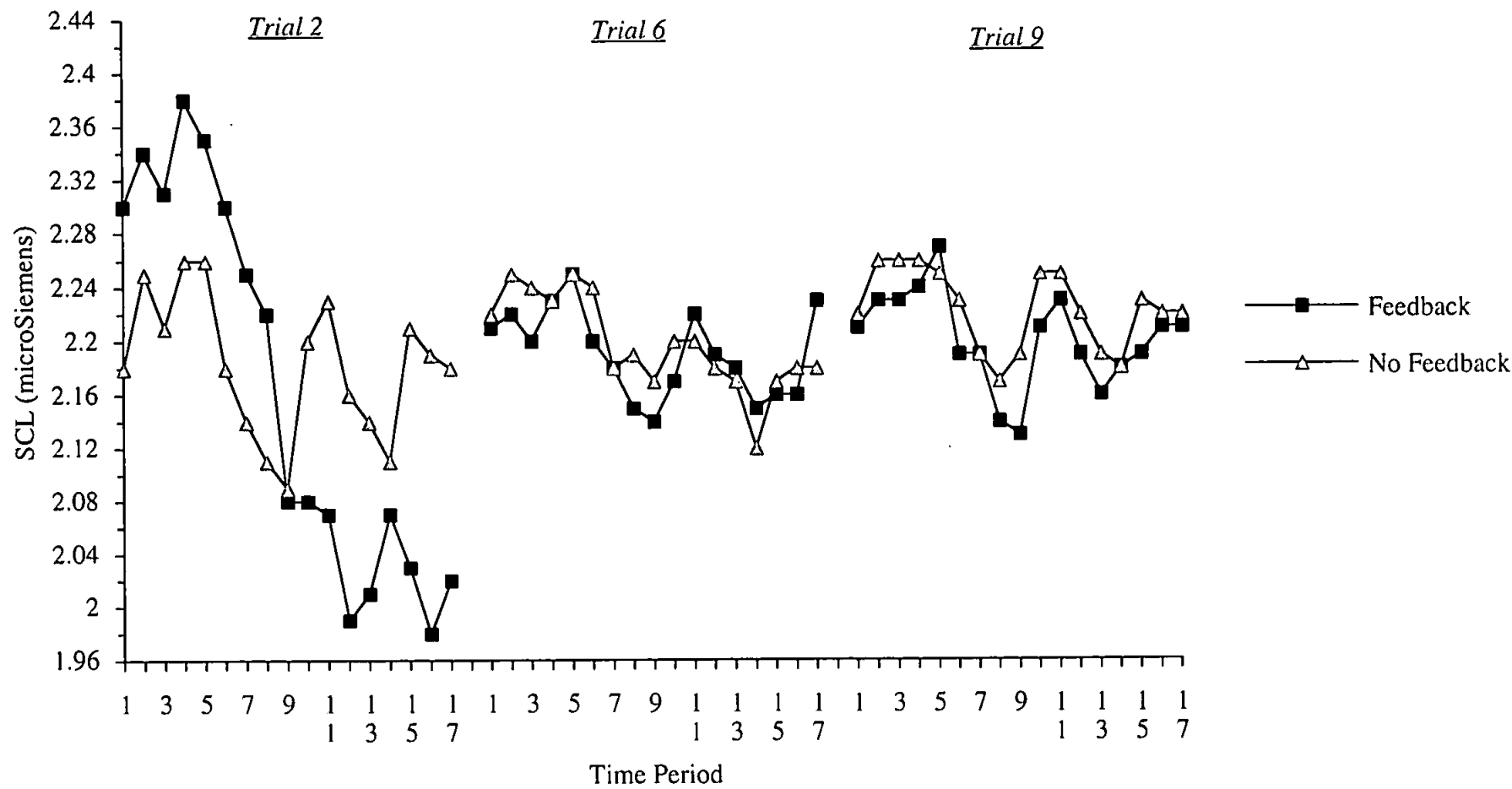


Figure 17. Mean skin conductance level as a function of groups, time periods and trials.





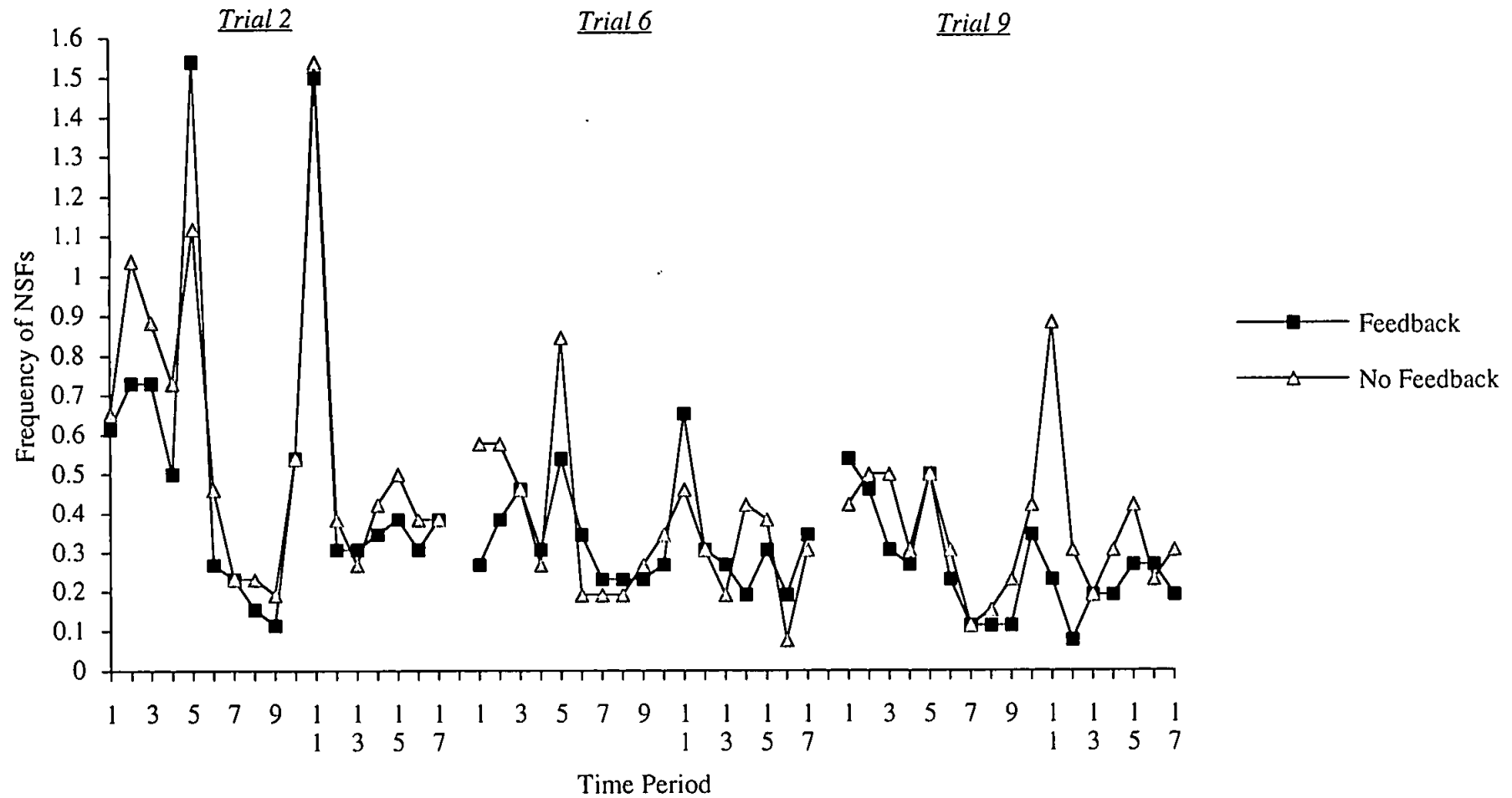
higher SCL compared with the NFb group across Periods 1 to 8 during Trial 2. However, during Periods 10 to 17 the NFb group produced higher SCL than the Fb group. During later trials the two groups revealed similar levels of activity.

3.3.2.2. Non-Specific Fluctuations. There was a highly significant difference across time periods ( $F(5, 270) = 13.01, p < .001$ ), across trials ( $F(2, 85) = 26.1, p < .001$ ) and a trials by time periods interaction ( $F(11, 543) = 4.1, p < .01$ ). As expected, further analysis of the interaction revealed similar results as the within-group analysis (Section 3.2.2.2). There was no main effect of condition or any interaction involving condition (all  $F_s < 1$ ). Group means across time periods and trials are shown in Figure 18. Inspection of Figure 18 reveals an inconsistent pattern of responding. During Trial 2, the NFb group produced a greater frequency of NSFs than the Fb group in Periods 2 and 3 compared with Period 5 in which the Fb group had a greater number of NSFs than the NFb group. During Trial 6, the NFb group produced greater activity than the Fb group during Periods 1, 2 and 5 compared with Period 11 in which the Fb group had a greater number of NSFs than the NFb group. During Trial 9, there was little difference between the two groups, with the exception of Period 11 in which the NFb group revealed a greater frequency of NSFs compared with the Fb group.

#### 3.4. Performance Data

Performance data was analysed in a two-way MANOVA with feedback as a between-subject factor and trials as a repeated measure. Performance was significantly better ( $F(1, 50) = 5.8, p < .05$ ) for the Fb group than for the NFb group (12.9% and 9.5 % correct respectively). There was also a significant difference across trials ( $F(5, 272) = 6.11, p < .05$ ), but no feedback by trials interaction ( $F(5, 272) = 1.48$ ). Post-hoc comparison of means across trials revealed Trials 1 and 3 to have a lower percentage of correct responses compared with Trials 4, 6 ( $p < .05$ ), 7 ( $p < .01$ ), 8, 9 ( $p < .05$ ), 10 and 11 ( $p < .01$ ). Trials 2 and 5 produced a lower percentage of correct responses than Trials 7 ( $p < .01$ ), 10 ( $p < .05$ ) and 11 ( $p < .01$ ), compared with Trials 4 and 6 which produced a lower percentage of correct responses than Trials 7 ( $p < .01$ ) and 11 ( $p < .05$ ). Trial 8 produced a lower number of correct responses than Trials 7 ( $p < .01$ ), 10 ( $p < .05$ ) and 11

Figure 18. Frequency of non-specific fluctuations as a function of groups, time periods and trials.



( $p < .01$ ), whereas, Trials 9 and 10 produced a lower number of correct responses compared with Trials 7 and 11 ( $p < .01$ ). Group means across trials are shown in Figure 19.

These results are more easily understood with reference to task difficulty. The easy trials: Trials 1, 7 and 10, produced the greatest percentage of correct responses (mean % = 17.3). The intermediate trials: Trials 4, 9 and 10, produced the second greatest number of correct responses (mean % = 10.9). The difficult trials: Trials 3, 6 and 8, produced the third greatest number of correct responses (mean % = 5.1), and the two very difficult trials: Trials 2 and 5, produced the smallest number of correct responses (mean % = 0.9).

### 3.5. Perceived Control Data

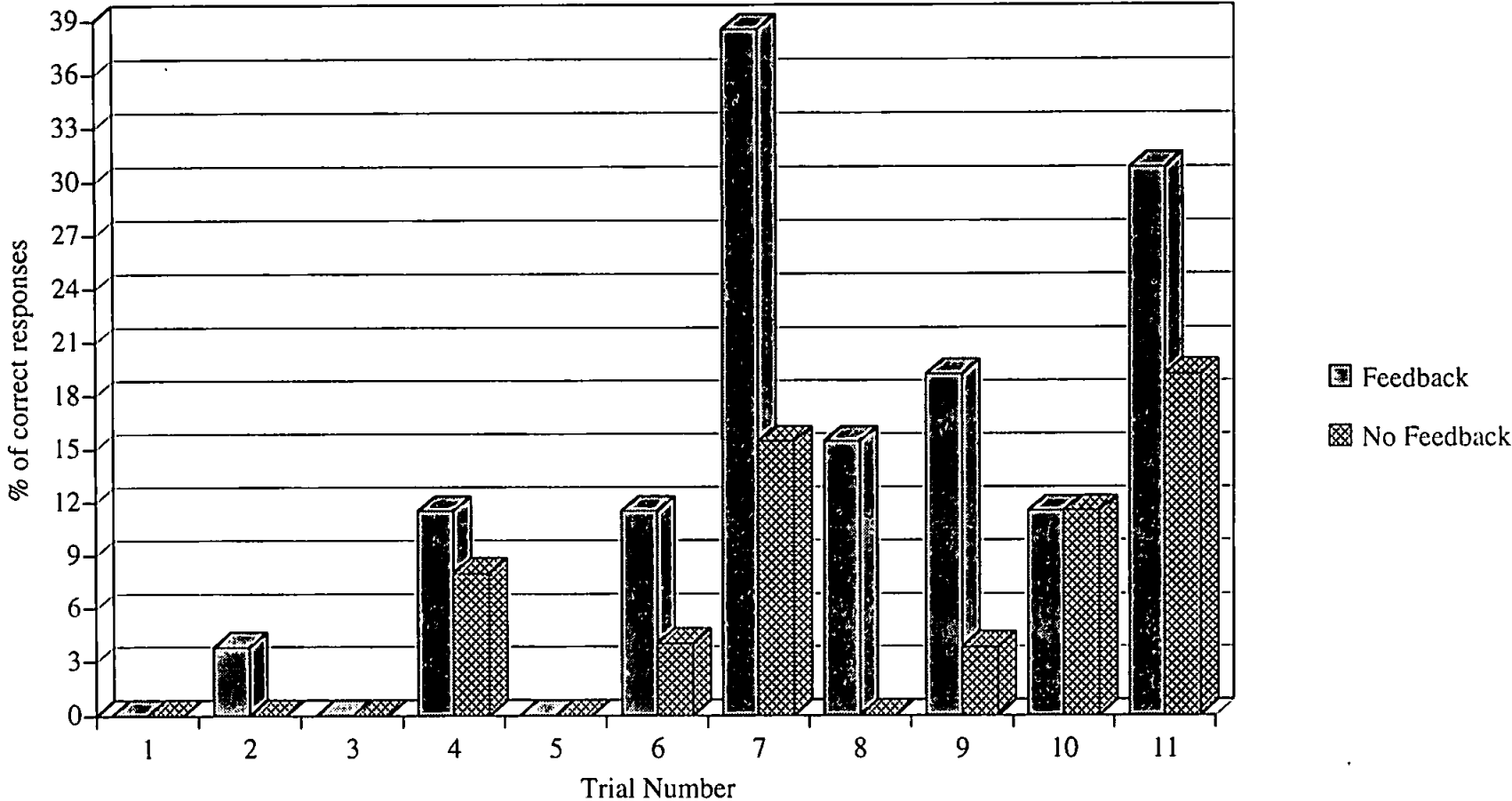
Perceived control data was analysed in a two-way MANOVA with feedback as a between-subject factor and trials as a repeated measure. There were no significant differences between groups, across trials or, any interaction between groups and trials (all  $F_s < 1$ ). Feedback and no feedback groups showed a mean response of 4 across all trials (from 1 "Feel I have complete control" to 5 "Feel I have no control").

## 4. DISCUSSION

In this experiment, receipt of feedback on a complex, externally paced problem-solving task did not result in any significant changes in cardiovascular or electrodermal activity compared with a no feedback group. However, in keeping with the literature reviewed in Chapter 3, Section 2.1, feedback did lead to an increase in performance on the task compared with a condition where no feedback was available. Contrary to prediction, however, the provision of feedback did not result in performance decrements across the course of the task, rather feedback increased levels of performance in later trials compared with the no feedback group.

Although there were no significant differences between the two experimental groups in terms of autonomic activity, detailed examination of the data suggests very

Figure 19. Mean percentage of correct responses as a function of groups and trials.



limited support for differential effects as a function of feedback, particularly in relation to the cardiovascular system. During Trial 2, all three cardiac measures produced a similar magnitude and pattern of responding in both feedback and no feedback conditions. In later trials, those subjects in receipt of feedback showed greater levels of HR responding during the first half of each trial, especially across Periods 1 to 5, compared with the no feedback group. There was little difference between the two groups during the second half of the trial. Measures of electrodermal activity were more inconsistent in their pattern of responding. In terms of SCL during Trial 2, the feedback group showed higher levels of responding than the no feedback group, but only in the first half of the trial (Time Periods 1 to 8). During the second half (Time Periods 10 to 17), the pattern was reversed with the feedback group showing lower levels of activity. In later trials, there were no differential effects as a function of feedback. Closer examination of the frequency of NSF's data revealed no meaningful pattern of results, with the exception that the no feedback group produced greater levels of activity across selected time periods of all three trials compared to the feedback condition. These findings suggest that the presence of feedback might lead to a dissociation between electrodermal and cardiovascular parameters, with a non-significant increase in levels of HR under feedback conditions compared with a stabilisation or decrease of electrodermal measures.

There are a number of possible reasons for the mainly insignificant results. The first concerns subject's performance on the task, with particular reference to levels of task difficulty. The work of Obrist and colleagues (Light & Obrist, 1983; Obrist, Gaebelein, Teller, Langer, Grignolo, Light & McCubbin, 1978) implies that tasks which are impossibly hard (i.e. performance at chance levels), lead to less sustained cardiovascular responses compared with moderately difficult tasks, because the former discourages subjects from continuing to exert their maximum effort. Indeed, the situation fosters "giving up" (Obrist *et al.*, 1978). However, the tasks employed by Obrist and co-workers were very different from that used in the present study (i.e. sensory-motor versus cognitive), and it is possible that manipulating levels of difficulty in cognitive tasks has a markedly different effect in terms of psychophysiological responses. This would be supported by those studies which have manipulated difficulty in mental arithmetic tasks, and found that HR increases were sustained in impossible conditions (e.g. Carroll, Turner

& Hellawell, 1986). Self-report measures reflected this sustained activity with high levels of reported engagement and arousal. One possible factor to explain these inconsistencies was suggested to be the duration of the impossible condition. That is, during Carroll *et al.*'s study the impossible trials lasted for only 4 min compared with the 14 min duration employed by Obrist and colleagues (1978). It may be that insufficient time was given in the former study in which to foster a sense of "giving up", and that if the impossible trials had continued subjects would have become less engaged and/or aroused.

In the present experiment, the overall failure rate averaged across all subjects was extremely high (88.8%), and the task was of a relatively long duration (19 min). Thus, the conclusions reached by Obrist *et al.* (1978), that impossible conditions foster "giving up", might partially explain the non-significant results in this study. Although it is not possible, based on an explanation of this type, to understand why subject's performance should increase across trials. Intuitively, it would seem that reduced effort should be reflected in poorer performance. These results are, however, in accordance with others studies (e.g. Light & Obrist, 1983; Obrist *et al.*, 1978) in which subjects in the impossible condition showed no slowing in reaction time compared with easy or difficult groups, even though the former reported giving up more often.

The overall failure rate, when examined across experimental conditions, reveals that although subjects in receipt of feedback produced significantly higher levels of performance than the no feedback group, the failure rate was extremely high in both conditions. Those subjects in the no feedback group had a failure rate of 90.5%, or, 27 successful trials and 259 failure trials compared with 87.1%, or, 37 successful and 249 failure trials in the feedback group. With this degree of difficulty, it seems likely that the majority of subjects, irrespective of experimental condition, simply gave up or guessed at the possible answer. An account supported by informal post-task questions. In terms of this particular task, therefore, it may not have been feedback *per se* that was unimportant, but rather the 'unrealistic' levels of task difficulty.

Examination of the performance data in terms of the feedback manipulation revealed no significant interaction between difficulty and availability of feedback. However, there were some interesting differences between feedback and no feedback conditions. The greatest difference emerged during easy trials, with subjects in receipt of feedback

producing lower failure rates (76.9%) than the no feedback group (88.5%). In the intermediate and difficult trials, feedback subjects again produced a lower failure rate (85.9 & 91% respectively) compared to the no feedback group (92.2 & 98.7% respectively), but the difference was not so great. During very difficult trials, there was little difference between feedback and no feedback conditions (100 & 98.1% respectively). Although these results did not reach significance, they do suggest that feedback may be more beneficial, in terms of increasing or sustaining performance, in easy compared to difficult or very difficult tasks. Indeed, if this study had employed a definition of 'difficulty' in accordance with previous studies (e.g. Obrist *et al.*, 1978), in which easy and impossible trials were defined as those with 98.7% and 4.6% success rates respectively, a significant difference may have emerged. Instead, in the light of the extremely low success rates given above, it is likely that subjects in the present study perceived all trials to be equally difficult/impossible.

It is not possible to say whether any changes as a function of feedback were masked or distorted by unrealistic difficulty levels. Past studies such as that by Fowles, Fisher and Tranel (1982) in which they manipulated availability of feedback and levels of difficulty (percentage success) suggest otherwise. With feedback having little effect on HR responding, irrespective of success rate (10, 50 or 90%). Instead, the most important factor in determining cardiac changes was the availability of monetary incentives. When subjects received money as a result of good performance, the authors found that, again irrespective of success rate, levels of HR activity were greater than for those subjects who received feedback only. Furthermore, the magnitude of this effect was dependent upon the magnitude of the incentive (Tranel, Fisher & Fowles, 1982). In terms of the present experiment, these findings suggest that even if levels of difficulty had been lower, feedback effects would still have been insignificant unless some type of incentive had also been available in order to maintain continued task involvement. Presumably this, in turn, would have depended upon the perceived value of attaining that incentive. High value incentives are more likely to have promoted better involvement/engagement on the task than low value ones. The outcome in the present experiment (i.e. the intrinsic pleasure of achievement) was obviously not of a high enough value compared to the extrinsic monetary reward available in Fowles' study.

However, in terms of the current study the results presented by Fowles and colleagues are only indicative, due to a number of methodological differences. For example, in the present experiment, feedback was provided on a trial-by-trial basis following performance. In Fowles' study feedback was presented only after several responses, against a background of continuous motor activity. Moreover, the feedback given was in the form of a high or low frequency tone, and not "success" or "failure" stimuli as in this study. The meaning of the feedback may therefore have differed in the two experiments. For example, the psychological effect of "failure" or losing a trial may not be equivalent to a high frequency tone. Thus, the feedback provided by Fowles and colleagues may have been less salient than that used in this study.

In addition, the task employed in Fowles' study was very different to the one used in the current experiment (i.e. serial reaction-time versus cognitive). It is uncertain what effect incentives might have in terms of complex, problem-solving tasks. The literature reviewed in Chapter 3, Section 2.1, suggests that feedback effects are mediated by incentives, such as, goal or target setting. Indeed, numerous studies have reported that the availability of goals is necessary before feedback can improve or sustain performance, and that without goals feedback has little effect. A combination of both feedback and goal-setting is therefore optimal for improving performance. This relationship is related, in turn, to that of task difficulty. For example, Locke, Shaw, Saari and Latham (1981) suggest that the motivational effect of goal-setting is dependent upon the difficulty of achieving the goal. When the perceived likelihood of success in attaining or trying for a goal is higher, the goal is more likely to be 'worked for' than when perceived likelihood is low. Expectations of success may be related to self-perceptions about ability on the task in question and/or the degree of perceived experimenter control. For example, if subject's believe that they cannot attain or at least approach the goal, and that even if they exerted more effort they would not improve their performance, this may have important effects in terms of mediating levels of motivation and presumably, physiological activation.

If this is applied to the present study, it may help to explain differences in cardiovascular responding during the first and second halves of later trials. Indeed, feedback may have initially (i.e. during the first half of the trial) sustained subject's motivation to succeed at the task, because the task itself was easier. Subjects had to



remember the positions of only four 'planes' with a visual representation of the air traffic control map available throughout. This increase in motivation may have led, in turn, to the increase in cardiovascular activity relative to the no feedback group. During the second half of the trial, however, the task was more difficult. The map was no longer available to subjects and, in addition, subjects had to remember the positions of the first four planes, add the next four, and verbalise their answer. Thus, it is possible that the majority of subjects simply gave up at this point or guessed at the possible answer, leading to a decrease in overall motivation and HR levels in those subjects receiving feedback bringing them down to the levels of the no feedback group. Indeed, this would be partially supported by the performance data with success rates, averaged across all subjects and trials, being higher in the first half of the trial (28.8%) compared with the second half (9.6%).

There is a second factor confounded with task difficulty which may also help to explain the insignificant results of this study, and that is lack of perceived control. Literature reviewed in Chapter 4, Section 3.1.1, suggests that the availability of control or perception of control is an important mediating variable in psychophysiological responding to behavioural challenge. With control or perceived control resulting in sustained increases in cardiovascular activity compared with a situation where no control is available. These effects, in turn, are mediated by levels of difficulty. That is, only when subjects are uncertain whether they can or cannot achieve the controlling response (i.e. on difficult tasks) are cardiovascular increases sustained compared with a no control condition. When achieving the controlling response is certain (i.e. on easy tasks), cardiac levels are lower or similar to those of no control subjects.

In the current study, ratings of perceived control made on a trial-by-trial basis indicated that subjects believed they had little or no control over the task i.e. that they could not succeed. This was probably due to two main factors, both of which were a direct result of the levels of difficulty employed. Firstly, subjects may have perceived that they lacked the ability to attain or even approach the set goal. A factor confounded by the nature of the task in which a major component involved individual's visual-spatial abilities. An ability which differed greatly between subjects. Secondly, subjects may have perceived that external factors (i.e. the experimenter) were controlling whether or not they succeeded at

the task, and that however well they performed the outcome of each trial would be similar. This was later confirmed by informal post-task questions.

In terms of the effect feedback had on the lack of perceived control, trial-by-trial ratings revealed no significant differences between the feedback and no feedback groups. It is possible, however, that the presence or absence of feedback may have altered subject's perceptions if feelings of control had been measured in a less crude and simplistic manner. Work carried out by Foushee, Davis, Stephan and Bernstein (1980), would support such a suggestion. Foushee *et al.* (1980) found that the availability of feedback altered individual's perception of the possibility of control. Subjects in receipt of feedback reported significantly greater perceived control over aversive stimuli compared with a no feedback group. Thus, to the degree that performance feedback, in the form of information concerning success or failure, reduces ambiguity about the effectiveness of performance, it may have an ameliorative effect on feelings of lack of control and improve subsequent performance, even when actual physical control is absent. Conversely, subjects who receive no performance feedback may show an increase in lack of perceived control.

Presumably, differences in the perception of control might also lead to changes in autonomic function. Literature reviewed in Chapter 3, Section 2.2.2., indicated that subjects in receipt of performance feedback produced increased cardiovascular activity compared to a no feedback group, but only when there was no opportunity to control aversive consequences of the event. When subjects could actively control whether or not they received aversive stimuli, the availability of feedback made little difference in terms of changes in cardiovascular activity (Light & Obrist, 1980). Caution is required in interpreting these results, however, as the methodology employed by Light and Obrist was entirely different to that used in this study. For example, the former employed an unsignalled shock avoidance reaction time task with aversive consequences, monetary incentives and actual physical control compared with the cognitive task, no aversive consequences, no incentives and perceived control in the present study. As has already been mentioned, the type, magnitude and perceived value of incentives may alter an individual's perception of the task which, in turn, may lead to changes in physiological function. The other discrepancies mentioned above may also markedly alter the pattern of psychophysiological responses, in such a way that cannot be easily anticipated.

There are a number of other factors which may help to account for the insignificant results of this study. One which will be briefly discussed is the type of feedback employed, which was confounded with both task difficulty and perceived control. The literature reviewed in Chapter 3, Section 2.1 suggests that the type of feedback - RF or KR - will determine whether or not it changes subject's performance and in what direction. The literature indicates that KR, which provides subjects with information about some aspect of performance which may be used to control subsequent performance, sustains that performance in complex, problem-solving tasks. Conversely, RF which provides information only to the level of 'success-failure' or 'right-wrong', may be of little use in this type of task because it provides no directive information. These differential effects are not due to differences in raising performance levels, however, as both KR and RF can be just as effective. Instead, differences arise in how the two types of feedback affect performance decrements. The literature suggests that differential effects occur through changes in level of motivation (i.e. the desire to do well), across the course of a task. In particular, RF leads to a performance decrement, because it cannot help subjects acquire knowledge about the properties of complex tasks. Moreover, in contrast to KR, RF does not lead to a simultaneous increase in perceived control or predictability over the situation, as it provides minimal information as to the nature of the response.

However, this is not consistent with the performance data of the current study, in which RF to the level of 'success-failure' led to significantly increased performance compared with a no feedback condition. Moreover, these effects were seen across the course of the experiment, with higher performance levels in later trials. It would appear therefore that RF did not produce a performance decrement, and that feedback in the form of RF can be effective in complex, decision-making tasks.

These findings are difficult to explain, particularly in association with the autonomic data in which there were no significant differences between the presence or absence of feedback. It may be that the two types of feedback - RF and KR - differentially effect levels of performance and physiological activity. Unfortunately, there are no studies which bear directly on this issue. Furthermore, the literature relating to physiological activity, reviewed in Chapter 3, Section 2.2.2, is inconclusive. The results of one study in which a complex matching task was employed indicated that RF, to the level of 'right-

wrong', does not significantly alter cardiovascular or electrodermal activity compared to no feedback conditions (Grilly, 1978). The authors conclusions were similar to those made in the performance literature; namely, that feedback which alters physiological function should consist of 'cognitive material' instead of response-orientated material, which enables subjects to perceive not only that their response was incorrect, but *why* it was incorrect.

If the current study had provided KR type feedback, in which subjects were told how and why they failed, perhaps the initial difference between feedback and no feedback groups seen in later trials, may have been sustained. Presumably, this would have occurred by sustaining an individual's level of motivation and promoting the 'desire to do well' through changes in a subject's perception of their own ability or competency at the task and/or the degree of perceived control. During the present study, by providing only RF combined with extremely high levels of task difficulty, feedback provided minimal additional information compared to the no feedback condition. Thus, to the majority of subjects feedback was probably irrelevant. Moreover, feedback may have had a negative impact in this particular task. For example, overall task difficulty was so high that feedback subjects were constantly (on 249 out of 286 trials) receiving 'failure' feedback. It seems unlikely that this degree of negative feedback had any significant incentive or motivational effects compared with the no feedback condition. Instead, the presence of feedback may have led to a lowering in overall motivation and a 'desire to do well'. Thus, the results of this study are difficult to generalise. However, it is possible, as indicated by the performance data, that RF may significantly alter autonomic function during complex tasks if levels of task difficulty are set at a more 'realistic' level (i.e. 50% success rate). Although these effects would depend, in turn, upon other mediating factors such as, the availability, type and magnitude of incentives and whether or not perceived or actual control was present.

There were additional findings of this study which, although not associated with the feedback manipulation, were of some interest. These included the physiological response patterns associated with the task. The task, as predicted, elicited strong autonomic activity with relatively large increases in cardiac levels over baseline, especially in relation to average (18 bpm) and minimum HR (26 bpm). Maximum HR also showed an increase

but not of the same magnitude (7 bpm). In addition, the results of the within-group analyses revealed highly significant differences in levels of activity across the various time periods of the task, in both electrodermal and cardiovascular systems.

In general, all three cardiovascular measures revealed similar clear-cut patterns of responding across time periods. At the beginning of each trial when subjects visually received the initial positions of the four planes (Time Period 2), and were asked for their first response (Time Period 4) HR showed a steady increase, reaching a peak when subjects were required to give their first response (Time Period 5). From this accelerative peak there followed an interval characterised by an anticipatory deceleration or inhibition, during the thirty seconds after subject's first response (Time Periods 5 to 9). Until the point in time when subjects received the visual cue to respond a second time (Time Period 9). At this point accelerative processes took over, but were of a smaller magnitude than the first accelerative response. Following the cue for subjects to give their second response (Time Period 10), there was a steady deceleration or stabilisation whilst subject's in the feedback condition received information concerning success or failure. Following this, both average and minimum HR produced a small increase whilst all subjects gave their ratings of perceived control, up until the point where they received a cue warning them of the start of the next trial (Time Periods 14 to 17). Maximum HR showed a similar pattern, but with an initial deceleration after receipt of feedback.

The pattern of responding for SCL was similar to that of the cardiovascular measures. However, frequency of NSFs revealed a slightly different pattern, with a steady number of NSFs between the beginning of the trial up until the 5 sec before subjects were asked for their first response (Time Periods 1 to 3). There was then a decrease, followed by a sharp increase in the number of response as subjects prepared to give their answer (Time Periods 4 & 5). From this peak, there was a significant decrease in NSFs during the interval between subject's first response and when the cue was presented for them to give their second response (Time Periods 5 to 9). At this point there was an increase in the number of NSFs of a similar magnitude to subject's first response. Following the cue for subjects to give their second response (Time Period 12) and up until the end of the trial (Time Period 17), levels of activity remained at a relatively stable level. The observed disassociation between measures of SCL and frequency of NSFs, was not unexpected.

Previous work, such as that by Kilpatrick (1972), indicates that measures of NSFs and electrodermal levels are relatively independent, with the latter being most sensitive to changes in cognitive processing (see Raskin, 1973 for a review).

One possible explanation for the changes in the pattern of physiological activity across time periods, may have been in terms of variations in motor output. For example, increases in autonomic activity centred around Time Periods 4, 5, 10 and 11 may have been due, not to psychological or motivational factors, but to subjects verbalising their responses and the corresponding interference with normal breathing patterns. Changes in motor activity, such as, O<sub>2</sub> consumption were not measured during the study so it is not possible to eliminate such an interpretation. However, there were significant changes in the magnitude of responding between the first (Time Periods 1 to 9) and second (Time Periods 9 to 17) halves of each trial (with the exception of the frequency of NSFs data), even though subjects were required to verbalise answers in both halves. Thus, it seems likely that psychological factors made a significant contribution to the observed effects.

Those psychological processes which may be involved in this type of behavioural challenge, and may also help to explain the significant differences between the first and second halves of each trial, have been mentioned previously. For example, the majority of subjects had some degree of perceived control, however minimal, in the first half of each trial when the amount of information and the cognitive demand on them was less. Levels of task involvement were, therefore, at their greatest during this time. This was reflected in higher levels of task performance and a corresponding increase in levels of cardiac activity accompanied by a similar increase in both tonic SCL and frequency of NSFs. However, during the second half of the trial the task itself was more difficult with an increase in the total amount of information presented to subjects, and an increased demand on visuo-spatial abilities, leading to a state of maximised uncertainty of success or failure accompanied by an increase in perceived lack of control. At some point, this 'information overload' led to the majority of subjects giving up on that particular trial. This, in turn, led to a decrease in overall motivation reflected by a lower level of task performance and a corresponding shift to lower levels of physiological activity. Furthermore, these differences between the two halves might be expected to be greater in later trials, a result confirmed, although not significantly, by the data.

There were also slight, non-significant, differences between the three cardiac measures - average, minimum and maximum HR - in terms of response patterning during the task, although these were not as large as anticipated by the literature reviewed in Chapter 5, Section 2.2.1. Indeed, this literature indicated that the three response elements might respond differently to the types of demands exerted upon the subject. For example, it was suggested that maximum HR would be most elevated at the most psychological stressful points; i.e. when the degree of task involvement or effort was at its greatest whereas changes in minimum HR would be reflected in greater decreases when perceptual-motor responding was at a minimal. In the current study, as mentioned earlier, all three measures followed similar patterns of responding across the various time periods of the task, with only small exceptions. For example, there was a slight disassociation between average and maximum HR during periods of high effort/motor output (i.e. Time Periods 4-5 and 10-11). That is, average HR peaked during the first of the two periods (Time Periods 4 & 10) compared to maximum HR which peaked later (Time Periods 5 and 11). Presumably, this was due to the influence of minimum HR on the former measure. In addition, there was a slight difference between the two measures during the 5 sec after subjects in the feedback condition had received information concerning success or failure (i.e. Time Period 14). With a decrease in levels of average HR compared to an increase in maximum HR. However, the greatest disassociation between the measures occurred between minimum and maximum HR, as would be expected by work reviewed in Chapter 5, Section 2.2.1. This was due to minimum HR producing a less clear-cut pattern of responding during periods of high effort/motor output (Time Periods 1-5 and 9-11) and those of low effort/motor output (Time Periods 5-9 and 11-17). Indeed, there were only small increases followed by, a slight decrease or stabilisation during these two types of periods. Additionally, as seen previously in average HR, minimum HR produced a decrease in levels of activity during Time Period 14 compared with maximum HR which produced an increase.

In general, these results do not support differential effects of this type of task on the three HR measures chosen to represent cardiovascular activity in this study. These findings were disappointing. Indeed, it had been hoped that by employing a difficult, externally-paced, information handling task, significant differences would emerge between the

measures which would help to elucidate on potential differences in neural innervation. No such conclusions could be made based on the available data. However, it is hoped that by measuring the three elements in future studies employing different tasks and methodologies, potential differences might emerge.

To summarise: although the results provided support for task influences on both cardiovascular and electrodermal response patterning the findings concerning feedback effects were contradictory. The results indicated that the presence of feedback was an important mediating factor both in increasing and sustaining performance levels on a complex, decision-making task. However, feedback was not effective in augmenting physiological activity in situations in which the task was very difficult and the feedback was predominantly that of failure.



## CHAPTER 7

### EXPERIMENT 2

#### 1. INTRODUCTION

In the previous experiment, feedback had little predictive value. In contrast to Weiss' (1971b) experiment, indication of success or failure did not signal the probability of an aversive outcome. It may be that any benefit of feedback may be derived from changes in the predictability of an aversive event. However, as discussed in Chapter 2 the effects of changes in predictability are not clear-cut, and need to be examined further before investigating any interaction with feedback.

The conflicting results in the literature may result from methodological difficulties. For example, the majority of predictability studies have introduced an additional confound associated with response interference. This occurs because of the presence of a warning stimulus in the predictable condition which itself elicits an autonomic response. This means that any comparisons between predictable and unpredictable conditions are confounded by the response elicited by the physical properties of the warning stimulus. Although some researchers have tried to overcome this problem, they have done so with only limited success (see Chapter 2, Section 3.1).

Thus, the main aim of this study was to manipulate temporal predictability whilst minimising any confounding effects due to response interference. This was achieved by employing a paradigm in which predictable subjects received the aversive event preceded by a warning stimulus on all trials whereas, unpredictable subjects received this combination on selected trials only. On the remaining trials, subjects in the unpredictable condition received numerous combinations; either, two aversive events occurring one after the other, the aversive event followed by the warning stimulus, or, the warning stimulus followed by a second identical warning stimulus. Only those trials in which all subjects received identical experimental stimuli were analysed (see Section 2.3).

An additional aim of the experiment was to assess the effects of warning signal duration on predictability. The preference behaviour literature reviewed in Chapter 2, Section 3.2, indicates that certain experimental parameters such as the length of warning

stimuli help to initially increase and then maintain preference for predictability in aversive situations. That is, preference behaviour increases as the length of the warning signal increases. Unfortunately, there are no studies to date which have manipulated this parameter and examined the effects on human physiological function. Nevertheless, it may be that the differential effects of warning signal duration can help to differentiate the theoretical explanations put forward to explain the supposedly beneficial effects of predictability; namely, the Safety Signal (Seligman, 1968), Preparatory Response (Perkins, 1968) and Preception (Lykken, 1962) Theories.

According to Seligman's safety analysis, the presence of a warning signal is a perfect predictor of the aversive event while the absence of the signal is a perfect predictor of safety. Thus, this analysis would predict fear during the warning signal and the aversive event itself and no fear during their absence. In contrast, during situations where no predictor of safety is present (i.e. in unpredictable conditions), the safety signal hypothesis suggests that subjects are in constant fear. Predictions based on a safety analysis would therefore be in terms of greater autonomic activity to unpredictable compared with predictable conditions during the safe periods (i.e. the inter-stimulus and inter-trial intervals) but similar reactions to the danger periods (i.e. the warning signal and aversive event). In contrast, the two preparatory analyses suggest that the presence of a warning signal preceding the onset of an aversive event allows the elicitation of a response acting to mitigate its impact. Thus, these theories predict lower autonomic activity during predictable aversive events compared to unpredictable ones. Neither of these theories make any specific predictions with regard to activity during the warning signal, or, anticipatory intervals. However, it might be suggested that greater autonomic activity should occur with unpredictability because with unpredictable events the subject is anticipating a more painful event since they are not able to prepare for it .

With regard to warning signal duration, the safety-signal hypothesis would predict that subjects should prefer short over long warning signals with the latter leading, in turn, to greater levels of anticipatory physiological activity. This is because in the short warning condition, the safe periods (i.e. during signal absence) are more easily identifiable and longer. Subjects in long warning conditions spend more of their time in the presence of the signal (i.e. danger periods) which means that they will remain in chronic fear over a longer

period of time, leading to a higher state of anticipatory physiological arousal compared to subjects in the short warning condition. The opposite is suggested by the preparatory theories. That is, individuals should prefer and show less psychophysiological activity at event impact in the long warning compared to short warning conditions, because the former situation allows subjects to prepare more adequately for that event (see Chapter 2, Section 2). Furthermore, based on suggestions made in the previous paragraph, long warning conditions may also lead to lower physiological responding during anticipatory periods, compared to short warning situations.

These predictions are only tentative, however, since both the safety signal and preparatory explanations have been subject to numerous criticisms on both conceptual and methodological grounds, mainly because the predictions made by each of the theories can be adjusted in accordance with the findings of the empirical data. For example, although the preparatory theories would predict lower psychophysiological stress in long warning conditions, the opposite assumption can also be made; namely, that preparatory responses must be precisely timed to coincide with the aversive event and conditions which allow for this (i.e. short warning signals) will be associated with lower psychophysiological stress than conditions which do not (i.e. long warning signals).

## 2. METHOD

### 2.1. Subjects

The subjects were 36 undergraduate volunteers (28 females and 8 males), with an age range of 18 - 40 years. All subjects were randomly allocated to one of four experimental conditions; predictable/long warning (PL), predictable/short warning (PS), unpredictable/long warning (UPL) or unpredictable/short warning (UPS). The only restriction to the random allocation was that there were equal numbers of the smaller, male population in each of the four groups.

## 2.2. Apparatus

The method of recording SC and HR was the same as that employed in Experiment 1. Presentation of the warning stimuli and slides was controlled by a BBC, model B microcomputer, situated in the experimenter's room. The warning stimuli consisted of the numbers 5, 4, 3, 2, 1 which were displayed on a colour monitor of 15 x 13.5 inches situated in the subject's room. This was positioned 31 inches from the subject and slightly to their right. Image size was 1.5 x 1.5 inches. For those subjects in the long warning groups, each number was displayed on the monitor for a duration of 5 sec. For those in the short warning groups, each number was displayed for 1 sec duration.

The slides consisted of 13 colour photographs of mutilation and injury taken from The International Standard Affective Slide Set, available from P. Lang's Laboratory. Slides were projected onto a ground glass screen 43 inches directly in front of the subject by a Kodak Carousel projector. Image size was 20 x 30 inches.

A two way intercom system was in operation throughout. It was situated 8 inches from the subject and directly in front of them.

## 2.3. Design

In the PL and PS groups, the experimental procedure consisted of the warning stimulus, a countdown from 5 to 1, with the duration of each figure depending on whether subjects were assigned to the long or short warning groups (see Section 2.2). Following this, there was a 5 sec inter-stimulus interval (ISI) before the onset of the slide. Slide duration was 10 sec. In the UPL and UPS groups, subjects received four different types of trial: Trial Type 1 (TT1), trials in which the slide was preceded by the warning stimulus: Trial Type 2 (TT2), trials in which two slides occurred one after the other: Trial Type 3 (TT3), trials in which a slide was followed by the warning stimulus: Trial Type 4 (TT4), trials in which the warning stimulus was followed by a second identical warning stimulus. Subjects received the four trial types in the same order; TT1 in Trials 1, 5, 8 and 13: TT2, in Trials 2, 7 and 10: TT3 in Trials 3, 9 and 12 and TT4 in Trials 4, 6 and 11. Duration of

the warning stimulus was the same as in the PL and PS conditions. Slide duration was 10 sec. In all trials, a 5 sec ISI occurred before the second half of the trial.

#### 2.4. Procedure

The seating and accommodation of subjects was the same as in Experiment 1. After electrode attachment, subjects were informed of the procedure to follow (see appendix). Subjects in the predictable groups were told that they would receive a warning consisting of the numbers 5, 4, 3, 2, 1 on the monitor shortly before the onset of the slide. Those subjects in the unpredictable groups were told that the numbers 5, 4, 3, 2, 1 and the slide would occur randomly to one another. The experimental procedure commenced with a 5 min resting period. Following this, subjects in each of the groups received 13 trials with an inter-trial interval (ITI) of 25, 30 or 35 sec. The mean ITI was 30 sec. After the subject had completed 13 trials, the experimenter unwired and debriefed the subject.

#### 2.5. Scoring

2.5.1. Cardiovascular activity. The measurement of HR was the same as in Experiment 1. All three cardiovascular measures (avHR, mnHR and mxHR) were assessed during the last 60 sec of the 5 min pre-task baseline in order to examine any pre-manipulation between-group differences.

Between-group differences in cardiovascular activity were assessed across Trials 1, 5, 8 and 13, as these were the only trials in which the predictable and unpredictable groups received exactly the same experimental procedure. Trial 1 was analysed separately from Trials 5, 8 and 13. Each trial was divided into 5 separate time periods: Period 1, consisted of the 0-5 sec after warning onset: Period 2, was the 0-5 sec before onset of the slide (ISI): Period 3, was the 0-10 sec after slide onset: Period 4, was the 25 sec ITI divided into five, 5 sec blocks: Period 5, involved a measure of the cardiac response profile obtained for each post-stimulus second up to ten seconds following slide onset. The cardiac response profile of Period 5 was then calculated as the difference between each post-stimulus second and the pre-stimulus mean (Period 2) and was expressed as changes in bpm. Uncorrected and

corrected HR are reported for all three cardiovascular measures across Time Periods 1 to 4. Measurement of corrected HR was the same as in Experiment 1. Period 5 was examined for mean change in avHR across Trials 5, 8 and 13 only.

2.5.2. Electrodermal activity. Tonic SCL and frequency of NSF's were assessed during the last 60 sec of the 5 min pre-task baseline. They were measured following the procedure of Experiment 1.

Between-group differences in electrodermal activity were assessed across Trials 1, 5, 8 and 13. Trial 1 was analysed separately from Trials 5, 8 and 13. For tonic SCL, each trial was divided into five time periods: Period 1, was the 0-5 sec after warning onset: Period 2, consisted of the 0-5 sec before onset of the slide (ISI): Period 3, was the 0-5 sec after slide onset: Period 4 was the 5-10 sec after slide onset and Period 5 consisted of the 25 sec ITI divided into five, 5 sec blocks. The frequency of NSF's was assessed during Period 5 only.

Any artefact free changes in SC greater than 0.02  $\mu$ S which occurred from 1-5 sec after stimulus onset were considered a response evoked by the stimulus. SCR amplitude was subjected to a square-root transformation prior to analysis in order to reduce the skew. Between-group comparisons of SCR amplitude were assessed during two time periods; Period 1 was the 1-5 sec after warning onset and Period 2 consisted of the 1-5 sec after slide onset.

### 3. RESULTS

#### 3.1. Baseline Activity

For all measures, differences between groups were assessed during the last 60 sec of the baseline in a 2-way ANOVA with predictability (predictable/unpredictable) and duration (long/short) as between subject factors. Group means and standard deviations for cardiovascular and electrodermal baseline activity are shown in Tables 11 and 12 respectively.

3.1.1. Cardiovascular Activity. There were no differences between groups in terms of avHR or mnHR (both  $F_s < 1$ ). Maximum HR revealed a main effect of predictability ( $F(1, 32) = 10.6, p < .01$ ). The P groups had higher HR than the UP groups (102.6 and 92.9 bpm respectively). There were no main effects or interactions involving duration for any of the three measures (all  $F_s < 1$ ).

3.1.2. Electrodermal Activity. There were no main effects or interactions involving NSF<sub>s</sub> or tonic SCL (all  $F_s < 1$ ).

### 3.2. Between-Groups Analysis: Trial 1

#### 3.2.1. Cardiovascular Activity

For all measures, Time Periods 1 to 3 were each assessed in a two-way ANOVA with predictability and duration as between subject factors. Time Period 4 was analysed in a three-way MANOVA with the above between subject factors and time (5 levels) as a repeated measure. Group means and standard deviations for uncorrected HR during Trial 1 are shown in Table 11.

##### 3.2.1.1. Uncorrected HR

3.2.1.1.1. Time Period 1. There was a significant effect of predictability involving avHR ( $F(1, 32) = 4.8, p < .05$ ) and mxHR ( $F(1, 32) = 5.3, p < .05$ ). The P groups had higher HR than the UP groups (avHR = 86.7 and 78.3 bpm; mxHR = 94 and 83.7 bpm respectively). MnHR produced no significant effect of predictability ( $F < 1$ ). There were no main effects or interactions involving duration across any of the three measures (all  $F_s < 1$ ).

3.2.1.1.2. Time Period 2. There were no main effects of predictability involving avHR or mnHR (both  $F_s < 1$ ), nor were there any significant effects or interactions involving duration for avHR ( $F(1, 32) = 1.8$ ) or mnHR ( $F(1, 32) = 2.1$ ). MxHR produced a significant effect of predictability ( $F(1, 32) = 4.7, p < .05$ ). The P groups had higher HR

Table 11. Mean cardiovascular activity during the pre-task baseline and time periods of Trial 1. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Group			
	PL	PS	UPL	UPS
Pre-Task Baseline				
AvHR	81.5 (9.56)	84.2 (12.8)	78.5 (9.66)	79.9 (12.5)
MnHR	63.6 (11.9)	69.9 (10.8)	64.0 (11.0)	69.2 (11.2)
MxHR	102.0 (7.55)	103.0 (11.0)	95.1 (9.76)	90.7 (14.4)
Time Period 1				
AvHR	84.9 (15.7)	88.5 (9.01)	75.6 (11.4)	81.0 (7.89)
MnHR	79.0 (15.4)	76.7 (4.34)	70.0 (6.09)	75.8 (5.99)
MxHR	91.7 (15.2)	96.4 (12.0)	80.8 (17.3)	86.7 (6.17)
Time Period 2				
AvHR	80.6 (15.9)	82.6 (9.64)	76.5 (13.3)	84.6 (8.24)
MnHR	72.6 (15.6)	75.6 (10.9)	69.7 (5.98)	79.2 (7.45)
MxHR	95.9 (11.1)	91.5 (10.7)	81.4 (16.1)	89.9 (7.84)
Time Period 3				
AvHR	78.4 (15.3)	81.6 (10.3)	77.2 (9.66)	82.2 (9.38)
MnHR	71.4 (14.4)	72.4 (11.5)	69.2 (5.95)	73.3 (10.9)
MxHR	89.7 (14.0)	93.1 (8.16)	83.3 (17.4)	89.4 (8.58)
Time Period 4				
AvHR	82.5 (11.0)	80.8 (15.3)	79.4 (9.90)	75.1 (12.1)
MnHR	74.5 (7.98)	75.4 (17.7)	74.1 (12.0)	69.8 (6.78)
MxHR	92.9 (11.2)	87.8 (14.0)	85.9 (11.1)	81.0 (12.6)



than the UP groups (93.7 and 85.6 bpm respectively). There was no main effect of duration ( $F < 1$ ) or any interaction involving duration ( $F(1, 32) = 3.1$ ) for mxHR.

3.2.1.1.3. Time Period 3. There were no significant differences between groups involving any of the three measures (all  $F_s < 1$ ).

3.2.1.1.4. Time Period 4. There were no significant effects of predictability or duration for avHR or mnHR (all  $F_s < 1$ ), nor were there any effects of predictability ( $F(1, 32) = 3.3$ ) or duration ( $F(1, 32) = 1.8$ ) involving mxHR. There was no significant difference across time involving any of the measures: avHR ( $F(3, 65) = 2.5$ ), mnHR ( $F(3, 64) = 1.1$ ) or mxHR ( $F(3, 74) = 1.3$ ), nor were there any interactions involving time for the three measures (all  $F_s < 1$ ).

3.2.1.1.5. Summary. There was a significant effect of predictability during the warning stimulus (avHR and mxHR) and the ISI (mxHR only), due to the P groups having higher HR levels than the UP groups. There were no significant effects of predictability involving mnHR, nor were there any main effects or interactions involving duration across any of the three measures.

Further examination of the pattern of responding across Time Periods 1 to 4 reveals significant differences between the three cardiovascular measures (see Table 11). AvHR produced a slight, non-significant increase from the pre-task baseline to the warning stimulus ( $T(1, 35) = 1.25$ ). In contrast, there was a highly significant increase in mnHR to the warning stimulus ( $T(1, 35) = 4.54, p < .001$ ), and a highly significant decrease in mxHR ( $T(1, 35) = 4.55, p < .001$ ). Comparisons between the pre-task baseline and the 0-10 sec after onset of the slide, revealed similar levels of activity for avHR ( $T < 1$ ), a highly significant decrease for mxHR ( $T(1, 35) = 4.37, p < .001$ ), and a significant increase involving mnHR ( $T(1, 35) = 4.31, p < .001$ ). In addition, the difference in levels of activity between the warning stimulus and the 0-10 sec period after slide onset reached significance for mnHR only, with a small decrease to the slide ( $T(1, 35) = 2.03$ ). The remaining two measures showed similar levels of activity to the warning and slide onset: avHR ( $T(1, 35) = 1.54$ ) and mxHR ( $T < 1$ ).

3.2.1.2. Corrected HR. The results across all time periods were similar to those of uncorrected HR, unless stated otherwise. MxHR during Time Periods 1 and 2 produced no

significant effect of predictability (both  $F_s < 1$ ). This was due to the P and UP groups producing similar decreases in HR levels from the pre-task baseline (Time Period 1 = -8.6 and -9.15; Time Period 2 = -8.9 and -7.25 change in bpm respectively). During Time Period 2, mxHR produced a predictability by duration interaction ( $F(1, 32) = 6, p < .05$ ). Analysis of the simple main effects revealed that the UPS group had significantly smaller changes in HR than the UPL and PS groups. There were no significant differences between any of the remaining groups (Figure 20).

3.2.1.3. Cardiovascular Summary. The results of average and minimum corrected HR were similar to those of uncorrected HR, but, mxHR did produce slightly different results. However, these results have to be considered in the light of pre-task between-group differences in which the P groups had higher HR than the UP groups (Section 3.1.1). As might be expected, corrected mxHR during Time Periods 1 and 2 produced no significant difference between the P and UP groups. Time Period 2 produced a significant predictability by duration interaction, this was due to the UPS group producing only slight differences between the baseline period and Trial 1 whereas, the PS and UPL groups produced a marked decrease in levels of activity from the baseline to Trial 1. It would appear, therefore, that differences between the predictability groups during Trial 1 can be accounted for by differential levels of activity in the pre-task baseline. Corrected mxHR during Time Periods 3 and 4 produced results very similar to uncorrected HR.

### 3.2.2. Electrodermal Activity

Tonic SCL was assessed in each of Time Periods 1 to 4 in a two way ANOVA with predictability and duration as between subject factors. SCL and frequency of NSFs during Period 5 were analysed in a three factor MANOVA with the above between subject factors and time as a repeated measure. Transformed scores for SCR amplitude were analysed in a two-way ANOVA with the above between subject factors. Group means and standard deviations for electrodermal activity during Trial 1 are shown in Table 12.

Figure 20. Mean change in maximum HR as a function of predictability and duration during Time Period 2, Trial 1.

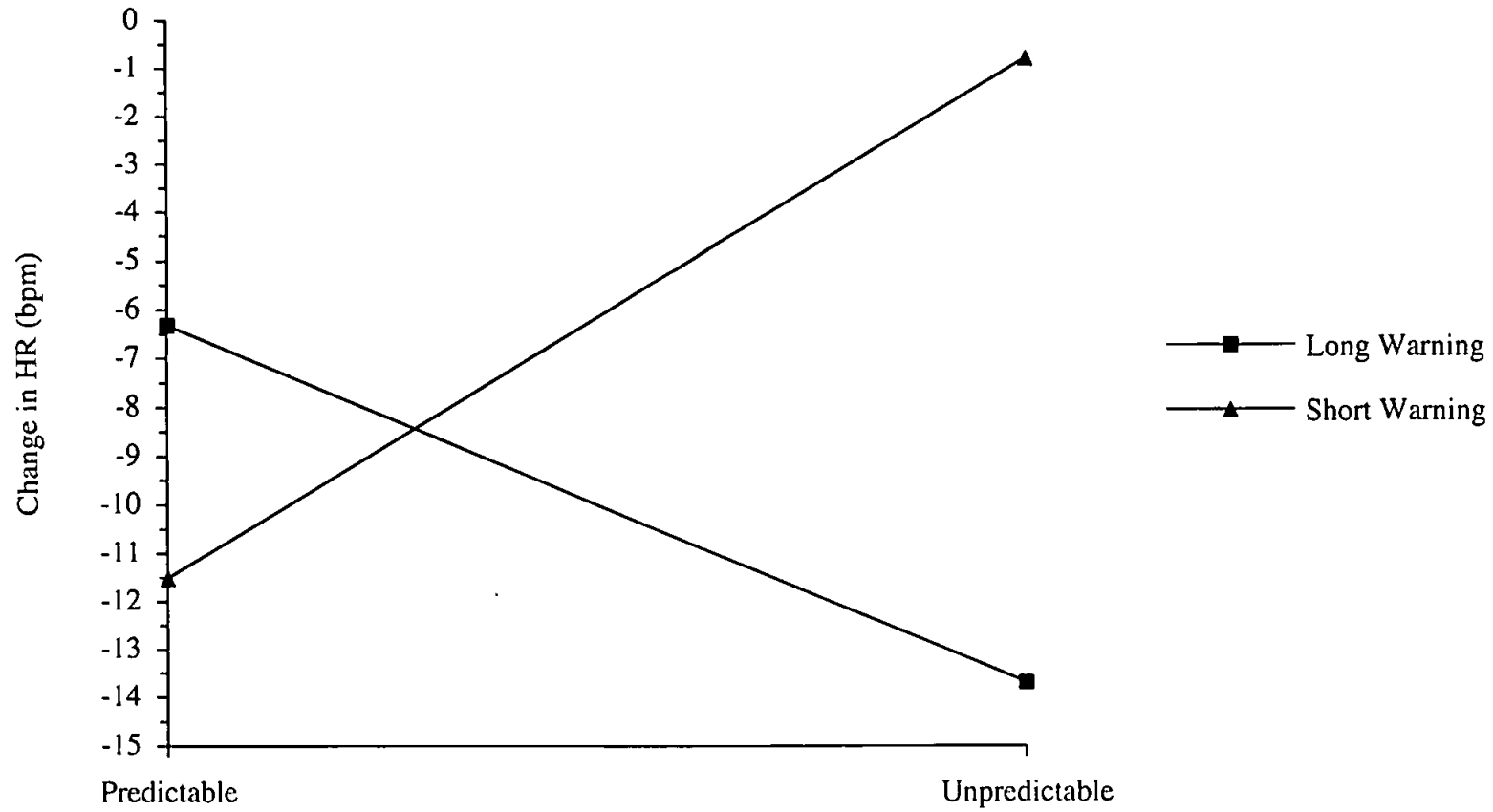


Table 12. Mean electrodermal activity during the pre-task baseline and time periods of Trial 1.  
Standard deviations are shown in parentheses.

Variables	Experimental Groups			
	PL	PS	UPL	UPS
Pre-Task Baseline				
Tonic SCL ( $\mu\text{S}$ )	1.67 (0.56)	1.54 (0.78)	1.56 (0.56)	1.78 (0.57)
Frequency of NSF's	2.67 (1.73)	2.45 (2.40)	2.33 (3.54)	2.89 (4.17)
Time Period 1				
Tonic SCL ( $\mu\text{S}$ )	1.70 (0.55)	1.78 (0.70)	1.58 (0.53)	1.79 (0.59)
SCR Amplitude ( $\sqrt{\mu\text{S}}$ )	0.20 (0.06)	0.00 (0.00)	0.00 (0.00)	1.41 (0.08)
Time Period 2				
Tonic SCL ( $\mu\text{S}$ )	1.78 (0.54)	1.79 (0.69)	1.60 (0.53)	1.80 (0.59)
Time Period 3				
Tonic SCL ( $\mu\text{S}$ )	1.78 (0.50)	1.80 (0.70)	1.62 (0.51)	1.82 (0.59)
SCR Amplitude ( $\sqrt{\mu\text{S}}$ )	0.26 (0.08)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)
Time Period 4				
Tonic SCL ( $\mu\text{S}$ )	1.85 (0.43)	1.88 (0.77)	1.62 (0.50)	1.98 (0.60)
Time Period 5				
Tonic SCL ( $\mu\text{S}$ )	1.80 (0.44)	1.86 (0.74)	1.60 (0.55)	1.98 (0.64)
Frequency of NSF's	0.24 (0.50)	0.40 (0.73)	0.15 (0.33)	0.13 (0.33)

### 3.2.2.1. Skin Conductance Level

3.2.2.1.1. Time Periods 1 to 4. There were no significant main effects or interactions (all  $F_s < 1$ ).

3.2.2.1.2. Time Period 5. There were no main effects or interactions involving predictability or duration (all  $F_s < 1$ ). There was a significant difference across time ( $F(1, 33) = 7.5, p < .01$ ), with significantly higher levels of activity during the first 5 sec of the ITI compared with the 10-15, 15-20 and 20-25 sec periods. The 5-10 sec period also produced significantly greater levels of tonic SCL compared with the 15-20 and 20-25 sec periods (1.84, 1.82, 1.79, 1.80 and 1.79  $\mu S$  for the 5-10, 10-15, 15-20 and 20-25 sec periods respectively).

3.2.2.2. Non-Specific Fluctuations. There were no significant effects of predictability ( $F(1, 32) = 2.9$ ) or duration ( $F < 1$ ), nor were there any differences across time or any interactions involving time (all  $F_s < 1$ ).

3.2.2.3. SCR Amplitude. There were no main effects or interactions involving Time Period 1 (all  $F_s < 1$ ) or 2 (all  $F_s (1, 32) = 2.8$ ).

### 3.3. Between-Groups Analysis: Trials 5, 8 & 13

#### 3.3.1. Cardiovascular Activity

For all measures, Time Periods 1 to 3 were each analysed in a three-way MANOVA with predictability and duration as between subject factors and trials (Trials 5, 8 and 13) as a repeated measure. Time Periods 4 and 5 were analysed in four-way MANOVA's with the above between subject factors and trials and time (5 and 10 levels respectively) as repeated measures. Group means and standard deviations for uncorrected HR during Time Periods 1 to 4 are shown in Table 13.

Table 13. Mean cardiovascular activity during time periods averaged across Trials 5, 8 and 13. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Group			
	PL	PS	UPL	UPS
Time Period 1				
AvHR	77.6 (11.5)	81.3 (9.89)	75.2 (10.5)	78.4 (9.92)
MnHR	70.7 (12.9)	74.6 (7.56)	69.4 (5.56)	72.9 (9.88)
MxHR	86.5 (11.0)	90.9 (9.00)	80.3 (10.9)	83.2 (11.1)
Time Period 2				
AvHR	77.6 (13.1)	80.7 (10.0)	73.4 (10.6)	75.3 (9.98)
MnHR	71.6 (14.1)	73.1 (8.98)	69.6 (6.66)	70.6 (9.90)
MxHR	85.4 (12.0)	89.5 (10.5)	79.5 (12.0)	80.9 (10.9)
Time Period 3				
AvHR	75.0 (13.0)	77.7 (9.99)	74.1 (10.9)	75.4 (9.20)
MnHR	67.8 (11.9)	69.0 (5.56)	67.5 (8.50)	68.2 (6.61)
MxHR	84.5 (13.9)	89.0 (10.9)	82.6 (13.5)	83.0 (11.6)
Time Period 4				
AvHR	80.3 (8.90)	77.1 (11.5)	75.6 (12.1)	74.9 (8.93)
MnHR	73.1 (13.9)	70.4 (10.0)	70.6 (8.98)	69.3 (7.01)
MxHR	88.2 (9.45)	83.5 (12.3)	82.0 (14.8)	80.6 (12.2)

### 3.3.1.1. Uncorrected Heart Rate

3.3.1.1.1. Time Period 1. There were no main effects or interactions involving predictability or duration for avHR or mnHR (all  $F_s < 1$ ). However, mxHR did produce a significant effect of predictability ( $F(1, 32) = 5, p < .05$ ). The P groups had higher HR compared with UP groups (89.0 and 81.7 bpm respectively). There was no significant effect of duration ( $F(1, 32) = 1.5$ ) involving mxHR. There was no significant difference across trials involving avHR ( $F(2, 41) = 3$ ) or mnHR ( $F < 1$ ), nor any interaction involving trials (all  $F_s < 1$ ). MxHR produced no significant difference across trials ( $F(2, 41) = 3.2$ ), nor any interaction involving trials (all  $F_s < 1$ ).

3.3.1.1.2. Time Period 2. There were no significant effects of predictability involving avHR ( $F(1, 32) = 1.7$ ) or mnHR ( $F(1, 32) = < 1$ ). However, mxHR did produce a significant effect of predictability ( $F(1, 32) = 4.4, p < .05$ ). The P groups had higher HR than the UP groups (87.4 and 80.2 bpm respectively). There were no significant main effects or interactions involving duration for any of the three measures (all  $F_s < 1$ ). There was no significant difference across trials for avHR ( $F(2, 47) = 1.8$ ), nor any interaction involving trials and predictability ( $F(2, 47) = 1.9$ ), trials and duration ( $F(2, 47) = 1.8$ ) or any three-way interaction ( $F < 1$ ). MnHR produced a significant difference across trials ( $F(2, 43) = 3.9, p < .05$ ), due to Trial 5 showing significantly higher HR compared with Trial 13. There were no other significant differences between trials (72.6, 71.7 and 69.4 bpm for Trials 5, 8 and 13 respectively). There was no significant interaction between trials and predictability ( $F(2, 43) = 1.4$ ), between trials and duration ( $F(2, 43) = 1.9$ ) or any three-way interaction ( $F < 1$ ). MxHR produced no significant main effect of trials or any interaction involving trials (all  $F_s < 1$ ).

3.3.1.1.3. Time Period 3. There were no main effects or interactions involving predictability or duration across any of the measures (all  $F_s < 1$ ). There was no significant difference across trials or any interactions involving trials for avHR (all  $F_s < 1$ ). However, mnHR did reveal a difference across trials ( $F(2, 43) = 6.3, p < .01$ ). Trial 5 produced significantly higher HR compared with Trial 13. There were no significant differences between any other trials (69.7, 68.7 and 66.2 bpm for Trials 5, 8 and 13 respectively). There were no other interactions involving trials for mnHR (all  $F_s < 1$ ). MxHR produced no significant difference across trials, nor any interaction involving trials (all  $F_s < 1$ ).

3.3.1.1.4. Time Period 4. There were no significant main effects of predictability involving avHR, mnHR (both  $F_s < 1$ ) or mxHR ( $F(1, 32) = 2.3$ ), nor were there any significant effects involving duration for any of the three measures (all  $F_s < 1$ ). AvHR produced no significant main effects or interactions involving trials or time (all  $F_s < 1$ ). MnHR produced no significant difference across trials or time, nor any interactions involving time ( $F < 1$ ). However, there was a predictability by duration by trials interaction involving mnHR ( $F(2, 44) = 3.8, p < .05$ ). Analysis of the simple simple main effects revealed that the PL group had significantly higher HR than the PS, UPL and UPS groups during Trials 5 and 8. There were no significant differences between groups during Trial 13. The PL and UPS groups produced a significant decrement in HR levels during Trial 13 compared with the PS and UPL groups which produced similar levels of activity across all three trials. The PL group during Trials 5 and 8 produced significantly higher HR compared with the UPS group during Trial 13 (Figure 21). MxHR produced no significant difference across trials or time, nor any interactions involving trials and time (all  $F_s < 1$ ).

3.2.1.1.5. Time Period 5. The UP groups produced a significantly greater increase in HR than the P groups ( $F(1, 32) = 4.3, p < .05$ ) (0.19 and -2.1 change in bpm respectively). There was a significant difference across time ( $F(4, 103) = 3.6, p < .001$ ) and a duration by trials by time interaction ( $F(6, 143) = 2.8, p < .05$ ). Analysis of the simple simple main effects revealed, firstly for Trial 5, that the Long Warning (LW) groups had greater increases in HR compared with the Short Warning (SW) groups during Seconds 6 and 9. There were no significant differences between groups during any of the remaining seconds. Analysis of Trial 8 revealed that the SW groups had a greater increase in HR than the LW groups during Second 9 whereas, there were no significant differences between groups during the remaining seconds. Analysis of Trial 13 revealed that the LW groups had greater increases in HR compared with the SW groups during Seconds 2, 3 and 10 whereas, during Seconds 5 and 6 the SW groups produced greater increases in HR than the LW groups (Figure 22).

3.2.1.1.6. Cardiovascular Summary. During the warning stimulus (Time Period 1), the P groups produced higher HR levels compared with the UP groups in terms of mxHR. However, there were no other main effects or interactions involving mxHR, nor were there any differences in terms of avHR or mnHR during this period.



Figure 21. Mean minimum HR as a function of groups and trials during Time Period 4.

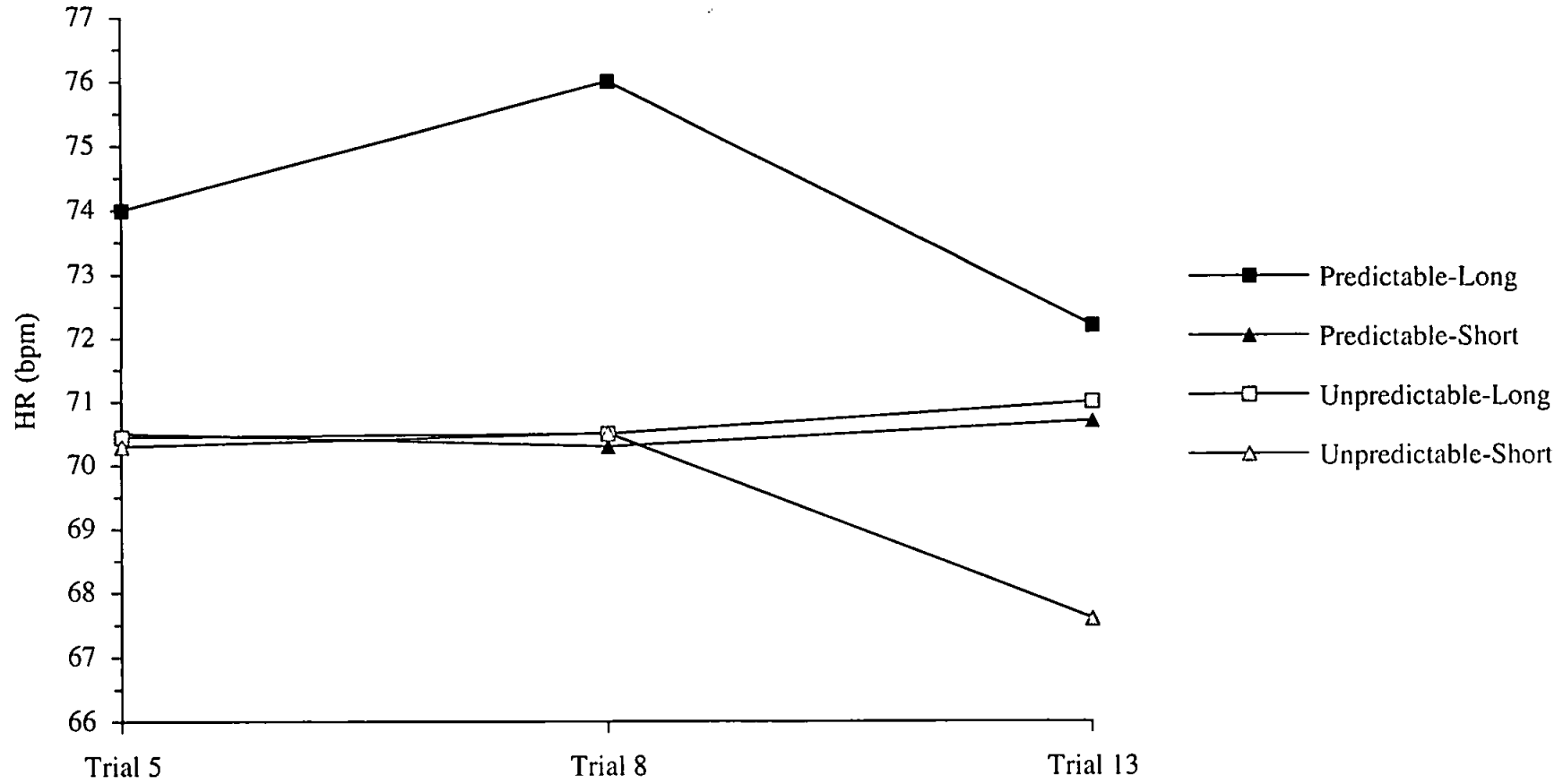
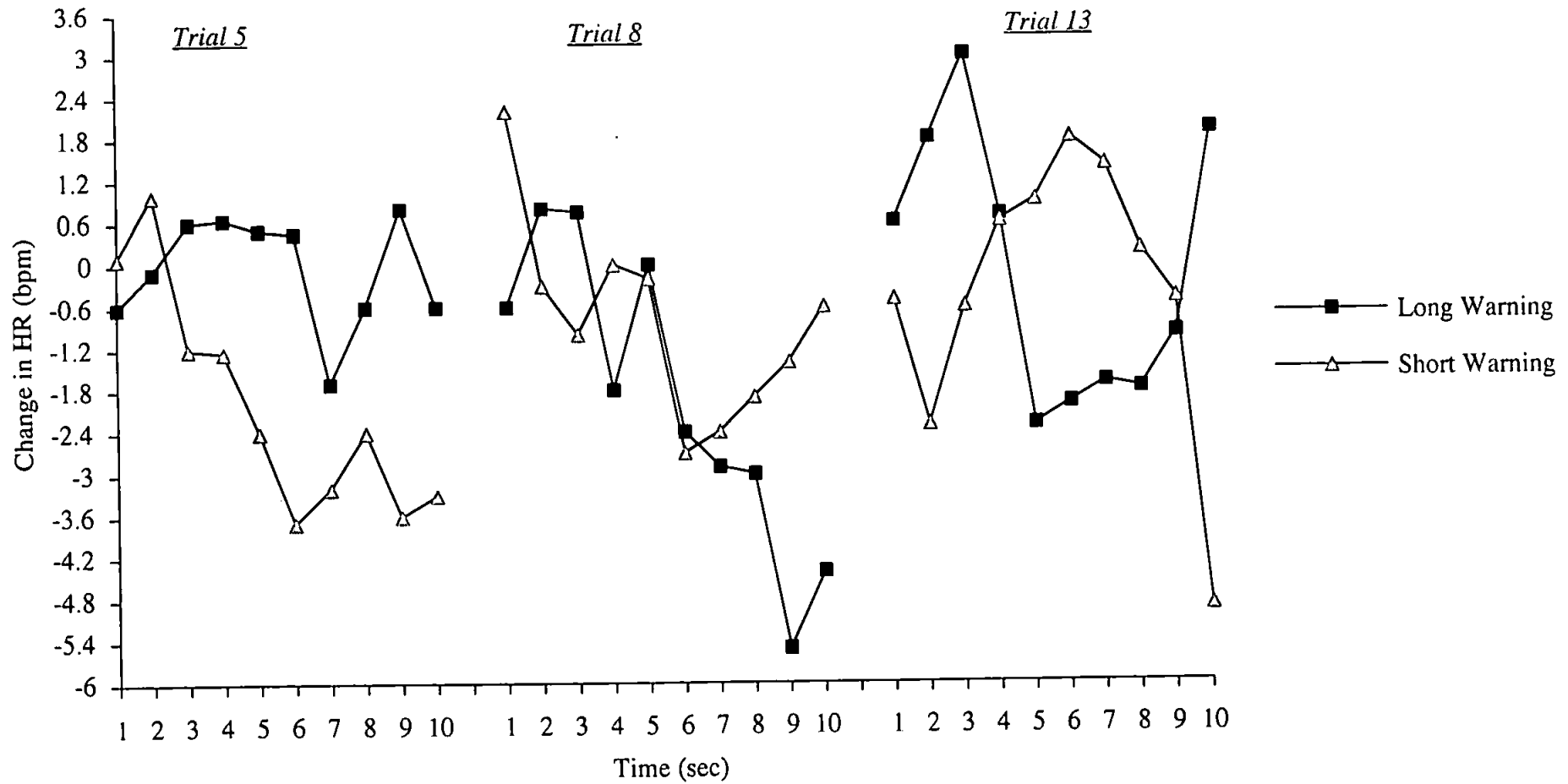


Figure 22. Mean change in average HR as a function of duration, trials and time during Time Period 5.



During the ISI (Time Period 2), the P groups again had higher mxHR levels compared with the UP groups. There were no other differences between groups in terms of mxHR during this period. For mnHR, however, Trial 5 produced higher HR than Trial 13. MnHR produced no other significant effects during the ISI, and nor were there any in terms of avHR.

Again, during slide duration (Time Period 3) mnHR produced a decrement in levels of activity across trials. However, there were no other significant effects involving mnHR during this period. In addition, avHR and mxHR revealed no main effects or interactions.

During the ITI (Time Period 4) mnHR produced the only significant effects. The PL group displayed higher HR than the remaining three groups during Trials 5 and 8 compared with no significant differences between groups in Trial 13 whereas, there were no significant differences between the PS, UPL and UPS groups.

Analysis of the cardiac response profile (Time Period 5) revealed that the UP groups had a greater increase in HR compared with the P groups. There was also a predictability by trials by time interaction. However, further analysis of this interaction did not reveal any meaningful pattern of results.

3.3.1.2. Corrected Heart Rate. The results of corrected HR were similar to those of uncorrected HR, unless stated otherwise. During Time Periods 1 and 2, mxHR produced no significant effect of predictability (both  $F_s < 1$ ). Further inspection of the means revealed that the UP and P groups showed similar decreases in HR levels from the baseline (Time Period 1 = -13.6 and -11.2 change in bpm; Time Period 2 = -15.2 and -12.7 change in bpm for P and UP respectively).

### 3.3.2. Electrodermal Activity

Tonic SCL (Time Periods 1 to 4) and transformed SCR amplitude (Time Periods 1 and 2) were assessed using a three-way MANOVA with predictability and duration as between subject factors and trials as a repeated measure. SCL and frequency of NSFs during Time Period 5 were examined using a four factor MANOVA with the above between subject factors and trials and time as repeated measures. Group means and

standard deviations for electrodermal activity during Time Periods 1 to 5 are shown in Table 14.

3.3.2.1. Skin Conductance Level. There were no significant main effects or interactions involving any of the time periods (all  $F_s < 1$ ).

3.3.2.2. Non-Specific Fluctuations. There were no main effects involving predictability or duration (all  $F_s < 1$ ), and nor was there any interaction involving the two factors ( $F(1, 32) = 2.1$ ). There were no significant main effects or any interactions involving trials or time (all  $F_s < 1$ ).

3.3.2.3. SCR Amplitude. Time Period 1 produced no significant effect of predictability ( $F < 1$ ), duration ( $F(1, 32) = 2.5$ ) or any interaction between the two factors ( $F(1, 32) = 1.6$ ). There was no significant difference across trials ( $F(2, 34) = 2.5$ ), or any interaction involving trials (all  $F_s(2, 34) < 1$ ). Time Period 2 produced no significant effects or interactions involving predictability or duration (all  $F_s(1, 32) = 1.9$ ). There was no significant difference across trials or any interaction involving trials ( $F < 1$ ).

#### 4. DISCUSSION

The major aim of this experiment was to investigate the physiological effects of predictability by supplying half of the subjects with information about the occurrence of an aversive event. In addition, the role of warning stimulus duration in augmenting or attenuating these effects was assessed. A number of predictions had been made concerning the effects of predictability on autonomic activity which were based on literature reviewed in Chapter 2, Section 3.3. These predictions were not substantiated by data from this experiment, with the exception of the cardiac response profile which produced a main effect of predictability due to the unpredictable groups showing higher HR levels than predictable groups. Manipulation of warning stimulus duration led to no significant changes in autonomic function in either predictable or unpredictable conditions, with the exception of data from the inter-trial interval in which the predictable-long group had

Table 14. Mean electrodermal activity during time periods averaged across Trials 5, 8 and 13.  
Standard deviations are shown in parentheses.

Variables	Experimental Groups			
	PL	PS	UPL	UPS
Time Period 1				
Tonic SCL ( $\mu\text{S}$ )	1.72 (0.53)	1.76 (0.74)	1.56 (0.54)	1.89 (0.54)
SCR Amplitude ( $\sqrt{\mu\text{S}}$ )	0.01 (0.07)	0.16 (0.10)	0.11 (0.40)	0.14 (0.09)
Time Period 2				
Tonic SCL ( $\mu\text{S}$ )	1.74 (0.54)	1.76 (0.74)	1.56 (0.56)	1.89 (0.52)
Time Period 3				
Tonic SCL ( $\mu\text{S}$ )	1.72 (0.53)	1.79 (0.75)	1.57 (0.55)	1.88 (0.54)
SCR Amplitude ( $\sqrt{\mu\text{S}}$ )	0.01 (0.06)	0.10 (0.10)	0.00 (0.00)	0.00 (0.00)
Time Period 4				
Tonic SCL ( $\mu\text{S}$ )	1.72 (0.54)	1.79 (0.78)	1.57 (0.53)	1.89 (0.50)
Time Period 5				
Tonic SCL ( $\mu\text{S}$ )	1.71 (0.56)	1.76 (0.73)	1.59 (0.51)	1.88 (0.50)
Frequency of NSF's	0.12 (0.33)	0.19 (0.50)	0.16 (0.33)	0.06 (0.33)

significantly higher minimum HR compared to predictable-short or unpredictable groups.

In general, the findings concerning the beneficial effects of predictability were not conclusive in either cardiovascular or electrodermal systems, with only the cardiac response curve revealing a significant reduction in activity as a function of predictability. However, further examination of the data reveals that this effect was probably not due to differences in response to the aversive stimuli itself - both predictable and unpredictable groups showed similar levels of activity to the slide - but was a result of differences in levels of responding during the pre-stimulus period. Indeed, during the inter-stimulus interval subjects in the unpredictable groups produced lower levels of cardiovascular activity compared to the predictable groups. Although these differences were not statistically significant, they did influence the pattern of responding when employed in calculating the cardiac response curve. That is, the predictable groups showed a decrease from pre-stimulus HR levels to impact of the aversive stimuli whereas, subjects in the unpredictable condition produced similar levels of activity in both periods. These results appear to suggest that predictability augments cardiac activity during anticipatory periods compared to unpredictable conditions, but has little or no effect during impact of the aversive event.

Indeed, this interpretation would be supported by the only other significant findings. That is, higher cardiac activity during the warning stimulus and inter-stimulus intervals of Trials 5, 8 and 13 in predictable compared to unpredictable groups, particularly in relation to maximum HR. In addition, higher minimum HR in the predictable-long group during the inter-trial intervals of Trials 5 and 8 compared to the predictable-short and unpredictable groups. There were no significant differences between the predictable-short and unpredictable groups during any of the trials. However, these results and particularly those associated with maximum HR, have to be interpreted in the light of the pre-task baseline data, in which the predictable groups showed higher maximum HR levels compared to the unpredictable groups. The significant differences between the conditions in the experimental trials appears to be largely due to these pre-manipulation differences. Indeed, this interpretation would be supported by firstly, corrected HR analyses which accounted for individual differences in end-organ activity and found no significant effects of predictability during the anticipatory periods. Secondly, by the Trial 1 data which

showed higher levels of maximum HR activity during the warning signal and inter-stimulus intervals in the predictable compared with unpredictable groups, even though all subjects had received identical experimental stimuli up to that point.

The mainly insignificant findings in both electrodermal and cardiovascular systems during the present experiment, although in line with a number of past studies (see Chapter 2, Section 3.3.2), are probably a result of several methodological problems. The most important being the employment of stimulus intensities which were too 'mild'. This was not intentional. On the contrary, pilot work revealed that subjects found the slides both 'distressing' and 'aversive'. Moreover, previous work employing mutilation slides found significantly large increases in cardiac activity at event impact (e.g. Klorman & Ryan, 1980). However, the cardiovascular data in the present experiment revealed a decrease in HR from pre-task baseline levels to slide onset during Trial 1, in both average (1.2 bpm) and maximum (8.8 bpm). Minimum HR produced an increase in responding to the slide, although this was relatively small (4.9 bpm). Thus, it may not have been the manipulation of predictability *per se* that was unimportant in this study, but rather the use of uniform, mild stimuli that were not sufficiently aversive for all subjects. This, in turn, may have meant that subjects did not care whether or not they received information concerning that event. Indeed, it has been suggested in the preference behaviour literature involving both human and nonhuman subjects (see Chapter 2, Section 3.2), that the beneficial effects of predictability only emerge in high intensity conditions and that at medium or low intensities there is little difference between predictable and unpredictable groups. Furthermore, it has been suggested that individual differences in stimulus threshold levels are so great, that stimulus intensity should be set at a level determined as aversive by each individual subject. A procedure not employed in this experiment.

There were other experimental parameters employed in the current study which may have been less than optimal for the production of changes as a function of predictability. Numerous studies, particularly in the nonhuman preference literature (see Chapter 2, Section 3.2.1), have indicated that parameters such as, duration of the aversive event and warning signal and the length of inter-trial and inter-stimulus intervals significantly change preference behaviour. For example, it has been found that preference behaviour changes as a function of the length of time between the warning signal and aversive event, with a

significant preference only emerging at short inter-stimulus intervals (i.e. 0.5 to 1.0 sec). No significant differences were found with longer intervals (i.e. 3.5 to 5.0 sec) (e.g. Furedy, 1975). The length of inter-stimulus interval employed in the present study was relatively long (5 sec), in order to allow response interference effects to be controlled for, and it may be that this, in combination with other factors some of which were mentioned above, precluded any significant effects of predictability in this particular experiment.

In terms of the effects of warning signal duration on autonomic activity, the results were largely insignificant. Indeed, there were no significant differences between long and short warning conditions in electrodermal or cardiovascular systems during anticipatory or impact periods, with the exception of minimum HR during the inter-trial interval. This was due to subjects in the predictable-long group producing greater cardiac activity than any of the remaining groups during Trials 5 and 8. There were no significant differences between any of the groups in Trial 13. There was also a difference in the cardiac response profile between long and short warning conditions across trials and time (seconds). However, further inspection of the data revealed no meaningful pattern of results.

Overall, these findings fail to confirm the results of past preference studies, in which preference behaviour increased with increasing duration of the warning signal (e.g. Perkins, Seymann, Levis & Spencer, 1966). Furthermore, they cannot help differentiate between the theoretical explanations put forward to account for the supposedly beneficial effects of predictability; namely, the Safety Signal (Seligman, 1975), Preparatory Response (Perkins, 1968) or Preception (Lykken, 1962) Theories, all of which have a precondition that temporal predictability of an aversive stimulus leads to lower physiological activity. Nevertheless, the one significant finding - that predictable/long conditions lead to an increase in levels of minimum HR compared to both predictable/short and unpredictable situations - corresponds with the findings of Weiss (1971a). He found that those animals in a progressive-signal (long warning) condition developed greater amounts of gastric ulceration than those in a signal (short warning) condition when control was absent. However, in conditions where animals could control the aversive event there was relatively little difference between the two warning conditions. Weiss concluded that the most critical variable in the development of pathology was the ability to perform a coping response rather than the presence or absence of a warning signal. In the present



study, although subjects in the predictable groups may have had a weak sense of perceived control in that predictability provides the individual with information about when an event is going to happen, none of the experimental groups had objective or physical control. Thus, according to Weiss, the beneficial effects of predictability could not be realised as subjects were unable to perform an actual coping response. Indeed, this type of interpretation would be supported by past studies involving human volunteers, which suggest that control must be present before predictability has any effect (e.g. Davis and Levine, 1982). However, it is not certain from the experimental data available, whether control is the important factor in this situation, or, whether control and predictability interact to produce an effect. This is because the majority of predictability studies have confounded predictability of an aversive event with control over that event. That is, those subjects in the predictable condition have more predictability *and* more perceived control than the unpredictable group. Unfortunately, data from the present study cannot help clarify these issues because availability of control was not manipulated. However, these and related questions will be dealt with in subsequent experimental chapters.

To summarise: the predictions concerning the beneficial effects of predictability and any changes in these effects as a function of warning signal duration have remained unconfirmed. As a result of these insignificant findings no conclusions could be made concerning possible theoretical interpretations of the data. It has been suggested that a number of stimulus and design factors may account for the discrepancies between the current findings and those reported previously in both the human and nonhuman literature.

## CHAPTER 8

### EXPERIMENT 3

#### 1. INTRODUCTION

The aim of this experiment was to examine the effects of temporal predictability using a similar paradigm to that employed in Chapter 7, whilst avoiding the problems produced by what turned out to be a mild aversive stimuli. This was achieved by employing stimuli (white noise) and levels of intensity (100 dB) which have been found in past studies, to elicit autonomic changes as a function of predictability (see Chapter 2, Section 3.3.2).

The aims of this study were somewhat more limited than the previous study in order to maximise the power of the experiment in examining the effects of temporal predictability. Nevertheless, it was hoped that any differential responding during anticipatory and impact periods would help elucidate on the different theories of predictability (see Chapter 7, Section 1). According to Seligman's (1968) safety signal analysis, the predictable condition should elicit lower levels of psychophysiological activity during the safe periods (i.e. the inter-stimulus and inter-trial intervals), compared to unpredictable situations with similar reactions to the danger periods (i.e. the warning signal and aversive event). On the other hand, the Preparatory Response (Perkins, 1968) and Preception (Lykken, 1962) Theories predict lower psychophysiological activity during predictable aversive events compared to unpredictable ones but make no specific regarding activity during either the warning signal, or, anticipatory periods. However, as mentioned in Chapter 7 Section 1, it could be suggested that greater autonomic activity should occur in unpredictable situations because in these conditions the subject is anticipating a more painful event since they cannot prepare for it.

### 2.1. Subjects

The subjects were 40 undergraduate volunteers (32 females and 8 males), with an age range of 18 - 40 years. All subjects were randomly allocated to one of two experimental conditions; predictable (P) or unpredictable (UP). The only restriction to the random allocation was that there were equal numbers of the smaller, male population in each of the two groups.

### 2.2. Apparatus

An IBM compatible computer was used to run the measurement software supplied by RC Electronics. The software controlled recording of all physiological responses via a Grass Model 7 polygraph. EKG was recorded from electrodes placed on the inner side of each wrist, with a ground on the left forearm. EKG was then recorded using a 7P1 F preamplifier. The output from the driver amplifier was used to trigger a 7P4 F tachograph which provided a measure of HR in beats per minute (bpm). Tachograph output was recorded by the IBM compatible computer. Recording of SC was made using Ag-AgCl electrodes filled with 0.05M NaCl electrolyte, and attached to masked areas of the distal phalanx of the index and second fingers of the left hand. A constant voltage of 0.5V was applied across the electrodes, and conductance was recorded with a 7P1 F preamplifier sensitivity equal to 0.02  $\mu$ S. The total area from which conductance was recorded was 0.35cm<sup>2</sup>. The output was then recorded by the IBM compatible computer.

Presentation of the warning stimuli and white noise were controlled by a BBC, model B microcomputer, situated in the experimenter's room. The warning stimuli and presentation of the warning stimuli was identical to that employed in Experiment 2, with the exception that each number was displayed on the monitor for a duration of 1 sec for all groups. The white noise was of 1 sec duration, and 100 dB intensity. It was produced by the BBC microcomputer, and presented via Eagle International, SE 5 stereophonic

headphones. Noise intensity was calibrated using a audiometer (Amplaid SLM 13 model) placed between the headphones.

A two way intercom system was in operation throughout. It was situated 8 inches from the subject and directly in front of them.

### 2.3. Design

The experimental design was identical to that employed in Experiment 2, with the exception that the aversive stimulus was 1 sec of white noise. In addition, the duration of the warning stimulus was 5 sec in both predictable and unpredictable conditions.

### 2.4. Procedure

The seating and accommodation of subjects was the same as in Experiment 1. After electrode attachment, subjects were informed of the procedure to follow (see appendix). Subjects in the predictable condition were told that they would receive a warning consisting of the numbers 5, 4, 3, 2, 1 on the monitor shortly before noise onset. Those subjects in the unpredictable condition were told that the 5, 4, 3, 2, 1 and the noise would occur randomly to one another. All subjects received a 1 min rest period followed by, the range correction stimulus consisting of 1 sec, 100 dB white noise. After a further 5 min rest period, subjects were presented with 13 trials with an inter-trial interval (ITI) of 25, 30 or 35 sec. The mean ITI was 30 sec. After the subject had completed 13 trials, the experimenter unwired and debriefed the subject.

### 2.5. Scoring

2.5.1. Cardiovascular activity. The measurement of HR was the same as in Experiment 1. All three cardiovascular measures (avHR, mnHR and mxHR) were assessed during the 5 min pre-task baseline in order to examine any pre-task between-group differences.

Between-group differences were assessed across Trials 1, 5, 8 and 13 as these were the only trials in which predictable and unpredictable subjects received identical

experimental stimuli. Trial 1 was analysed separately from Trials 5, 8 and 13. Each trial was divided into 5 separate time periods: Period 1, consisted of the 0-5 sec after warning onset: Period 2, was the 0-5 sec before onset of the noise (ISI): Period 3 was the 0-6 sec after noise onset: Period 4, was the 25 sec ITI divided into five, 5 sec blocks: Period 5, involved a measure of the cardiac response profile. Measurement of the cardiac response profile was the same as in Experiment 2. Uncorrected and corrected HR are reported for all three cardiovascular measures across Time Periods 1 to 4. Measurement of corrected HR was the same as in Experiment 1. Period 5 was examined for change in avHR across Trials 5, 8 and 13 only.

2.5.2. Electrodermal activity. Tonic SCL and frequency of NSF's were assessed during the 5 min pre-task baseline. They were measured following the procedure of Experiment 1.

Between-group differences in electrodermal activity were assessed across Trials 1, 5, 8 and 13. Trial 1 was analysed separately from Trials 5, 8 and 13. For the analysis carried out on tonic SCL, each trial was divided into three periods: Period 1, was the 0-5 sec after warning onset: Period 2, was the 0-5 sec before onset of the noise (ISI) and Period 3 consisted of the 5-25 sec of the ITI divided into four, 5 sec blocks. The frequency of NSF's was assessed during the 5-25 sec of the ITI only.

The criteria and measurement of evoked responses were the same as in Experiment 2. All evoked responses were range-corrected (Lykken & Venables, 1971) in terms of subject's response to the range-correction stimulus which was presented before the pre-task baseline. All range-corrected responses were then subjected to a square-root transformation. Between-group comparisons of range-corrected SCR amplitude were assessed during two time periods: Period 1, included the 1-5 sec after warning onset and Period 2 the 1-5 sec after noise onset.

### 3.1. Baseline Activity

With all measures, differences between groups were assessed across the 5 min baseline. These 5 min were divided into five, 60 sec periods and a two-way MANOVA was employed with predictability (predictable/unpredictable) as a between subject factor and time (5 levels) as a repeated measure. Group means and standard deviations for cardiovascular and electrodermal baseline activity are shown in Tables 15 and 16 respectively.

3.1.1. Cardiovascular Activity. There were no significant effects of predictability across any of the measures (all  $F_s < 1$ ). There was a significant difference across time for avHR ( $F(2, 80) = 5.8, p < .01$ ), mnHR ( $F(3, 123) = 3, p < .05$ ) and mxHR ( $F(4, 135) = 10.4, p < .001$ ). Multiple comparison of means, firstly for avHR, revealed significantly higher HR during the 0-60 and 180-240 sec periods compared with the 60-120 sec period. There were no significant differences between the remaining periods (81.4, 78.6, 79.5, 80.6 and 80.5 bpm for the 0-60, 60-120, 120-180, 180-240 and 240-300 sec periods respectively). MnHR revealed significantly higher HR during the 240-300 sec period compared with the 60-120 sec period. None of the other comparisons were significant (69.4, 68.3, 69.5, 69.5 and 70.8 bpm for the 0-60, 60-120, 120-180, 180-240 and 240-300 sec periods respectively). MxHR produced significantly higher HR during the 0-60 sec period compared with the remaining four, 60 sec periods. The remaining comparisons did not reach significance (97.0, 90.5, 90.9, 92.7 and 91.0 bpm for the 0-60, 60-120, 120-180, 180-240 and 240-300 sec periods respectively). There were no significant interactions involving time across any of the three measures (all  $F_s < 1$ ).

3.1.2. Electrodermal Activity. There were no significant main effects of predictability involving tonic SCL ( $F(1, 38) = 3.2$ ) or frequency of NSF's ( $F(1, 38) = 1.5$ ). There was a highly significant main effect of time for SCL ( $F(2, 72) = 19, p < .001$ ), due to significantly higher levels of responding during the 0-60 sec period compared with the

remaining four, 60 sec periods (3.09, 2.94, 2.94, 2.95 and 2.92  $\mu$ S for the 0-60, 60-120, 120-180, 180-240 and 240-300 sec periods respectively). Analysis of NSFs also produced a difference across time ( $F(3, 95) = 5.3, p < .01$ ), with a significantly greater frequency of NSFs during the 0-60 sec period compared with the 180-240 and 240-300 sec periods. The 60-120 sec period also revealed a significantly higher frequency of NSFs than the 240-300 sec period (4.58, 4.30, 3.90, 3.50 and 3.23 for the 0-60, 60-120, 120-180, 180-240 and 240-300 sec periods respectively). There were no significant interactions involving time for either of the two electrodermal measures (all  $F_s < 1$ ).

### 3.2. Between-Groups Analysis: Trial 1

#### 3.2.1. Cardiovascular Activity

For all measures, Time periods 1 to 3 were each assessed in a one-way ANOVA with predictability as a between subject factor. Time Period 4 was analysed in a two-way MANOVA with the above between-subject factor and time (5 levels) as a repeated measure. Group means and standard deviations for uncorrected HR during Trial 1 are shown in Table 15.

3.2.1.1. Uncorrected HR. There were no significant differences between groups involving any of the time periods (all  $F_s < 1$ ). During Time Period 4 there were no significant differences across time involving avHR ( $F(3, 98) = 2.5$ ) or mnHR ( $F(3, 95) = 1.3$ ). However, mxHR did produce a highly significant difference across time ( $F(3, 122) = 9.2, p < .001$ ). Comparison of means revealed that the 0-5 sec period had significantly higher HR levels compared with the remaining four, 5 sec periods (94.8, 90.0, 89.8, 89.4 and 88.7 bpm for the 0-5, 5-10, 10-15, 15-20 and 20-25 sec periods respectively). There were no interactions involving time for any of the three measures (all  $F_s < 1$ ).

Further examination of the pattern of responding across Time Periods 1 to 4 revealed significant differences between the three cardiovascular measures (see Table 15). AvHR showed similar levels of activity during the pre-task baseline and the warning stimulus ( $T < 1$ ). In contrast, mxHR produced a significant decrease in activity to the

Table 15. Mean cardiovascular activity during the pre-task baseline and time periods of Trial 1. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Groups	
	Predictable	Unpredictable
Pre-Task Baseline		
AvHR	80.3 (14.9)	79.9 (11.4)
MnHR	69.9 (14.3)	69.7 (11.0)
MxHR	93.0 (15.4)	91.8 (12.9)
Time Period 1		
AvHR	80.4 (16.1)	79.6 (12.2)
MnHR	74.8 (15.9)	72.1 (9.89)
MxHR	86.3 (16.7)	86.0 (12.9)
Time Period 2		
AvHR	79.7 (16.2)	77.2 (11.6)
MnHR	73.9 (15.5)	71.9 (10.6)
MxHR	85.7 (16.5)	83.9 (14.5)
Time Period 3		
AvHR	85.3 (16.6)	85.2 (12.6)
MnHR	75.7 (17.3)	77.2 (11.8)
MxHR	95.0 (16.3)	93.2 (14.9)
Time Period 4		
AvHR	83.4 (16.7)	85.4 (13.9)
MnHR	76.6 (18.2)	79.5 (12.7)
MxHR	89.7 (16.4)	91.3 (14.4)



warning stimulus ( $T(1, 39) = 5.1, p < .001$ ), and mnHR a significant increase ( $T(1, 39) = 3.7, p < .01$ ). All three measures showed a significant increase in activity from the baseline to noise onset; avHR ( $T(1, 39) = 4.1, p < .001$ ), mnHR ( $T(1, 39) = 4.7, p < .001$ ) and mxHR ( $T(1, 39) = 2.4, p < .05$ ). In addition, further comparisons revealed a significant increase in HR levels from the warning stimulus to noise onset; avHR ( $T(1, 39) = 6.0, p < .001$ ), mnHR ( $T(1, 39) = 2.8, p < .05$ ) and mxHR ( $T(1, 39) = 6.4, p < .001$ ).

3.2.1.2. Corrected HR. The results of corrected HR were similar to those of uncorrected HR, unless stated otherwise. During Time Period 1, mnHR produced a significant effect of predictability ( $F(1, 38) = 4.9, p < .05$ ). The P group had greater increases in HR from the pre-task baseline compared with the UP group (4.3 and 1.2 change in bpm respectively).

### 3.2.2. Electrodermal Activity

Tonic SCL was assessed during Periods 1 and 2 in a one-way ANOVA with predictability as a between subject factor. SCL during Period 3 was analysed in a two factor MANOVA with the above between subject factor and time as a repeated measure. Frequency of NSFs and transformed scores for SCR amplitude were assessed in a one-way ANOVA as above. Group means and standard deviations for electrodermal activity during Trial 1 are shown in Table 16.

3.2.2.1. Skin Conductance Level. There were no significant effects of predictability involving Time Period 1 ( $F(1, 38) = 3.4$ ), Time Period 2 ( $F(1, 38) = 3.6$ ) or Time Period 3 ( $F < 1$ ). During Time period 3, there was a highly significant difference across time ( $F(1, 48) = 55.95, p < .001$ ). Multiple comparison of means revealed that the 5-10 sec period had significantly higher levels of activity than the 10-15, 15-20 and 20-25 sec periods. The 10-15 sec period produced significantly higher levels of responding compared with the 15-20 and 20-25 sec periods. There were no significant differences between the remaining periods (3.35, 3.21, 3.14 and 3.11  $\mu$ S for the 5-10, 10-15, 15-20 and 20-25 sec periods respectively). There was no significant interaction involving time during Time Period 3 ( $F(1, 48) = 1.5$ ).

Table 16. Mean electrodermal activity during the pre-task baseline and time periods of Trial 1. Standard deviations are shown in parentheses.

Variables	Experimental Groups	
	Predictable	Unpredictable
Pre-Task Baseline		
Tonic SCL ( $\mu\text{S}$ )	2.59 (0.92)	3.35 (1.64)
Frequency of NSF's	3.33 (3.02)	4.47 (3.42)
Time Period 1		
Tonic SCL ( $\mu\text{S}$ )	2.67 (0.99)	3.51 (1.76)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu\text{S}$ )	0.104 (0.10)	0.103 (0.12)
Time Period 2		
Tonic SCL ( $\mu\text{S}$ )	2.79 (1.01)	3.68 (1.83)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu\text{S}$ )	0.052 (0.78)	0.074 (0.10)
Time Period 3		
Tonic SCL ( $\mu\text{S}$ )	2.80 (0.93)	3.60 (1.77)
Frequency of NSF's	0.95 (1.09)	1.55 (1.40)

3.2.2.2. Non-Specific Fluctuations. There was no significant effect of predictability ( $F(1, 38) = 2.3$ ).

3.2.2.3. SCR Amplitude. There were no significant effects of predictability during Time Periods 1 or 2 (both  $F_s < 1$ ).

### 3.3. Between-Groups Analysis: Trials 5, 8 and 13

#### 3.3.1. Cardiovascular Activity

For all measures, Time Periods 1 to 3 were each assessed in a two-way MANOVA with predictability as a between subject factor and trials (Trials 5, 8 and 13) as a repeated measure. Time Periods 4 and 5 were analysed in a three factor MANOVA with the same between subject factor as above and trials and time (5 and 10 levels respectively) as repeated measures. Group means and standard deviations for uncorrected HR during Time Periods 1 to 4 are shown in Table 17.

##### 3.3.1.1. Uncorrected HR

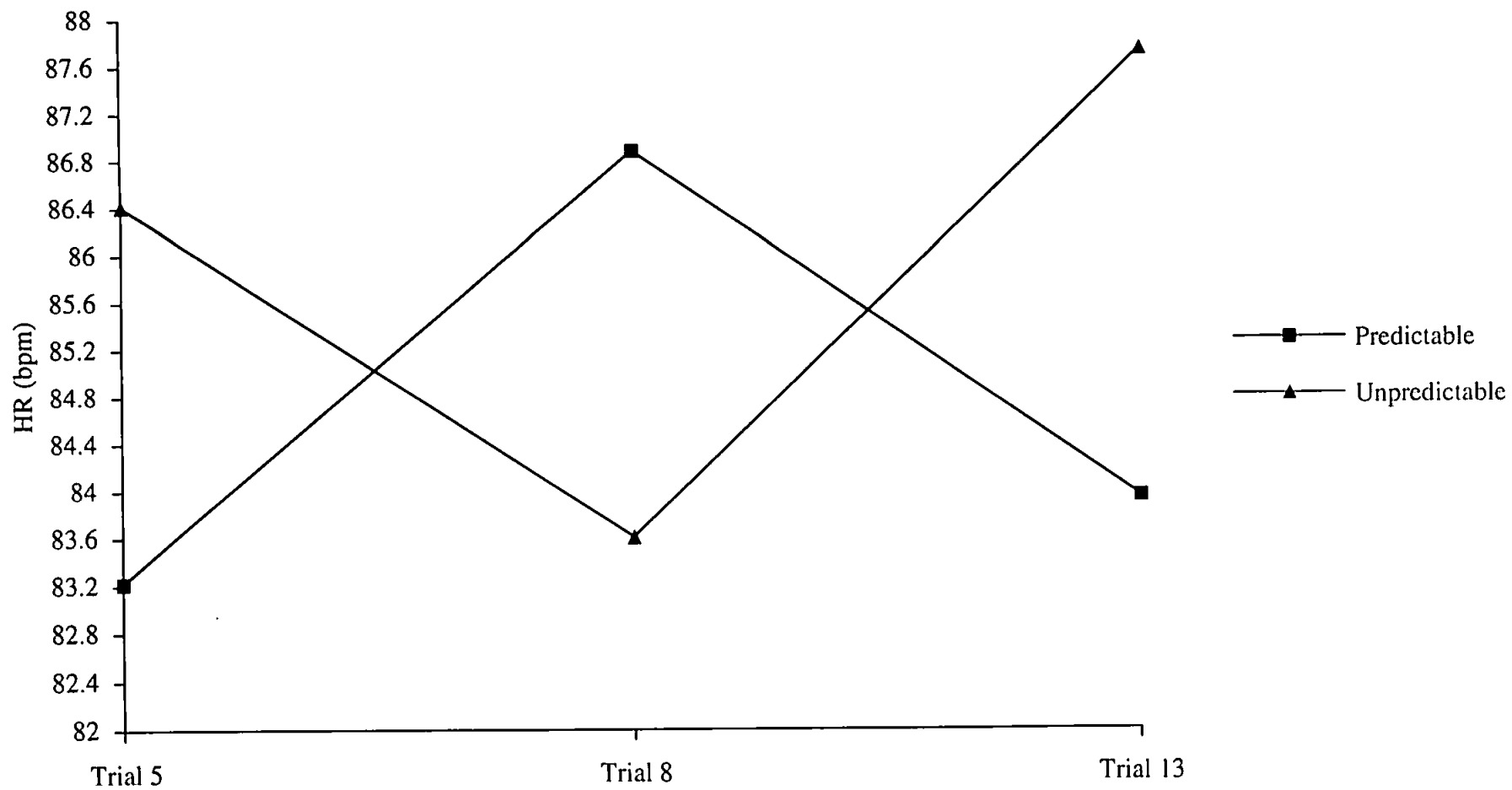
3.3.1.1.1. Time Period 1. There were no significant main effects of predictability involving any of the three measures (all  $F_s < 1$ ). There was no difference across trials or any interaction involving trials for any of the measures (all  $F_s < 1$ ).

3.3.1.1.2. Time Period 2. There were no significant effects of predictability involving any of the three measures (all  $F_s < 1$ ). There was no significant difference across trials involving any of the three measures (all  $F_s < 1$ ), nor were there any interactions involving trials for avHR ( $F(2, 61) = 1.8$ ) or mnHR ( $F < 1$ ). MxHR produced a predictability by trial interaction ( $F(2, 66) = 3.9, p < .05$ ). Simple main effects revealed that the UP group had significantly higher HR levels than the P group during Trial 13, compared with no significant difference between the two groups in earlier trials (Figure 23).

Table 17. Mean cardiovascular activity during time periods averaged across Trials 5, 8 and 13. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Groups	
	Predictable	Unpredictable
Time Period 1		
AvHR	79.4 (13.4)	81.6 (14.2)
MnHR	72.3 (12.8)	76.3 (13.2)
MxHR	85.7 (13.9)	86.9 (14.9)
Time Period 2		
AvHR	78.5 (13.4)	80.7 (13.5)
MnHR	73.2 (13.5)	75.7 (13.9)
MxHR	84.7 (13.9)	85.9 (13.4)
Time Period 3		
AvHR	77.6 (12.2)	82.3 (14.5)
MnHR	71.5 (11.8)	76.2 (14.6)
MxHR	84.4 (13.9)	89.4 (14.8)
Time Period 4		
AvHR	77.9 (13.0)	80.3 (13.3)
MnHR	72.6 (12.5)	74.6 (12.5)
MxHR	83.4 (14.1)	84.9 (14.8)

Figure 23. Mean maximum HR as a function of groups and trials during Time Period 2.



3.3.1.1.3. Time Period 3. There were no main effects of predictability involving avHR ( $F(1, 38) = 1.4$ ), mnHR ( $F < 1$ ) or mxHR ( $F(1, 38) = 1.4$ ). There were no significant differences across trials for avHR, mnHR (both  $F_s < 1$ ) or mxHR ( $F(2, 56) = 1.7$ ), nor was there any interactions involving trials for avHR ( $F(1, 48) = 1.5$ ), mnHR ( $F < 1$ ) or mxHR ( $F(2, 56) = 1.7$ ).

3.3.1.1.4. Time Period 4. There were no significant effects of predictability involving any of the measures (all  $F_s < 1$ ), nor any significant difference across trials involving avHR ( $F(1, 53) = 1.4$ ), mnHR ( $F(2, 65) = 1.8$ ) or mxHR ( $F(1, 53) = 1.7$ ). There was a significant difference across time involving avHR ( $F(3, 121) = 5, p < .01$ ) and mxHR ( $F(3, 122) = 7.9, p < .001$ ), and a predictability by time interaction for avHR ( $F(3, 121) = 4.7, p < .01$ ) and mxHR ( $F(3, 122) = 5.3, p < .01$ ). Analysis of the simple main effects revealed, firstly for avHR, that the UP group had significantly higher HR than the P group during the 0-5 and 20-25 sec periods of the ITI. There were no significant differences between groups during any of the remaining 5 sec periods (Figure 24). MxHR revealed that the UP group had significantly higher HR than the P group during the 0-5 sec period of the ITI. There were no significant differences between the two groups during the remaining periods of the ITI (Figure 25). There were no other interactions involving time for avHR or mxHR (all  $F < 1$ ). There was no significant difference across time involving mnHR ( $F(3, 113) = 1.3$ ), nor a predictability by time ( $F(3, 113) = 2$ ) or any trial by time interactions (all  $F_s(6, 207) = 1.4$ ).

3.3.1.1.5. Time Period 5. There was no significant main effect of predictability or any difference across time involving mean change in avHR (all  $F_s < 1$ ). However, there was a predictability by time interaction ( $F(4, 153) = 3.6, p < .01$ ). The UP group had a significantly greater increase in HR than the P group during Seconds 1, 2 and 3. During Seconds 4 to 10 there were no significant differences between the two groups (Figure 26). There was a significant difference across trials ( $F(2, 71) = 3.7, p < .05$ ). Further comparison of means revealed a significant decrement in levels of activity over trials (1.34, -0.32 and -0.81 change in bpm for Trials 5, 8 and 13 respectively). However, there were no significant interactions involving trials (all  $F_s < 1$ ).

3.3.1.1.6. Summary. During the warning stimulus (Time Period 1) and the white noise (Time Period 3) there were no significant differences between the groups involving

Figure 24. Average HR as a function of groups and time during Time Period 4.

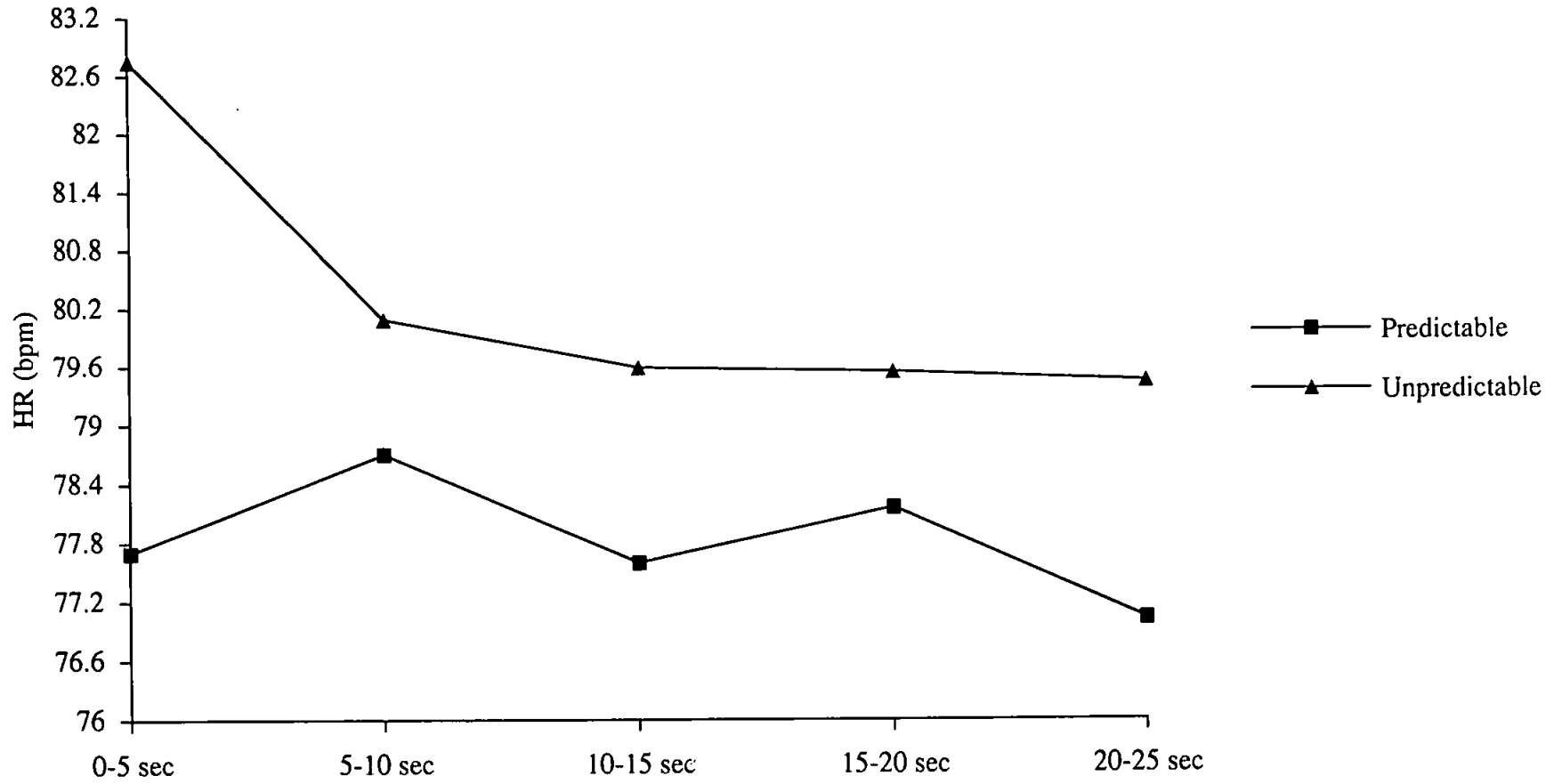


Figure 25. Mean maximum HR as a function of groups and time during Time Period 4.

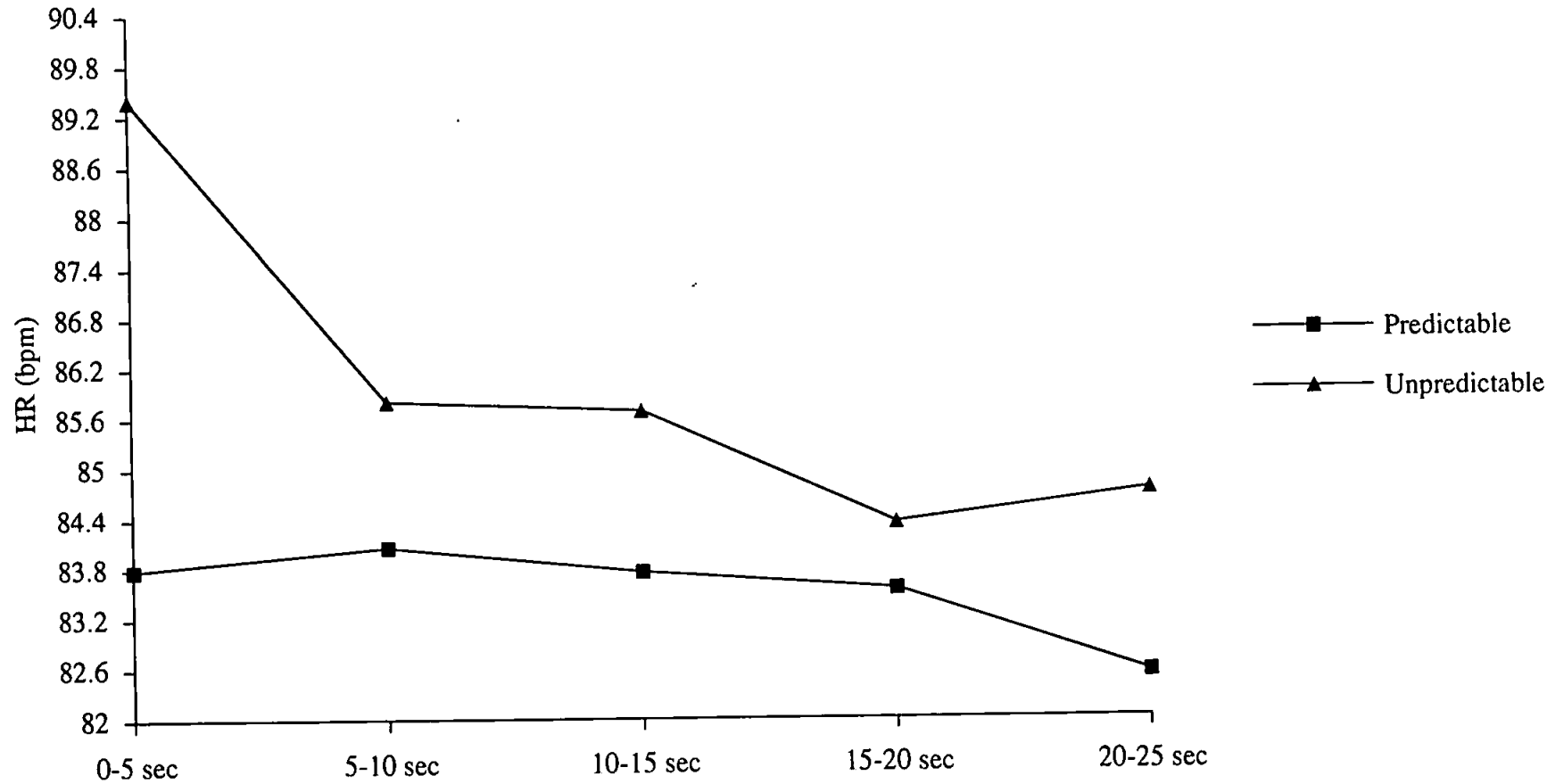
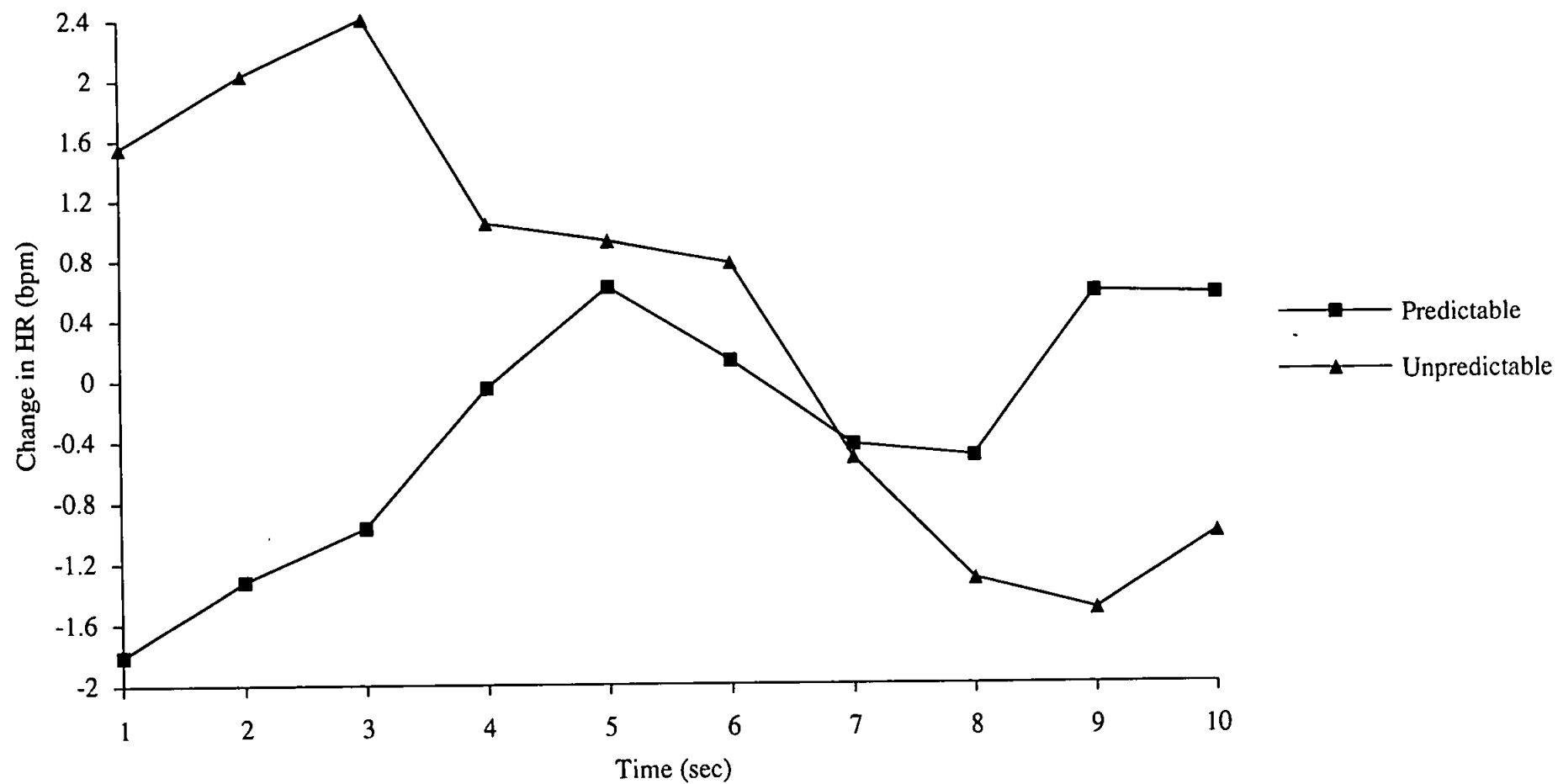




Figure 26. Mean change in average HR as a function of groups and time during Time Period 5.



any of the cardiovascular measures. However, visual inspection of the means revealed that the UP group produced higher HR compared with the P group across all three measures.

During the ISI (Time Period 2), mxHR produced a predictability by trial interaction which was due to the UP group producing higher HR than the P group during Trial 13. There were no significant differences between the two groups in the early trials. There were no other significant effects or interactions during this period involving any of the three measures. However, as seen previously an inspection of the means reveals that the UP group produces higher HR than the P group across all measures.

During the ITI (Time Period 4), both avHR and mxHR revealed a predictability by time interaction. The interaction was due to the UP group producing significantly higher HR than the P group during the 0-5 sec and 20-25 sec (avHR only) periods. However, the UP group also had non-significantly higher HR during the remaining 5 sec periods. There were no other main effects or interactions during this period involving any of the cardiovascular measures.

The cardiac response profile (Time Period 5) revealed a predictability by time interaction. The UP group had significantly greater increases in HR compared with the P group during Seconds 1, 2 and 3 after noise onset.

3.3.1.2. Corrected HR. The results involving corrected HR were similar to those of uncorrected HR, unless stated otherwise. During Time Period 3, there were significant effects of predictability involving avHR ( $F(1, 38) = 6.2, p < .05$ ) and mxHR ( $F(1, 38) = 7.8, p < .01$ ). The UP group had significantly greater increases in avHR and smaller decreases in mxHR compared with the P group (avHR = 2 and -3.21 change in bpm respectively; mxHR = -0.514 and -7.58 change in bpm).

### 3.3.2. Electrodermal Activity

Tonic SCL was assessed during Time Periods 1 and 2 using a two-way MANOVA with predictability as a between subject factor and trials as a repeated measure. SCL during Time Period 3 was analysed using a three-way MANOVA with the above between subject factor and trials and time as repeated measures. Frequency of NSFs and transformed

Table 18. Mean electrodermal activity during time periods averaged across Trials 5, 8 and 13. Standard deviations are shown in parentheses.

Variables	Experimental Groups	
	Predictable	Unpredictable
Time Period 1		
Tonic SCL ( $\mu$ S)	2.63 (0.91)	3.33 (1.61)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.196 (0.11)	0.278 (0.15)
Time Period 2		
Tonic SCL ( $\mu$ S)	2.66 (0.93)	3.34 (1.59)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.139 (0.95)	0.137 (0.12)
Time Period 3		
Tonic SCL ( $\mu$ S)	2.62 (0.90)	3.34 (1.56)
Frequency of NSF's	0.48 (0.79)	0.72 (1.01)

scores for SCR amplitude were assessed in a two-way MANOVA as above. Group means and standard deviations for electrodermal activity during Time Periods 1 to 3 are shown in Table 18.

3.3.2.1. Skin Conductance Level. There were no significant effects of predictability involving Time Period 1 ( $F(1, 38) = 3$ ), Time Period 2 ( $F(1, 38) = 2.8$ ) or Time Period 3 ( $F(1, 38) = 3$ ). There was no significant difference across trials or any interaction involving trials during Time Periods 1 or 2 (all  $F_s < 1$ ). During Time Period 3 there were no significant differences across trials or time (all  $F_s < 1$ ). However, there was a trials by time interaction ( $F(4, 139) = 5.5, p < .01$ ). Multiple comparison of means revealed that the 5-10 sec period during Trial 5 had significantly higher HR compared with the 10-15, 15-20 and 20-25 sec periods during Trial 5, and all periods of Trials 8 and 13. The 10-15, 15-20 and 20-25 sec periods during Trial 5 and the 5-10 sec periods of Trials 8 and 13 produced significantly higher HR than the 15-20 and 20-25 sec periods during Trials 8 and 13 (Figure 27). There were no significant interactions between predictability and trials ( $F(1, 50) < 1$ ), predictability and time ( $F(1, 51) = 2.9$ ) or predictability by trials by time ( $F(4, 139) = 1.7$ ).

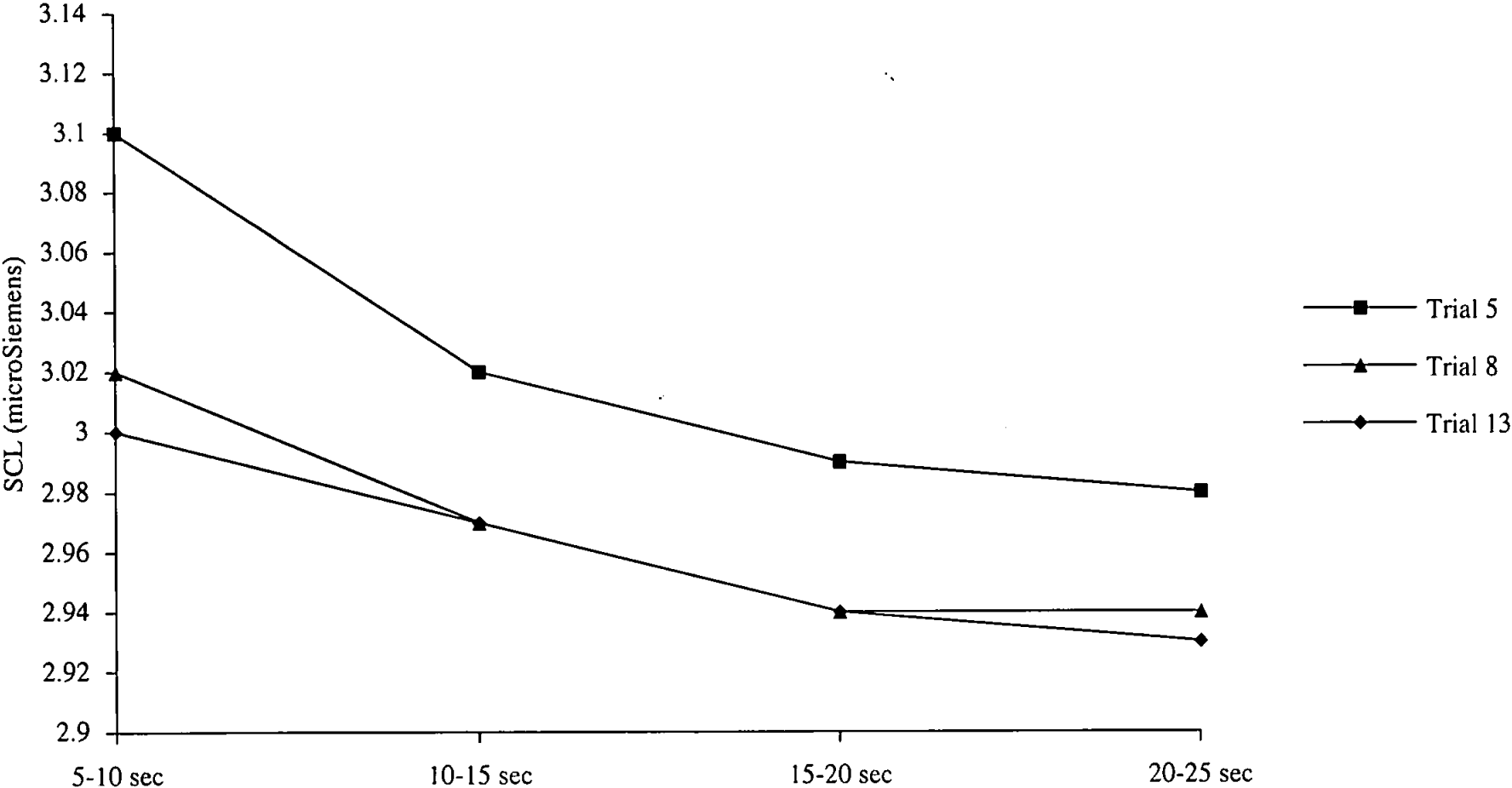
3.3.2.2. Non-Specific Fluctuations. There were no significant main effects of predictability ( $F < 1$ ) or trials ( $F(2, 75) = 1.9$ ), nor was there a predictability by trials interaction ( $F(2, 75) = 1.6$ ).

### 3.3.2.3. SCR Amplitude

3.3.2.3.1. Time Period 1. There was a main effect of predictability ( $F(1, 38) = 4.2, p < .05$ ), due to the UP group producing greater levels of responding than the P group (0.278 and 0.196  $\sqrt{\text{range-corrected } \mu\text{S}}$  respectively). There was no significant difference across trials ( $F < 1$ ), nor any predictability by trials interaction ( $F(2, 71) = 2.7$ ).

3.3.2.3.2. Time Period 2. There was no significant effect of predictability ( $F < 1$ ). There was a highly significant difference across trials ( $F(2, 70) = 23.4, p < .001$ ), with Trial 13 producing significantly greater levels of responding compared with Trial 5. There were no significant differences between the other trials (0.098, 0.144 and 0.173  $\sqrt{\text{range-}}$

Figure 27. Mean skin conductance level as a function of time and trials during Time Period 3.



corrected  $\mu S$  for Trials 5, 8 and 13 respectively). There was no significant interaction involving trials ( $F(2, 70) < 1$ ).

3.3.2.4. Electrodermal Summary. There were no significant differences between groups in terms of tonic SCL or frequency of NSFs. However, an inspection of the SCL means across all time periods revealed that the UP group had higher levels of responding compared with the P group. Similarly, NSFs means reveal that the UP group has higher levels of responding than the P group during Trials 5 and 8. Range-corrected SCR Amplitude revealed a significant difference between groups during the warning stimulus (Time Period 1), with the UP group producing greater levels of responding than the P group.

#### 4. DISCUSSION

In this experiment, receipt of information warning of the occurrence of an aversive event led to a significant reduction in cardiovascular activity. Temporal predictability had little effect upon the electrodermal system, with the exception of response amplitudes during the warning stimulus in which the unpredictable group showed higher levels of responding compared to the predictable group. In general, the significant effects of this experiment are in line with findings from the human and nonhuman literature reviewed in Chapter 2, Sections 3.3.1 and 3.3.2 respectively. They are also in accordance with much of the preference behaviour literature (see Chapter 2, Section 3.2).

Further examination of the cardiovascular data indicates that there were significant effects of predictability during both the anticipatory (i.e. the inter-stimulus and inter-trial intervals) and impact (i.e. during the event and the cardiac response curve) periods of Trials 5, 8 and 13, particularly in relation to average and maximum HR. For example, during the first anticipatory period - the inter-stimulus interval - maximum HR produced changes across trials as a function of predictability, with subjects in the unpredictable condition showing higher levels of responding compared to the predictable group during Trial 13. A similar pattern emerged in Trial 5, although this failed to reach significance. During the second anticipatory period - the inter-trial interval - the unpredictable group

produced higher average and maximum HR levels across the 25 sec interval, compared to subjects in the predictable condition. However, the difference between the two groups only reached significance in the 0-5 and 20-25 sec (average HR only) periods. During the impact period, both average and maximum corrected HR produced significant effects of predictability, due to subjects in the unpredictable group having higher levels of activity than those in the predictable condition. A similar pattern of responding was observed in the cardiac response curve, where the unpredictable group produced a greater increase in HR during the 0-6 sec after stimulus onset, compared to the predictable condition. Further comparisons revealed that this difference between predictable and unpredictable groups only reached significance during the 0-3 sec period. Moreover, the pattern of responding was reversed during the 8-10 sec period, with subjects in the predictable condition producing greater HR increases compared to the unpredictable group.

Although the results mentioned above were the only findings to reach statistical significance in terms of cardiac activity, further inspection of the data reveals that the unpredictable condition had greater HR levels compared to the predictable group throughout the time periods of Trials 5, 8 and 13, and across all three cardiac measures. Moreover, contrary to the findings of the previous study, the results reported here were not due to any pre-experimental intra-individual variance in end-organ activity. Indeed, there was little difference between predictable and unpredictable conditions either in the pre-task baseline, or during the time periods of Trial 1.

The one anomalous result, a significant effect of predictability for corrected minimum HR during the warning stimulus of Trial 1, was due to subjects in the predictable condition producing a greater increase in activity from the baseline to warning stimulus, compared to the unpredictable group. This result is difficult to explain as all subjects had received identical experimental stimuli up to this point. Indeed, the only difference between predictable and unpredictable groups was in terms of the pre-experimental instructions, in which subjects in the former condition were told that a warning signal would precede each aversive event whereas, the unpredictable group were told that the warning signal and white noise would occur randomly to one another. It is difficult to understand why these particular instructional differences would lead to an increase in minimum HR activity under predictable conditions. Furthermore, the remaining two cardiac measures showed similar

levels of responding during the warning stimulus, in both predictable and unpredictable conditions. There were no other significant results for minimum HR, corrected or uncorrected, during the time periods of Trial 1, and where any non-significant differential responding did emerge it was due to the unpredictable group having higher HR levels compared to the predictable condition (i.e. Time Periods 3 and 4).

In terms of the electrodermal system, there were no significant effects as a function of predictability during either anticipatory or impact periods, with the exception of the response to the warning stimulus during Trials 5, 8 and 13, in which the unpredictable group produced greater response amplitudes compared to the predictable condition. However, detailed examination of the other electrodermal measures - tonic SCL and frequency of NSFs - also revealed greater activity in unpredictable compared to predictable conditions, across all time periods. These latter findings should, however, be considered in the light of the pre-task baseline results which, although not statistically significant, do indicate that subjects in the unpredictable group had higher electrodermal activity than the predictable subjects. A similar pattern also emerged during the time periods of Trial 1, before subjects had received any differential stimuli. Thus, it would appear that the effects of predictability, at least with respect to tonic SCL and the frequency of NSFs, are due to individual differences in end-organ activity, and not to experimental manipulation *per se*.

The one significant result, involving response amplitudes to the warning stimulus, should be considered in the light of these additional electrodermal findings. That is, although the three measures have been shown in the past to be relatively independent (see Kilpatrick, 1972), it is probable that they consist of similar underlying physiological mechanisms. Thus, the differential effects of predictability in the response amplitude data may be a result of pre-manipulation differences. Unfortunately, since response amplitudes were not measured in the pre-task baseline, it is not possible to clarify the role of intra-individual variance during the experimental trials.

In terms of a theoretical interpretation of the data, the cardiovascular results lend tentative support to preparatory activity explanations. This is because responding in the predictable condition was reduced across both the anticipatory periods, and at event impact. As mentioned in Section 1, the two preparatory theories suggest a reduction in autonomic activity during the event itself but do not make any specific predictions regarding activity



during the anticipatory periods. However, it has also been indicated, that both the preparatory response and preception explanations could easily be expanded to include anticipatory responding. That is, unpredictability may lead to increased anticipatory physiological activity, since subjects are waiting for a more aversive event (i.e. one they cannot adequately prepare for). However, theoretical interpretation of the data from the present study should be treated with caution, and merely as an indication of the possible mechanisms underlying the effects of predictability. Firstly, because the electrodermal measures showed little reduction in activity as a function of predictability. It is therefore debatable whether either safety or preparation was indeed beneficial in this particular experiment. Secondly, because the study was not designed specifically to examine theoretical explanations, indeed this was only secondary to the main aim of the experiment. That is, whether or not temporal predictability leads to a reduction in psychophysiological activity.

The major problem with the experiment reported in Chapter 7 appeared to be that the aversive stimulus used was too mild. In the present study, this was rectified at least to some extent, by employing stimuli set at levels of intensity which in the past have been found to elicit changes in autonomic activity as a function of predictability (e.g. Baltissen & Weimann, 1991). Indeed, there were significant increases in cardiac activity. However, these increases were not overly large; i.e. average (5.3 bpm), minimum (6.7 bpm) and maximum (1.7 bpm) HR. It is unclear why the stimuli employed in the present study failed to produce greater changes in physiological activity, although it is possible that the use of a between-subject design and stimuli calibrated in terms of dB rather than sensation level may have attenuated the subjective loudness of the experimental stimuli. Thus, although the chosen stimulus intensities should have been intense enough to elicit a substantial increase in autonomic activity to the noise, the combination of these moderating variables may have attenuated stimulus characteristics sufficiently to preclude a relatively large increase in activity across all subjects.

In addition, it may be that the duration of the aversive stimuli (i.e. 1 sec) was not of sufficient length to allow a major cardiac response to develop. Furthermore, the event was over so quickly that subjects may not have been greatly concerned as to whether or not they received information concerning stimulus onset. Indeed, this would be supported by

findings from the preference for predictability literature, in which no clear preference emerged with short duration events (i.e. 0.05 sec). However, when longer (i.e. 5 sec) durations were employed, significant preferences did emerge. This type of explanation should be treated with caution, however, since a number of studies have found strong preferences for predictability with stimulus durations as low as 0.5 sec (see Chapter 2, Section 3.2).

One variable which was not mentioned in the previous study, but which may be of importance, is the type of warning stimulus employed. For example, in both this and the previous experiment, the warning signal was a visual countdown from 5 to 1, whereas, in the vast majority of past studies which have found beneficial effects of predictability, the stimulus employed was either a light or auditory tone. It may be that the latter are better predictors, or, are in some way preferred by subjects compared to the type of countdown procedure employed here. Unfortunately, there are no studies to date which have involved countdown procedures and measured human physiological function. It is therefore not possible to clarify what effect different kinds of warning stimuli may have.

Finally, one factor which was mentioned in association with the findings of the previous study, may also be of relevance here; namely, availability of control. It was suggested that a possible reason why there were few significant effects of predictability in Chapter 7 might be because subjects did not have the opportunity to control the aversive event, and that this same control is a prerequisite for any beneficial effects of predictability. As in the previous study, this experiment did not independently manipulate actual control and, therefore, it is not possible to draw any firm conclusions on the interrelationship between predictability of an event and control over that same event. For example, it may be that if subjects in the predictable condition had been able to control either the initiation, duration or termination of the white noise, then the significant effects observed during this experiment in terms of cardiovascular activity may have been sustained across all time periods, and for all three HR measures. Additionally, there may well have emerged more significant differences in the electrodermal system. The possible relationship between temporal predictability and control will be discussed in the next experimental chapter.

## CHAPTER 9

### EXPERIMENT 4

#### 1. INTRODUCTION

In Chapters 7 and 8, it was suggested that the non-availability of control may have played an important role in the effects of predictability. The potential relationships between temporal predictability and control were discussed in some detail in Chapter 4. Three such relationships were introduced, all of which have received some empirical support. First, the effects of predictability may be reducible to the effects of added control. Second, the effects of control may be reducible to the added predictability associated with control. Such a suggestion would be supported by the Safety Signal (Seligman, 1968), Relevant Feedback (Weiss, 1971a) and Preparatory Response (Perkins, 1968) Theories which suggest that control provides the individual with predictive information about the aversive event. This information leads to stress reduction by providing relevant feedback, increased safety signals or more accurately timed preparatory responses.

Finally, predictability and control may be independent. That is, control produces effects over and above those of additional predictability, and *vice versa*. This is in line with the minimax hypothesis (Miller, 1979), which suggests that control conditions insure lower maximum future danger than no control conditions. The Minimax Theory also assumes that the effects of predictability and control are additive. Indeed, that the effects are beneficially additive. That is, situations where both factors are present lead to the greatest reduction in psychophysiological stress. Where one or other factor is available, intermediate levels of psychophysiological stress result. However, the effects of predictability and those of control are not always beneficial. For example, temporal predictability has been shown to augment psychophysiological stress compared to unpredictability (see Chapter 2). It may well be that the opportunity to control leads to a reduction in the deleterious effects of predictability in some situations, whilst in others availability of control adds to these effects. Unfortunately, there are no studies to date which have investigated this relationship.

However, it may be that the effects of predictability and control are not additive. In aversive situations, either control *or* predictability may be sufficient to lower psychophysiological stress with no additional benefit being derived from the presence of both factors. Evidence supporting this comes from those studies in which subjects who received controllable aversive stimuli, predictable aversive stimuli, or aversive stimuli that were both predictable and controllable displayed similar levels of psychophysiological stress and measures of performance. Only subjects who lacked both control and predictability over the aversive stimulus displayed significantly greater performance decrements and psychophysiological stress than subjects exposed to no aversive stimuli (e.g. Burger & Arkin, 1980) (see Chapter 4).

The aim of this experiment was to explore the possible relationship between temporal predictability and control. The paradigm used was similar to that in the preceding study in which differential effects of predictability were produced. Availability of control was manipulated using a technique employed in perceived control studies (e.g. Geer, Davison & Gatchel, 1970) in which the opportunity to control led to a reduction in autonomic and subjective activity compared with a no control condition. This technique involved a purely instructional manipulation of control. That is, subjects in the control condition were informed that they could control the duration of the noise depending on their performance in a reaction time task, whereas, subjects in the no control condition were not told of any relationship between performance and noise duration. However, none of the subjects could actually alter noise duration. Subjects in the control condition merely believed they could (i.e. they had perceived instrumental control (Miller, 1979)). Employing this type of design meant that exposure to the aversive stimuli was identical across all groups, which alleviated the disadvantages associated with yoked control designs (see Chapter 4, Section 1.1).

An additional aim of the experiment was to differentiate between theoretical explanations for any beneficial effects of control and/or predictability. The Safety Signal (Seligman, 1968), Relevant Feedback (Weiss, 1971a) and Preparatory Response (Perkins, 1968) Theories (the predictability theories) have in common the proposition that availability of control only provides additional predictability of the aversive event. Thus, when the two factors are manipulated independently, as in the present experiment, the predictability

theories predict that the effects of control should not be greater than when the aversive stimulus is perfectly predictable. In contrast, Miller's (1979) Minimax Theory suggests that control provides an individual with more than just extra predictability, and in situations where the two variables are manipulated independently, the effects of control should be greater than predictability alone. Specific predictions made by the theory are that subjects should show less anticipatory (i.e. during the warning signal, inter-stimulus and inter-trial interval) physiological arousal in situations with control because the maximum danger presented by the aversive event is lower than when the event is uncontrollable. The hypothesis makes no predictions regarding event impact, as it proposes no mechanism whereby expectations concerning the maximum danger presented by the event affect responses to that event once it occurs. However, responding at impact may be reduced in control situations, if it is assumed that when subjects have a controlling response available they experience less anticipatory arousal, and against this background of relaxation, the aversive event leads to less physiological arousal at impact. The Minimax Theory also predicts that the effects of predictability and control are additive. That is, where both factors are present the greatest physiological activity should be observed whereas, where either predictability or control is available intermediate levels should result.

## 2. METHOD

### 2.1. Subjects

The subjects were 56 undergraduate volunteers (44 females and 12 males), with an age range of 18 - 40 years. All subjects were randomly allocated to one of four experimental conditions; predictable/control (PC); predictable/no control (PNC); unpredictable/control (UPC) or unpredictable/no control (UPNC). The only restriction to the random allocation was that there were equal numbers of the smaller, male population in each of the four groups.

## 2.2. Apparatus

The method of recording SC and HR was the same as that employed in Experiment 3, with the exception that SC was recorded from the non-preferred hand and HR was measured using a finger plethysmograph (Type PPS) attached to the fourth finger of the non-preferred hand. The warning stimuli and presentation of the warning stimuli were identical to that employed in Experiment 3, with the exception that it consisted of the numbers 3, 2, 1 only. The white noise was of 3 sec duration, and 98 dB.

A reaction time button of 17 by 17 mm was situated on the subject's lap. All reaction time data was recorded by the BBC microcomputer.

A two way intercom system was in operation throughout. It was situated 8 inches from the subject and directly in front of them.

## 2.3. Design and Procedure

The experimental design was a 2 (control/no control) x 2 (predictable/unpredictable) x 13 (trials) repeated measures between subject.

The seating and accommodation of subjects was the same as in Experiment 1. After electrode attachment, subjects were informed of the procedure to follow (see appendix). Those subjects in the control groups were told that they could reduce the duration of the white noise from 5 to 2 secs if their reaction time to the message "Press button now" was within certain limits set by the computer. However, if their reaction time was not within these limits then the duration of the noise would be 5 sec. In contrast, subjects in the no control groups were told to press the button as quickly as possible, but were not told of any relationship between reaction time and duration of the noise. None of the subjects, either in control or no control conditions, could actually control the duration of the noise. However, by assuring that all subjects pressed the reaction time button any confounding effects associated with differential motor activity were controlled for. Subjects in the predictable and unpredictable conditions also received differing pre-task instructions. Those subjects in the predictable groups were told that they would receive a warning

consisting of the numbers 3, 2, 1 shortly before noise onset. Subjects in the unpredictable groups were told that the warning and white noise would occur randomly.

All subjects then received a 1 min rest period followed by, the range correction stimulus which consisted of 98 dB white noise, 3 sec in duration. Following this, there was a 3 min rest period. All subjects then received 13 trials with an inter-trial interval of 25, 30 or 35 sec. The mean ITI was 30 sec. The experimental trials were identical to that employed in Experiment 3, except that the warning signal consisted of the numbers 3, 2, 1 only. Each number was displayed on the subject's monitor for 1 sec. In addition, the white noise was of 3 sec duration. 1 sec after noise onset the message "Press button now" appeared on the subject's monitor. The message remained on the screen until subjects pressed the reaction time button, and for up to a maximum of 2 sec thereafter.

After the subject had completed 13 trials, the experimenter unwired and debriefed the subject.

## 2.4. Scoring

2.4.1. Cardiovascular activity. The measurement of HR was the same as in Experiment 1. All three cardiovascular measures (avHR, mnHR and mxHR) were assessed during the 3 min pre-task baseline in order to examine any pre-task between-group differences.

Between-group differences in cardiovascular activity were assessed across Trials 1, 5, 8 and 13. Trial 1 was analysed separately from Trials 5, 8 and 13. Each trial was divided into 6 separate time periods: Period 1, consisted of the 0-3 sec after warning onset: Period 2, was the 0-5 sec before onset of the noise (ISI): Period 3 was the 0-1 sec after noise onset: Period 4, was the 1-3 sec after noise onset: Period 5 was the 25 sec ITI divided into five, 5 sec blocks: Period 6, involved a measure of the cardiac response profile. Measurement of the cardiac response profile was the same as in Experiment 2. Uncorrected and corrected HR are reported for all three cardiovascular measures across Time Periods 1 to 5. Measurement of corrected HR was the same as in Experiment 1. Period 6 was examined for change in avHR across Trials 5, 8 and 13 only.

2.4.2. Electrodermal activity. Tonic SCL and frequency of NSF's were assessed during the 3 min pre-task baseline. They were measured following the procedure of Experiment 1.

Between-group differences in electrodermal activity were assessed across Trials 1, 5, 8 and 13. Trial 1 was analysed separately from Trials 5, 8 and 13. For tonic SCL, each trial was divided into three periods: Period 1, was the 0-3 sec after warning onset: Period 2, was of the 0-5 sec before onset of the noise (ISI) and Period 3 consisted of the 5-25 sec of the ITI divided into four, 5 sec blocks. The frequency of NSF's was assessed during the 5-25 sec of the ITI only.

The criteria, measurement, range-correction and transformation of evoked responses were the same as in Experiment 3. Between-group comparisons of range-corrected SCR amplitude were assessed during three time periods: Period 1, was the 1-5 sec after warning onset: Period 2 consisted of the 1-5 sec after noise onset and Period 3 was the 1-5 sec after noise offset.

2.4.3. Reaction Time Data. Between group differences in reaction time (RT) were assessed across Trials 1, 5, 8 and 13. Trial 1 was analysed separately from Trials 5, 8 and 13.

### 3. RESULTS

#### 3.1. Baseline Activity

With all measures, differences between groups were assessed across the 3 min baseline. These 3 min were divided into three, 60 sec periods and a three-way MANOVA was employed with control (control/no control) and predictability (predictable/unpredictable) as between subject factors and time (3 levels) as a repeated measure. Group means and standard deviations for cardiovascular and electrodermal baseline activity are shown in Tables 19 and 20 respectively.

3.1.1. Cardiovascular Activity. There were no significant effects of control for avHR ( $F(1, 52) = 2$ ), mnHR ( $F(1, 52) = 2.5$ ) or mxHR ( $F < 1$ ), and nor were there any effects



of predictability involving avHR, mnHR (both  $F_s < 1$ ) or mxHR ( $F(1, 52) = 1.7$ ). There were no control by predictability interactions involving avHR ( $F(1, 52) = 2.2$ ) or mnHR ( $F < 1$ ). However, there was a significant control by predictability interaction for mxHR ( $F(1, 52) = 4.6, p < .05$ ). Analysis of the simple main effects revealed that the PC and UPNC groups had higher HR than the PNC group. The UPNC group also produced higher HR than the UPC group and, to a smaller extent the PC group. However, none of these comparisons were significant (Figure 28). There was a significant difference across time for avHR ( $F(2, 76) = 9.1, p < .001$ ), mnHR ( $F(2, 99) = 5.3, p < .01$ ) and mxHR ( $F(2, 88) = 27.8, p < .001$ ). Post-hoc comparison of means revealed, firstly for avHR, that the 0-60 sec period had significantly higher HR than the 60-120 and 120-180 sec periods. The 120-180 sec period produced significantly higher HR levels than the 60-120 sec period (84.3, 82 and 83.2 bpm for the 0-60, 60-120 and 120-180 sec periods respectively). Analysis of mnHR revealed that the 120-180 sec period had significantly higher HR than the 0-60 and 60-120 sec periods (70.3, 70.5 and 72.3 bpm for the 0-60, 60-120 and 120-180 sec periods respectively). MxHR revealed a significant decrement in levels of activity across the 60 sec periods (100.7, 94.8 and 95.2 bpm for the 0-60, 60-120 and 120-180 sec periods respectively). There were no significant interactions involving time for any of the three measures (all  $F_s < 1$ ).

3.1.2. Electrodermal Activity. There were no significant differences between groups in terms of either of the electrodermal measures (all  $F_s < 1$ ). There was a significant difference across time for SCL ( $F(1, 65) = 20.5, p < .001$ ). The 0-60 sec period produced significantly higher SCL than the 60-120 and 120-180 sec periods (3.19, 2.99 and 3  $\mu$ S respectively). There were no significant interactions involving time for SCL (all  $F_s < 1$ ). There was no significant difference across time involving NSF $s$  ( $F < 1$ ), nor any predictability by time ( $F(2, 85) = 1.4$ ) or control by time ( $F(2, 85) = 1.6$ ) interactions.

3.1.3. Summary. There were no significant differences between groups in terms of cardiovascular or electrodermal measures, with the exception of mxHR which produced a control by predictability interaction.

Figure 28. Mean maximum HR as a function of groups during the pre-task baseline.

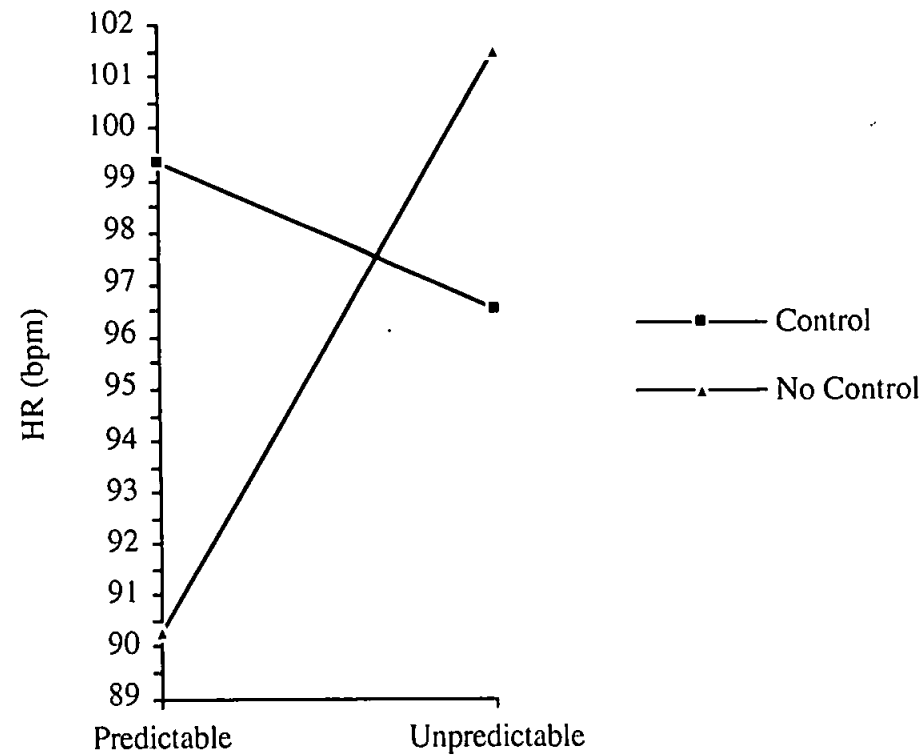
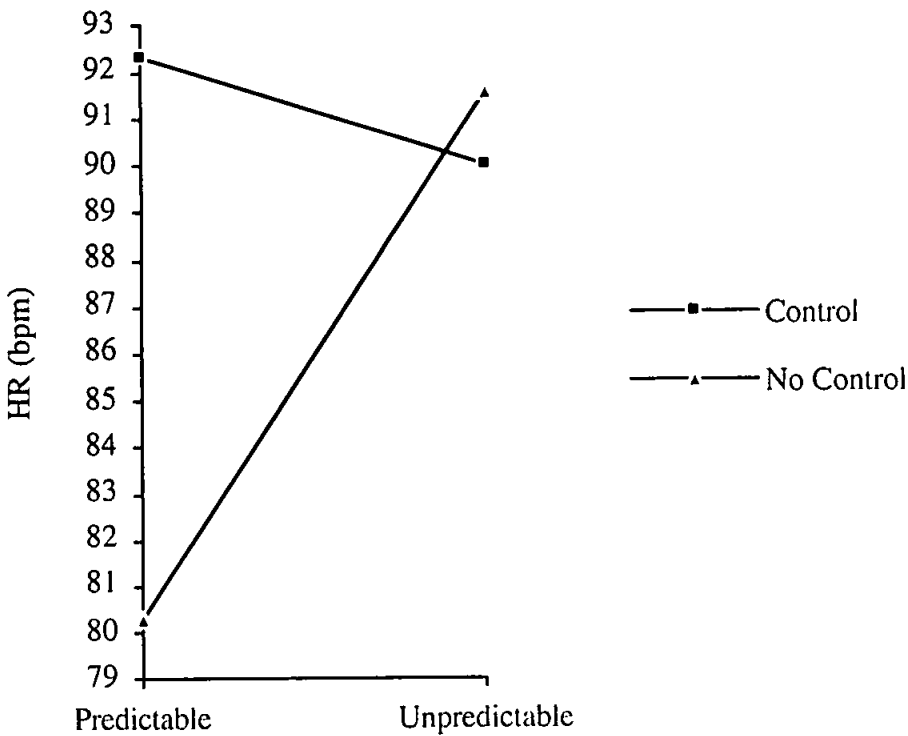


Figure 29. Mean maximum HR as a function of groups during Time Period 1, Trial 1.



### 3.2. Between-Groups Analysis: Trial 1

#### 3.2.1. Cardiovascular Activity

For all measures, Time periods 1 to 4 were each assessed in a two-way ANOVA with control and predictability as between subject factors. Time Period 5 was analysed in a three-way MANOVA with the above between-subject factors and time (5 levels) as a repeated measure. Group means and standard deviations for uncorrected HR during Trial 1 are shown in Table 19.

##### 3.2.1.1. Uncorrected HR

3.2.1.1.1. Time Period 1. There were no significant main effects of control involving avHR ( $F(1, 52) = 3$ ), mnHR or mxHR (both  $F_s(1, 52) = 2.3$ ), nor were there any significant effects of predictability involving avHR ( $F(1, 52) = 1.6$ ), mnHR ( $F(1, 52) = 2.4$ ) or mxHR ( $F(1, 52) = 1.7$ ). There were no control by predictability interactions involving avHR ( $F(1, 52) = 2.9$ ) or mnHR ( $F(1, 52) = 1.7$ ). However, mxHR did produce a control by predictability interaction ( $F(1, 52) = 4.1, p < .05$ ). Analysis of the simple main effects revealed that the PC and UPNC groups had significantly higher HR than the PNC group. The UPC group also produced higher HR than the PNC group, although this did not reach significance. There were no significant differences between the three remaining groups (Figure 29).

3.2.1.1.2. Time Period 2. There were no significant effects of control involving avHR ( $F(1, 52) = 1.9$ ), mnHR ( $F(1, 52) = 3.1$ ) or mxHR ( $F(1, 52) = 1.3$ ), nor were there any main effects of predictability involving avHR ( $F(1, 52) = 1.2$ ), mnHR ( $F < 1$ ) or mxHR ( $F(1, 52) = 2.3$ ). There were no significant two-way control by predictability interactions involving avHR ( $F(1, 52) = 3.4$ ) or mnHR ( $F(1, 52) = 1.9$ ). However, mxHR did produce a control by predictability interaction ( $F(1, 52) = 4.2, p < .05$ ). Analysis of the simple main effects revealed similar results as Time Period 1. They are therefore not reported in detail here.

3.2.1.1.3. Time Period 3. There were significant effects of control involving all three measures: avHR ( $F(1, 52) = 5.6, p < .05$ ), mnHR ( $F(1, 52) = 5.9, p < .05$ ) and

Table 19. Mean cardiovascular activity during the pre-task baseline and time periods of Trial 1. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Groups			
	PC	PNC	UPC	UPNC
Pre-Task Baseline				
AvHR	86.2 (11.3)	76.9 (11.0)	84.8 (12.0)	85.0 (14.2)
MnHR	72.3 (13.5)	66.5 (10.4)	74.5 (11.2)	70.7 (12.6)
MxHR	99.3 (11.0)	90.3 (12.3)	96.6 (12.0)	101.5 (15.5)
Time Period 1				
AvHR	86.1 (11.6)	74.8 (12.4)	84.7 (12.6)	84.5 (12.6)
MnHR	79.3 (11.9)	70.1 (12.2)	80.1 (12.6)	79.4 (12.4)
MxHR	92.3 (11.4)	80.3 (13.2)	90.0 (13.2)	91.6 (13.6)
Time Period 2				
AvHR	86.1 (9.79)	75.9 (11.6)	83.7 (11.0)	85.1 (14.2)
MnHR	80.8 (10.9)	71.1 (11.5)	78.9 (10.9)	77.7 (12.8)
MxHR	91.2 (9.68)	80.9 (11.2)	89.5 (11.8)	92.4 (15.0)
Time Period 3				
AvHR	90.7 (12.0)	76.6 (11.5)	88.3 (13.7)	85.5 (15.9)
MnHR	88.7 (12.6)	76.7 (11.5)	86.6 (13.8)	83.0 (16.1)
MxHR	92.9 (11.2)	78.6 (11.6)	90.2 (13.5)	88.6 (16.1)
Time Period 4				
AvHR	94.1 (9.86)	81.3 (11.1)	92.0 (10.4)	92.3 (15.3)
MnHR	89.5 (9.00)	75.8 (10.3)	87.2 (11.6)	84.7 (14.5)
MxHR	98.6 (11.2)	86.6 (12.0)	96.1 (10.5)	98.3 (15.1)
Time Period 5				
AvHR	90.5 (10.5)	80.8 (11.3)	90.4 (10.6)	88.4 (14.5)
MnHR	84.4 (10.4)	75.5 (10.3)	85.2 (9.29)	86.6 (12.4)
MxHR	95.8 (10.3)	85.9 (12.4)	95.3 (12.1)	95.3 (15.6)

mxHR ( $F(1, 52) = 5, p < .05$ ). The C groups produced significantly higher HR than the NC groups (avHR = 89.5 and 81.1 bpm; mnHR = 87.7 and 78.8 bpm; mxHR = 91.5 and 83.6 bpm respectively). There were no significant effects of predictability involving any of the measures (all  $F$ s  $< 1$ ). There were no control by predictability interactions involving avHR ( $F(1, 52) = 2.4$ ), mnHR ( $F(1, 52) = 2.1$ ) or mxHR ( $F(1, 52) = 3.2$ ).

3.2.1.1.4. Time Period 4. There were main effects of control involving avHR ( $F(1, 52) = 4.1, p < .05$ ), mnHR ( $F(1, 52) = 7, p < .01$ ) and mxHR ( $F(1, 52) = 4.2, p < .05$ ). The C groups produced higher HR levels than the NC groups (avHR = 93.1 and 86.8 bpm; mnHR = 88.4 and 80.2 bpm; mxHR = 97.4 and 92.4 bpm respectively). There were no significant effects of predictability for any of the measures (all  $F$ s  $< 1$ ). There were control by predictability interactions involving avHR ( $F(1, 52) = 4.3, p < .05$ ) and mxHR ( $F(1, 52) = 4.7, p < .05$ ). Analysis of the simple main effects for both measures revealed similar results as Time Period 1. The PC and UPNC groups had significantly higher HR than the PNC groups. The UPC group also had higher HR than the PNC group, although this was not significant. There was no significant control by predictability interaction involving mnHR ( $F(1, 52) = 3.4$ ).

3.2.1.1.5. Time Period 5. MnHR produced a significant effect of control ( $F(1, 52) = 4, p < .05$ ). The C groups had higher HR than the NC groups (84.8 and 78.5 bpm respectively). However, there were no significant effects of control involving avHR ( $F(1, 52) = 3.4$ ) or mxHR ( $F(1, 52) = 2.2$ ). There were no significant effects of predictability involving any of the three measures (all  $F$ s  $< 1$ ), nor were there any control by predictability interactions involving avHR ( $F(1, 52) = 1.5$ ), mnHR ( $F < 1$ ) or mxHR ( $F(1, 52) = 2.1$ ). There was a significant difference across time for avHR ( $F(3, 166) = 11.1, p < .001$ ), mnHR ( $F(3, 161) = 4.4, p < .05$ ) and mxHR ( $F(3, 166) = 10.8, p < .001$ ). Post-hoc comparison of means revealed, firstly for avHR, that the 0-5 and 5-10 sec periods had significantly higher HR than the remaining periods. The 0-5 sec period also produced significantly higher HR than the 5-10 sec period (90.2, 88.5, 87.0, 86.2 and 85.8 bpm for the 0-5, 5-10, 10-15, 15-20 and 20-25 sec periods respectively). MnHR revealed that the 0-5 sec period had significantly higher HR than the 10-15, 15-20 and 20-25 sec periods. The 5-10 sec period produced significantly higher HR than the 20-25 sec period (83.5, 82.2, 81.5, 81.1 and 79.9 bpm for the 0-5, 5-10, 10-15, 15-20 and 20-25 sec periods

respectively). MxHR revealed that the 0-5 and 5-10 sec periods had significantly higher HR than all the remaining periods (96.2, 94.1, 92, 91.6 and 91.5 bpm for the 0-5, 5-10, 10-15, 15-20 and 20-25 sec periods respectively). There were no interactions involving time for any of the three cardiovascular measures (all  $F < 1$ ).

3.2.1.1.6. Summary. There were no significant differences between groups during the warning stimulus and the ISI (Time Periods 1 and 2), with the exception of mxHR which produced a control by predictability interaction. This was due to the PC, UPNC and, to a smaller extent the UPC group producing significantly higher HR than the PNC group.

During the 0-1 and 1-3 sec after noise onset (Time Periods 3 and 4), the C groups produced higher HR levels than the NC groups across all three measures. In addition, during Time Period 4 both avHR and mxHR produced a control by predictability interaction. Further analysis revealed a similar pattern as seen in Time Periods 1 and 2 for mxHR.

During the ITI (Time Period 5) mnHR produced a significant effect of control, due to the C groups demonstrating higher HR levels than the NC groups. There were no other significant differences between groups during this period, across any of the cardiovascular measures.

Further examination of the cardiovascular data across Time Periods 1 to 5, reveals a dissimilar pattern of responding for the three HR measures (see Table 19). For example, avHR produced similar levels of activity during the pre-task baseline and the warning stimulus ( $T(1, 55) = 1.6$ ) whereas, mxHR showed a significant decrease to the warning ( $T(1, 55) = 9.6, p < .001$ ), and mnHR a significant increase ( $T(1, 55) = 7.6, p < .001$ ). A similar pattern emerged between the baseline and the 0-1 sec after noise onset; both avHR and mnHR produced a significant increase to the noise ( $T(1, 55) = 2.1, p < .05$ ) and ( $T(1, 55) = 9.8, p < .001$ ) respectively, whilst mxHR showed a significant decrease ( $T(1, 55) = 7.0, p < .001$ ). Further comparisons between the warning stimulus and the 0-1 sec after noise onset revealed similar levels of activity for mxHR ( $T < 1$ ) compared with a significant increase to the noise involving both avHR ( $T(1, 55) = 3.0, p < .05$ ) and mnHR ( $T(1, 55) = 6.3, p < .001$ ). This pattern of activity was more marked when comparing the warning stimulus and the 1-3 sec after noise onset, with highly significant increases to the

noise in all three measures: avHR ( $T(1, 55) = 9.1, p < .001$ ), mnHR ( $T(1, 55) = 7.2, p < .001$ ) and mxHR ( $T(1, 55) = 8.2, p < .001$ ).

3.2.1.2. Corrected HR. The results involving corrected HR were similar to those of uncorrected HR, unless stated otherwise. During Time Period 1, there was a significant main effect of predictability involving mnHR ( $F(1, 52) = 4.7, p < .05$ ). The UP groups had higher HR levels than the P groups (6.25 and 3.59 change in bpm respectively). In addition, during Time Periods 1 and 2 mxHR produced no control by predictability interaction (both  $F_s < 1$ ). During Time Periods 3, 4 and 5 there were no significant effects of control involving avHR (Time Periods 3 and 4), mnHR (Time Periods 3, 4 and 5) or mxHR (Time Periods 3 and 4) (all  $F_s < 1$ ). In addition, during Time Period 4 there were no control by predictability interactions involving avHR or mxHR (both  $F_s < 1$ ).

3.2.1.3. Corrected HR Summary. During the warning stimulus (Time Period 1), corrected mnHR produced an effect of predictability due to the UP groups producing a greater increase in levels of activity from the baseline period compared with the P groups. In addition, during Time Period 1 and the ISI (Time Period 2) corrected mxHR produced no warning by control interaction. Further inspection of the means revealed that all four groups produced a similar decrease in levels of activity from the pre-task baseline (Time Period 1 = -6, -7.9, -5 and -7.9, Time Period 2 = -7.1, -7.3, -5.5 and -7.1 change in bpm for the PC, PNC, UPC and UPNC groups respectively).

During the 0-1 and 1-3 sec periods after noise onset and the ITI (Time Periods 3, 4 and 5) corrected mnHR produced no main effect of control, due to both the C and NC groups producing similarly marked increases in levels of activity from the pre-task baseline (Time Period 3 = 12.5 and 14.1, Time Period 4 = 13.2 and 10.8, Time Period 5 = 9.6 and 9.1 change in bpm respectively). During Time Periods 3 and 4, both corrected avHR and mxHR produced no significant effects of control. Further inspection of means revealed a similar pattern as seen previously, with the C and NC groups producing similar increases in levels of activity from the pre-task baseline (e.g. Time Period 3; avHR = 7.6 and 5.8; mxHR = 0.7 and -1.4 change in bpm respectively). In addition, both avHR and mxHR produced no significant control by predictability interactions during Time Period 4. All four

groups produced similar increases in HR from the baseline (avHR = 7.2, 4.5, 7.9 and 7.2, mxHR = -0.3, 1.6, -1.1 and 1.2 change in bpm for the PC, PNC, UPC and UPNC groups respectively).

In general, it would appear that the significant effect of control during Time Periods 3, 4 and 5 was mainly due to pre-task differences between the C and NC groups, and not a result of experimental manipulation. The significant control by predictability interaction involving all three measures throughout Trial 1, also appears to be due to pre-task differences, with the PNC group producing markedly lower HR across all time periods. Corrected HR, however, revealed no control by predictability interaction due to all four groups producing similar changes in HR from the baseline period.

### 3.2.2. Electrodermal Activity

Tonic SCL was assessed during Time Periods 1 and 2 in a two-way ANOVA with control and predictability as between subject factors. SCL during Time Period 3 was analysed in a three factor MANOVA with the above between subject factors and time (4 levels) as a repeated measure. Frequency of NSFs and transformed scores for SCR Amplitude were assessed in a two-way ANOVA as above. Group means and standard deviations for electrodermal activity during Trial 1 are shown in Table 20.

3.2.2.1. Skin Conductance Level. There were no significant effects of control or predictability across any of the three time periods (all  $F_s < 1$ ). There was a highly significant difference across time during Time Period 3 ( $F(1, 70) = 70.9, p < .001$ ). Comparison of means revealed a significant decrement in levels of activity across the 20 sec of the ITI (3.5, 3.34, 3.25 and 3.19  $\mu S$  for the 5-10, 10-15, 15-20 and 20-25 sec periods respectively). There were no interactions involving time during Time Period 3 (all  $F_s < 1$ ).

3.2.2.2. Non-Specific Fluctuations. There were no significant effects of control or predictability, nor any interactions involving the two factors (all  $F_s < 1$ ).



Table 20. Mean electrodermal activity during the pre-task baseline and time periods of Trial 1. Standard deviations are shown in parentheses.

Variables	Experimental Groups			
	PC	PNC	UPC	UPNC
Pre-Task Baseline				
Tonic SCL ( $\mu$ S)	3.00 (1.27)	3.27 (1.67)	2.69 (1.15)	3.27 (1.63)
Frequency of NSF's	4.59 (3.01)	4.97 (4.20)	5.14 (4.13)	3.67 (2.75)
Time Period 1				
Tonic SCL ( $\mu$ S)	3.10 (3.10)	3.37 (3.37)	2.90 (2.90)	3.35 (3.35)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.14 (0.24)	0.12 (0.08)	0.06 (0.12)	0.06 (0.08)
Time Period 2				
Tonic SCL ( $\mu$ S)	3.34 (1.32)	3.66 (2.21)	3.02 (1.38)	3.57 (1.75)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.02 (0.09)	0.07 (0.12)	0.01 (0.07)	0.02 (0.06)
Time Period 3				
Tonic SCL ( $\mu$ S)	3.30 (1.31)	3.58 (1.98)	2.94 (1.39)	3.45 (1.56)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.27 (0.21)	0.28 (0.19)	0.19 (0.12)	0.19 (0.13)
Frequency of NSF's	0.61 (1.17)	0.98 (1.21)	0.84 (1.60)	0.77 (1.10)

Table 21. Mean cardiovascular activity during time periods averaged across Trials 5, 8 and 13. Standard deviations are shown in parentheses.

Variables HR (bpm))	Experimental Groups			
	PC	PNC	UPC	UPNC
Time Period 1				
AvHR	85.0 (11.5)	74.3 (11.9)	80.4 (10.2)	83.1 (10.6)
MnHR	80.4 (11.1)	69.5 (11.6)	75.3 (10.0)	77.5 (12.3)
MxHR	89.6 (11.4)	79.5 (12.0)	85.8 (11.1)	89.2 (14.3)
Time Period 2				
AvHR	83.7 (9.99)	75.4 (10.9)	79.6 (10.3)	83.0 (13.8)
MnHR	77.8 (10.0)	69.3 (10.4)	75.1 (10.0)	76.9 (13.2)
MxHR	89.7 (10.6)	81.6 (12.9)	85.2 (11.4)	89.9 (14.6)
Time Period 3				
AvHR	82.2 (10.6)	72.3 (11.1)	81.2 (11.8)	82.8 (13.0)
MnHR	80.4 (11.8)	70.5 (11.2)	79.1 (11.3)	79.9 (12.9)
MxHR	84.1 (10.0)	74.2 (11.3)	83.3 (12.5)	85.4 (13.5)
Time Period 4				
AvHR	84.3 (10.6)	73.2 (10.8)	83.2 (10.4)	85.5 (13.3)
MnHR	80.5 (10.9)	69.7 (10.5)	79.8 (10.2)	81.6 (13.2)
MxHR	88.1 (10.7)	76.8 (11.8)	86.4 (11.1)	89.1 (13.2)
Time Period 5				
AvHR	84.4 (9.89)	75.2 (11.8)	80.6 (9.00)	83.2 (12.7)
MnHR	78.9 (10.0)	70.1 (11.8)	75.3 (8.89)	76.7 (10.7)
MxHR	89.5 (10.8)	80.6 (13.0)	86.2 (9.61)	89.8 (15.4)

Figure 30. Average HR as a function of groups during Time Period 1.

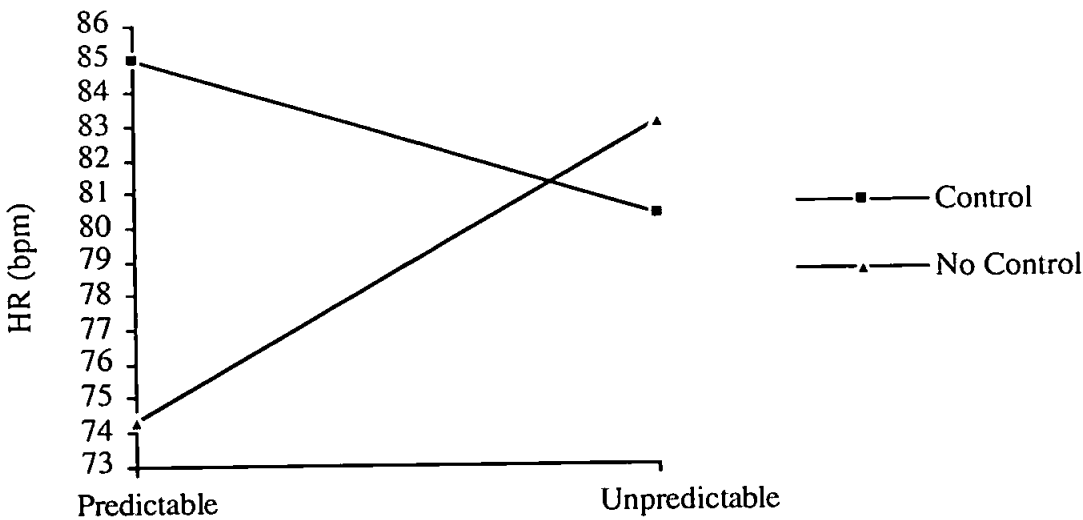


Figure 31. Mean minimum HR as a function of groups during Time Period 1.

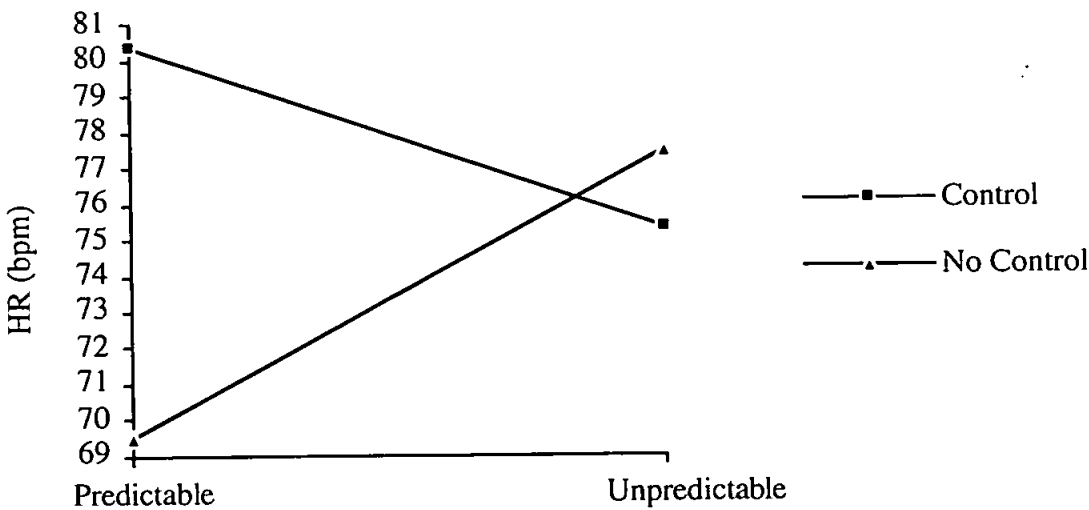
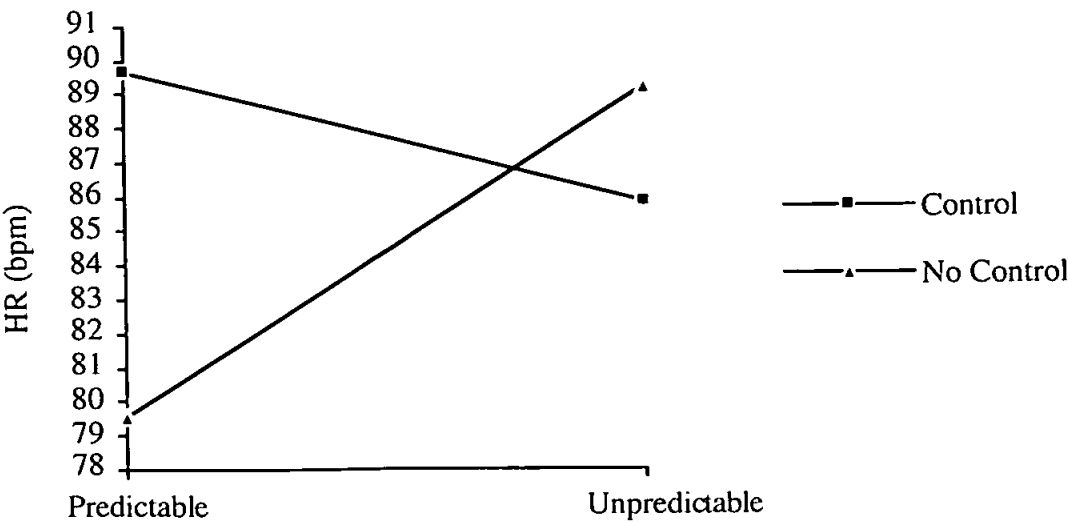


Figure 32. Mean maximum HR as a function of groups during Time Period 1.



the UPC group. The PC and UPNC groups revealed similar levels of activity. The UPC group had higher HR than the PNC group. However, none of these comparisons were statistically significant. There was no significant difference across trials or any interaction involving trials for avHR (all  $F_s < 1$ ). MnHR produced no significant effect of trials ( $F < 1$ ), no control by trials ( $F(2, 100) = 2.7$ ), predictability by trials ( $F(2, 100) = 1.5$ ), or any three-way interaction ( $F < 1$ ). MxHR produced no significant difference across trials ( $F < 1$ ), or a predictability by trials interaction ( $F(2, 100) = 2$ ). However, there was a significant control by trials interaction ( $F(2, 100) = 3.2, p < .05$ ). Simple main effects revealed that the C groups had significantly higher HR levels than the NC groups during Trials 8 and 13. There was no significant difference between groups during Trial 5 (Figure 33). MxHR produced no three-way interaction ( $F < 1$ ).

3.3.1.1.2. Time Period 2. There were no main effects of control or predictability involving any of the measures (all  $F_s < 1$ ). There was a control by predictability interaction involving avHR ( $F(1, 52) = 4, p < .05$ ) (Figure 34) and mxHR ( $F(1, 52) = 4.2, p < .05$ ) (Figure 35). Analysis of the simple main effects for both measures revealed that the PC and UPNC group had higher HR than the PNC group and, to a lesser extent the UPC group. However, none of these comparisons reached significance. MnHR produced no predictability by control interaction ( $F(1, 52) = 3.5$ ). There was no significant difference across trials involving avHR ( $F(2, 103) = 2$ ), nor was there a predictability by trials interaction ( $F < 1$ ). However, there was a two-way control by trials interaction ( $F(2, 103) = 3.1, p < .05$ ). The C groups had significantly higher HR during Trial 5 compared with the NC groups. There were no significant differences between groups during later trials (Figure 36). AvHR produced no three-way interaction ( $F < 1$ ). There was no main effect of trials or any interaction involving trials for mnHR or mxHR (all  $F_s < 1$ ).

3.3.1.1.3. Time Period 3. There were no significant effects of control involving avHR ( $F(1, 52) = 2$ ), mnHR ( $F(1, 52) = 2.5$ ) or mxHR ( $F(1, 52) = 1.6$ ), nor were there any significant main effects of predictability involving avHR ( $F(1, 52) = 2.6$ ), mnHR ( $F(1, 52) = 2$ ) or mxHR ( $F(1, 52) = 2.9$ ). There were no control by predictability interactions involving any of the three measures; avHR ( $F(1, 52) = 3.8$ ), mnHR ( $F(1, 52) = 3.5$ ) and mxHR ( $F(1, 52) = 3.9$ ). There was no significant difference across trials or any

Figure 33. Mean maximum HR as a function of control and trials during Time Period 1.

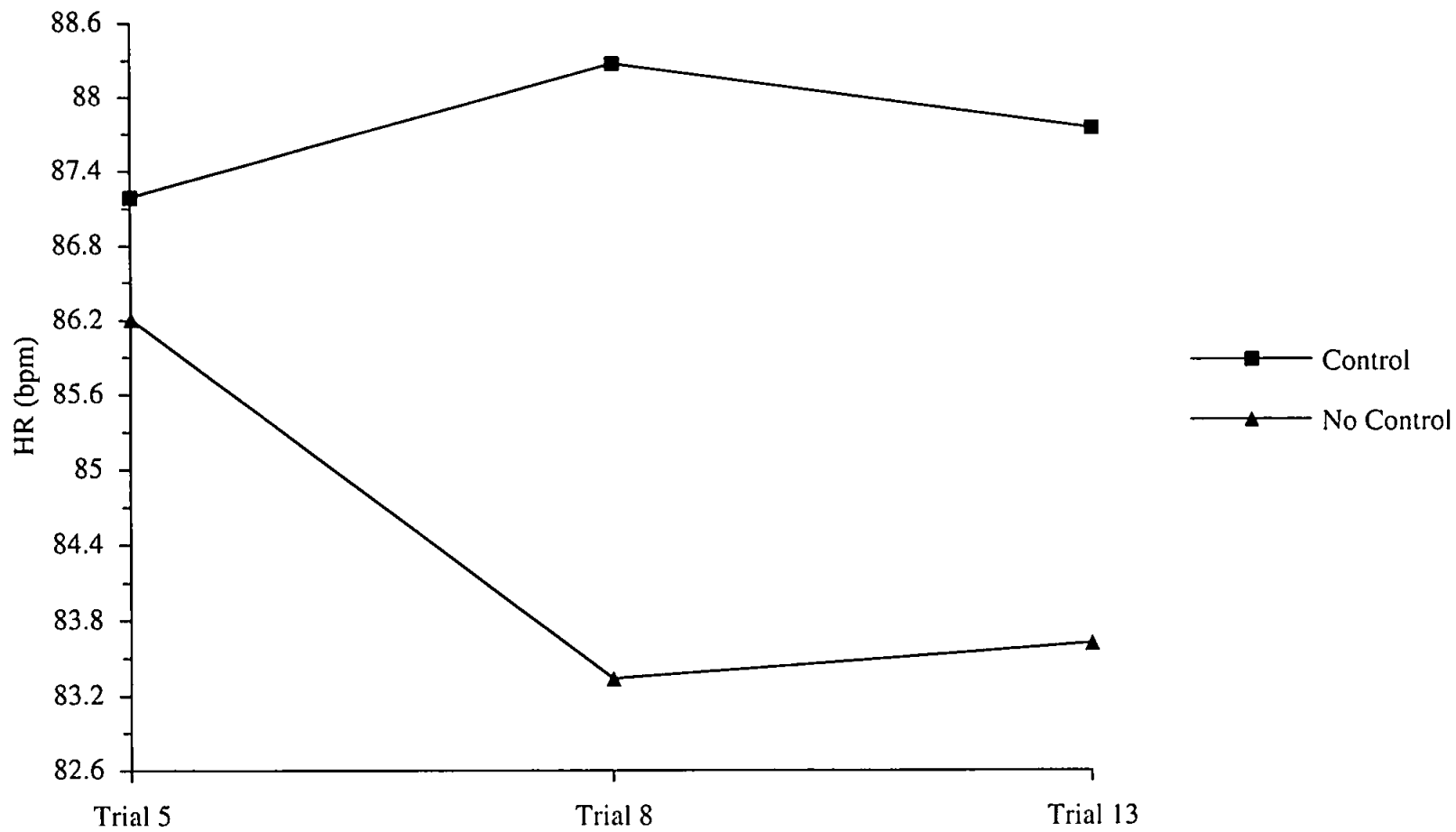


Figure 34. Average HR as a function of groups during Time Period 2.

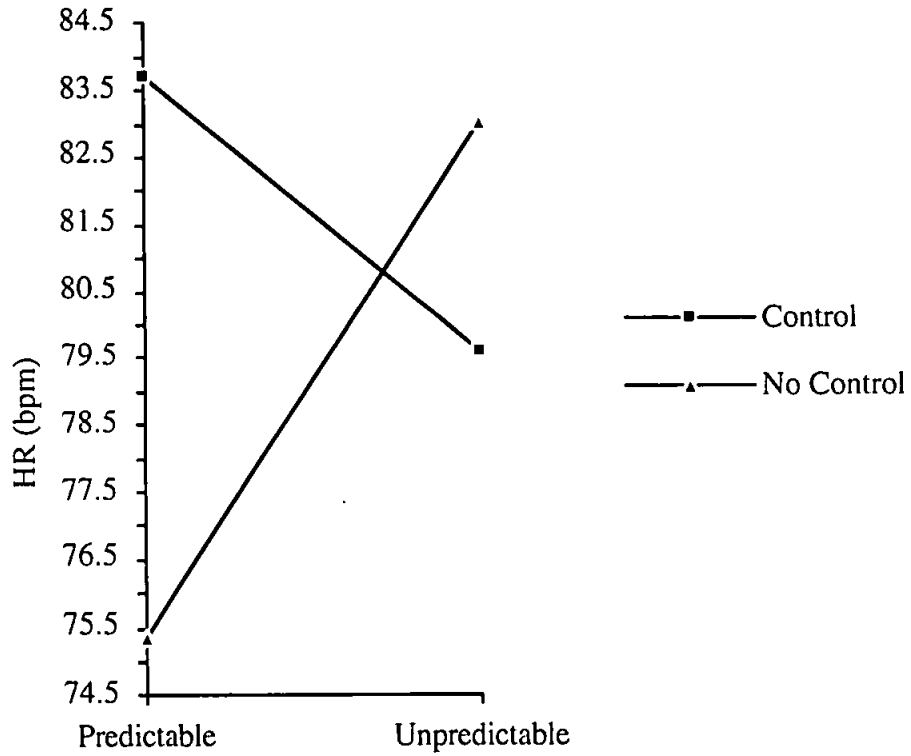


Figure 35. Mean maximum HR as a function of groups during Time Period 2.

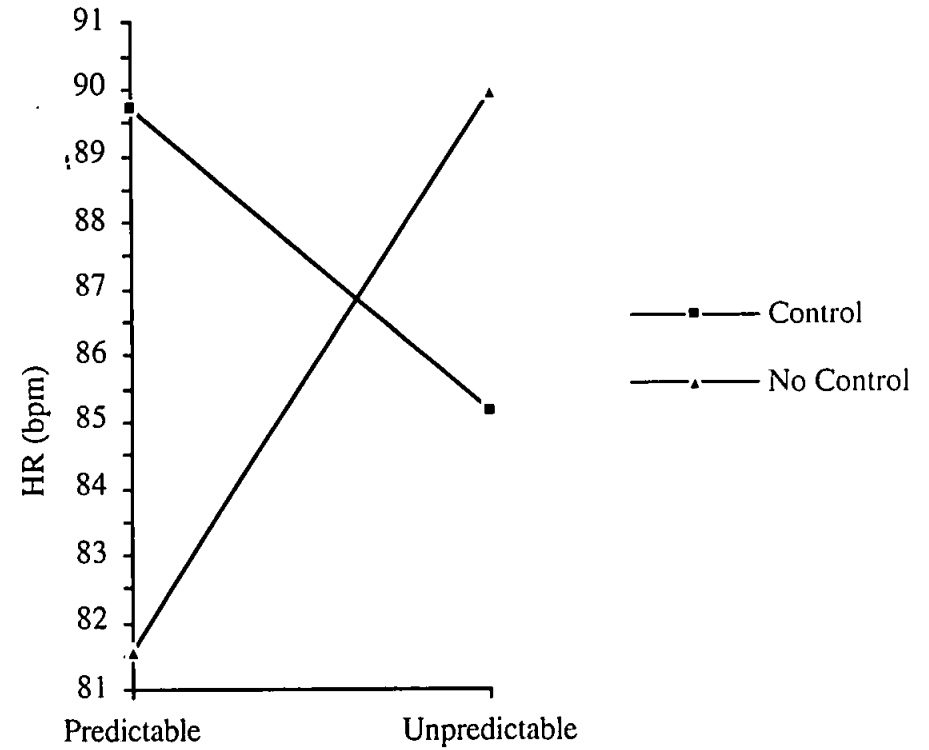
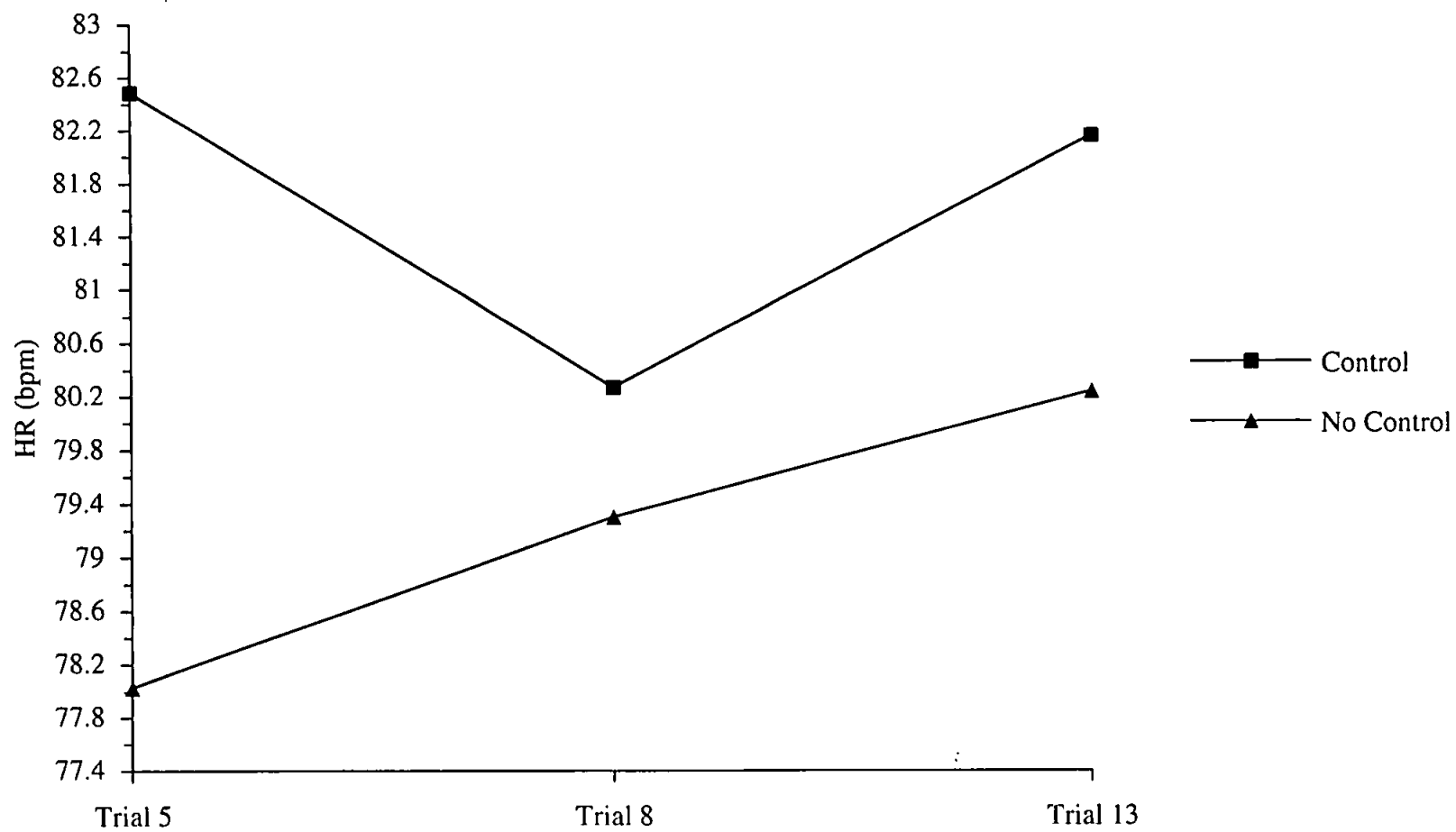


Figure 36. Average HR as a function of control and trials during Time Period 2.



control by trials interaction involving any of the measures (all  $F_s < 1$ ). However, there was a predictability by trials interaction for avHR ( $F(2, 94) = 5.7, p < .01$ ), mnHR ( $F(2, 101) = 4.4, p < .05$ ) and mxHR ( $F(2, 88) = 6.1, p < .01$ ). Analysis of the simple main effects for mnHR, revealed that the UP groups had significantly higher HR levels than the P groups during Trials 5 and 8, compared with no significant difference between the groups in Trial 13. Analysis of avHR and mxHR revealed that the UP groups produced significantly higher HR than the P groups across all three trials. Group means across trials are plotted in Figures 37, 38 and 39 for avHR, mnHR and mxHR respectively. There were no three-way interactions involving any of three HR measures (all  $F_s < 1$ ).

3.3.1.1.4. Time Period 4. There were no significant effects of control involving avHR ( $F(1, 52) = 2.3$ ), mnHR ( $F(1, 52) = 2.6$ ) or mxHR ( $F(1, 52) = 2$ ), nor were there any significant effects of predictability for avHR ( $F(1, 52) = 3.7$ ) or mxHR ( $F(1, 52) = 3$ ). However, there was a significant effect of predictability involving mnHR ( $F(1, 52) = 4.1, p < .05$ ). The UP groups had higher HR than the P groups (80.7 and 76.6 bpm respectively). There were significant control by predictability interactions involving avHR ( $F(1, 52) = 5.3, p < .05$ ), mnHR ( $F(1, 52) = 5, p < .05$ ) and mxHR ( $F(1, 52) = 5.2, p < .05$ ). Simple main effects for all three measures revealed a similar pattern of responding. The PNC group had markedly lower levels of activity than any other group, but only the PNC-UPNC comparison reached significance (see Figures 40, 41 and 42 for avHR, mnHR and mxHR respectively). There were no significant differences across trials for avHR or mnHR (both  $F_s < 1$ ). However, mxHR did reveal a significant difference across trials ( $F(2, 85) = 5.5, p < .01$ ). There were no control by trials interactions involving avHR ( $F(2, 93) = 2.6$ ), mnHR ( $F(2, 98) = 2$ ) or mxHR ( $F < 1$ ), although all three measures produced a predictability by trials interaction: avHR ( $F(2, 93) = 6.9, p < .01$ ), mnHR ( $F(2, 98) = 7.7, p < .01$ ) and mxHR ( $F(2, 85) = 3.2, p < .05$ ). Analysis of the simple main effects revealed a similar pattern across avHR and mxHR, with the UP groups producing significantly higher HR levels than the P groups across all three trials. MnHR revealed the UP groups to have significantly higher HR than the P groups across Trials 5 and 13, compared with no significant difference between the two groups in Trial 8 (see Figures 43, 44 and 45 for avHR, mnHR and mxHR respectively). There were no three-way interactions involving any of the HR measures (all  $F_s < 1$ ).



Figure 37. Average HR as a function of predictability and trials during Time Period 3.

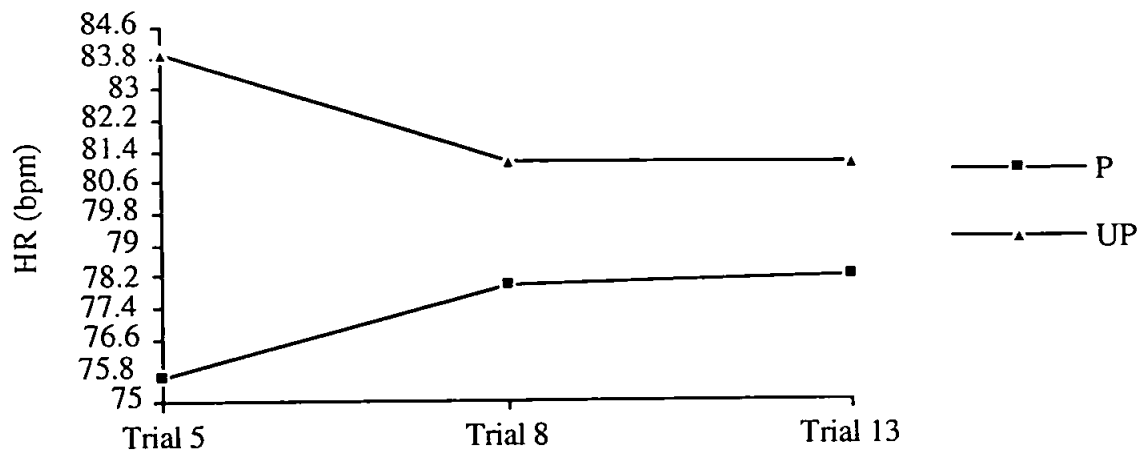


Figure 38. Mean minimum HR as a function of predictability and trials during Time Period 3.

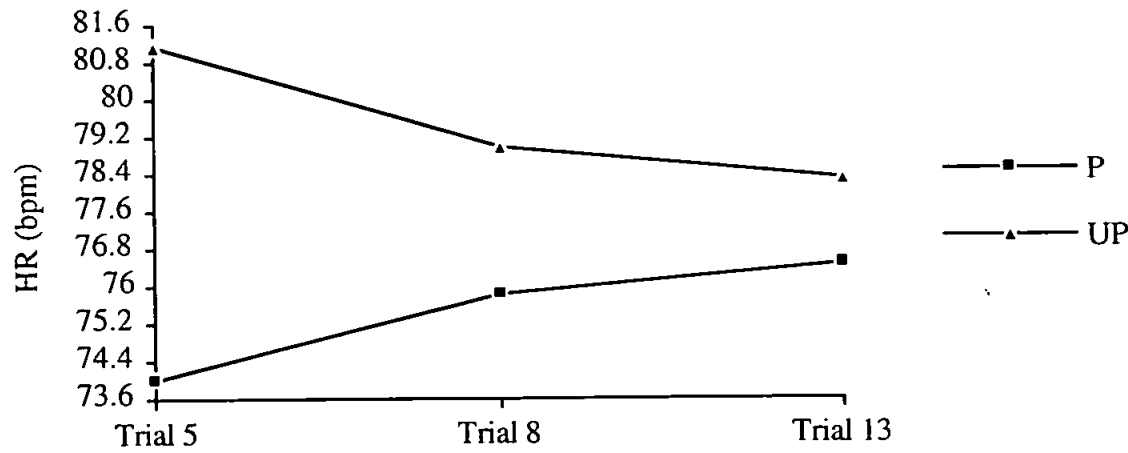


Figure 39. Mean maximum HR as a function of predictability and trials during Time Period 3.

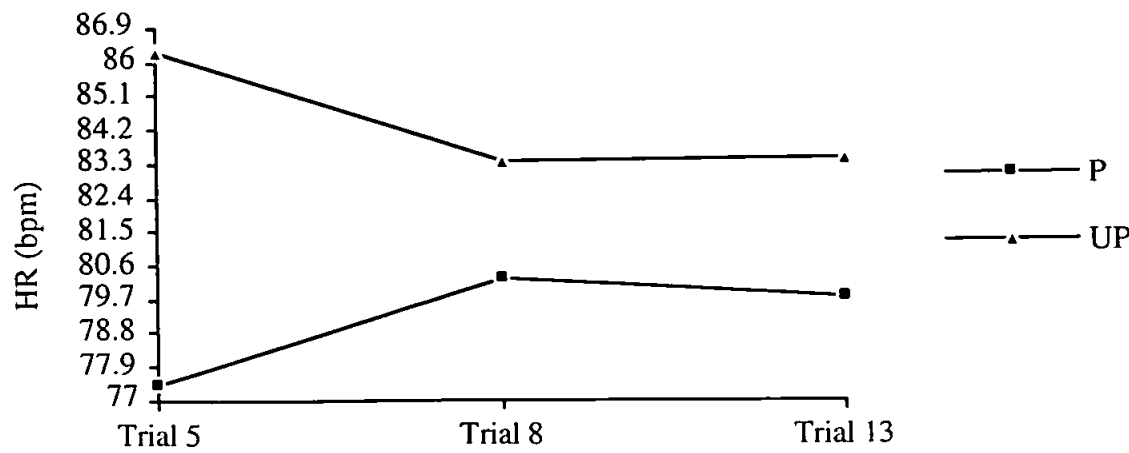


Figure 40. Average HR as a function of groups during Time Period 4.

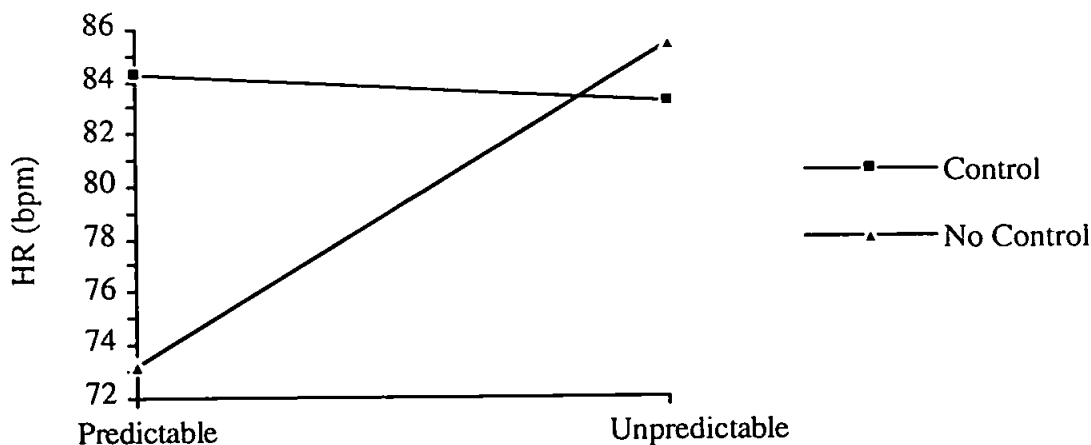


Figure 41. Mean minimum HR as a function of groups during Time Period 4.

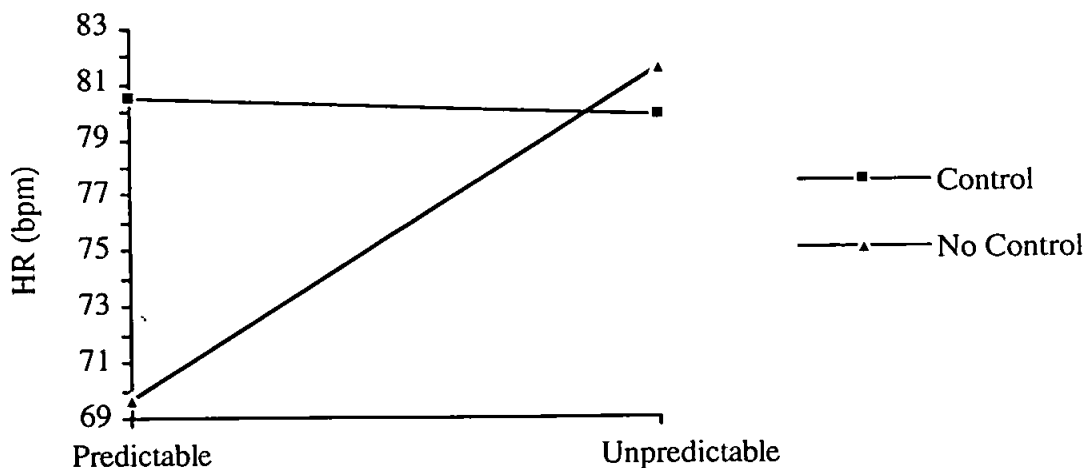


Figure 42. Mean maximum HR as a function of groups during Time Period 4.

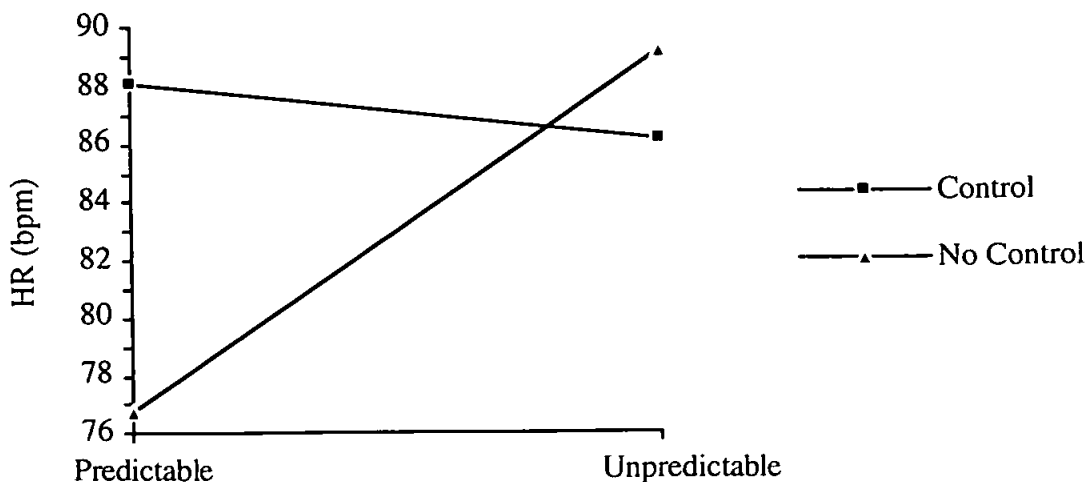


Figure 43. Average HR as a function of predictability and trials during Time Period 4.

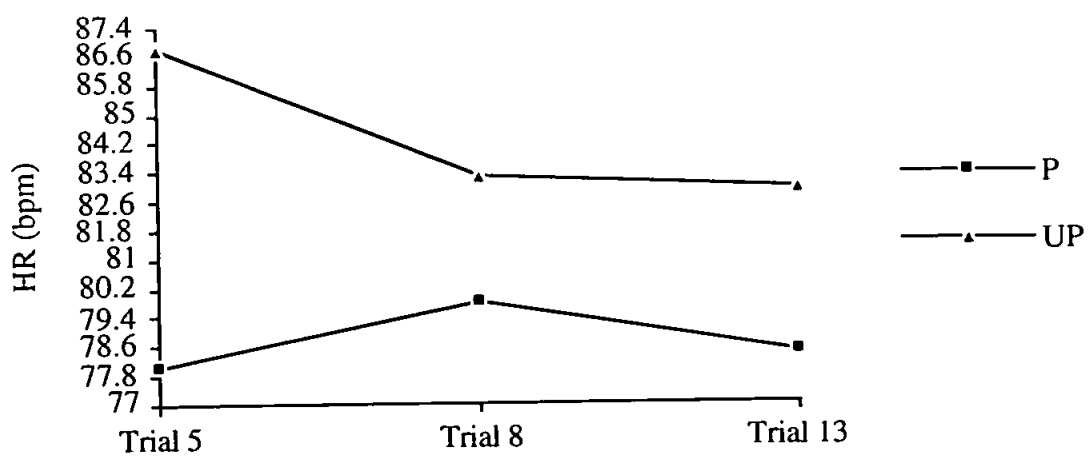


Figure 44. Mean minimum HR as a function of predictability and trials during Time Period 4.

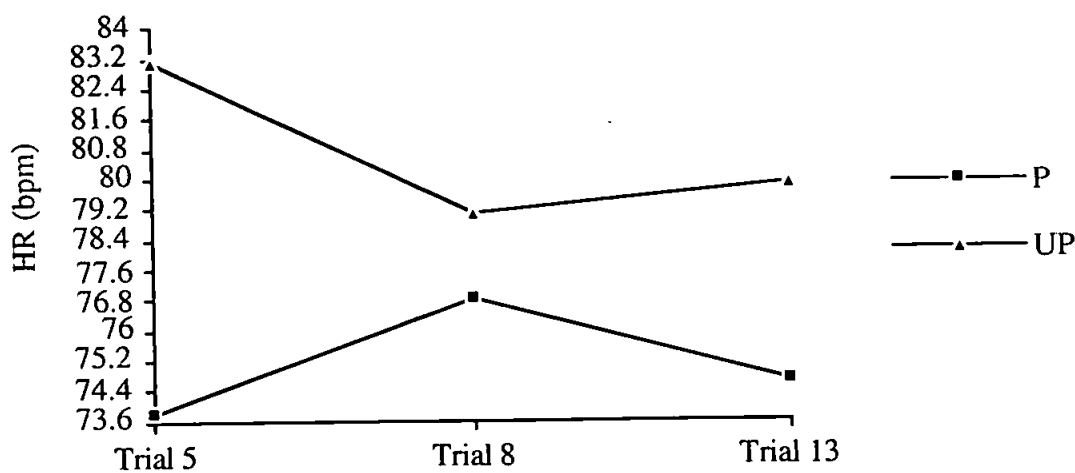
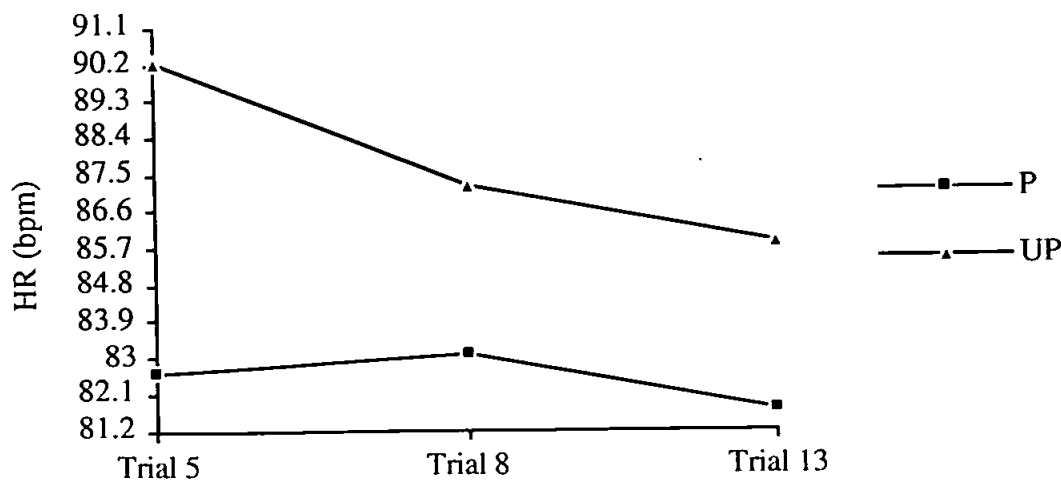


Figure 45. Mean maximum HR as a function of predictability and trials during Time Period 4.



3.3.1.1.5. Time Period 5. There were no significant effects of control or predictability involving avHR (all  $F$ s < 1). However, there was a control by predictability interaction ( $F(1, 52) = 4.1, p < .05$ ). In addition, avHR produced a significant difference across trials ( $F(2, 93) = 4.1, p < .05$ ) and a control by trials interaction ( $F(2, 93) = 4.6, p < .05$ ), but no predictability by trials ( $F(2, 93) = 2$ ) or three-way ( $F(2, 93) = 1.6$ ) interaction. There was a significant difference across time ( $F(4, 194) = 5.5, p < .01$ ), a control by predictability by time interaction ( $F(4, 194) = 2.6, p < .05$ ) and a four-way control by predictability by trials by time interaction ( $F(6, 323) = 2.6, p < .05$ ). Analysis of the simple main effects revealed (Figure 46), firstly for Trial 5, that the PC, UPC and UPNC groups had higher HR than the PNC group across the 25 sec of the ITI. The PC group also produced higher HR levels than the UPC group during the 10-15, 15-20 and 20-25 sec periods, and higher HR than the UPNC group during the 5-10 and 15-20 sec periods. The UPNC group produced greater levels of activity than the UPC group during the 10-15 and 20-25 sec periods. During Trial 8, the PC, UPC and UPNC groups produced higher HR than the PNC group across the 25 sec of the ITI. The PC group also revealed higher HR levels than the UPC group over all 25 sec, and higher HR than the UPNC group during the 5-10 and 20-25 sec periods. The UPNC group produced greater levels of activity compared with the UPC group during the 10-15 and 15-20 sec periods. During Trial 13, the PC and UPNC groups produced higher HR than the PNC group over the 25 sec, compared with the UPC group which produced higher HR than the PNC group during the 0-5, 5-10, 10-15 and 20-25 sec periods. The PC and UPNC groups produced higher HR than the UPC groups across the 25 sec. All of these comparisons were statistically significant.

MnHR revealed no significant effect of control ( $F(1, 52) = 1.7$ ) or predictability ( $F < 1$ ), nor a control by predictability interaction ( $F(1, 52) = 3.3$ ). There was no significant difference across trials or a predictability by trials interaction (all  $F$ s < 1). However, there was a control by trials interaction ( $F(2, 95) = 3.3, p < .05$ ). There was no significant difference across time, nor any control by time or predictability by time interactions (all  $F$ s < 1). MnHR did reveal a control by predictability by trials by time interaction ( $F(6, 297) = 2.7, p < .05$ ). Simple main effects revealed (Figure 47), firstly for Trial 5, that the PC, UPC and UPNC groups had higher HR than the PNC group over the 25 sec of the

Figure 46. Average HR as a function of groups, trials and time during Time Period 5.

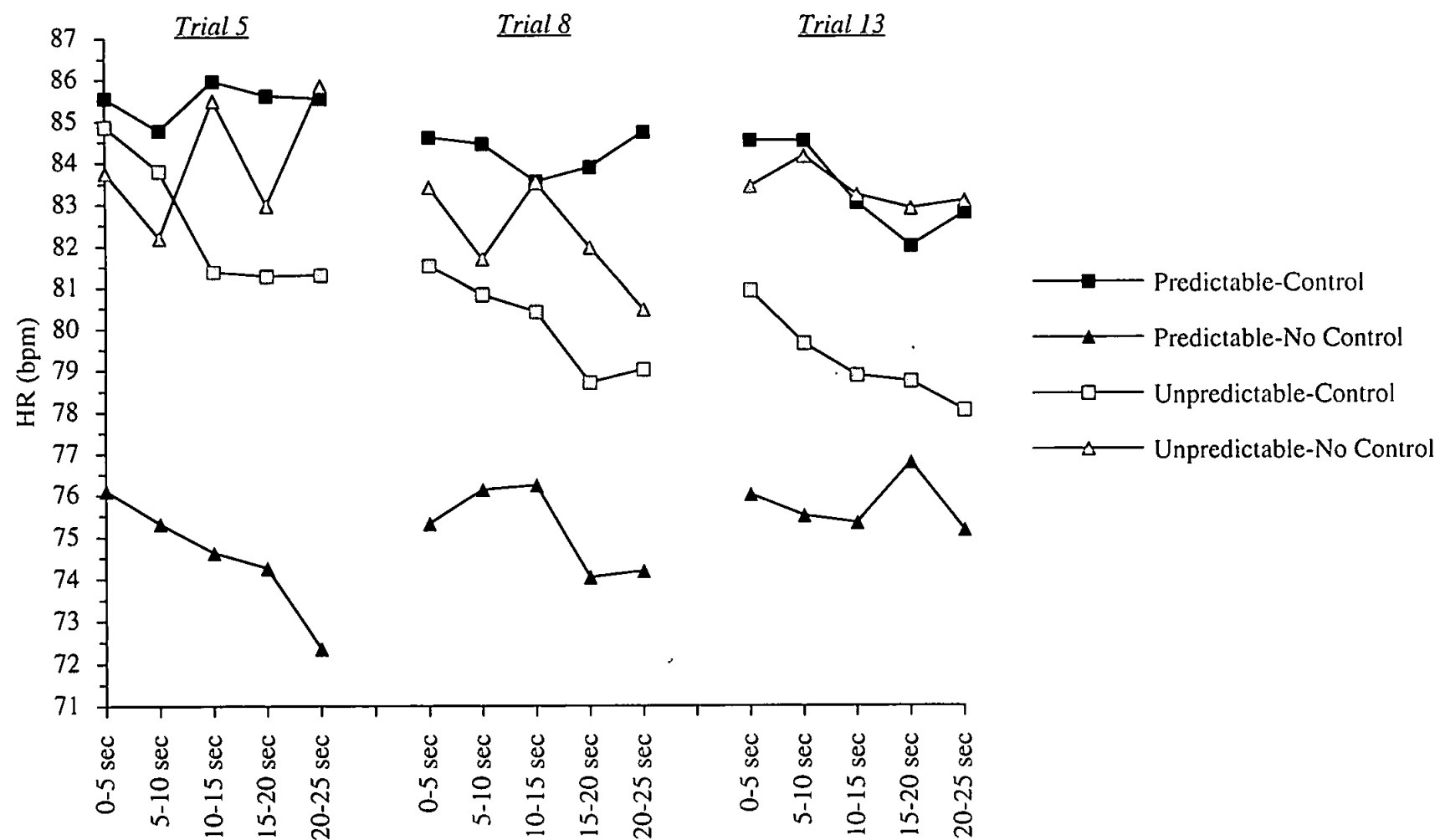
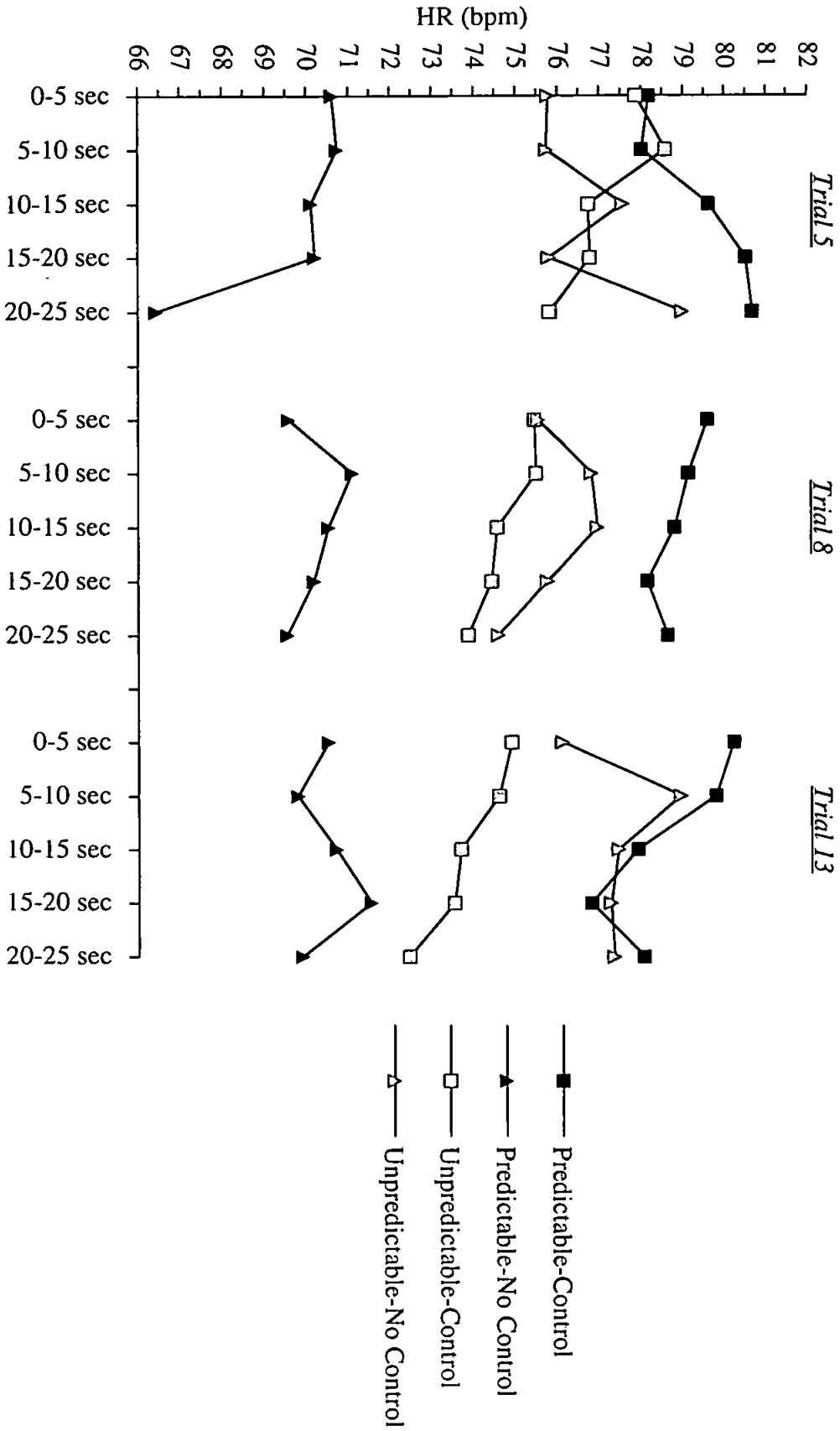


Figure 47. Mean minimum HR as a function of groups, trials and time during Time Period 5.



ITI. The PC group also revealed higher HR levels than the UPC group during the 10-15, 15-20 and 20-25 sec periods, and higher HR than the UPNC group during the 0-5, 5-10 and 15-20 sec periods. During the 5-10 sec period the UPC group produced higher HR than the UPNC group, compared with the 20-25 sec period in which the reverse occurred. During Trial 8, the PC, UPC and UPNC groups produced higher levels of responding than the PNC group over the 25 sec. The PC group also produced higher HR than the UPC group over all five sec periods, and higher HR levels than the UPNC group during the 0-5, 5-10, 15-20 and 20-25 sec periods. During the 10-15 sec period the UPNC group produced higher HR than the UPC group. During Trial 13, the PC, UPC and UPNC groups produced greater levels of activity than the PNC group over all 25 sec. The PC group also produced higher HR than the UPC group over the 25 sec, and higher HR than the UPNC group during the 0-5 sec period. The UPNC group produced higher HR levels than the UPC group during the 5-10, 10-15, 15-20 and 20-25 sec periods. All of these comparisons reached significance.

MxHR revealed no significant main effects of control or predictability (both  $F_s < 1$ ). However, there was a control by predictability interaction ( $F(1, 52) = 4.1, p < .05$ ). Simple main effects revealed that the PC and UPNC groups produced markedly higher HR than the PNC group, and to a smaller extent higher HR than the UPC group. The UPC group produced higher HR levels than the PNC group. However, none of these comparisons reached significance (Figure 48). There was a significant difference across trials ( $F(2, 98) = 5.8, p < .01$ ), a predictability by trials interaction ( $F(2, 98) = 4, p < .05$ ), but no control by trials ( $F(2, 98) = 2.3$ ) or three-way ( $F(2, 98) = 1.9$ ) interaction. Analysis of the simple main effects for the predictability by trials interaction revealed that the UP groups produced significantly higher HR levels than the P groups during Trial 5, compared with no significant difference between the two groups in later trials (Figure 49). There was a highly significant difference across time involving mxHR ( $F(3, 167) = 11.1, p < .001$ ), due to the 0-5 sec period producing significantly higher HR than the remaining periods. There were no significant differences between the 5-10, 10-15, 15-20 and 20-25 sec periods (88.3, 86.5, 86.6, 85.4 and 85.9 bpm for the 0-5, 5-10, 10-15, 15-20 and 20-25 sec periods respectively). There were no other interactions involving either trials or time for mxHR (all  $F_s < 1$ ).

Figure 48. Mean maximum HR as a function of groups during Time Period 5.

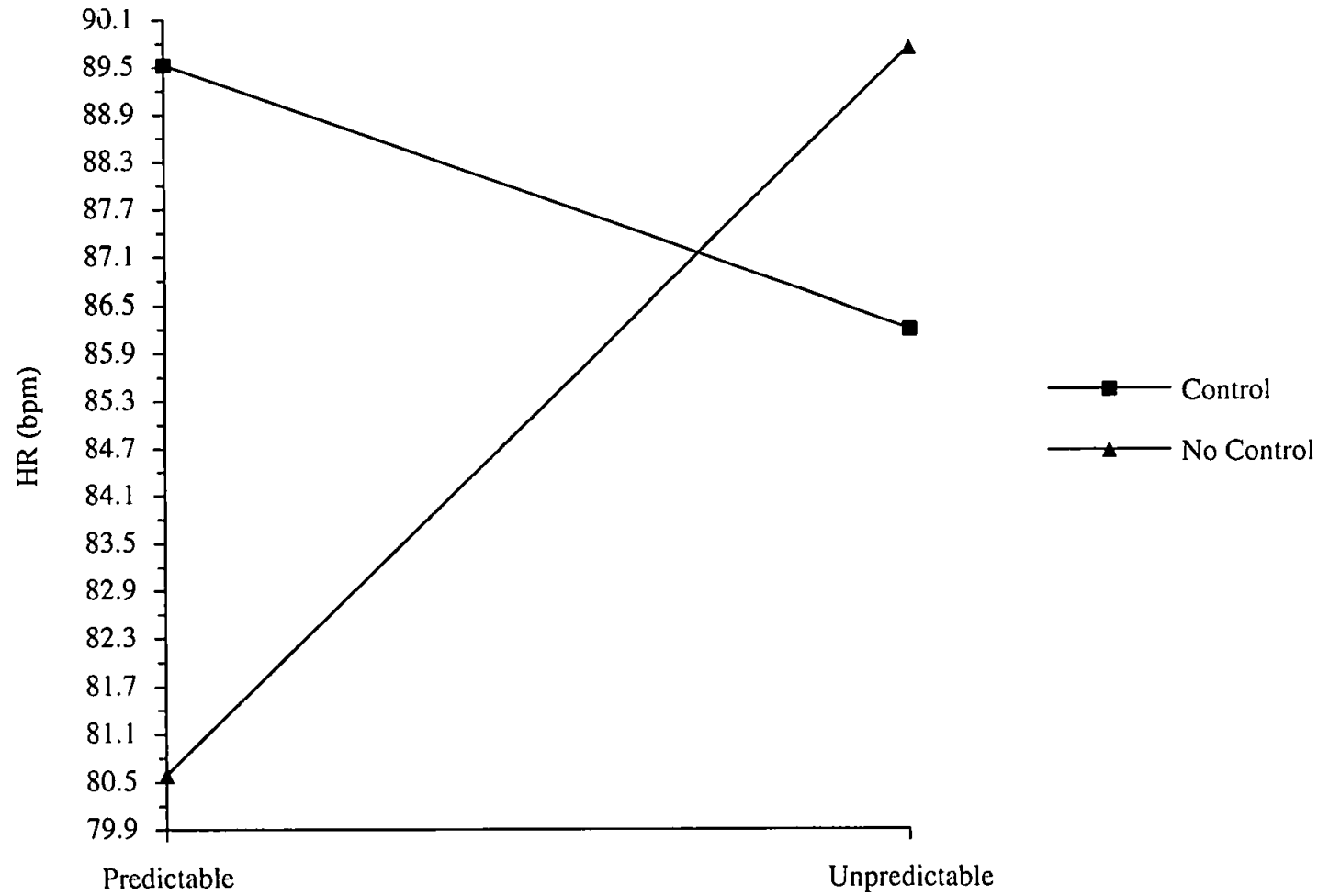
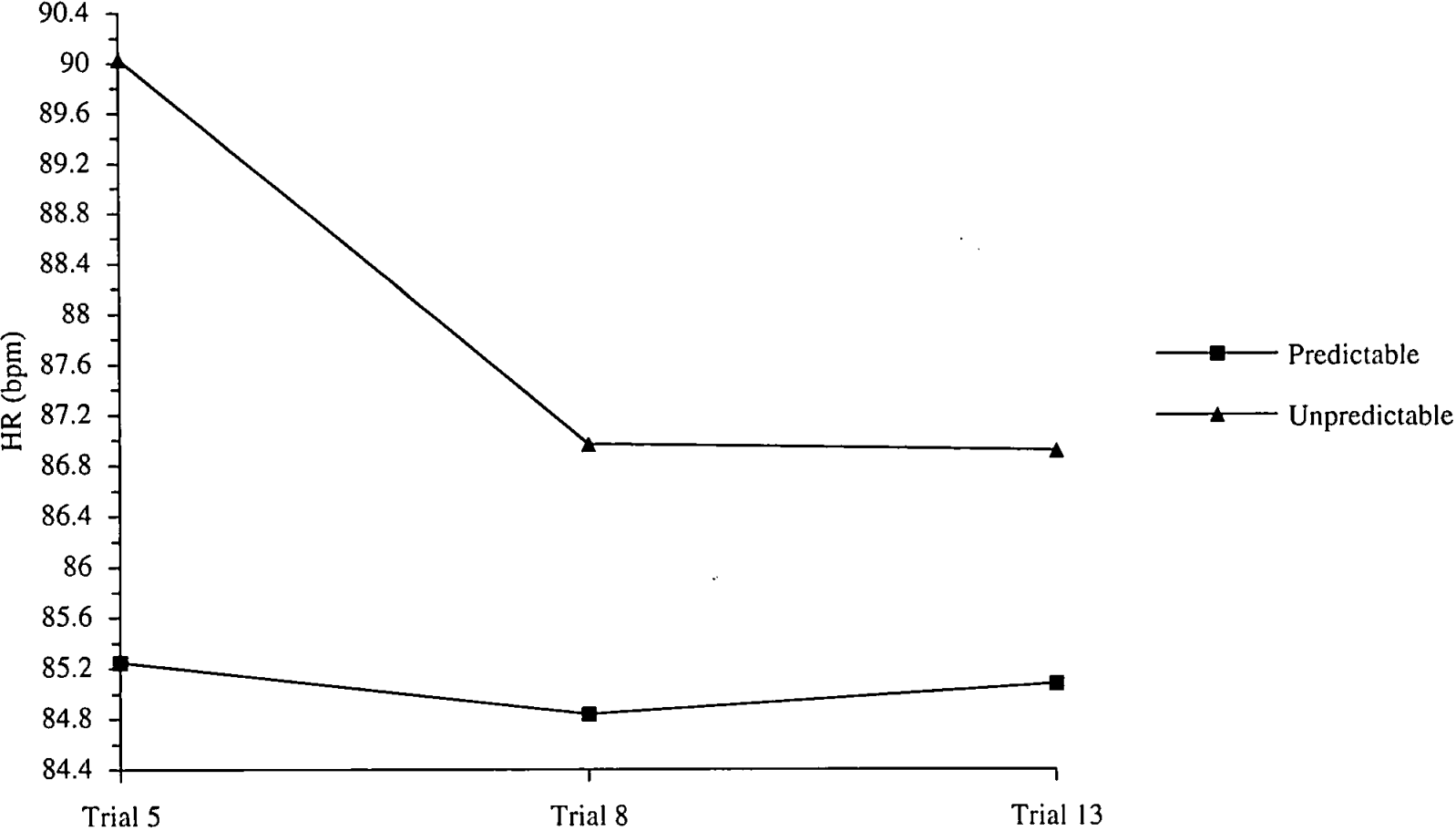




Figure 49. Mean maximum HR as a function of predictability and trials during Time Period 5.



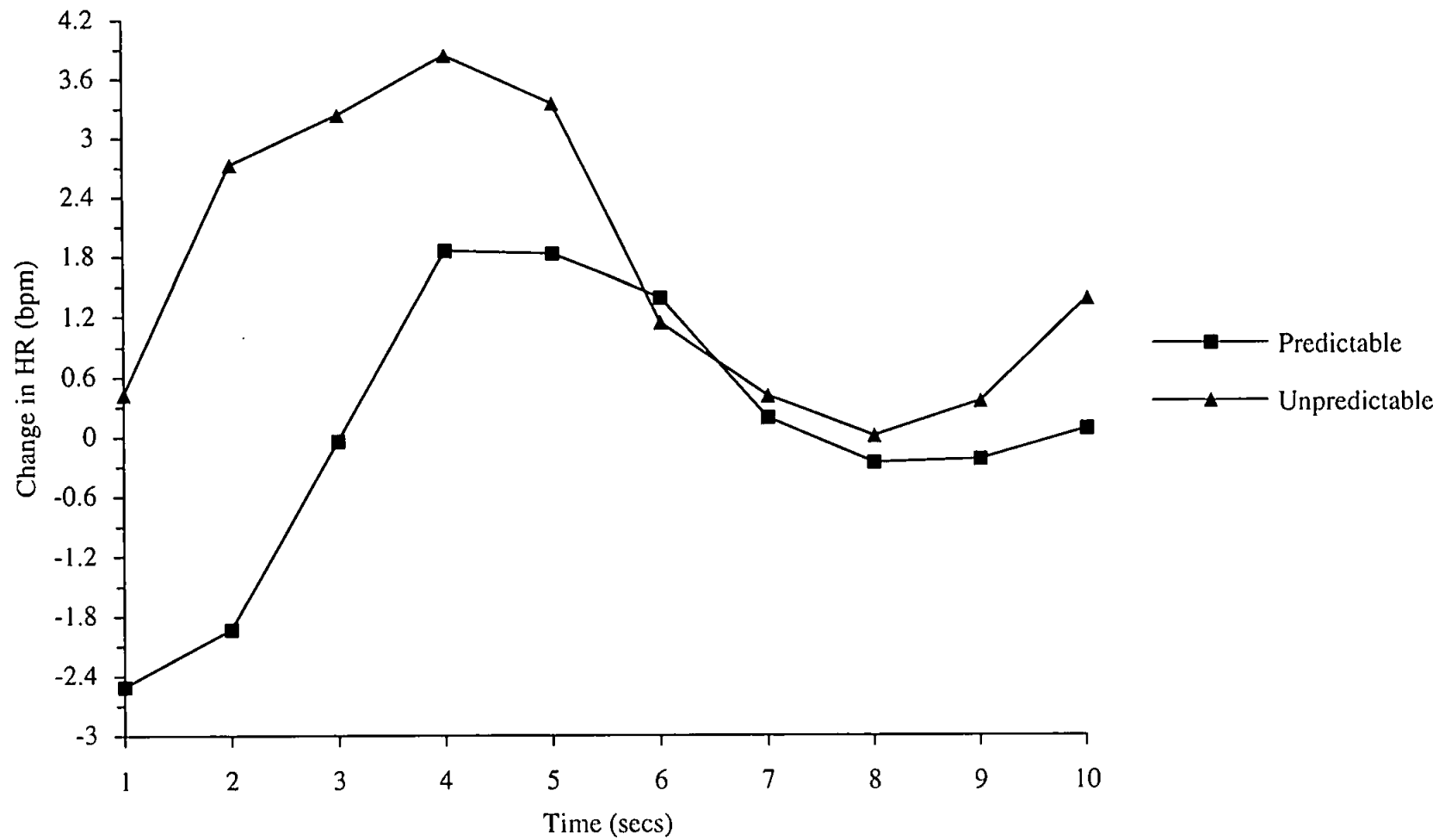
3.3.1.1.6. Time Period 6. There was a significant effect of predictability involving mean change in avHR ( $F(1, 52) = 4.1, p < .05$ ). The UP groups produced a greater increase in avHR than the P groups (1.69 and 0.038 change in bpm respectively). There was no significant effect of control ( $F(1, 52) = 3.4$ ), nor any control by predictability interaction ( $F < 1$ ). There was a significant difference across trials ( $F(2, 100) = 3.5, p < .05$ ), due to a significant decrement in levels of activity across trials (1.65, 1.14 and -0.26 change in bpm for Trials 5, 8 and 13 respectively). There were no control by trials ( $F(2, 100) = 1.9$ ), predictability by trials ( $F(2, 100) = 2.9$ ) or three-way ( $F(2, 100) = 2.1$ ) interactions. There was a significant difference across time ( $F(4, 199) = 7.9, p < .01$ ) and a predictability by time interaction ( $F(4, 199) = 3.5, p < .05$ ). Analysis of the simple main effects revealed that the UP groups had significantly greater change in avHR than the P groups during the 1-3 sec after noise onset. However, during the 4-10 sec there were no significant differences between the two groups (Figure 50). There were no other interactions involving either trials or time (all  $F$ s  $< 1$ ).

3.3.1.1.7. Summary. There were significant predictability by control interactions for all three measures during the warning stimulus (Time Period 1), and for avHR and mxHR during the ISI (Time Period 2). The PNC, and to a lesser extent the UPC group, had lower HR levels than the PC and UPNC groups. However, none of these comparisons reached significance. In addition, the C groups produced markedly higher mxHR than the NC groups on Trials 8 and 13, during Time Period 1. During Time Period 2, the C groups had higher avHR levels than the NC groups, although this reached significance during Trial 5 only.

During Time Period 3, the UP groups produced higher HR than the P groups across Trials 5 and 8 (mnHR) and Trials 5, 8 and 13 (avHR and mxHR). During Time Period 4, the UP groups had higher HR levels than the P groups in Trials 5 and 13 (mnHR) and Trials 5, 8 and 13 (avHR and mxHR). In addition, during Time Period 4, the PC, UPC and UPNC groups had higher HR levels than the PNC group (in all three measures), although only the latter comparison was significant.

During Time Period 5, the PC, UPC and UPNC groups produced significantly higher HR (avHR and mnHR) than the PNC group across the ITI of all three trials. The PC group had higher HR than the UPC group across the 10-15, 15-20 and 20-25 sec

Figure 50. Mean change in average HR as a function of predictability and time during Time Period 6.



periods of Trial 5 and across all 25 sec of Trials 8 and 13. The PC group also had higher HR than the UPNC groups during during Trials 5 and 8 but not during Trial 13. AvHR revealed the UPNC group to have greater levels of activity than the UPC group during selected periods of Trials 5 and 8, and across all 25 sec of Trial 13 whereas, for mnHR the UPNC group had higher HR than the UPC during Trial 13 only. With mxHR, the effects were less pronounced. The PC, UPC and UPNC groups showed similar HR levels to each other and, produced higher HR than the PNC group. However, these comparisons were not statistically significant. In addition, mxHR produced a predictability by trials interaction. The UP groups had higher HR than the P groups, although this difference was only significant during Trial 5.

During the cardiac response profile (Time Period 6), there was a predictability by time interaction due to the UP groups producing a significantly greater increase in HR during the 1-3 sec after noise onset.

3.3.1.2. Corrected HR. The results involving corrected HR were similar to those of uncorrected HR, unless otherwise stated.

3.3.1.2.1. Time Period 1. There were no significant control by predictability interactions involving avHR or mxHR (both  $F_s < 1$ ). MnHR did produce a two-way interaction ( $F(1, 52) = 8.6, p < .01$ ), although further analysis revealed a different pattern of responding than for uncorrected mnHR. That is, the PC and UPNC groups had significantly greater increases in HR from the pre-task baseline compared to the UPC group, and to a smaller extent the PNC group. The PNC group also had greater HR increases compared to the UPC group, although this difference did not reach significance (Figure 51). MxHR produced a control by trials ( $F(2, 96) = 3.2, p < .05$ ) interaction. However, as seen previously, analysis of the simple main effects revealed a different pattern of responding than uncorrected mxHR. The NC groups had smaller HR decreases from the baseline compared to the C groups across all trials, although the difference between groups reached significance during Trials 5 and 8 only (Figure 52).

3.3.1.2.2. Time Period 2. There was a significant main effect of control involving avHR ( $F(1, 52) = 4.1, p < .05$ ) and mnHR ( $F(1, 52) = 4.2, p < .05$ ). The NC groups produced a smaller decrease in avHR and a greater increase in mnHR compared with the C

Figure 51. Mean change in minimum HR as a function of groups during Time Period 1.

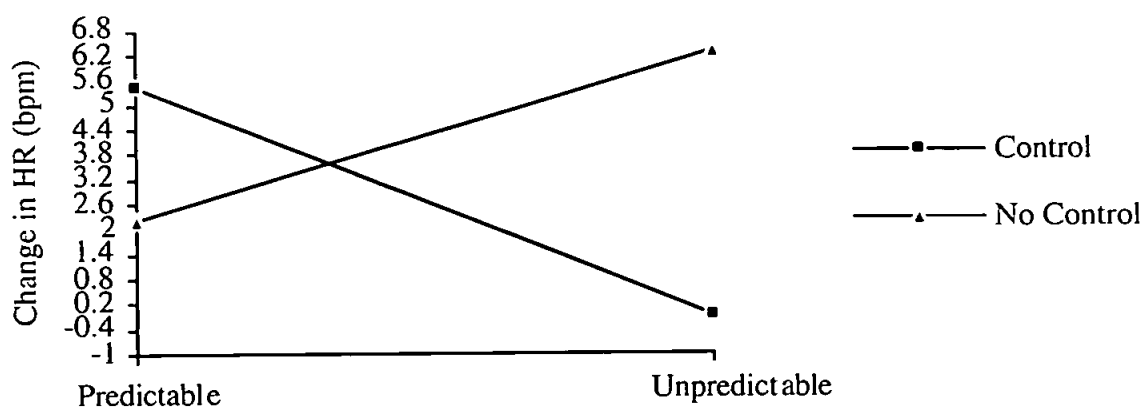


Figure 52. Mean change in maximum HR as a function of control and trials during Time Period 1.

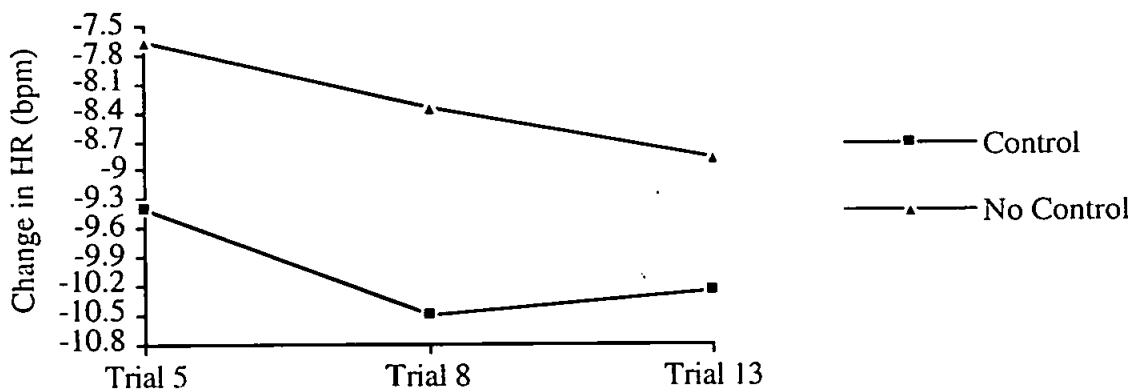
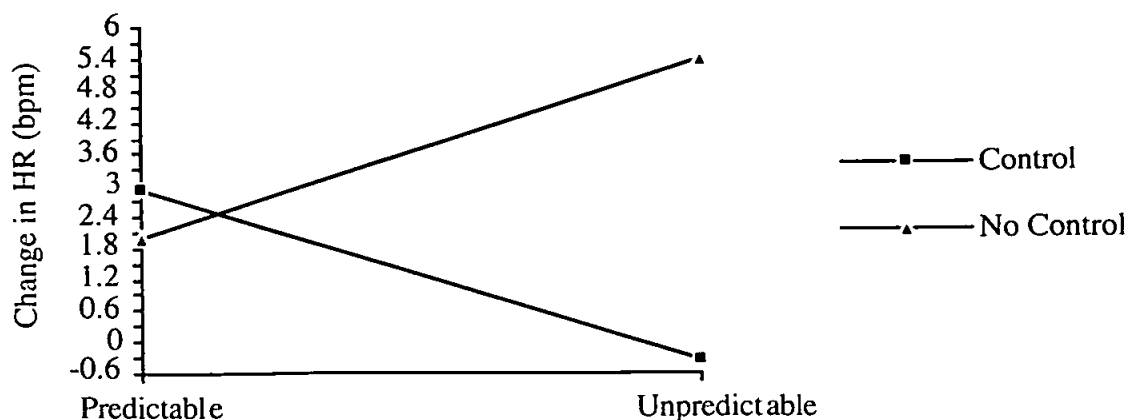


Figure 53. Mean change in minimum HR as a function of groups during Time Period 2.



groups (avHR = -1.77 and -3.87 change in bpm; mnHR = 3.68 and 1.29 change in bpm respectively). There was also a significant control by predictability interaction involving mnHR ( $F(1, 52) = 7.4, p < .01$ ). The PC and UPNC groups produced significantly greater increases in HR than the UPC group. The UPNC group also had greater HR increases compared to the PC and PNC groups, although this failed to reach significance (Figure 53). There were no significant control by predictability interactions involving avHR or mxHR (both  $F_s < 1$ ). AvHR produced a significant control by trials ( $F(2, 103) = 3.1, p < .05$ ) interaction. However, analysis of the simple main effects revealed a different pattern of responding than uncorrected avHR. The NC groups had significantly smaller HR decreases from the pre-task baseline compared to the C groups during Trials 8 and 13. There were no significant differences during Trial 5 (Figure 54).

3.3.1.2.3. Time Period 3. There was a significant control by predictability interaction involving mnHR ( $F(1, 52) = 4.3, p < .05$ ). The UPNC group produced greater increases in HR than the PNC, UPC and PC groups, but only the first two comparisons reached significance (Figure 55). In addition, all three measures produced a predictability by trials interaction; avHR ( $F(2, 94) = 5.7, p < .01$ ), mnHR ( $F(2, 101) = 4.4, p < .05$ ) and mxHR ( $F(2, 88) = 6.1, p < .01$ ). As seen previously, analysis of the simple main effects revealed a different pattern of responding than for uncorrected HR, with significantly greater increases (mnHR) or smaller decreases (avHR & mxHR) in UP compared to P groups during Trial 5. There were no significant differences in Trials 8 or 13 (see Figures 56, 57 & 58 for avHR, mnHR and mxHR respectively).

3.3.1.2.4. Time Period 4. There were significant main effects of predictability involving avHR ( $F(1, 52) = 4.6, p < .05$ ) and mnHR ( $F(1, 52) = 4.7, p < .05$ ). The UP groups produced smaller decreases in avHR and greater increases in mnHR than the P groups (avHR = -0.32 and -3.09 change in bpm; mnHR = 7.2 and 3.98 change in bpm respectively). There were no control by predictability interactions involving avHR or mxHR (both  $F_s < 1$ ). However, mnHR did produce a two-way interaction ( $F(1, 52) = 8.8, p < .01$ ), although analysis of the simple main effects revealed a different pattern of responding than uncorrected mnHR. That is, the UPNC group had significantly greater increases in HR from the pre-task baseline compared to the remaining three groups. The PC and UPC also had greater HR increases compared to the PNC group, although this

Figure 54. Mean change in average HR as a function of control and trials during Time Period 2.

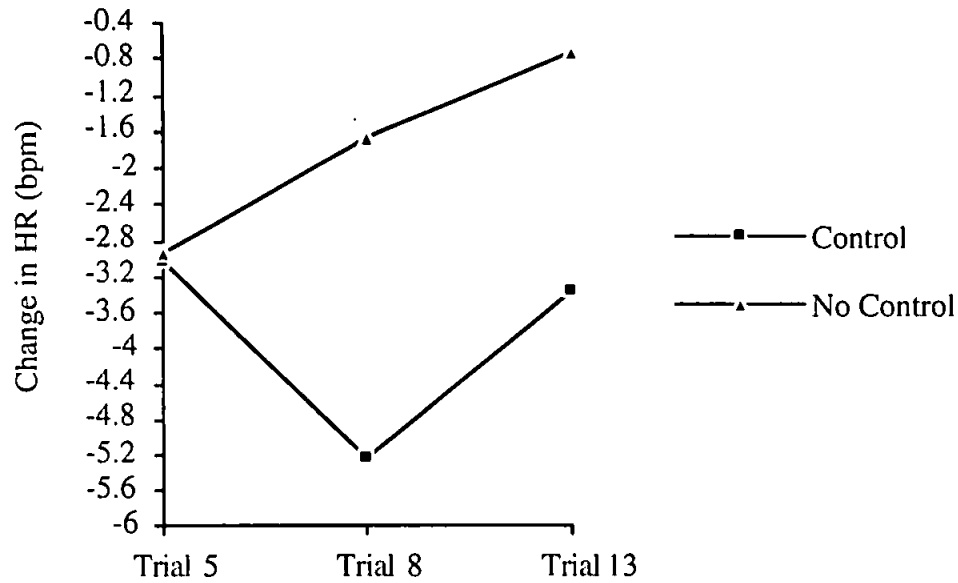


Figure 55. Mean change in minimum HR as a function of groups during Time Period 3.

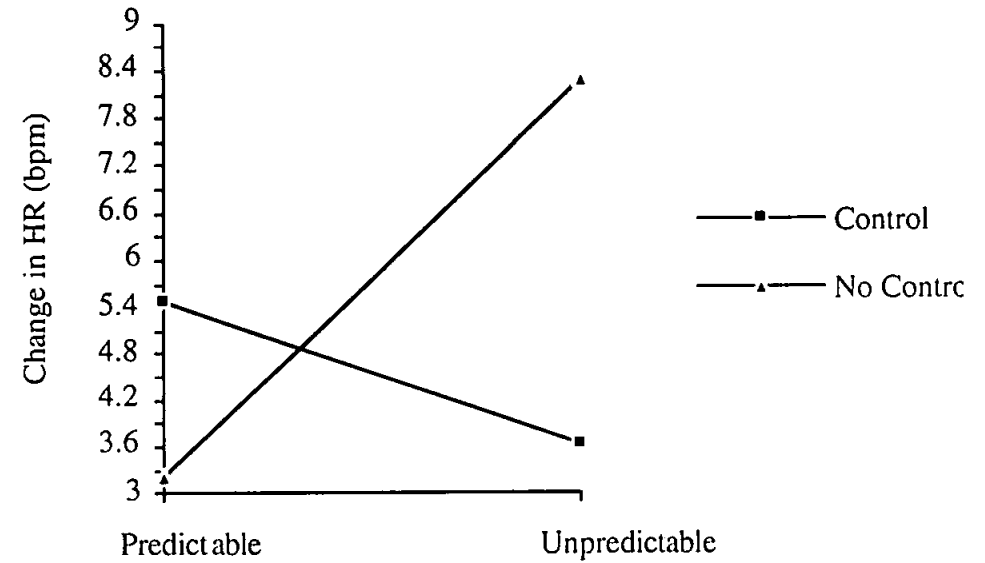


Figure 56. Mean change in average HR as a function of predictability and trials during Time Period 3.

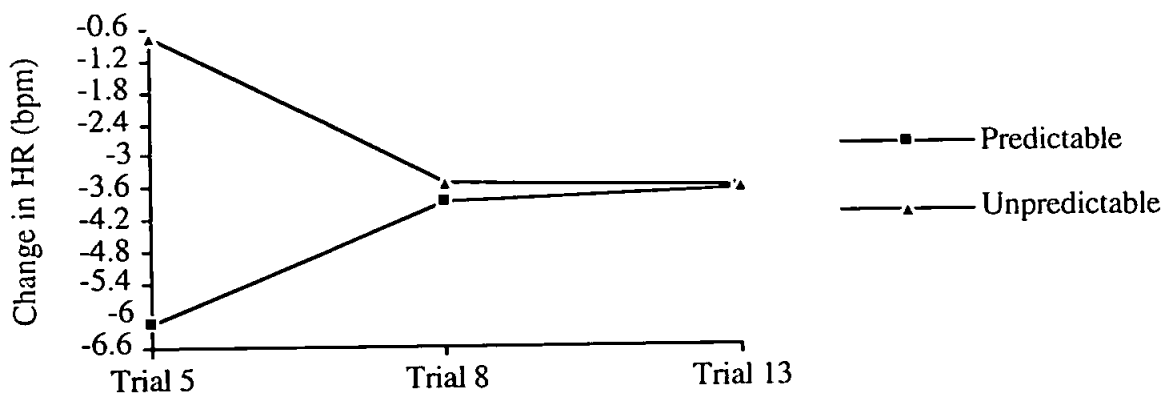


Figure 57. Mean change in minimum HR as a function of predictability and trials during Time Period 3.

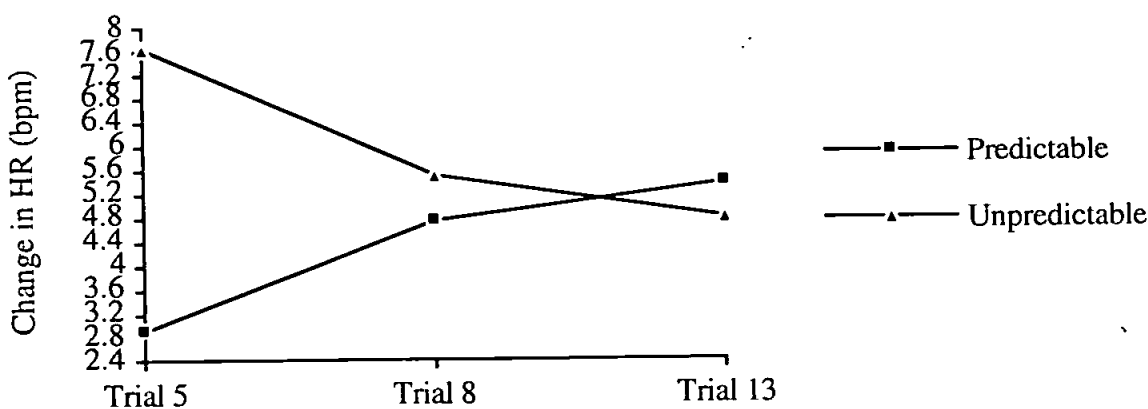


Figure 58. Mean change in maximum HR as a function of predictability and trials during Time Period 3.

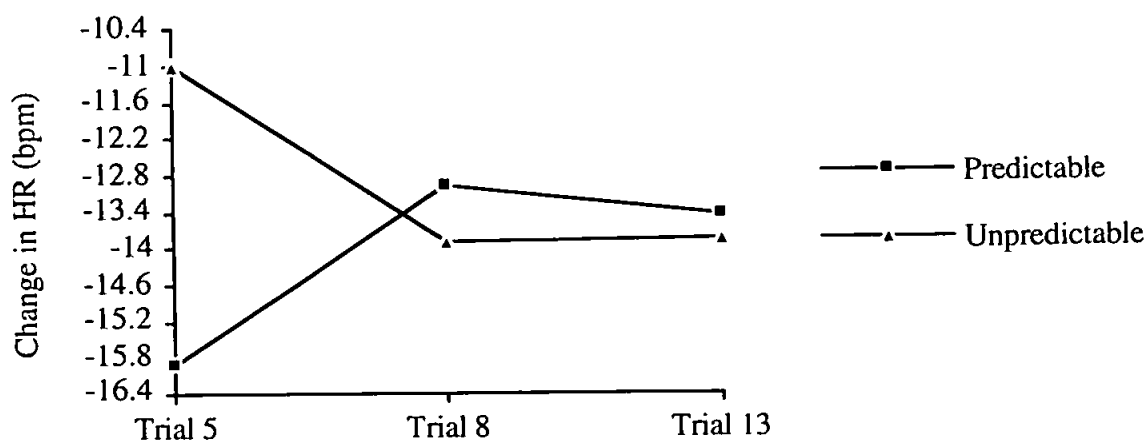
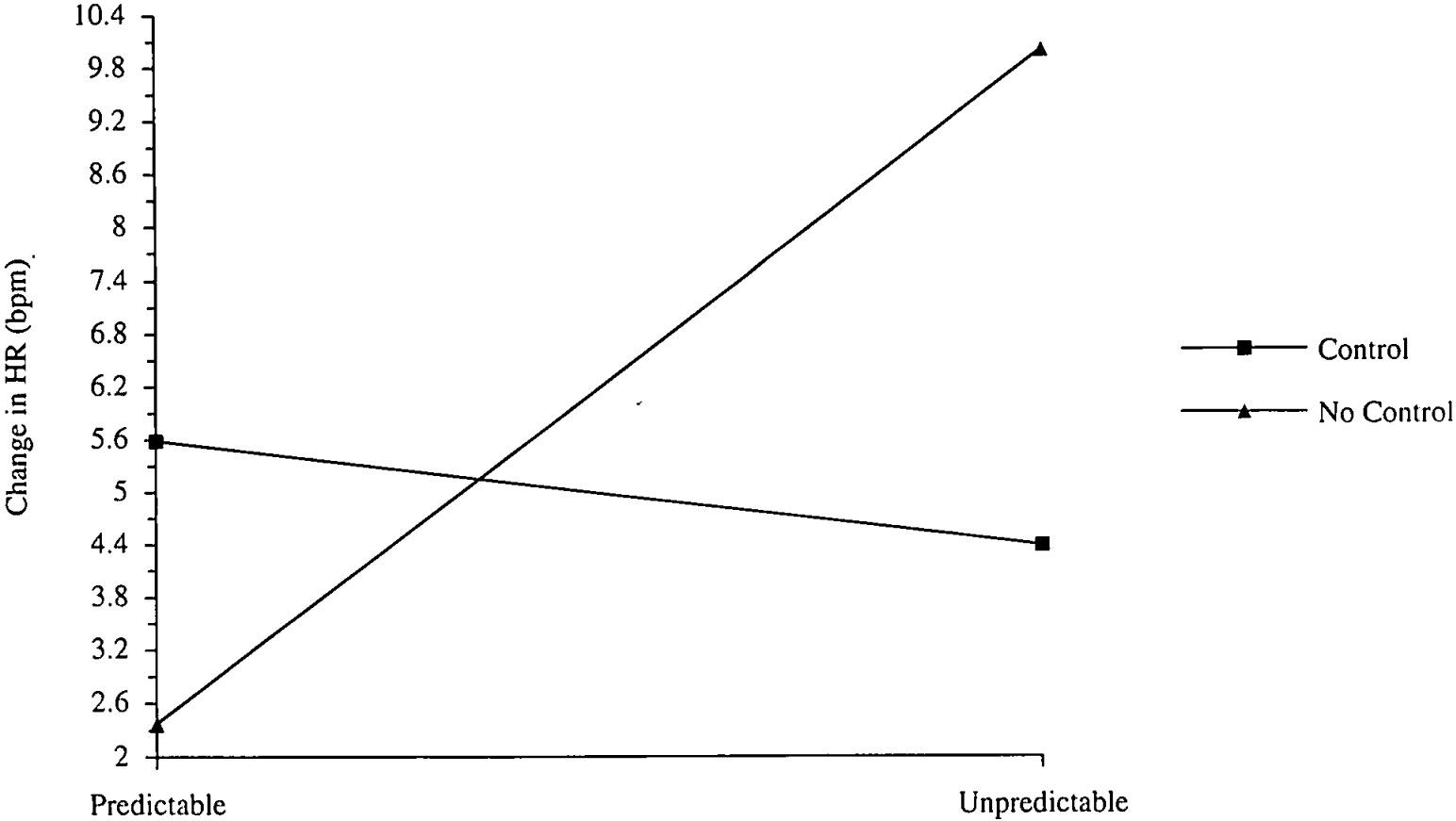




Figure 59. Mean change in minimum HR as a function of groups during Time Period 4.



failed to reach significance (Figure 59). In addition, all three measures produced a predictability by trials interaction; avHR ( $F(2, 93) = 6.9, p < .01$ ), mnHR ( $F(2, 98) = 7.7, p < .01$ ) and mxHR ( $F(2, 85) = 3.2, p < .05$ ). However, as before analysis of the simple main effects revealed a different pattern of responding than for uncorrected HR, with the UP groups producing significantly greater increases (avHR & mnHR) or smaller decreases (mxHR) compared to the P groups during Trial 5. There were no significant differences between the groups during later trials (see Figures 60, 61 & 62 for avHR, mnHR and mxHR respectively).

3.3.1.2.5. Time Period 5. There were no three (avHR) or four-way (avHR & mnHR) interactions during this period (all  $F_s < 1$ ). AvHR and mxHR produced no predictability by control interactions (both  $F_s < 1$ ), although there was a two-way interaction involving mnHR ( $F(1, 52) = 6.1, p < .05$ ). Analysis of the simple main effects revealed that the UPNC group had greater increases in HR compared to the PC, PNC and UPC groups, although only the latter comparison reached significance. The PC and PNC groups also had greater HR increases compared to the UPC group, although this difference was not significant (see Figure 63). There were significant control by trials interactions involving both avHR ( $F(2, 93) = 4.6, p < .05$ ) and mnHR ( $F(2, 95) = 3.3, p < .05$ ). However, analysis of the simple main effects revealed a different pattern of responding than for uncorrected HR. For avHR, the NC groups produced smaller decreases during Trials 8 and 13 compared to the C groups, although only the latter comparison reached significance (Figure 64). Simple main effects for the mnHR interaction revealed greater increases in the NC groups during Trials 8 and 13. There were no significant differences during Trial 5 (Figure 65). There was a significant predictability by trials interaction involving mxHR ( $F(2, 98) = 4, p < .05$ ). However, as seen previously, simple main effects revealed a different pattern of responding than for uncorrected HR. That is, the UP groups produced smaller HR decreases from the pre-task baseline compared to the P groups in Trials 8 and 13, although only the latter comparison reached significance (Figure 66).

3.3.1.2.6. Corrected HR Summary. During the warning stimulus, the ISI, the 1-3 sec after noise onset and the ITI (Time Periods 1, 2, 4 and 5), there were no predictability by control interactions involving corrected avHR or mxHR. Further inspection of means

Figure 60. Mean change in average HR as a function of predictability and trials during Time Period 4.

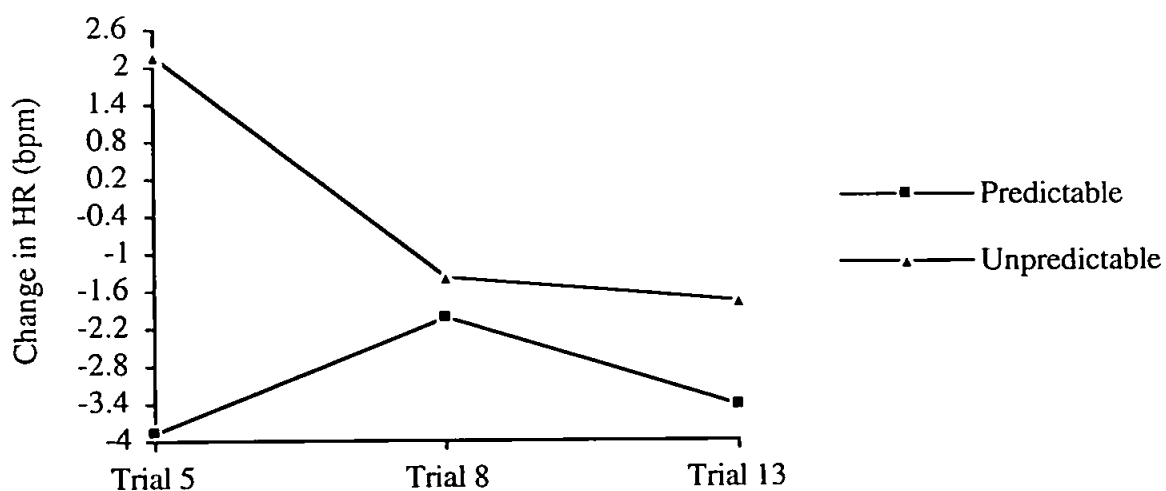


Figure 61. Mean change in minimum HR as a function of predictability and trials during Time Period 4.

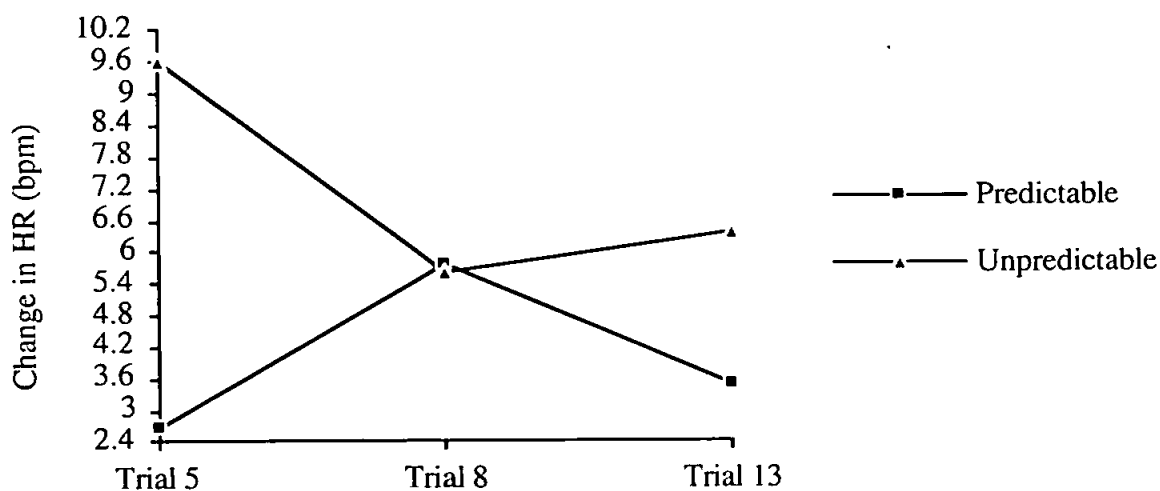


Figure 62. Mean change in maximum HR as a function of predictability and trials during Time Period 4.

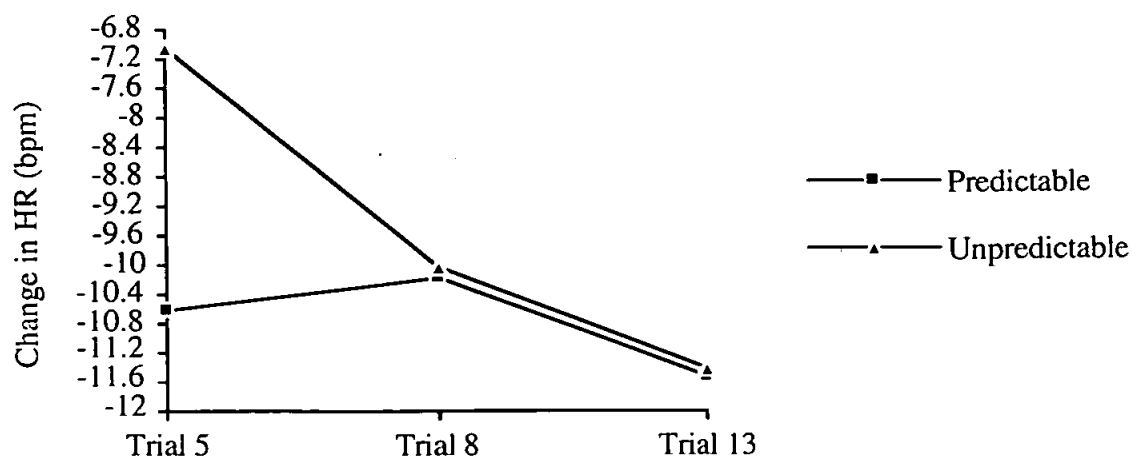


Figure 63. Mean change in average HR as a function of groups during Time Period 5.

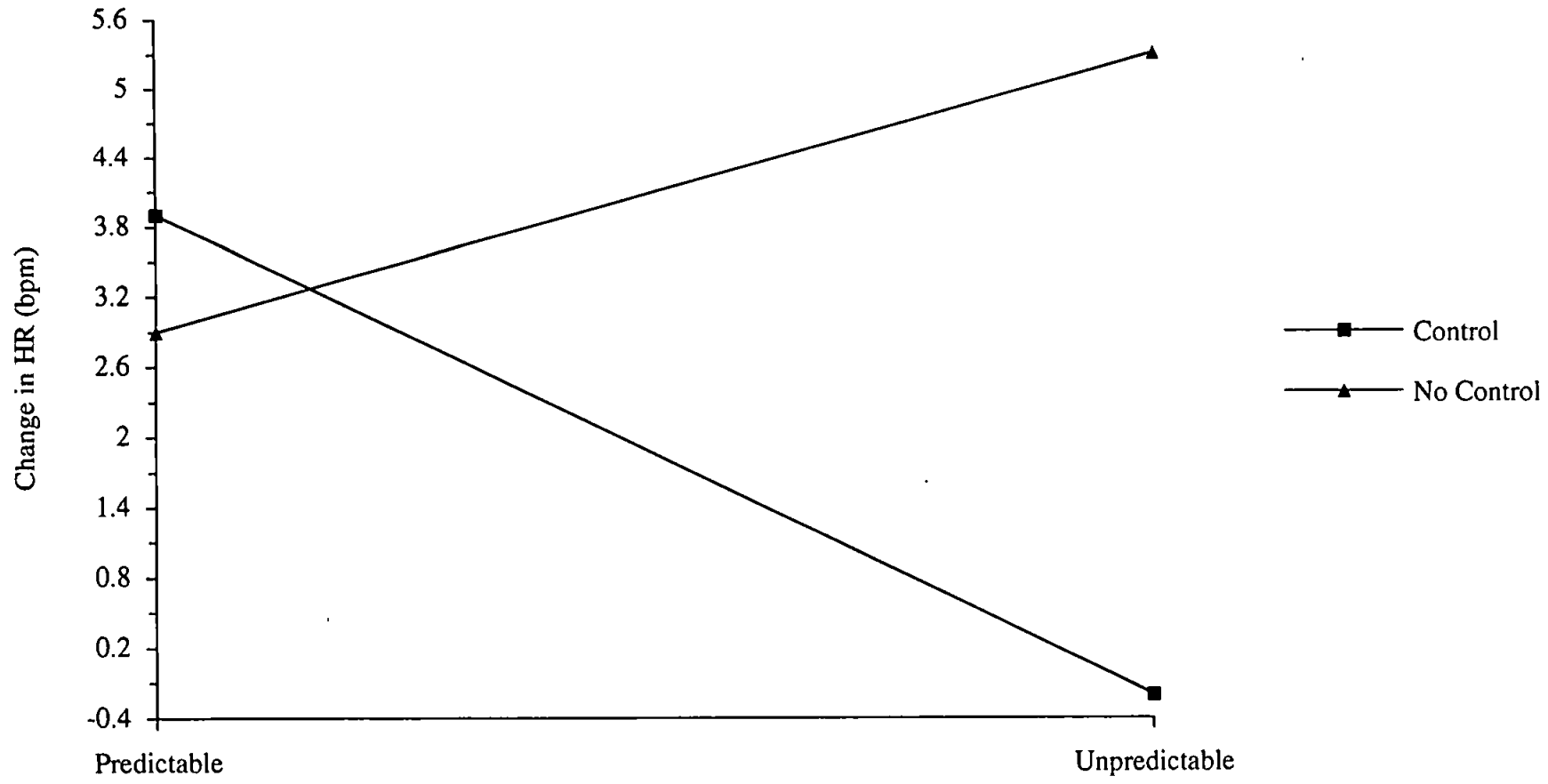


Figure 64. Mean change in average HR as a function of control and trials during Time Period 5.

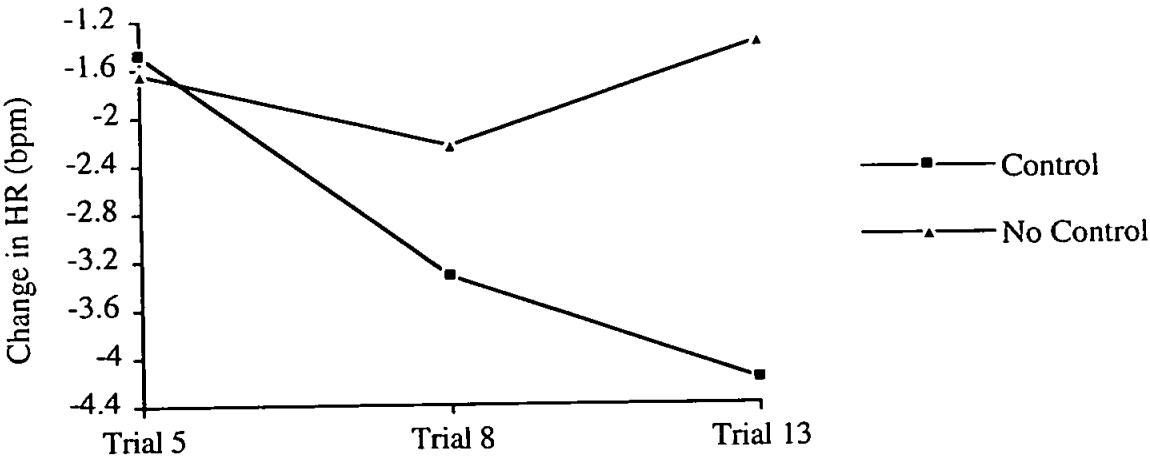


Figure 65. Mean change in minimum HR as a function of control and trials during Time Period 5.

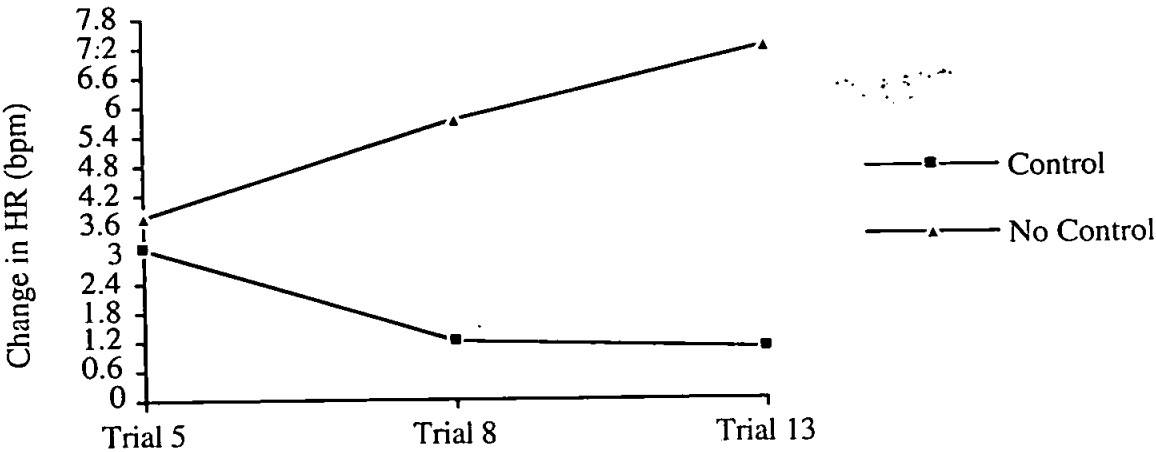


Figure 66. Mean change in maximum HR as a function of predictability and trials during Time Period 5.



revealed that all four groups produced a similar decrease in levels of activity from the pre-task baseline (e.g. Time Period 1: avHR = -2.9, -2.5, -3.7 and -2.0 and mxHR = -8.7, -8.7, -9.2 and -10.2 change in bpm for the PC, PNC, UPC and UPNC groups respectively). However, corrected mnHR produced a significant control by predictability interaction across all time periods. This was due to the UPNC group producing greater HR increases from the pre-task baseline compared with the PC, PNC and UPC groups, although in general only the latter comparison was significant. The PC group also had significantly greater increases in HR compared with the UPC group, although this reached significance during Time Periods 1 and 2 only. None of the other differences were significant.

During Time Periods 1 (mxHR), 2(avHR) and 5 (avHR & mnHR), the data produced significant control by trials interactions. In contrast to the uncorrected data, this was due to the NC groups producing greater increases in avHR and mnHR and smaller decreases in mxHR compared to the C groups during selected trials. In addition, during Time Period 2, corrected avHR and mnHR produced a significant main effect of control. This was due to the C groups producing a greater decrease in avHR and a smaller increase in mnHR from the baseline period compared to the NC groups.

During Time Periods 3 and 4, all three measures produced a significant predictability by trials interaction. Unlike the uncorrected data, however, the UP groups produced greater increases in avHR and mnHR and smaller decreases in mxHR compared to the P groups, during Trial 5 only. In later trials there were no significant differences between the groups. MxHR also produced a significant predictability by trials interaction during Time Period 5, with the UP groups producing smaller HR decreases compared to the P groups during Trials 8 and 13, although only the latter comparison reached significance. In addition, during Time Period 4, corrected avHR and mnHR produced a significant main effect of predictability. The UP groups had similar avHR and marked increases in mnHR from the baseline period compared with the P groups which revealed a decrease in levels of activity.

In summary, it would appear that the control by predictability interaction involving both uncorrected avHR and mxHR was mainly due to differential levels of activity before experimental manipulation began, whereas, the two-way control by predictability

interaction involving corrected mnHR appeared to be due to the UPNC group producing a greater increase in levels of activity from the pre-task baseline. In addition, there is fairly strong evidence to suggest that the NC and UP groups produced greater increases in HR levels compared with the C and P groups respectively, for all three measures.

### 3.3.2. Electrodermal Activity

Tonic SCL, frequency of NSFs and range-corrected SCR Amplitude were assessed during Time Periods 1 and 2 in a three-way MANOVA with control and predictability as between subject factors and trials as a repeated measure. SCL during Time Period 3 was analysed in a four factor MANOVA with the above between subject factors and trials and time (4 levels) as repeated measures. Group means and standard deviations for electrodermal activity across Time Periods 1 to 3 are shown in Table 22.

3.3.2.1. Skin Conductance Level. There were no significant differences between groups involving any of the time periods (all  $F_s < 1$ ). There were no significant differences across trials or any interaction involving trials for Time Periods 1, 2 or 3 (all  $F_s < 1$ ). However, Time Period 3 did reveal a highly significant difference across time ( $F(1, 64) = 70.9, p < .001$ ) and a trials by time interaction ( $F(3, 161) = 2.9, p < .05$ ). Analysis of the simple main effects revealed that Trial 5 had significantly higher HR than Trial 13 during the 5-10 sec period. During the 10-15 sec period, Trials 5 and 13 produced significantly higher HR than Trial 8. During the 15-20 and 20-25 sec periods there were no significant differences between trials (Figure 67).

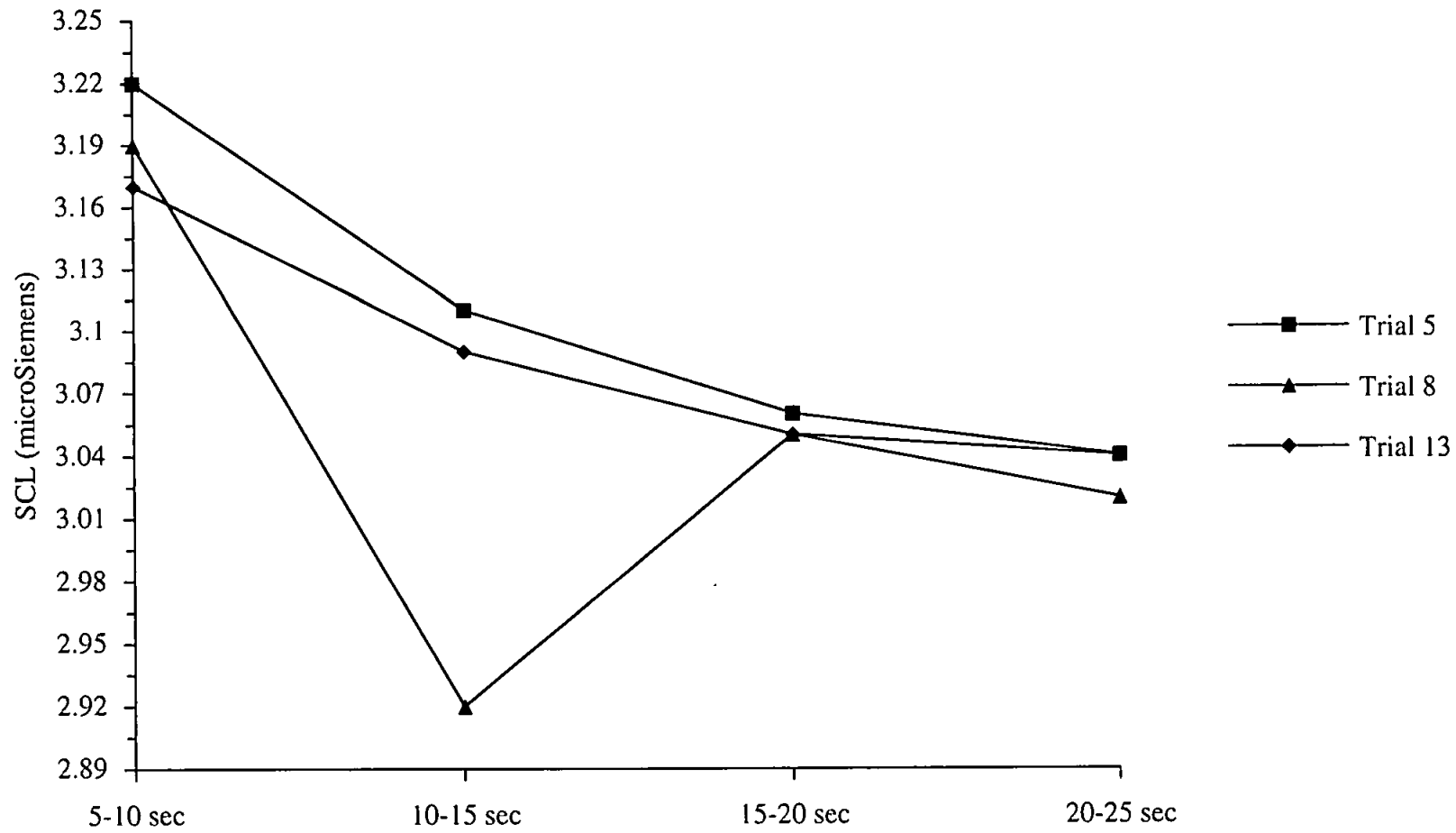
3.3.2.2. Non-Specific Fluctuations. There were no significant differences between groups (all  $F_s < 1$ ). There was a significant difference across trials ( $F(2, 93) = 5.5, p < .01$ ). Further comparisons revealed that Trials 5 and 8 produced significantly greater frequency of NSFs compared with Trial 13. There was no significant difference between Trials 5 and 8 (1.04, 0.93 and 0.55 for Trials 5, 8 and 13 respectively). There were no interactions involving trials (all  $F_s < 1$ ).

Table 22. Mean electrodermal activity during time periods averaged across Trials 5, 8 and 13.  
Standard deviations are shown in parentheses.

Variables	Experimental Groups			
	PC	PNC	UPC	UPNC
Time Period 1				
Tonic SCL ( $\mu$ S)	2.96 (1.28)	3.19 (1.60)	2.74 (1.28)	3.35 (1.52)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.17 (0.20)	0.29 (0.23)	0.19 (0.12)	0.19 (0.13)
Time Period 2				
Tonic SCL ( $\mu$ S)	3.04 (1.34)	3.22 (1.52)	2.74 (1.29)	3.34 (1.57)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.17 (0.28)	0.14 (0.14)	0.04 (0.13)	0.05 (0.09)
Time Period 3				
Tonic SCL ( $\mu$ S)	3.00 (1.27)	3.22 (1.60)	2.76 (1.30)	3.39 (1.47)
SCR Amplitude ( $\sqrt{\text{range-corrected}}$ $\mu$ S)	0.37 (0.28)	0.31 (0.23)	0.21 (0.13)	0.23 (0.14)
Frequency of NSFfs	0.67 (1.02)	1.05 (1.28)	0.91 (1.21)	0.74 (0.90)



Figure 67. Mean Skin Conductance Level as a function of trials and time during Time Period 3.



### 3.3.2.3. SCR Amplitude.

3.3.2.3.1. Time Period 1. There was a main effect of predictability ( $F(1, 52) = 4.7, p < .05$ ). The P groups had higher levels of responding than the UP groups (0.293 and 0.192  $\sqrt{\text{range-corrected } \mu S}$  respectively). There was no significant effect of control, nor any control by predictability interaction (all  $F_s < 1$ ). There was no significant difference across trials ( $F(1, 69) = 1.6$ ), nor any control by trials ( $F(1, 69) = 2.9$ ), predictability by trials or three-way (both  $F_s < 1$ ) interactions.

3.3.2.3.2. Time Period 2. There was a significant effect of predictability ( $F(1, 52) = 5.8, p < .05$ ). The P groups had higher levels of responding than the UP groups (0.156 and 0.046  $\sqrt{\text{range-corrected } \mu S}$  respectively). There was no significant main effect of control, nor any control by predictability interaction (both  $F_s < 1$ ). There was a significant difference across trials ( $F(2, 97) = 4.3, p < .05$ ). Further comparisons revealed that Trial 13 produced significantly higher levels of responding than Trial 5, and there were no significant differences between other trials (0.083, 0.105 and 0.114  $\sqrt{\text{range-corrected } \mu S}$  for Trials 5, 8 and 13 respectively). There were no interactions involving trials (all  $F_s < 1$ ).

3.3.2.3.3. Time Period 3. Again, there was a significant main effect of predictability ( $F(1, 52) = 5.3, p < .05$ ). The P groups had higher levels of responding than the UP groups (0.341 and 0.218  $\sqrt{\text{range-corrected } \mu S}$  respectively). There was no significant effect of control or a control by predictability interaction (both  $F_s < 1$ ). There was no significant difference across trials ( $F(2, 88) = 2.7$ ), nor any control by trials ( $F(2, 88) = 1.4$ ), predictability by trials ( $F(2, 88) = 2.4$ ) or three-way interaction ( $F(2, 88) = 1.9$ ).

3.3.2.4. Electrodermal Summary. There were no significant differences between groups in terms of tonic SCL or frequency of NSF's. However, further inspection of SCL means reveals a similar pattern across all time periods, with the NC groups producing greater levels of activity compared with the C groups. Transformed SCR Amplitude scores revealed significantly larger responses for the P groups, across all three time periods.

### 3.3.3. Reaction Time Data

RT scores were analysed in a three-way MANOVA with control and predictability as between subject factors and trials as a repeated measure. There were no significant effects of control ( $F < 1$ ) or predictability ( $F(1, 52) = 1.7$ ), nor a control by predictability interaction ( $F(1, 52) = 2.5$ ). There was a significant difference across trials ( $F(2, 97) = 10$ ,  $p < .001$ ). Further comparisons revealed that Trial 5 produced significantly slower RT's compared with Trials 8 and 13 (39.0, 31.8 and 32.9 msec for Trials 5, 8 and 13 respectively). Group means across trials are shown in Figure 68.

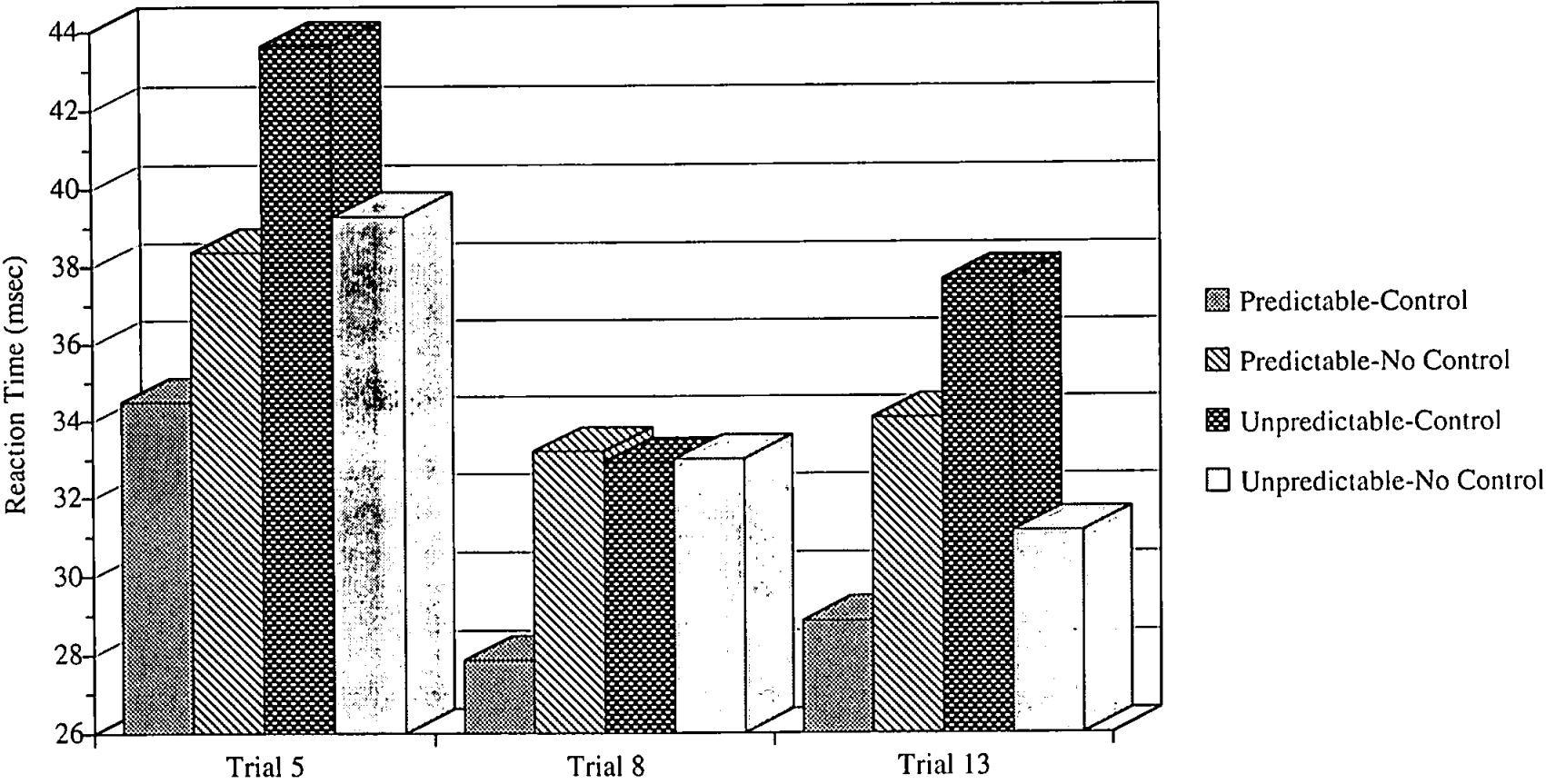
## 4.

## DISCUSSION

One particular set of findings from the cardiovascular data in this study which are of marked importance when considering any theoretical interpretation, were the significant differences between uncorrected and corrected HR during Trials 5, 8 and 13. Analyses of the former, revealed significant interactions between predictability and control during the warning stimulus, involving all three HR measures. Moreover, this interaction remained significant during the inter-stimulus and inter-trial intervals (average and maximum HR only), and the 1-3 sec period after noise onset. This was mainly due to the PNC group producing lower HR levels compared to the remaining groups across all five time periods. The PC and UPNC groups also produced higher HR than the UPC condition during the warning stimulus, and the inter-stimulus and inter-trial intervals. However, during the impact period these three groups showed similar levels of cardiac activity. It would appear, therefore, that where temporal predictability was present, the availability of control led to greater levels of HR activity than did no control, whereas, when the situation was unpredictable, the reverse occurred with higher HR in no control groups. However, changes in the ability to make a controlling response appear to be more important with predictable compared to unpredictable stimuli, since the difference between control-no control groups was markedly greater in the former condition.

There were also differences in the effect of noise impact. During the 1-3 sec after noise onset, the unpredictable groups produced significantly higher minimum HR

Figure 68. Mean response times as a function of groups and trials.



compared to the predictable groups. Furthermore, during the 0-1 and 1-3 sec after noise onset the unpredictable groups produced higher minimum HR during Trials 5 and 8 compared to the predictable conditions, and similarly during Trials 5, 8 and 13 for average and maximum HR. A similar pattern emerged in the cardiac response curve, with greater changes in average HR from the pre-stimulus period in unpredictable compared to predictable conditions during the first three seconds after noise onset.

Perceived ability to control also seemed to have an effect on uncorrected HR. It produced greater activity during the warning stimulus (maximum HR only), inter-stimulus (average HR only) and inter-trial (average and minimum HR) intervals in Trials 5 (average and minimum HR), 8 and 13 (maximum HR only).

However, these uncorrected HR results should be treated with caution, particularly in the light of significant differences between the uncorrected and corrected HR data. These differences appear to be due to the pattern of cardiovascular responding during the pre-task baseline in which the PNC group showed markedly lower activity levels compared to the remaining three experimental groups in all three HR measures. Furthermore, such an explanation would be supported by findings from the Trial 1 data in which uncorrected HR produced differential responding across all five time periods. As before, this was mainly due to the PNC group showing lower HR levels compared with the remaining three groups. However, analyses of corrected HR during Trial 1, in which pre-task differences were taken into account, produced no significant differences between any of the experimental conditions. Indeed, all four groups showed similar increases in average and minimum HR and smaller decreases in maximum HR from the pre-task baseline. Due to the problems associated with uncorrected HR, it was felt that any theoretical interpretation based solely on this data would be misleading. Consequently, the remaining discussion will focus entirely on results from the corrected HR data.

Corrected minimum HR seemed to produce the most interesting results across Trials 5, 8 and 13. During the warning stimulus, the UPNC, and to a lesser extent the PC group, had greater increases in minimum HR compared to the PNC condition which, in turn, increased more than did the UPC group. In the inter-stimulus interval the pattern of responding was similar, except that the PC group produced a decrease in level of activity from the warning stimulus to the inter-stimulus interval, whereas, responding in the PNC

group remained relatively unchanged during these two periods. The pattern of responding during the impact periods was similar to that observed to the warning stimulus, with the PC group producing greater increases in minimum HR compared to the PNC group. In addition, the UPC group produced a marked increase in HR to the noise, especially during the 1-3 sec after impact. Indeed, the increase was similar to that for the PC condition. During the inter-trial interval, the UPNC, and to a lesser extent the PC group produced a marked decrease in HR change from the 1-3 sec impact period to the inter-trial interval compared with the PNC condition which showed similar levels of activity during both periods. Consequently, there was little difference between them during the inter-trial interval. However, the UPC group produced an even more marked decrease in levels of activity from noise impact to the inter-trial interval, so it had significantly lower minimum HR change scores than the other three groups.

In terms of theoretical interpretations concerning the effects of control, these findings are difficult to reconcile with the predictability theories all of which predict no differential effects of control when predictability is present. As can be seen, differences did emerge, with the opportunity to control resulting in sustained or increased cardiac function compared to conditions of no control. Moreover, in unpredictable situations the reverse occurred with control conditions leading to a marked reduction in minimum HR change compared to situations where no control was available. It would appear, therefore, that control does have effects over and above those of predictability.

In terms of predictability *per se*, all three predictability theories would predict lower physiological activity during predictable compared to unpredictable conditions. The Safety-Signal Theory (Seligman, 1968) would predict that there would be greater autonomic activity in unpredictable compared to predictable conditions during safe periods (i.e. the inter-stimulus and inter-trial intervals) but similar reactions to the danger periods (i.e. the warning signal and aversive event). In contrast, the Preparatory Response Theory (Perkins, 1968) would predict lower autonomic activity during predictable aversive events compared to unpredictable ones. The theory makes no specific predictions with regard to activity during the warning signal, or, anticipatory (inter-stimulus and inter-trial) intervals. However, as suggested previously (see Chapters 7 and 8), unpredictable conditions might lead to greater autonomic activity during these periods if it is assumed that subjects are

anticipating a more painful event, since they are not able to adequately prepare for it. Finally, the Relevant Feedback Theory (Weiss, 1971a) would predict less anticipatory autonomic activity with predictability if the mechanism by which relevant feedback reduces arousal is because it provides a safety signal, as defined in Seligman's Theory. However, the relevant feedback theory makes no specific predictions concerning activity during the warning signal, or at event impact as, in terms of the latter period it proposes no mechanism whereby relevant feedback affects responses to the event once it occurs.

In general, the results involving the no control groups were in accordance with predictions made by the predictability theories, and much of the past predictability literature (see Chapter 2). That is, supplying information about the occurrence of an aversive event led to a reduction in minimum HR compared to situations of unpredictability. However, contrary to the Safety Signal Theory differential effects emerged during both the warning signal, and in the impact periods. During the inter-stimulus and inter-trial intervals, the predictable groups displayed lower minimum HR compared to unpredictable conditions, a finding predicted by both the safety-signal and relevant feedback explanations. An extended version of the Preparatory Response Theory can account for the differential effects observed during all five time periods.

In terms of the control groups, predictability had the opposite effect. That is, it produced higher minimum HR change scores to the warning signal and in the inter-stimulus and inter-trial intervals compared with the unpredictable condition. During the two impact periods, the predictable and unpredictable groups showed similar changes in minimum HR due to subjects in the latter condition producing greater increases to the noise. It is not clear why availability of control led to an augmentation of cardiac activity during predictable situations. Indeed, these results are not in accordance with the handful of past predictability studies which have included control, the majority of which have found that in situations where organisms have a controlling response available, predictability is less aversive (see Chapter 2, Section 3.3.1). This issue will be discussed in detail later in the chapter.

The alternative to the predictability theories in explaining the effects of control is the Minimax Theory, which suggests that the effects of control and predictability are independent. That is, control has effects above and beyond predictability. More

specifically, it predicts that control conditions should lead to lower physiological activity during the warning signal and anticipatory (i.e. inter-stimulus and inter-trial) intervals compared to no control conditions. An extended version of the minimax hypothesis (see Section 1) would also predict lower physiological arousal at impact when a controlling response is available. Furthermore, the Minimax Theory which states that the aversiveness of an event is determined by the maximum danger presented by that event to the individual, would predict that the effects of control and predictability are beneficially additive.

The availability of a controlling response did lead to effects over and above those of temporal predictability. Furthermore, these effects were generally additive. However, contrary to predictions made by the Minimax Theory adding control to predictability produced increased changes in minimum HR, particularly to the warning signal and, to a lesser extent, during the inter-trial interval. Indeed, predictability and control produced greater increases in minimum HR compared with either on its own across all time periods, with the exception of the 1-3 sec period after noise impact. During this period, the PC and UPC groups showed similar levels of activity, due to subjects in the latter condition producing greater increases in minimum HR from the pre-task baseline to the noise. The limited findings of the present study appear to suggest, therefore, that the effects of temporal predictability and perceived instrumental control are additive. However, they are not *beneficially* additive as would be predicted by the Minimax Theory.

These findings are contrary to predictions made by the alternative to the Minimax Theory which states that the effects of temporal predictability and control are not additive. Indeed, this alternative suggests that either control or predictability is sufficient to lower psychophysiological stress, and that no additional benefit can be derived from the presence of both factors. However, the findings of the present experiment suggest that although the situation where both temporal predictability and availability of control were absent did *generally* lead to the greatest increases in minimum HR change, compared to conditions in which predictability and/or control were available, this was not the case during the warning stimulus, or inter-trial interval. In these periods, the PC and UPNC groups displayed similar minimum HR change scores. Furthermore, contrary to predictions, the PC group produced greater minimum HR increases compared to the UPC and, to a lesser extent, the



PNC group during the warning stimulus, the 0-1 sec period after noise impact, and in the inter-stimulus and inter-trial intervals.

The relationship between the two intermediate conditions was also contrary to predictions put forward above. Namely, that the PNC and UPC groups would show similar levels of cardiac activity across all five time periods. A prediction also in line with the Minimax Theory which suggests that temporal predictability and availability of control are of equal importance in aversive situations. In this particular study, however, differences did emerge with the PNC group producing greater minimum HR change scores to the warning stimulus, and during the inter-stimulus and inter-trial intervals compared to the UPC group. Thus, situations where no controlling response was available appeared to be more arousing in terms of higher minimum HR activity, compared to situations which were temporally unpredictable. However, during the impact periods the two intermediate conditions displayed similar levels of cardiac activity. It would appear, therefore, that both predictability and perceived control are equally important in terms of attenuating the effects of a noxious event once it occurs.

It is unclear why a situation involving both temporal predictability and opportunity to control should lead to sustained, or even increased cardiovascular activity. Indeed, there appears to be no plausible explanation available in the predictability literature (see Chapter 2). However, one highly tentative explanation suggested by the control literature (see Chapter 4) may be that subjects in the PC group were merely trying harder to succeed at the task and this effort was facilitated, in turn, by the combination of precise timing as to the occurrence of the noxious event and a controlling response which could shorten the duration of that event from 5 to 2 sec. It is this interaction between temporal predictability and control which augmented HR activity compared to conditions where only prediction *or* control was available. The task performance data, which could be interpreted as a measure of 'effort', would partially support such an explanation. That is, the PC group produced markedly, although not significantly, faster reaction times across Trials 5, 8 and 13 compared to the remaining three groups.

It could be argued, that an alternative interpretation of the reaction time data might be in terms of differential motor activity. That is, the PC group produced the fastest reaction times which may, in turn, have led to higher HR levels through increased motor

activity. However, this would appear unlikely based on the performance of subjects in the UPNC group who produced slower reaction times compared to the PC group, yet displayed the greatest levels of HR activity.

Moreover, the findings from both the corrected average and maximum HR analyses would lend some support to an explanation in terms of sustained or increased effort. For instance, during the warning stimulus the no control groups produced significantly smaller decreases in maximum HR during Trials 5 and 8, compared to the control groups. However, there was no difference between the two conditions in Trial 13, due to the two no control groups showing a reduction in HR across trials, whereas, the control groups sustained or even increased maximum HR across trials. The control groups also produced a marked increase in average HR change scores between Trial 8 and 13 of the inter-stimulus interval, compared to the no control groups. Thus, it may be that in situations where subjects are given the opportunity to control efficiently by the addition of predictability, a certain level of task involvement or effort is maintained across trials.

It may be that in this particular experiment sustained or increased effort in the PC condition was mediated by high levels of behavioural uncertainty. Indeed, such an explanation would be partially supported by the work of Obrist and colleagues (see Obrist, 1981 for a review). For instance, Obrist, Gaebelein, Teller, Langer, Grignolo, Light and McCubbin (1978) found that in difficult active coping tasks (i.e. where control was contingent on task performance), uncertainty about task outcomes led to sympathetically-mediated increases in cardiovascular activity by evoking greater efforts directed at controlling task outcomes. Similarly, recent work by Phillips (1989) suggests that in situations where behavioural uncertainty is high, the constant striving to escape or cope with a noxious event may be more costlier in terms of metabolically expensive and inappropriate responses than conditions in which no control is available; i.e. situations in which subjects passively accept in order to cope. The only way in which subjects can resolve this uncertainty, Phillips argues, is by recognising a contingency between response and response outcome. If subjects do not establish this contingency then uncertainty will continue.

In terms of this experiment, subjects may not have learnt this contingency (response and response outcomes remained uncertain) for a number of reasons. Firstly,

subjects were not told the criteria by which they could succeed. Indeed, they were merely told that "... if your reaction time is within certain limits set by the computer then noise duration would be reduced from 5 to 2 sec". Thus, the number of possible responses permitted by the instructions were numerous. Secondly, subjects were not told whether or not their controlling had been successful. That is, they never received trial-by-trial performance feedback. It may be that subjects were uncertain that they had actually received the short duration noise (i.e. that their controlling had been successful), particularly as they never received the long duration noise which indicated that controlling had been unsuccessful. Thus, the manipulation of perceived control in this particular experiment relied upon subjects being able to successfully differentiate long and short duration aversive events. However, if trial-by-trial feedback had been made available this uncertainty may have been reduced or even eliminated; i.e. feedback could have facilitated the beneficial effects of having control<sup>2</sup>.

Nevertheless, a behavioural uncertainty interpretation for the pattern of responding observed in the PC condition should be treated with caution, since studies carried out by Obrist and co-workers involved only unsignalled conditions. In the present study, opportunity to control sustained or increased cardiac function only in predictable situations. In unpredictable conditions, control led to a reduction in cardiovascular activity. From a behavioural uncertainty perspective, it would be expected that unpredictability would be associated with higher levels of uncertainty than predictable situations since in the former condition subjects received no information warning of the occurrence of the noxious event. It remains unclear, therefore, why adding control to a signalled aversive situation augments cardiac function, whereas, adding control to an unsignalled event attenuates cardiovascular activity.

One conclusion would appear warranted based on data presented in this chapter. That is, the results from predictable situations and those from unpredictable ones are unlikely to be sufficiently incorporated into a single theory or hypothesis. In terms of the

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<sup>2</sup> It is uncertain to what extent subjects in the control conditions felt that they had control over noise duration mainly because psychological measures, such as, rating scales were not administered. Perhaps a more comprehensive estimate of perception of control would have been appropriate both in this and the remaining four studies. However, it was felt, when these studies were being designed, that there were no instruments in this particular area which would satisfactorily combat the associated methodological and conceptual difficulties.

former, it might be that subjects in the control group had a greater level of incentive (i.e. reduction in the duration of a noxious event) which, in turn, maintained continued effort and task involvement compared with those subjects in the no control condition.

Presumably, on the evidence of past studies described above, this sustained involvement or engagement on the task promoted, in turn, greater levels of cardiovascular activity compared with situations in which the magnitude of incentive was minimal; namely, situations in which subjects had no control over the aversive event. In terms of unpredictable situations, it might be argued that an explanation such as Miller's (1979) minimax hypothesis would be more appropriate. That is, individuals are motivated by a desire to minimise the maximum danger to themselves. Therefore, situations in which a controlling response is available (i.e. where individual's can put an upper limit on how aversive the situation can become) lead to a reduction in psychophysiological arousal compared to situations in which a subject believes that there is nothing he or she can do to reduce the level of discomfort. These issues will be discussed further in Chapter 11.

The significant findings from the electrodermal system - that the predictable groups produced greater response amplitudes compared to the unpredictable groups during the warning stimulus, and the 1-5 sec after noise onset and offset - appear inexplicable. Indeed, these results were surprising in the light of the highly significant findings in the opposite direction during Experiment 2, in which the unpredictable groups produced greater response amplitudes to the warning stimulus compared to the predictable groups. Furthermore, during Experiment 3 there were no significant differences between predictable and unpredictable conditions in terms of electrodermal activity. The present experiment employed a similar paradigm to both of these previous studies and it is, therefore, difficult to understand why the results should be so significantly different.

Furthermore, the results of the remaining electrodermal measures - tonic SCL and frequency of NSF's - produced no differential effects of predictability throughout Trials 5, 8 and 13. Indeed, further inspection of the group means for these two measures reveals that both the predictable and unpredictable conditions showed similar levels of electrodermal activity across all time periods. Marked independence between the three electrodermal measures has been reported in the psychophysiological literature (e.g. Kilpatrick, 1972), suggesting that many of the electrodermal components may represent

partially independent sources of information. However, this issue remains controversial (see Venables & Christie, 1980). Moreover, this type of explanation appears insufficient in the light of inconsistencies in the pattern of response amplitude responding across experiments. Consequently, it is suggested that the response amplitude data from this particular study should be treated with some caution.

Finally, in terms of the cardiovascular data from this experiment it was interesting that minimum HR produced significant differences between predictability and control conditions throughout, whereas, the effects in terms of average and maximum HR were more limited. These differences had been predicted earlier, see Chapter 5 Section 2.2.1, where it had been suggested that the three response elements might respond differently to behavioural demands exerted upon the subject. Indeed, the finding that the additive effects of temporal predictability and availability of control only emerged in minimum HR may indicate that the ability to lower cardiac activity, or 'relax' may be more important than the extent of HR acceleration, at least in tasks with aversive consequences. However, these findings appear contrary to those of Obrist and colleagues (see Obrist, 1981 for a review) which indicated that it was the extent of sympathetically-mediated increases in cardiac function which was important in aversive situations. Particularly since these exaggerated responses, which are in excess of metabolic demands, may predict the early stages of haemodynamic dysfunction. The significance of changes in peak deceleration or minimum HR reactivity sensitive to the degree of control and levels of behavioural uncertainty engendered by the situation is not yet clear. Moreover, although these data may indicate excessive, metabolically inappropriate vagal drive in the same manner as Obrist and co-workers referred to sympathetic effects, such a conclusion would seem premature. Firstly, because sympathetic influences were not blocked. Secondly, because although average and maximum HR did not produce statistically significant differences between predictable and control conditions, further inspection of groups means reveals a similar pattern of responding to minimum HR.

To summarise: the predictions concerning the independent, additive effects of temporal predictability and control were partially confirmed in the cardiovascular system. That is, availability of control did have effects over and above those of predictability and *vice versa*. Moreover, these effects were significantly different than where either

predictability or control was present. However, contrary to predictions, the effects of predictability and control were not beneficially additive. That is, the condition in which both factors were present did not lead to the greatest reduction in psychophysiological stress. It was suggested that a number of factors may account for these findings. In particular, the degree of effort and level of behavioural uncertainty engendered by the task, and the non-availability of trial-by-trial performance feedback. In general, the electrodermal system appeared insensitive to changes in the degree of predictability or amount of perceived control available.

CHAPTER 10  
EXPERIMENT 5

1. INTRODUCTION

The experiment described in Chapter 9 demonstrated that in situations which were temporally unpredictable, the perceived ability to control the duration of a noxious event reduced cardiac activity. Conversely, when combined with temporal predictability, perception of control augmented the cardiovascular response. The subsequent discussion of these results raised a number of important points concerning the nature of the prediction-control relationship, particularly the potential mediating role of behavioural uncertainty. That is, during the preceding study, task uncertainty was intentionally maximised by the absence of trial-by-trial performance feedback. It was suggested that if information had been provided informing individuals whether or not their controlling had been successful, subject's perception of the importance of control and/or predictability may have been significantly altered by reducing aspects of task uncertainty; namely, ambiguity surrounding the effectiveness of the individual's response. This, in turn, might have made the two factors more efficient in reducing psychophysiological stress. That is, the combination of temporal predictability and opportunity to control may always be more aversive unless the individual can gain feedback concerning the situation. This is in accordance with work by Weinberg and Levine (1980) which suggests that the negative effects of predictability can be attributed primarily to a lack of feedback (see Chapter 4).

According to a behavioural uncertainty perspective, where temporal predictability, control and feedback are manipulated independently, as in the present experiment, lower cardiovascular activity would be expected in predictable and unpredictable situations when feedback is made available, especially for those subjects who can control. The presence of information informing subjects whether or not their controlling has been successful should enhance the perception of control which should lead, in turn, to a reduction in both response and response outcome uncertainty. Where opportunity to control is not available, the predictions are less clear-cut. For instance, it could be argued that the presence of feedback should have little beneficial effect as subjects are unaware of any relationship

between task performance and event duration and, therefore, feedback reduces neither response or response outcome uncertainty. Alternatively, it could be that information concerning noise duration may reduce ambiguity about the aversive event which might, in turn, have an ameliorative effect on feelings of lack of control and reduce physiological function even when actual physical control is absent. This argument derives some support from findings in the nonhuman feedback literature reviewed in Chapter 3 Section 2.2.1 and, in particular, the work of Weiss (1971a, 1971b, 1971c). For example, Weiss (1971a) found that in unsignalled situations in which organisms had a controlling response available in order to avoid or escape electric shock, feedback led to a significant reduction in gastrointestinal pathology compared with no feedback conditions. However, where no controlling response was available, changes in the feedback-no feedback manipulation had no differential effects.

An alternative explanation for the relationship between predictability, control and feedback may be in terms of differential effort. Essentially, this approach was suggested by the work of Light and Obrist (1980) who found that feedback augmented cardiovascular activity in unsignalled reaction-time tasks when subjects had no opportunity to control shock. The authors suggested that feedback (in the form of reaction times) promoted a more active engagement in the task by providing an additional incentive; i.e. to beat their 'best' time. This enhanced motivation associated with an increase in active coping led to increased sympathetic activity. Enhanced effort or task involvement was especially prominent in situations where no control was available since subjects with control already had an incentive to be trying hard; namely, shock avoidance.

Thus, although feedback may reduce behavioural uncertainty compared with no feedback situations as suggested previously, feedback may, in addition, promote a greater degree of effort or task engagement by providing additional incentives. Indeed, this would be supported by findings from Chapter 6 in which information concerning success or failure led to significantly increased performance compared to no feedback conditions during a complex, decision-making task. Furthermore, these effects were seen across the course of the task, with higher performance levels in later trials. It was suggested, therefore, that feedback may sustain an individual's levels of motivation and promote the 'desire to do well' through changes in subject's perception of their own ability or



competency at the task and/or their degree of perceived control. The findings from both laboratory and applied feedback studies would support such an interpretation (Chapter 3, Sections 2.1.1 and 2.1.2. respectively).

According to a differential effort explanation, a number of testable predictions can be made during situations in which temporal predictability, control and feedback are manipulated independently. Namely, that the provision of performance feedback should augment cardiovascular activity compared to no feedback situations, and that these effects should be most pronounced in subjects with control. This is because feedback, concerning information about the success or otherwise of an individual's controlling response, provides additional incentives which, in turn, promotes a greater degree of effort or task involvement. Feedback might also have augmentative effects in situations where no controlling response is available, since it may enhance active coping mechanisms which are independent of the actual ability to control.

The aim of this final experiment was, therefore, to explore the possible relationships between feedback, temporal predictability and opportunity to control. Feedback concerning 'success-failure' only was provided (i.e. RF) as this had been found to be beneficial both in terms of increasing task performance and in maintaining that performance across the course of Experiment One. Although RF had had no significant effects on either electrodermal or cardiovascular responding during this particular study, it was suggested that this was due not to feedback *per se* but rather to the type of task employed (an externally paced, information-handling game involving extensive visual-spatial abilities), the level of task difficulty (88% failure rate) and the lack of any aversive consequences (Chapter 6). It was hoped that the present experiment would maximise any potential effects of RF by employing a more straightforward paradigm in which the opportunity to change stimulus duration was contingent on task performance.

In terms of predictability and control, the paradigm used was similar to that in the preceding study in which differential effects of predictability and control were produced, with one notable exception. Namely, the manipulation of the controlling response. During the previous experiment, it could be argued that the presence of the warning signal in the PC condition did not simply predict the occurrence of the noxious event, but that it also aided the prediction of the controlling response. Thus, the nature or meaning of control

might have been significantly different depending on whether subjects were allocated to the predictable or unpredictable condition. For instance, although subjects in both groups could not actually physically alter noise duration, the additional sense of control provided by predictability may have changed subject's perception of the task, for example, by giving an extra incentive to control. Essentially, therefore, the augmentation of cardiac activity observed in the PC condition during the previous study might have been due to the confounding effects of predictability on control rather than to the degree of effort, or behavioural uncertainty engendered by the situation. In the present study, the manipulation of temporal predictability was identical to the preceding experiment. However, in order to differentiate between predictability of event onset and control over event duration, subjects were informed that their reaction time to the word "START" presented at the beginning of each trial would determine the duration of the white noise, presented at the end of the trial. Therefore, the manipulation of temporal predictability and opportunity to control basically occurred in separate halves of each trial (see Section 2.3).

Following changes in the manipulation of control, an additional aim of the present study was to compare any relationship between prediction and control to that observed in Chapter 9. Potential relationships may, in turn, help to differentiate between theoretical explanations for the supposedly beneficial effects of control and/or predictability; namely, the Safety Signal (Seligman, 1968), Relevant Feedback (Weiss, 1971a), Preparatory Response (Perkins, 1968) or Minimax (Miller, 1979) Theories. Predictions made by each of these theories have been discussed previously (see Chapter 9, Section 1). Briefly, the former explanations (the predictability theories) have in common the proposition that availability of control only provides additional predictability of the aversive event. Thus, when the two factors are manipulated independently these theories predict that the effects of control should not be greater than when the aversive stimulus is perfectly predictable. In contrast, the Minimax Theory suggests that control provides an individual with more than just additional predictability, and in situations where the two variables are manipulated independently, the effects of control should be greater than predictability alone. Miller's (1979) minimax hypothesis also predicts that the effects of predictability and control are additive. That is, where both factors are present the greatest autonomic activity should

result whereas, where either predictability or control is available intermediate levels should be observed.

## 2. METHOD

### 2.1. Subjects

The subjects were 96 psychology undergraduates (76 females and 20 males), with an age range of 18 - 40 years. All subjects were randomly allocated to one of eight experimental conditions (see Section 2.3). The only restriction to the random allocation was that there were equal numbers of the smaller, male population in each of the four groups.

### 2.2. Apparatus

Throughout the experiment, the recording apparatus (HR, SC and Reaction Time (RT)) and warning stimuli were the same as those used in Experiment 4. In addition to delivering the warning stimuli, the BBC microcomputer was also employed to present the word "START" at the beginning of each trial and the feedback or no feedback message: "NOISE DURATION WILL BE (2 or 5) SECONDS" or "THE NOISE WILL FOLLOW". Both messages were displayed on a colour monitor of 15 x 13.5 inches situated in the subject's room. This was positioned 31 inches from the subject and slightly to their right. The white noise was 100 dB, 2 or 5 sec duration and was delivered as in Experiment 3.

A two way intercom system was in operation throughout. It was situated 8 inches from the subject and directly in front of them.

### 2.3. Design and Procedure

The experimental design was a 2 (predictable/unpredictable) x 2 (control/no control) x 2 (feedback/no feedback) x 8 (trials) repeated measures between subject. Subjects were randomly allocated to one of eight conditions; predictable/control/feedback (PCFb):

predictable/control/no feedback (PCNFb): predictable/no control/feedback (PNCFb):  
predictable/no control/no feedback (PNCNFb): unpredictable/control/feedback (UPCFb):  
unpredictable/control/no feedback (UPCNFb): unpredictable/no control/feedback  
(UPNCFb) or unpredictable/no control/no feedback (UPNCNFb).

The seating and accommodation of subjects was the same as in Experiment 1. After electrode attachment, subjects were informed of the procedure to follow (see appendix). Those subjects in the control groups were told that they could reduce the duration of the white noise from 5 to 2 sec if their reaction time to the word "START", presented on the subject's monitor at the beginning of each trial, was within certain limits set by the computer. However, if their reaction time was not within these limits then the duration of the noise would be 5 sec. Subjects in the no control groups were told to press the button as quickly as possible, but were not told of any relationship between reaction time and noise duration. Thus, by making all subjects press the reaction time button it was hoped that any differential motor activity would be controlled for. However, none of the subjects, in either the control or no control conditions, could actually control the duration of the noise (i.e. they had perceived instrumental control (Miller, 1979)). Subjects allocated to the feedback groups were also informed that after they had responded they would be told the duration of the noise. Those subjects in the no feedback groups were simply told that the noise would follow. Following this, subjects in the predictable groups were told that they would receive a warning consisting of the numbers 3, 2, 1 on the monitor 5 sec before onset of the white noise. Those subjects allocated to the unpredictable groups were simply told that they would receive a warning of 3, 2, 1 before noise onset on some trials, but not on others.

After these instructions had been given the experimenter left the room. The experimental procedure for all eight groups commenced with a one minute resting period. All subjects then received the first range-correction stimulus consisting of 2 sec, 100 dB white noise followed by 15 sec during which no stimuli were presented. Subjects then received the second range-correction stimulus consisting of 5 sec, 100 dB white noise. Following this, there was a 3 min rest period. After these 3 min the first trial began. At the beginning of each trial, all subjects received the message "START" on the monitor. The message remained on the VDU until subjects had pressed the reaction time button and for up to a maximum of 2 sec. After 2 sec, there followed a 5 sec wait during which nothing

happened. Following these 5 sec, those subjects in the feedback groups received the message "NOISE DURATION WILL BE (2 or 5) SECONDS" on the screen for a duration of 2 sec. Noise duration depended on the trial number (discussed fully later). Subjects in the no feedback groups received the message "THE NOISE WILL FOLLOW". Those subjects in the predictable groups then received 15 sec during which nothing happened (Inter-Stimulus Interval (1) (ISI (1))). After 15 sec, subjects received a warning of 3, 2, 1. Each number was displayed on the screen for a duration of 1 sec. Subjects then had a 5 sec wait during which nothing was displayed on the screen (Inter-Stimulus Interval (2) (ISI (2))). After 5 sec, the 100 dB white noise tone was presented. Noise duration depended on the trial number; during Trials 1, 4, 5, 6, 7 and 8 duration of the noise was 2 sec, whereas, in Trials 2 and 3 duration of the noise was 5 sec. Subjects in the unpredictable groups received two different types of trial. Firstly, trials in which the noise was preceded by a warning of 3, 2, 1 (Trial Type 1 (TT1)); secondly, trials where the noise was followed by a warning of 3, 2, 1 (Trial Type 2 (TT2)). All subjects received the two types of trial in the same order (see Table 23). Presentation and duration of the warning stimulus and noise was identical to the predictable group. In order to further augment unpredictability, subjects received different lengths of ISI's (1) and (2), varying between 5 and 25 sec, with a mean of 15 sec. All experimental parameters across predictable and unpredictable groups are shown in Table 23. The control and no control conditions differed only in the instructions they were given before the start of the experiment (see above). All subjects received 8 trials with an inter-trial interval of 15, 20 or 25 sec. The mean ITI was 20 sec. Following this, the experimenter re-entered the room and unwired the subject. The experimental procedure for all groups during the trials (1, 5 & 8) in which they received identical parameters is shown in Figure 69.

## 2.4. Scoring

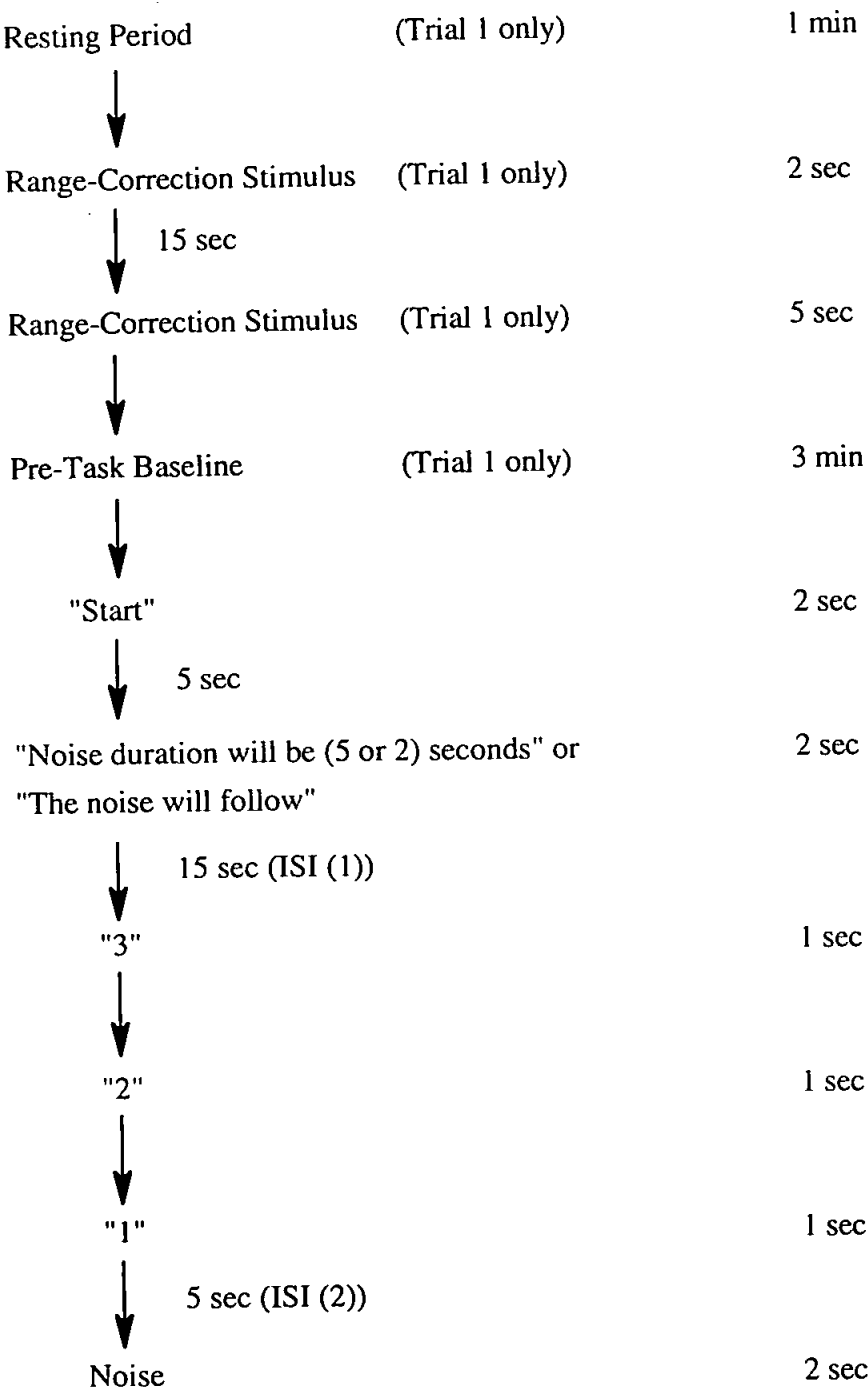
2.4.1. Cardiovascular activity. The measurement of HR was the same as in Experiment 1. All three cardiovascular measures (avHR, mnHR and mxHR) were assessed during the 3 min pre-task baseline in order to examine any pre-task between-group differences.

Between-group comparisons for all three cardiovascular measures were made

Table 23. Experimental parameters during Experiment 5.

Trial No	Trial Type		Noise Duration (sec)	ISI (1) (sec)		ISI (2) (sec)		Inter-Trial Interval (sec)
	P	UP		P	UP	P	UP	
1	TT1	TT1	2	15	15	5	5	20
2	TT1	TT1	5	15	5	5	25	15
3	TT1	TT2	5	15	25	5	25	15
4	TT1	TT1	2	15	20	5	10	25
5	TT1	TT1	2	15	15	5	5	20
6	TT1	TT2	2	15	5	5	25	20
7	TT1	TT1	2	15	20	5	20	25
8	TT1	TT1	2	15	15	5	5	20

Figure 69. Experimental Procedure for Trials 1, 5 & 8 during Experiment 5.



across Trials 1, 5 & 8 as these were the only trials where unpredictable groups received exactly the same experimental procedure as predictable groups. Trial 1 was analysed separately from Trials 5 and 8. Each trial was divided into 8 separate time periods (Table 24): Period 1, was the 0-5 sec before onset of the Fb/NFb message: Period 2, was the 0-2 sec after Fb/NFb message onset: Period 3, was the 0-15 sec between offset of the Fb/NFb message and onset of the warning stimulus (ISI (1)): Period 4, was the 0-3 sec after warning stimulus onset: Period 5, was the 0-5 sec between warning stimulus offset and onset of the noise (ISI (2)): Period 6, was the 0-2 sec after noise onset: Period 7, was the 20 sec ITI divided into four, 5 sec blocks: Period 8, involved a measure of the cardiac response profile. Measurement of the cardiac response profile was the same as in Experiment 2. Uncorrected and corrected HR are reported for all three cardiovascular measures across Time Periods 1 to 7. Measurement of corrected HR was the same as in Experiment 1. Period 8 was examined for change in avHR across Trials 5 and 8 only.

2.4.2. Reaction Time Data. Between group differences in reaction time (RT) were assessed across Trials 1, 5 and 8. Trial 1 was analysed separately from Trials 5 and 8.

### 3. RESULTS

#### 3.1. Baseline Activity

With all measures, differences between groups were assessed across the 3 min baseline. These 3 min were divided into three, 60 sec periods and a four-way MANOVA was employed with control (control/no control), predictability (predictable/unpredictable) and feedback (feedback/no feedback) as between subject factors and time (3 levels) as a repeated measure. Group means for cardiovascular activity during the pre-task baseline are shown in Table 25.

There were no differences between groups in terms of any of the three cardiovascular measures (all  $F$ s < 1). There was a significant difference across time for avHR ( $F(2, 149) = 6, p < .01$ ) and mxHR ( $F(2, 158) = 20.6, p < .001$ ). Post-hoc comparison of means revealed, for both avHR and mxHR, that the 0-60 sec period had



Table 24. Time Periods for cardiovascular activity during Experiment 5.

Time Period	Stimulus
1	0-5 sec between "START" offset and onset of Fb/NFb message
2	0-2 sec after Fb/NFb message onset
3	0-15 sec after Fb/NFb message offset and warning onset (ISI (1))
4	0-3 sec after warning onset
5	0-5 sec between warning offset and onset of the noise (ISI (2))
6	0-2 sec after noise onset
7	0-20 sec Inter-Trial Interval (ITI)
8	0-10 sec Cardiac Response Profile

Table 25. Mean cardiovascular activity during the pre-task baseline. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Groups							
	PCFb	PCNFb	PNCfB	PNCNFb	UPCFb	UPCfNB	UPNCfB	UPNCNFb
Pre-Task Baseline								
AvHR	81.4 (17.6)	83.3 (16.1)	79.5 (13.9)	86.0 (14.0)	81.3 (9.9)	83.7 (17.9)	77.7 (13.6)	79.3 (11.4)
MnHR	70.5 (16.2)	71.2 (15.6)	67.7 (7.1)	74.8 (15.9)	70.4 (9.3)	73.4 (17.5)	66.0 (11.7)	68.2 (9.7)
MxHR	93.6 (19.2)	97.0 (18.5)	93.8 (14.6)	99.4 (13.2)	96.0 (10.2)	96.3 (19.3)	91.1 (13.4)	91.4 (13.7)

significantly higher HR than the 60-120 and 120-180 sec periods. There were no significant differences between the 60-120 and 120-180 sec periods (avHR = 82.4, 80.8 and 81.3 bpm; mxHR = 98.0, 93.5 and 93.3 bpm for the 0-60, 60-120 and 120-180 sec periods respectively). There were no significant interactions involving time for avHR or mxHR (all  $F_s < 1$ ). There was no main effect or any interactions involving time for mnHR (all  $F_s < 1$ ).

### 3.2. Between-Groups Analysis: Trial 1

For all measures, Time periods 1 to 6 were each assessed in a three-way ANOVA with control, predictability and feedback as between subject factors. Time Period 7 was analysed in a four-way MANOVA with the above between-subject factors and time (4 levels) as a repeated measure. Group means for uncorrected HR across Time Periods 1 to 7 are shown in Table 26.

#### 3.2.1. Uncorrected HR.

3.2.1.1. Time Periods 1 to 6. There were no significant differences between groups in terms of avHR, mnHR or mxHR (all  $F_s < 1$ ).

3.2.1.2. Time Period 7. There were no significant effects of predictability involving any of the three measures (all  $F_s < 1$ ). AvHR revealed no main effect of control ( $F(1, 88) = 1.9$ ), nor did mnHR ( $F(1, 88) = 2.1$ ) or mxHR ( $F(1, 88) = 1.3$ ). There was no significant effect of feedback for avHR ( $F(1, 88) = 1.8$ ), mnHR ( $F(1, 88) = 3.1$ ) or mxHR ( $F < 1$ ). AvHR revealed a highly significant difference across time ( $F(3, 226) = 35.1, p < .001$ ), due to the 0-5 sec period producing significantly higher HR than the three remaining 5 sec periods (90.9, 86.9, 86.1 and 85.1 bpm for the 0-5, 5-10, 10-15 and 15-20 sec periods respectively). There were no interactions involving time for avHR (all  $F_s < 1$ ). MnHR revealed a significant difference across time ( $F(3, 235) = 20.1, p < .001$ ) and a two-way predictability by time interaction ( $F(3, 235) = 3.6, p < .05$ ). Analysis of the simple main effects revealed that the UP groups had slightly higher HR than the P groups during the 0-5 sec period following this, both P and UP groups produced a decrease in HR

Table 26. Mean cardiovascular activity during time periods of Trial 1. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Groups							
	PCFb	PCNFb	PNCfB	PNCNFb	UPCFb	UPCNFb	UPNCFb	UPNCNFb
Time Period 1								
AvHR	83.9 (16.9)	84.8 (17.6)	80.9 (12.5)	88.3 (14.7)	81.9 (9.76)	84.1 (18.8)	82.1 (15.3)	80.9 (13.0)
MnHR	78.4 (17.6)	77.6 (18.8)	73.6 (10.5)	81.5 (14.9)	77.2 (10.7)	78.4 (17.4)	76.1 (16.7)	74.8 (10.7)
MxHR	89.2 (17.5)	91.2 (17.0)	87.0 (13.4)	94.3 (14.5)	87.1 (10.2)	89.6 (20.1)	87.8 (14.7)	86.6 (16.0)
Time Period 2								
AvHR	82.1 (17.0)	80.3 (18.7)	75.6 (8.58)	83.0 (12.9)	77.9 (8.62)	81.1 (16.2)	78.0 (15.9)	79.7 (12.8)
MnHR	83.5 (17.4)	84.5 (15.8)	79.6 (13.6)	87.3 (15.6)	83.5 (10.2)	85.4 (17.0)	77.9 (13.9)	80.0 (12.3)
MxHR	82.1 (17.0)	80.3 (18.7)	75.6 (8.59)	83.0 (12.9)	77.9 (8.62)	81.1 (16.2)	78.0 (15.9)	79.7 (12.8)
Time Period 3								
AvHR	83.5 (17.4)	84.5 (15.8)	79.6 (13.6)	87.3 (15.6)	83.5 (10.2)	85.4 (17.0)	77.9 (13.9)	80.0 (12.3)
MnHR	77.8 (17.9)	75.5 (16.5)	72.1 (9.52)	79.5 (15.7)	76.9 (10.7)	77.5 (16.3)	70.4 (13.9)	72.6 (12.0)
MxHR	83.5 (17.4)	84.5 (15.8)	79.6 (13.6)	87.3 (15.6)	83.5 (10.2)	85.47 (17.0)	77.9 (13.9)	80.0 (12.3)

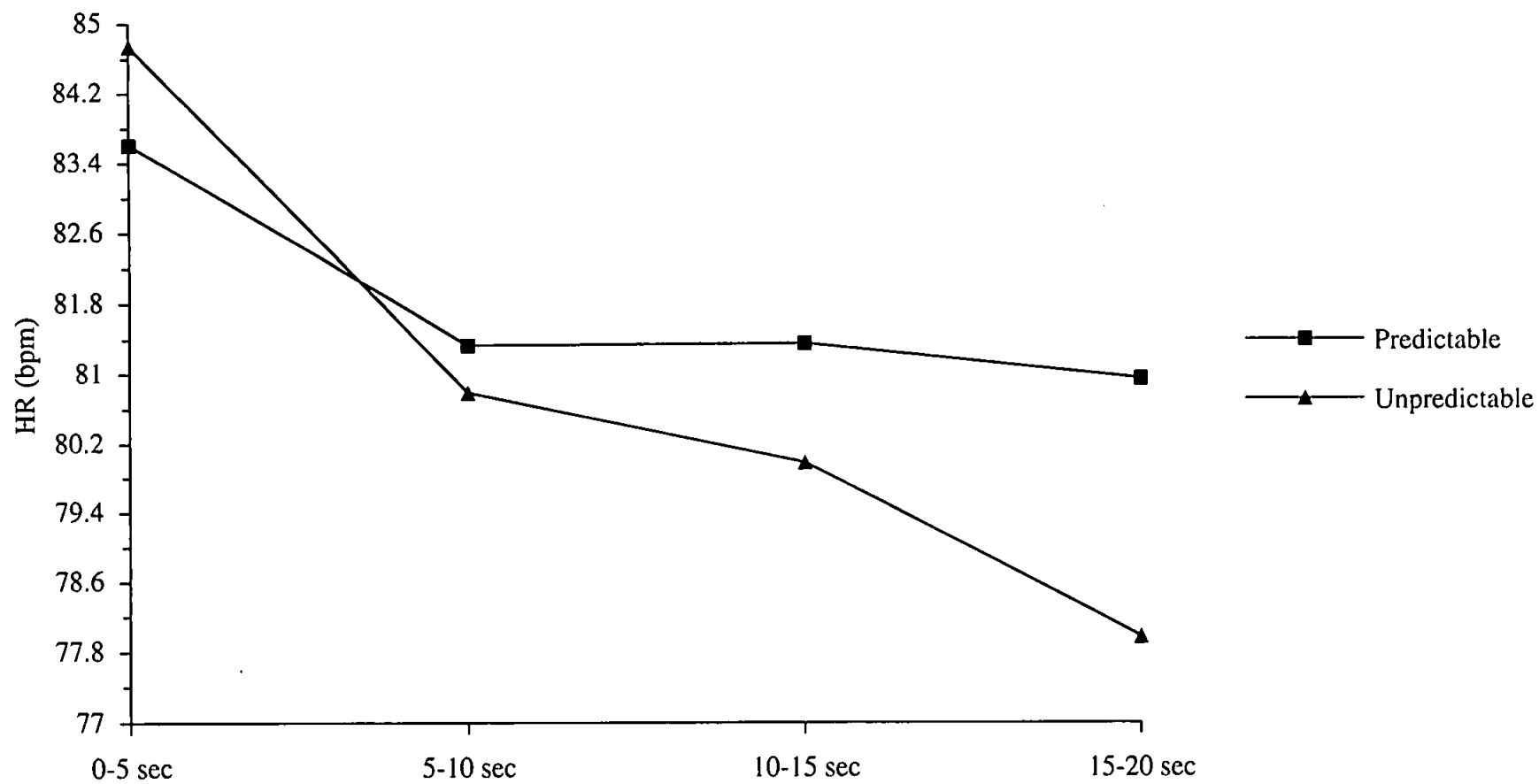
Time Period 4								
AvHR	85.1 (17.9)	87.5 (15.4)	82.0 (14.4)	89.6 (17.2)	82.1 (11.5)	86.7 (18.6)	82.5 (14.0)	82.5 (12.0)
MnHR	78.7 (19.5)	80.9 (16.3)	77.3 (14.8)	85.2 (17.5)	77.0 (11.2)	82.3 (18.2)	76.9 (13.7)	77.3 (12.0)
MxHR	92.5 (17.0)	95.3 (14.6)	87.5 (14.5)	93.9 (17.0)	86.5 (11.7)	92.3 (19.2)	87.3 (15.1)	87.4 (11.9)
Time Period 5								
AvHR	85.5 (17.7)	85.7 (16.1)	81.0 (15.0)	87.2 (19.0)	81.6 (10.3)	87.8 (19.2)	79.8 (16.0)	80.3 (12.4)
MnHR	77.7 (19.2)	79.5 (17.1)	75.7 (16.1)	82.4 (19.6)	76.7 (10.6)	83.7 (19.3)	74.9 (16.2)	74.7 (12.8)
MxHR	85.5 (17.7)	85.7 (16.1)	81.0 (15.0)	87.2 (19.0)	81.6 (10.3)	87.8 (19.2)	79.8 (16.0)	80.3 (12.4)
Time Period 6								
AvHR	88.6 (22.6)	89.6 (16.5)	85.3 (15.4)	89.3 (19.8)	85.7 (11.4)	90.0 (17.9)	82.0 (16.7)	84.8 (10.9)
MnHR	83.9 (22.9)	84.3 (16.5)	80.9 (15.7)	85.7 (21.0)	81.4 (12.3)	86.0 (18.2)	76.8 (16.4)	79.4 (10.4)
MxHR	93.2 (22.6)	95.3 (16.5)	91.4 (15.4)	93.5 (19.8)	90.3 (11.4)	94.8 (17.9)	88.2 (16.7)	90.6 (10.9)
Time Period 7								
AvHR	88.1 (19.1)	89.6 (14.2)	85.9 (10.5)	88.8 (18.6)	85.9 (15.5)	93.6 (15.9)	81.0 (10.3)	84.9 (13.2)
MnHR	81.4 (18.3)	83.6 (13.5)	78.4 (15.7)	83.9 (15.4)	80.5 (10.7)	88.7 (18.0)	74.6 (10.9)	79.8 (12.7)
MxHR	93.6 (18.8)	95.4 (14.9)	92.8 (15.2)	94.0 (16.4)	91.4 (11.5)	98.2 (18.6)	88.0 (11.8)	90.5 (13.9)

levels during the 5-10 sec period. The P groups then showed relatively stable HR levels during the 10-15 and 15-20 sec periods whereas, the UP groups produced a steady decrease in HR. The difference between the groups reached significance during the 15-20 sec period only (Figure 70). MnHR revealed no significant control by time ( $F(3, 235) = 2.4$ ), feedback by time or three-way interactions (all  $F_s < 1$ ). MxHR produced a highly significant difference across time ( $F(3, 226) = 25.3, p < .001$ ), due to the 0-5 sec period producing significantly higher HR than the three remaining 5 sec periods. The 5-10 sec period also had higher HR levels than the 10-15 and 15-20 sec periods whereas, there was no significant difference between the latter two periods (96.9, 93.0, 91.4 and 91.2 bpm for the 0-5, 5-10, 10-15 and 15-20 sec periods respectively). There were no interactions involving time for mxHR (all  $F_s < 1$ ).

3.2.1.3. Summary. There were no significant differences between groups across any of the time periods, with the exception of the ITI (Time Period 7). During the ITI, mnHR produced a predictability by time interaction which was due to the P groups having higher HR levels compared with the UP groups. However, the difference between the groups reached significance during the 15-20 sec period only.

Further examination of the pattern of responding across the pre-task baseline and Time Periods 1 to 7 revealed significant differences between the three cardiovascular measures (see Tables 25 & 26 respectively). For example, a significant increase from the pre-task baseline to the warning stimulus for both avHR ( $T(1, 95) = 5.4, p < .001$ ) and mnHR ( $T(1, 95) = 10.8, p < .001$ ). In contrast, mxHR produced a significant decrease from the baseline to warning onset ( $T(1, 95) = 3.4, p < .01$ ). Comparisons between the pre-task baseline and onset of the noise revealed a similar pattern: a significant increase for both avHR ( $T(1, 95) = 5.8, p < .001$ ) and mnHR ( $T(1, 95) = 10.3, p < .001$ ) compared with a non-significant decrease for mxHR ( $T < 1$ ). All three measures revealed a significant increase from the warning stimulus to noise onset: avHR ( $T(1, 95) = 3.2, p < .01$ ), mnHR ( $T(1, 95) = 3.8, p < .01$ ) and mxHR ( $T(1, 95) = 2.4$ ).

Figure 70. Mean minimum HR as a function of predictability and time during Time Period 7, Trial 1.



### 3.2.2. Corrected HR

3.2.2.1. Time Period 1. There were no main effects of predictability, control or feedback involving any of the three cardiovascular measures (all  $F_s < 1$ ). There were no predictability by control interactions involving avHR ( $F(1, 88) = 1.8$ ), mnHR ( $F < 1$ ) or mxHR ( $F(1, 88) = 2.4$ ), nor were there any significant predictability by feedback interactions involving avHR, mxHR (both  $F_s < 1$ ) or mnHR ( $F(1, 88) = 2.3$ ). There were no significant control by feedback interactions involving avHR, mnHR (both  $F_s < 1$ ) or mxHR ( $F(1, 88) = 2.2$ ), nor were there any three-way interactions (all  $F_s < 1$ ).

3.2.2.2. Time Period 2. There were no significant effects of predictability, control or feedback involving any of the three measures (all  $F_s < 1$ ). There was a significant predictability by control interaction involving avHR ( $F(1, 88) = 6.9, p = .01$ ) and mxHR ( $F(1, 88) = 7.5, p < .001$ ). Simple main effects revealed, firstly for avHR, that the PNC groups had significantly greater decreases in HR from the baseline period compared with the PC and UPNC groups. The UPC groups also had significantly greater decreases in HR levels compared with the UPNC groups. There were no other significant differences (Figure 71). MxHR revealed similar results to avHR, with the PNC groups producing significantly greater decreases in HR compared with the PC and UPNC groups (Figure 72). MnHR revealed no predictability by control interaction ( $F(1, 88) = 2.5$ ). There were no other interactions involving any of the measures (all  $F_s < 1$ ).

3.2.2.3. Time Period 3. There were no effects of predictability involving any of the measures (all  $F_s < 1$ ). MxHR produced a main effect of control ( $F(1, 88) = 7.1, p < .01$ ). The C groups showed smaller decreases in HR from the baseline period compared with the NC groups (-0.26 and -4.67 change in bpm respectively). There were no significant effects of control involving avHR ( $F(1, 88) = 2.8$ ) or mnHR ( $F < 1$ ). AvHR and mnHR produced no significant effect of feedback (both  $F_s < 1$ ), nor did mxHR ( $F(1, 88) = 2.1$ ). There were no other interactions involving any of the measures (all  $F_s < 1$ ).

3.2.2.4. Time Period 4. There were no significant effects of predictability or feedback involving any of the three measures (all  $F_s < 1$ ), nor were there any main effects of control involving avHR ( $F < 1$ ), mnHR ( $F(1, 88) = 1.4$ ) or mxHR ( $F(1, 88) = 2.2$ ). MxHR produced a two-way predictability by control interaction ( $F(1, 88) = 4.9, p < .05$ ). The PNC, UPC and UPNC groups had significantly greater decreases in HR levels from



Figure 71. Mean change in average HR as a function of groups during Time Period 2, Trial 1.

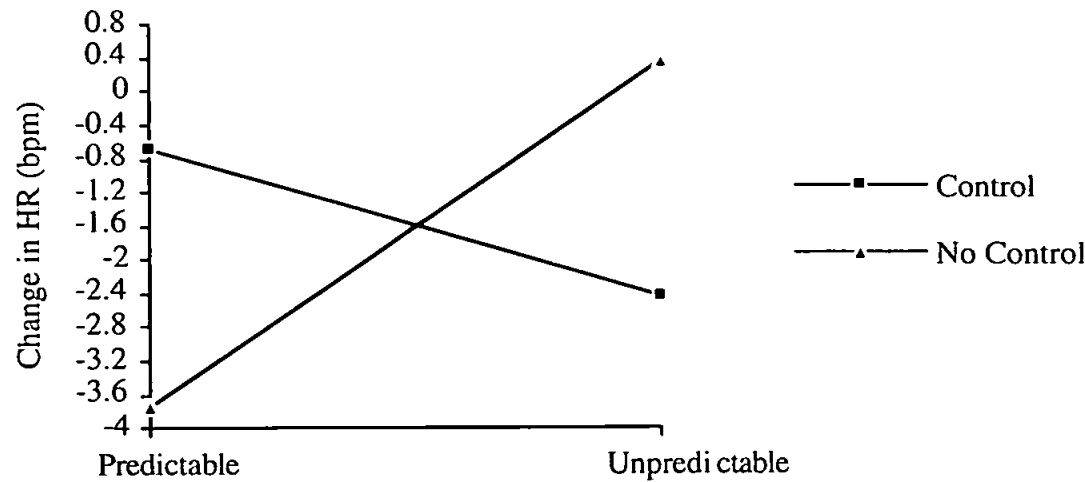


Figure 72. Mean change in maximum HR as a function of groups during Period 2, Trial 1.

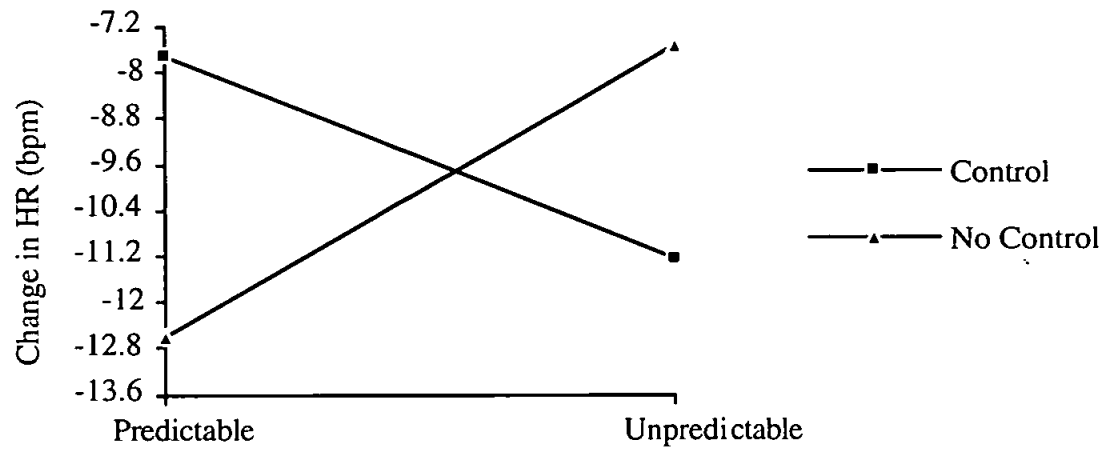
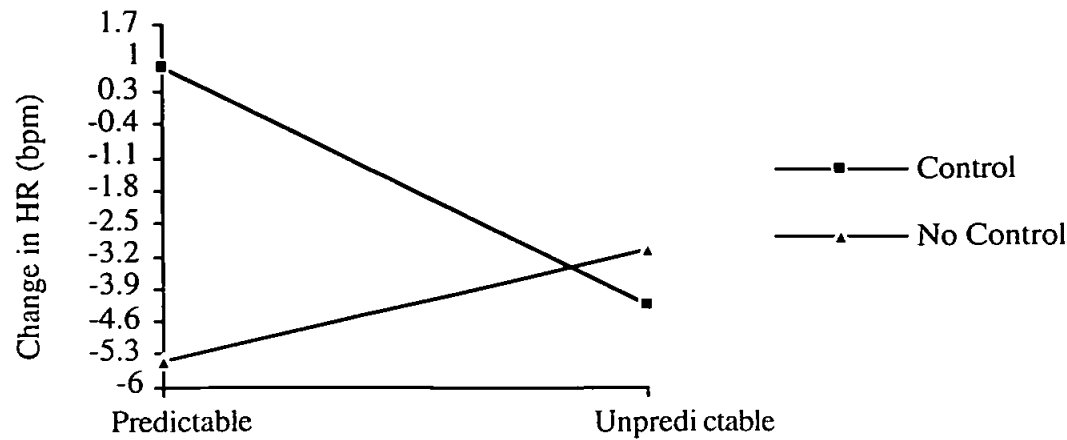


Figure 73. Mean change in maximum HR as a function of groups during Time Period 4, Trial 1.



the pre-task baseline compared with the PC group. There were no significant differences between the remaining groups (Figure 73). AvHR and mnHR produced no predictability by control interactions ( $F(1, 88) = 1.6$ ) and ( $F < 1$ ) respectively. There were no significant predictability by feedback interactions involving any of the three measures (all  $F_s < 1$ ). AvHR and mnHR produced no control by feedback interactions (all  $F_s < 1$ ), nor did mxHR ( $F(1, 88) = 3.7$ ). There were no three-way interactions involving any of the measures (all  $F_s < 1$ ).

3.2.2.5. Time Period 5. There were no significant main effects of predictability or feedback involving any of the cardiovascular measures (all  $F_s < 1$ ). There were no significant effects of control involving avHR ( $F(1, 88) = 1.7$ ), mnHR ( $F < 1$ ) or mxHR ( $F(1, 88) = 2.5$ ). There were no other interactions involving any of the measures (all  $F_s < 1$ ).

3.2.2.6. Time Period 6. There were no main effects or interactions involving any of the three measures (all  $F_s < 1$ ).

3.2.2.7. Time Period 7. The results were similar to those of uncorrected HR, they are therefore not reported in detail here.

3.2.3. Corrected HR Summary. Compared with uncorrected HR, corrected HR produced significant differences between groups involving all three measures. During Time Period 2, corrected avHR and mxHR produced a two-way predictability by control interaction. This was due to the PC and UPNC groups produced significantly smaller decreases in HR from the pre-task baseline compared with the remaining two groups. A similar pattern emerged for corrected mxHR during Time Period 4. The PC group produced an increase in HR from the baseline period compared with the remaining groups, which all produced decreases in HR.

During Time Period 3, corrected mxHR produced a significant effect of control in which the C groups had smaller decreases in HR levels from the pre-task baseline compared with the NC groups.

The results of Time Periods 1, 5, 6 and 7 were similar to uncorrected HR.

3.2.4. Reaction Time. Between-group differences in RT were assessed using a three-way ANOVA with control, predictability and feedback as between subject factors. There were no significant effects of control, predictability or feedback (all  $F_s < 1$ ), nor were there any predictability by control, predictability by feedback (both  $F_s < 1$ ) or control by feedback ( $F(1, 88) = 3.4$ ) interactions. The three-way interaction was not significant ( $F < 1$ ). Group means are shown in Figure 74.

### 3.3. Between-Groups Analysis: Trials 5 & 8.

For all measures, Time Periods 1 to 6 were each analysed in a four-way MANOVA with predictability, control and feedback as between subject factors and trials as a repeated measure. Time Periods 7 and 8 involved the same factors as above but included time (4 and 10 levels respectively) as a second repeated measure. Uncorrected HR means across Time Periods 1 to 7 are shown in Table 27.

#### 3.3.1. Uncorrected HR

3.3.1.1. Time Period 1. There were no significant differences between groups involving any of the measures (all  $F_s < 1$ ). AvHR and mnHR produced no significant difference across trials or any interactions involving trials (all  $F_s < 1$ ). MxHR produced no significant difference across trials, nor any predictability by trials (all  $F_s < 1$ ), control by trials ( $F(1, 88) = 3.7$ ), feedback by trials or any higher order interactions (all  $F_s < 1$ ).

3.3.1.2. Time Period 2. There were no main effects of predictability, control or feedback involving any of the three measures (all  $F_s < 1$ ). AvHR produced no significant differences across trials, nor any two-way interactions (all  $F_s < 1$ ). There were no significant predictability by control by trials ( $F < 1$ ), predictability by feedback by trials ( $F(1, 88) = 2.2$ ) or control by feedback by trials interactions ( $F(1, 88) = 2.3$ ) involving avHR. MnHR produced no significant difference across trials, nor any two-way interactions involving trials (all  $F_s < 1$ ). However, there were significant predictability by feedback by trials ( $F(1, 88) = 4.1, p < .05$ ) and control by feedback by trials interactions ( $F(1, 88) = 4, p < .05$ ). Analysis of the simple simple main effects, firstly for the predictability by feedback by trials interaction, revealed that during Trial 5 the PFb, PNFB

Figure 74. Mean response times as a function of groups during Trial 1.

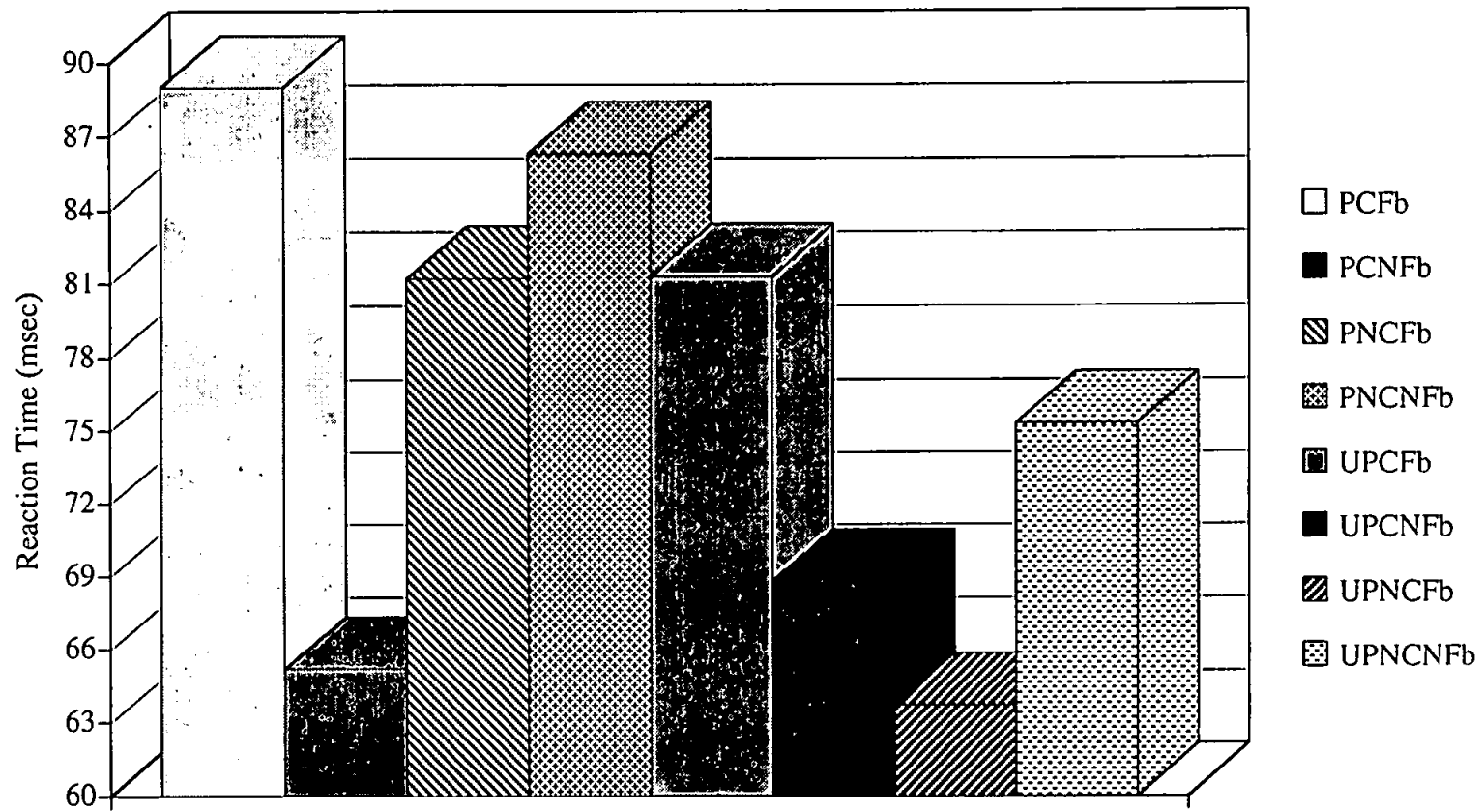


Table 27. Mean cardiovascular activity during time periods averaged across Trials 5 and 8. Standard deviations are shown in parentheses.

Variables (HR (bpm))	Experimental Groups							
	PCFb	PCNFb	PNCfB	PNCNFb	UPCFb	UPCNFb	UPNCFb	UPNCNFb
Time Period 1								
AvHR	80.8 (15.5)	81.3 (11.9)	81.6 (10.5)	85.6 (11.8)	80.0 (8.54)	82.8 (15.4)	78.3 (11.9)	78.0 (10.2)
MnHR	75.7 (14.4)	74.8 (11.9)	75.5 (10.9)	79.6 (9.98)	73.6 (9.73)	78.0 (14.3)	73.2 (11.0)	73.1 (10.1)
MxHR	86.6 (17.4)	86.7 (11.8)	87.0 (11.8)	91.2 (11.1)	86.3 (8.50)	86.7 (16.1)	82.9 (12.6)	82.9 (11.0)
Time Period 2								
AvHR	79.0 (14.1)	78.3 (11.9)	81.2 (13.9)	81.8 (9.37)	76.6 (8.42)	79.9 (15.1)	76.5 (10.1)	78.0 (8.11)
MnHR	74.3 (13.7)	74.5 (12.9)	76.3 (13.2)	77.7 (8.32)	73.3 (8.56)	77.0 (15.5)	72.2 (11.1)	74.4 (9.78)
MxHR	84.2 (15.9)	82.3 (11.8)	85.4 (13.5)	86.1 (10.1)	80.6 (9.16)	83.1 (14.9)	81.6 (12.5)	81.7 (9.00)
Time Period 3								
AvHR	80.3 (16.1)	81.6 (10.4)	80.6 (8.90)	85.0 (13.9)	79.4 (7.32)	82.5 (16.6)	74.9 (9.98)	77.7 (10.2)
MnHR	72.2 (14.8)	71.5 (12.0)	71.5 (6.90)	77.2 (9.78)	72.5 (7.97)	76.6 (16.2)	67.2 (9.99)	71.2 (9.67)
MxHR	88.0 (18.8)	90.8 (12.8)	89.7 (12.9)	94.0 (9.98)	86.9 (8.12)	89.4 (17.4)	83.9 (11.2)	86.0 (11.9)

Time Period 4								
AvHR	79.9 (15.8)	82.5 (13.4)	80.2 (8.98)	85.3 (11.8)	77.9 (7.33)	81.6 (16.1)	75.1 (9.91)	76.5 (10.1)
MnHR	73.2 (15.1)	75.9 (12.8)	73.9 (7.21)	80.5 (12.4)	73.6 (8.00)	78.1 (16.6)	70.5 (9.93)	72.2 (9.90)
MxHR	86.4 (16.7)	88.9 (13.8)	85.9 (10.2)	90.6 (10.9)	82.7 (7.82)	85.6 (15.9)	79.6 (9.99)	81.2 (11.1)
Time Period 5								
AvHR	79.3 (14.9)	82.9 (10.7)	77.7 (7.89)	85.0 (9.98)	78.1 (8.11)	81.6 (16.5)	73.5 (8.70)	76.9 (10.2)
MnHR	73.4 (14.1)	75.4 (10.1)	71.9 (6.70)	79.8 (10.9)	72.2 (9.55)	76.7 (16.9)	69.0 (8.93)	72.2 (10.9)
MxHR	85.4 (17.1)	90.1 (12.0)	84.4 (9.90)	90.6 (10.1)	84.0 (8.44)	87.5 (15.3)	78.7 (9.99)	82.5 (10.7)
Time Period 6								
AvHR	80.8 (15.9)	79.7 (13.4)	79.2 (9.61)	83.1 (10.5)	77.9 (8.90)	82.5 (17.1)	74.4 (9.55)	78.3 (10.8)
MnHR	75.7 (15.4)	75.3 (14.1)	74.9 (9.21)	80.0 (10.5)	73.1 (9.21)	79.1 (16.1)	70.9 (9.11)	74.8 (10.1)
MxHR	86.6 (17.0)	84.1 (13.5)	83.1 (10.2)	86.4 (10.8)	82.6 (8.40)	86.3 (17.1)	77.7 (10.4)	81.8 (11.9)
Time Period 7								
AvHR	81.7 (15.8)	80.2 (11.9)	80.3 (9.09)	84.2 (14.8)	77.5 (8.90)	80.1 (8.78)	73.8 (9.50)	76.9 (10.0)
MnHR	75.2 (13.9)	74.7 (11.3)	73.8 (9.29)	79.4 (14.1)	72.3 (8.91)	75.5 (8.66)	68.3 (9.51)	72.2 (9.70)
MxHR	88.5 (17.0)	86.1 (12.3)	87.1 (8.61)	89.0 (15.0)	83.2 (10.9)	84.7 (9.89)	79.8 (11.9)	81.9 (10.9)

and UPNFb groups had significantly higher HR levels than the UPFb group. There were no significant differences between the remaining three groups during Trial 5, nor were there any in Trial 8. Further comparison of means across trials reveals that the UPFb group had significantly higher HR during Trial 8 compared with Trial 5 whereas, there were no significant differences across trials involving the remaining groups (Figure 75).

Simple simple main effects of the control by feedback by trials interaction, firstly for Trial 5, revealed that the NCNFb group had significantly higher HR than the CFb and NCFb groups. There were no significant differences between the remaining groups. During Trial 8, the CNFb group produced significantly greater HR activity than the CFb group whereas, there were no significant differences between the NC groups. Further comparison of means across trials revealed that the NCFb group had significantly higher HR during Trial 8 compared with Trial 5 whereas, there were no significant differences across trials involving the remaining three groups (Figure 76). MxHR produced no significant effect of trials or any interaction involving trials (all  $F_s < 1$ ).

3.3.1.3. Time Period 3. There were no significant effects of predictability involving avHR ( $F(1, 88) = 1.9$ ), mnHR ( $F < 1$ ) or mxHR ( $F(1, 88) = 2.5$ ), nor were there any main effects of control involving any of the three measures (all  $F_s < 1$ ). None of the measures produced an effect of feedback; avHR ( $F(1, 88) = 1.5$ ), mnHR ( $F(1, 88) = 2.2$ ) and mxHR ( $F(1, 88) = 1.2$ ). There were no predictability by control interactions involving avHR ( $F(1, 88) = 1.9$ ), mnHR ( $F(1, 88) = 3.1$ ) or mxHR ( $F(1, 88) = 1.2$ ), nor were there any significant predictability by feedback or control by feedback interactions involving any of the cardiovascular measures (all  $F_s < 1$ ). AvHR and mxHR produced no significant difference across trials or any interactions involving trials (all  $F_s < 1$ ). MnHR produced no significant effect of trials, nor any predictability by trials (all  $F_s < 1$ ), control by trials ( $F(1, 88) = 2.5$ ), feedback by trials or any higher order interactions (all  $F_s < 1$ ).

3.3.1.4. Time Period 4. There were no significant main effects of predictability for avHR ( $F(1, 88) = 3.1$ ) or mnHR ( $F < 1$ ), although the effect did reach significance for mxHR ( $F(1, 88) = 5.4, p < .05$ ). The P groups had higher HR than the UP groups (87.9 and 82.2 bpm respectively). There were no effects of control involving any of the three measures (all  $F_s < 1$ ), nor were there significant effects of feedback involving avHR ( $F(1,$

Figure 75. Mean minimum HR as a function of predictability, feedback and trials during Time Period 2.

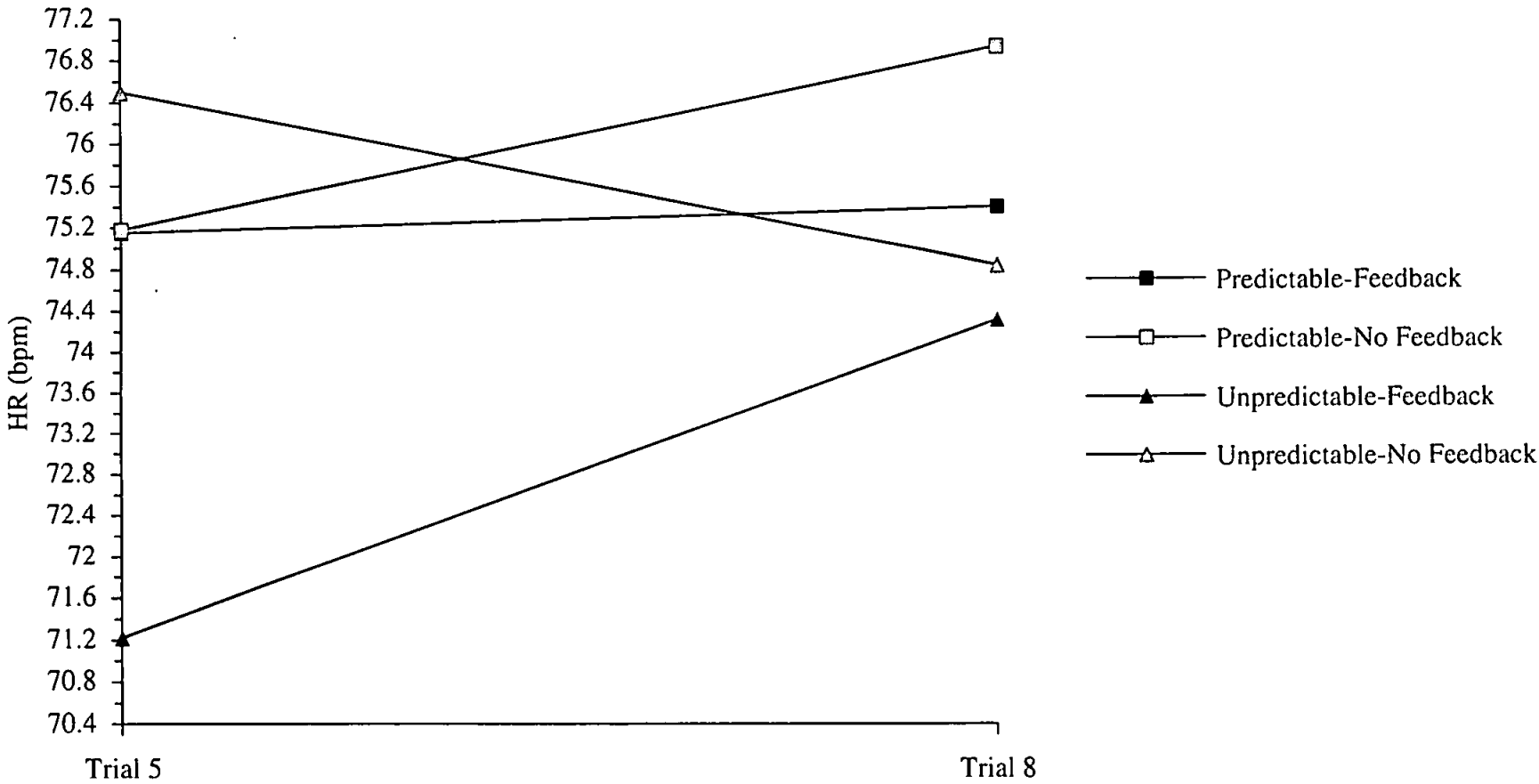
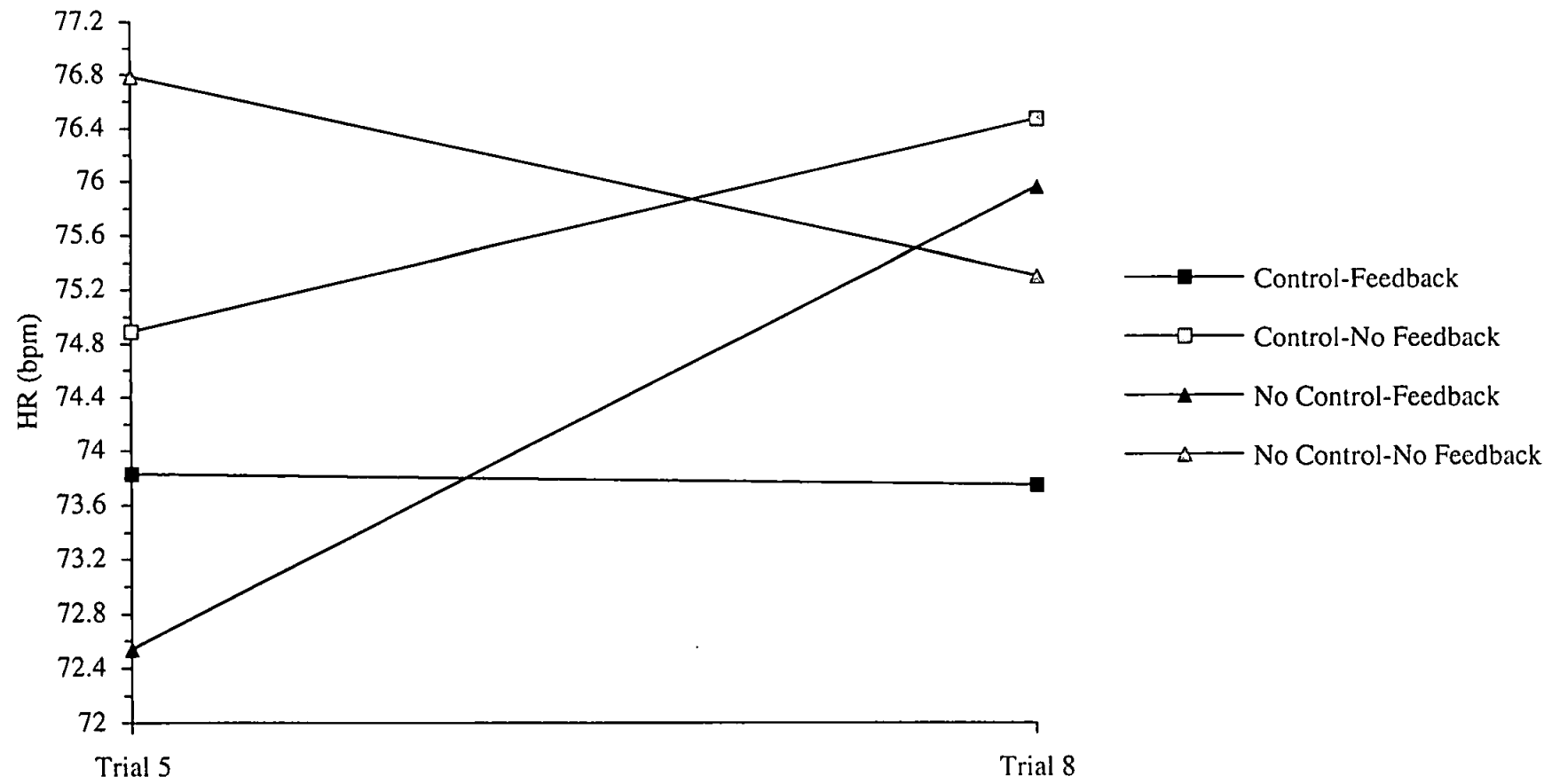




Figure 76. Mean minimum HR as a function of control, feedback and trials during Time Period 2.



88) = 1.9), mnHR ( $F(1, 88) = 2.7$ ) or mxHR ( $F(1, 88) = 1.5$ ). None of the measures revealed a difference across trials or any interaction involving trials (all  $F_s < 1$ ).

3.3.1.5. Time Period 5. There were no significant effects of predictability involving avHR ( $F(1, 88) = 2.9$ ), mnHR ( $F(1, 88) = 1.4$ ) or mxHR ( $F(1, 88) = 3.7$ ), nor were there any effects of control involving the three measures (all  $F_s < 1$ ). There were significant effects of feedback for avHR ( $F(1, 88) = 4, p < .05$ ), mnHR ( $F(1, 88) = 4.1, p < .05$ ) and mxHR ( $F(1, 88) = 4.0, p < .05$ ). The NFb groups had higher HR than the Fb groups (avHR = 81.6 and 77.1 bpm; mnHR = 76.0 and 71.6 bpm; mxHR = 87.7 and 83.1 bpm respectively). There were no significant two-way predictability by control interactions involving any of the three measures; avHR ( $F(1, 88) = 1.2$ ), mnHR ( $F(1, 88) = 1.5$ ) and mxHR ( $F(1, 88) = 1.1$ ), nor were there any predictability by feedback or control by feedback interactions (all  $F_s < 1$ ). There were no significant differences across trials, nor any predictability by trials or feedback by trials interactions involving any of the three measures (all  $F_s < 1$ ). There was a control by trials interaction involving avHR ( $F(1, 88) = 7.5, p < .01$ ), mnHR ( $F(1, 88) = 4.6, p < .05$ ) and mxHR ( $F(1, 88) = 8.2, p < .01$ ). Simple main effects revealed a similar pattern across all three measures, with the C groups producing significantly higher HR levels than the NC groups during Trial 8 compared with no significant difference during Trial 5. Further comparison of means across trials revealed no significant differences between Trials 5 and 8 involving the C groups whereas, the NC groups produced significantly higher HR during Trial 5 compared with Trial 8. Group means across trials are shown in Figures 77 to 79 for avHR, mnHR and mxHR respectively. AvHR produced no predictability by control by trials ( $F(1, 88) = 1.99$ ), predictability by feedback by trials ( $F(1, 88) = 2.9$ ), control by feedback by trials or any four-way interaction (all  $F_s < 1$ ). MnHR produced no predictability by control by trials, control by feedback by trials (all  $F_s < 1$ ), predictability by feedback by trials ( $F(1, 88) = 2.4$ ) or four-way interaction ( $F < 1$ ). MxHR produced no predictability by control by trials ( $F(1, 88) = 3.1$ ), predictability by feedback by trials ( $F(1, 88) = 1.8$ ), control by feedback by trials or a four-way interaction (all  $F_s < 1$ ).

3.3.1.6. Time Period 6. There were no significant effects of predictability or control involving any of the three measures (all  $F_s < 1$ ), nor were there any main effects of feedback involving avHR ( $F(1, 88) = 1.4$ ), mnHR ( $F(1, 88) = 2.5$ ) or mxHR ( $F < 1$ ).

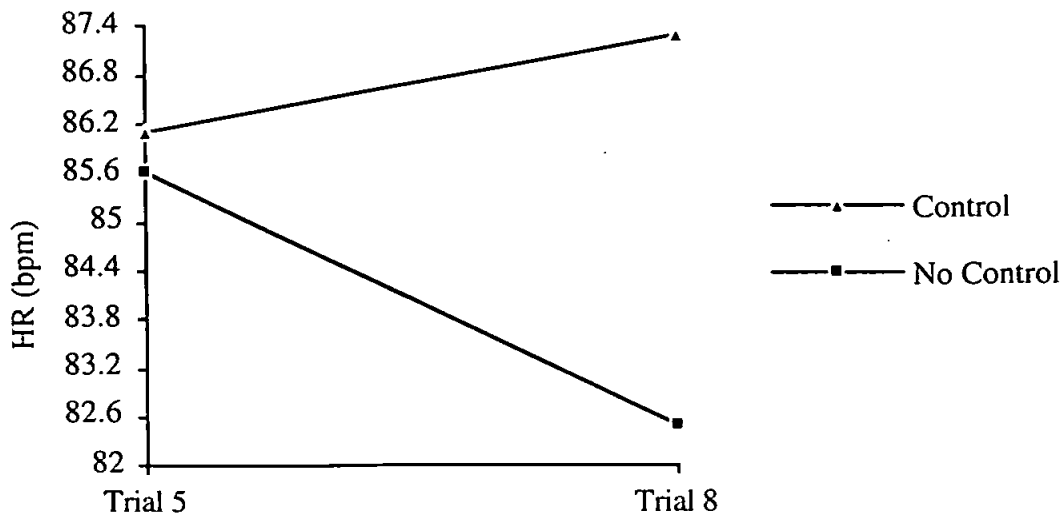
Figure 77. Average HR as a function of control and trials during Time Period 5.



Figure 78. Mean minimum HR as a function of control and trials during Time Period 5.



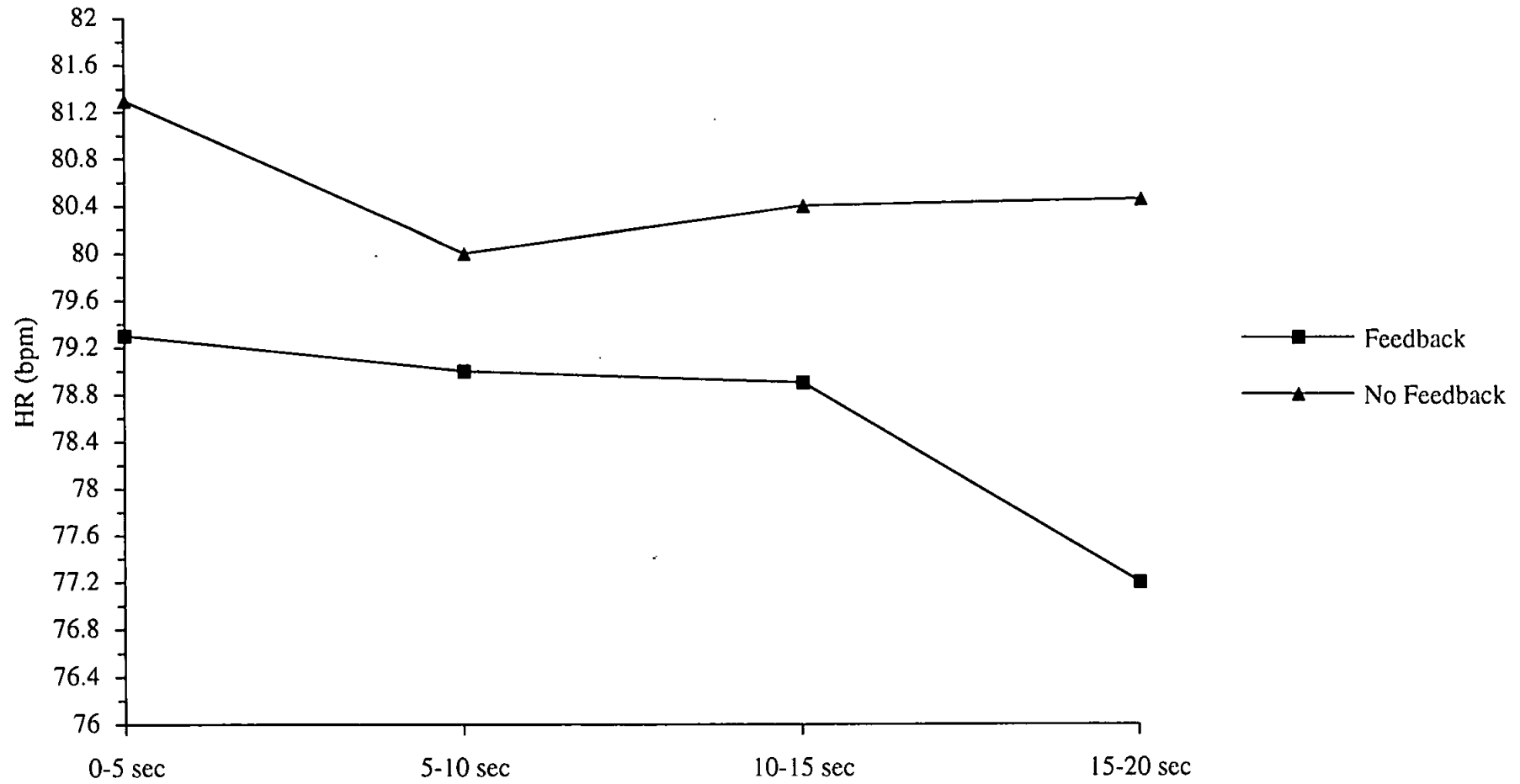
Figure 79. Mean maximum HR as a function of control and trials during Time Period 5.



None of the measures produced a significant difference across trials or any other interactions involving trials (all  $F_s < 1$ ).

3.3.1.7. Time Period 7. There were significant effects of predictability involving avHR ( $F(1, 88) = 4.3, p < .05$ ) and mxHR ( $F(1, 88) = 5, p < .05$ ). The P groups had higher HR than the UP groups (avHR = 81.7 and 77.1 bpm; mxHR = 87.7 and 82.4 bpm respectively). MnHR revealed no significant effect of predictability ( $F(1, 88) = 3.1$ ). There were no main effects of control or feedback involving any of the three measures (all  $F_s < 1$ ). AvHR and mnHR revealed no difference across trials or any interaction involving trials (all  $F_s < 1$ ). MxHR revealed no significant difference across trials ( $F(1, 88) = 3.6$ ), nor any interaction involving trials (all  $F_s < 1$ ). AvHR produced a significant difference across time ( $F(3, 236) = 6.3, p < .01$ ) and a feedback by time interaction ( $F(3, 236) = 2.7, p < .05$ ). Simple main effects revealed the NFb groups had higher HR levels than the Fb groups across all four 5 sec periods, although this reached significance during the 0-5, 10-15 and 15-20 sec periods only. Further comparison of means revealed that the Fb groups had significantly greater HR levels during the 0-5, 5-10 and 10-15 sec periods compared with the 15-20 sec period. There were no significant differences across the first three periods. The NFb group produced higher HR during the 0-5 sec compared with the 5-10 sec period whereas, there were no significant differences between the remaining periods (Figure 80). There were no other interactions involving time for avHR (all  $F_s < 1$ ). MnHR revealed no significant difference across time ( $F(2, 208) = 2.7$ ), however, there was a trials by time interaction ( $F(3, 223) = 4.5, p < .01$ ). Simple main effects revealed, firstly for Trial 5, that the 0-5 sec period had significantly greater HR activity than the remaining three, 5 sec periods. The 5-10 and 10-15 sec periods also had significantly higher levels of activity than the 15-20 sec period. During Trial 8, there were no significant differences between the four, 5 sec periods. Further comparison of means across trials revealed that during the 0-5 sec period Trial 5 produced significantly higher HR than Trial 8 whereas, during the 5-10 and 10-15 sec periods there were no significant differences between the trials. During the 15-20 sec period, Trial 8 produced higher HR than Trial 5 (Figure 81). There were no other interactions involving mnHR (all  $F_s < 1$ ). MxHR produced a main effect of time ( $F(3, 257) = 7, p < .01$ ). Further comparison of means revealed that the 0-5

Figure 80. Average HR as a function of feedback and time during Time Period 7.



3.2.2.3. SCR Amplitude. There were no main effects of control involving any of the three time periods (all  $F_s < 1$ ), or predictability during Time Period 1 ( $F(1, 52) = 3.1$ ), Time Period 2 ( $F(1, 52) = 2.4$ ) or Time Period 3 ( $F(1, 52) = 3.8$ ). There were no two-way control by predictability interactions during any of the time periods (all  $F_s < 1$ ).

3.2.3. Reaction Time. Between-group differences in RT were assessed using a two-way ANOVA with predictability and control as between subject factors. There were no significant effects or any interaction (all  $F_s < 1$ ) (66.1, 67.9, 72.4 and 57.3 msec for the PC, PNC, UPC and UPNC groups respectively).

### 3.3. Between-Groups Analysis: Trials 5, 8 & 13.

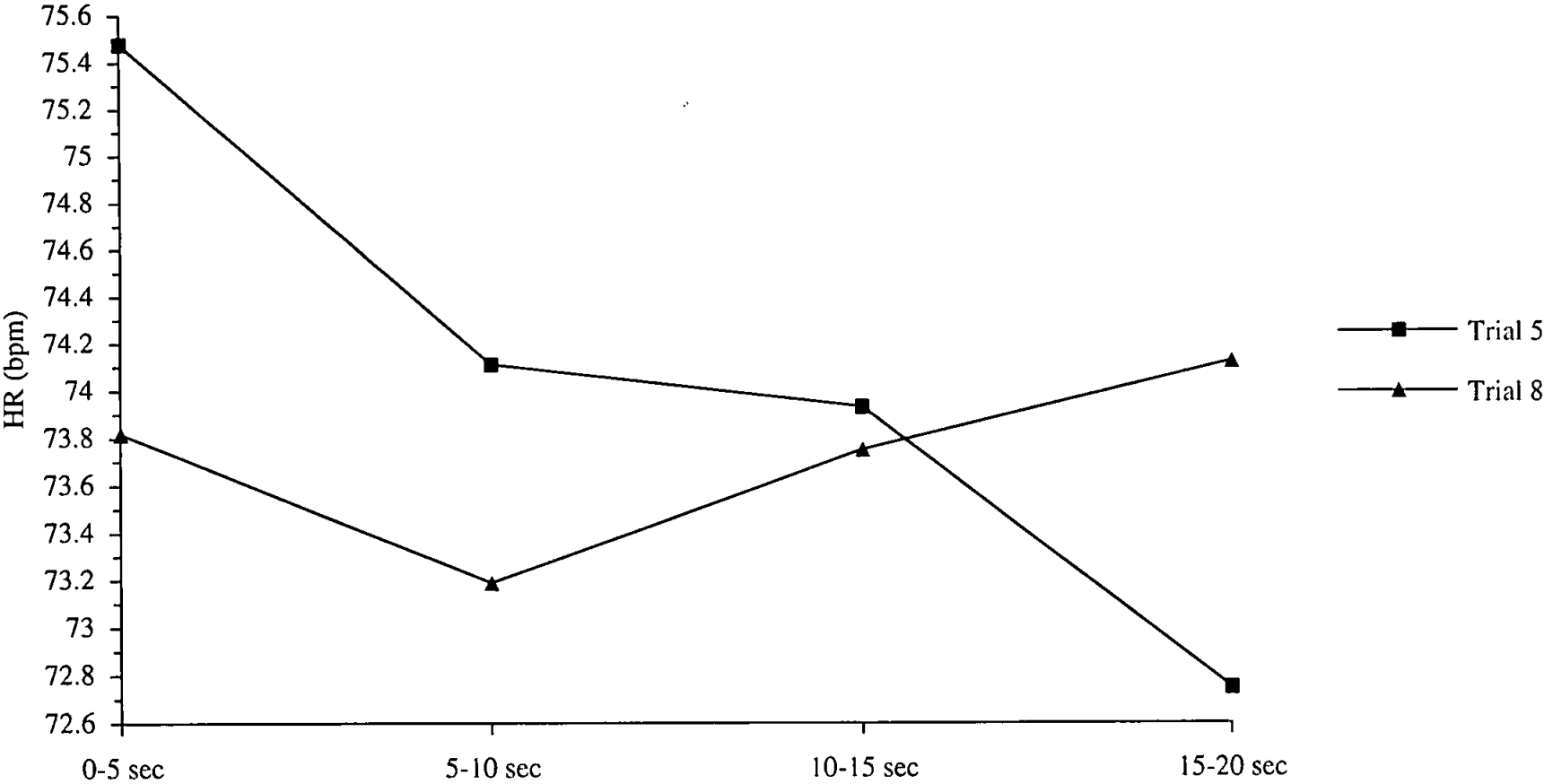
#### 3.3.1. Cardiovascular Activity

For all measures, Time Periods 1 to 4 were each analysed in a three-way MANOVA with control and predictability as between subject factors and trials (Trials 5, 8 & 13) as a repeated measure. Time Periods 5 and 6 were assessed in a four-way MANOVA with the same between subject factors as above and trials and time (5 and 10 levels respectively) as repeated measures. Group means and standard deviations for uncorrected HR across Time Periods 1 to 5 are shown in Table 21.

##### 3.3.1.1. Uncorrected HR

3.3.1.1.1. Time Period 1. There were no significant effects of control involving any of the three measures; avHR ( $F(1, 52) = 1.7$ ), mnHR ( $F(1, 52) = 2.2$ ) and mxHR ( $F(1, 52) = 1.1$ ), nor were there any significant effects of predictability involving any of the measures (all  $F_s < 1$ ). There were significant control by predictability interactions involving avHR ( $F(1, 52) = 4.7, p < .05$ ) (Figure 30), mnHR ( $F(1, 52) = 4.9, p < .05$ ) (Figure 31) and mxHR ( $F(1, 52) = 4.5, p < .05$ ) (Figure 32). Analysis of the simple main effects revealed a similar pattern for all three cardiovascular measures, with the PC and UPNC groups producing markedly higher HR than the PNC group and to a lesser extent

Figure 81. Mean minimum HR as a function of trials and time during Time Period 7.



and 5-10 sec periods produced significantly higher HR than the 15-20 sec period. There were no significant differences between the remaining periods (86.1, 85.3, 85.0 and 83.8 bpm for the 0-5, 5-10, 10-15 and 15-20 sec periods respectively). There were no interactions involving trials or time for mxHR (all  $F$ s < 1).

3.3.1.8. Time Period 8. There were no significant effects of predictability ( $F < 1$ ) or control ( $F(1, 88) = 3.2$ ) involving mean change in avHR. There was a significant effect of feedback ( $F(1, 88) = 8.1, p < .01$ ), two-way predictability by feedback ( $F(1, 88) = 5.1, p < .05$ ) and control by trials interactions ( $F(1, 88) = 7.1, p < .01$ ) and a predictability by control by feedback by trials interaction ( $F(1, 88) = 5.4, p < .05$ ). Further inspection of the four-way interaction (Figure 82) revealed, firstly for Trial 5, that the PCFb and PNCfB groups had greater increases in HR from the pre-stimulus period compared with the remaining groups, although none of these comparisons reached significance. During Trial 8, all groups produced greater increases in HR compared with the PCNFb group, although only the PCNFb-PCFb and PCNFb-PNCfB comparisons reached significance. The PCFb and PNCfB groups also had greater HR levels than the PNCNFb, UPCfB, UPNCfB, UPNCfB and UPNCNFb groups, although this did not reach statistical significance. Further comparisons across trials revealed no significance differences involving any of the groups. There was a significant difference across time ( $F(4, 352) = 9.7, p < .01$ ), a predictability by time ( $F(4, 352) = 4.2, p < .01$ ) and a predictability by control by trials by time interaction ( $F(5, 417) = 2.6, p < .05$ ). Further inspection of the four-way interaction (Figure 83) revealed, firstly for Trial 5, that the UPNC group had significantly greater HR than all remaining groups during Second 1. In addition, during Second 1 the UPC group produced a significantly greater increase in HR compared with the PNC group. During Seconds 2 and 3 the PC, UPC and UPNC groups had significantly greater HR than the PNC group. In addition, during Second 2, the UPNC group produced significantly greater increases in HR compared to the UPC group. During Seconds 4 and 5 there were no significant differences between any of the groups. The PC and PNC groups had significantly greater increases in HR during Seconds 6, 7, 8, 9 and 10 compared with the UPC group. In addition, during Seconds 8 and 9 the PC and PNC groups also produced significantly greater HR compared to the UPNC group. During Second 10, the PC group had a significantly greater increase in HR compared with the three remaining groups



Figure 82. Mean change in average HR as a function of groups and trials during Time Period 8.

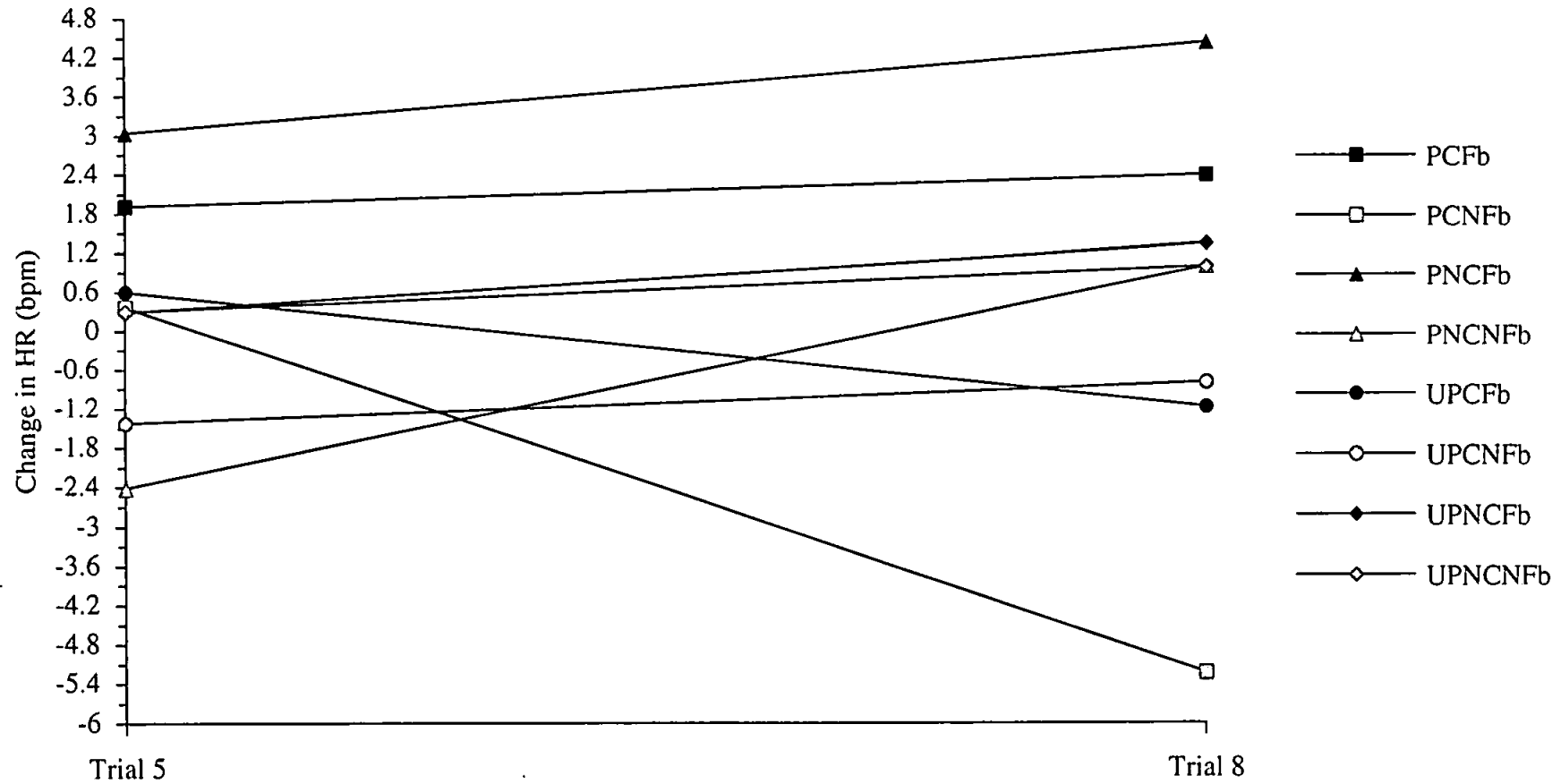
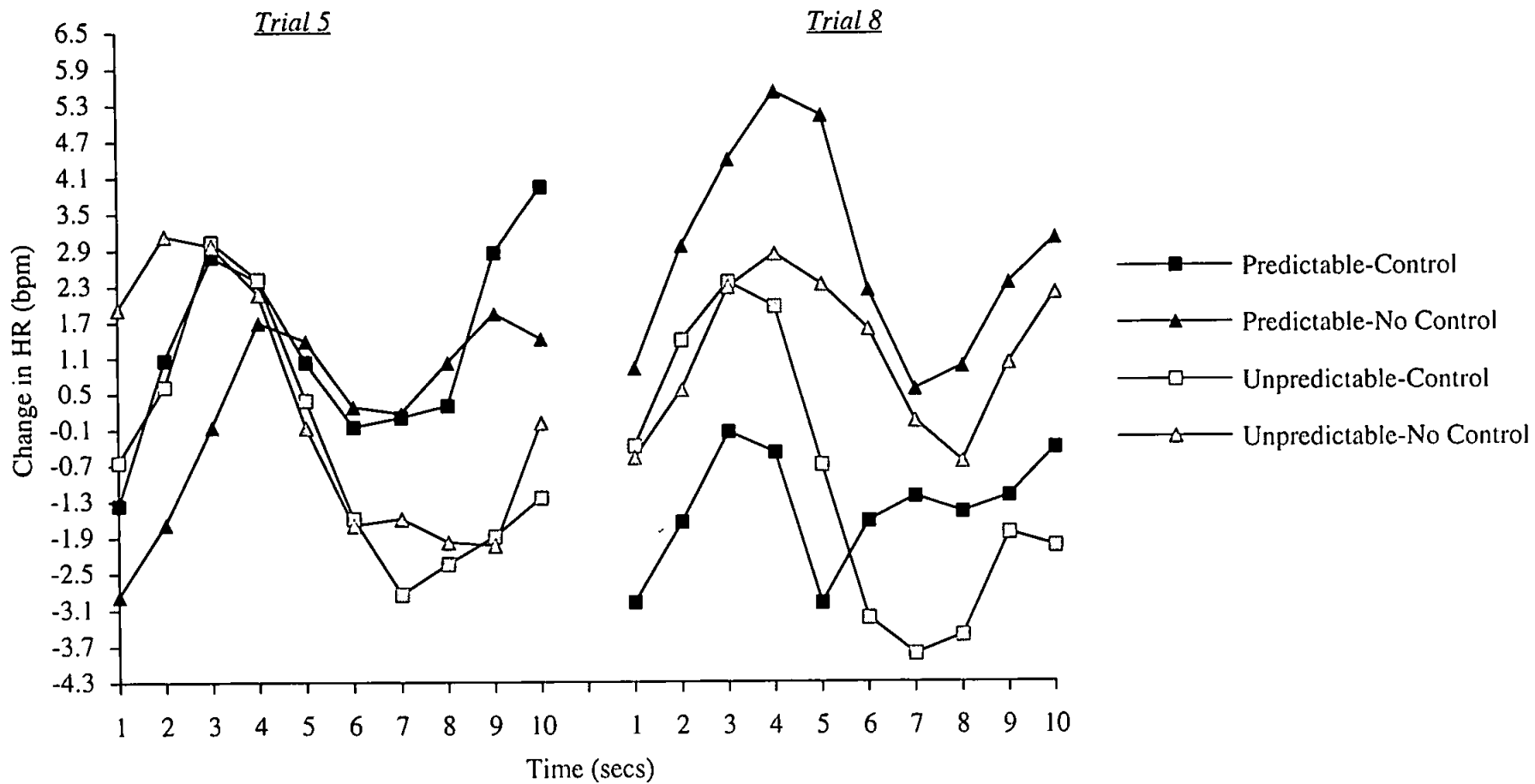


Figure 83. Mean change in average HR as a function of predictability, control, trials and time during Time Period 8.



whereas, the PNC group produced a significantly greater increase in HR levels than the UPC group only. None of the remaining comparisons were significant.

During Trial 8, the PNC, UPC and UPNC groups had greater increases in HR levels compared with the PC group during Seconds 1 to 5. In addition, during Seconds 2, 4 and 5 the PNC group produced a greater increase in HR compared with the UPNC group. The PNC group had a greater increase in HR than the UPC group during Seconds 4 and 5. During Seconds 6, 8, 9 and 10 the PNC group produced greater increases in HR compared with the PC group whereas, during Seconds 6, 9 and 10 the UPNC group had greater increases in HR levels than the PC group. During Seconds 5 to 10 the PNC and UPNC groups produced greater increases in HR compared with the UPC group. All of these comparisons reached significance. There were no other significant differences.

Further comparisons across trials revealed, firstly for Second 1, no difference between Trials 5 and 8 involving the PC and UPC groups. The PNC group had significantly higher HR during Trial 8 compared with Trial 5 whereas, the UPNC group produced significantly higher HR levels during Trial 5 than Trial 8. During Second 2, the PC and UPNC groups had significantly higher HR during Trials 5 compared with Trial 8 whereas, the PNC group had significantly higher HR during Trial 8. The UPC group produced no significant difference across trials. During Seconds 3, 4 and 5, the PC group had significantly higher HR during Trial 5 compared with Trial 8 whereas, for the PNC group the pattern was reversed. During Seconds 3 and 4, there were no significant differences across trials for the UPC or UPNC groups. During Seconds 5 and 6, the UPNC group produced significantly higher HR levels during Trial 8 compared with Trial 5. During Seconds 7 and 8, there were no significant differences across trials involving any of the groups. In Second 9, the PC group produced significantly higher HR during Trial 5 compared with Trial 8 whereas, for the UPNC group the pattern was reversed. The PNC and UPC groups revealed no significant differences across trials. During Second 10, the PC group had significantly higher HR levels during Trial 5 compared with Trial 8. There were no significant differences across trials involving the remaining three groups.

3.3.1.9. Summary. During Time Period 2, mnHR produced a predictability by feedback by trials and a control by feedback by trials interaction. Further analysis of the former interaction revealed that the UPFb group had significantly lower HR levels than the

remaining groups during Trial 5. Analysis of the control by feedback by trials interaction revealed that the NCNFB group produced significantly higher HR compared with the CFb and NCFb groups during Trial 5. During Trial 8, the CNFB group had significantly higher HR levels than the CFb group.

Analysis of Time Periods 4 (mxHR only) and 7 (avHR and mxHR) revealed main effects of predictability, due to the P groups producing significantly higher HR levels than the UP groups.

During Time Period 5, all three cardiovascular measures produced a main effect of feedback and a two way control by trials interaction. The feedback effect was due to the NFb groups producing significantly higher HR levels than the Fb groups. Further analysis of the control by trials interaction across all three measures, revealed that the C groups had significantly higher HR compared with the NC groups during Trial 8. There was no significant difference between the groups during Trial 5.

There were no significant differences between groups involving any of the measures during Time Period 6. However, in Time Period 7, avHR produced a feedback by time interaction. This was due to the NFb groups producing higher HR levels compared with the Fb groups across the 20 sec ITI, although the difference only reached significance during the 0-5, 10-15 and 15-20 sec periods.

During the cardiac response profile (Time Period 8), analysis of avHR change produced a predictability by control by feedback by trials interaction. Further analysis of the interaction revealed that the PCFb and PNCfb group had greater increases in HR compared with all the remaining groups during Trials 5 and 8, although none of these comparisons reached significance. The remaining groups produced similar levels of activity during both trials, with the exception of the PCNFb group during Trial 8 which showed the greatest decrease in HR levels. In addition, during Time Period 8 there was a four-way predictability by control by trials by time interaction. During Trial 5, the UPNC group showed the greatest increase in avHR during the 1-3 sec after noise onset compared with the PNC group which produced the greatest decrease in HR. However, during the 5 to 10 sec after noise onset, the two P groups produced the greatest increases compared with the UP groups which both produced a decrease in avHR. During Trial 8 a different pattern

emerged, with the NC groups producing the greatest increases in avHR during the 4 to 10 sec after noise onset compared with the C groups.

3.3.2. Corrected HR. The results involving all three measures were similar to those of uncorrected HR unless otherwise stated.

3.3.2.1. Time Period 2. Corrected mnHR produced significant predictability by feedback by trials ( $F(1, 88) = 4.1, p < .05$ ) and control by feedback by trials ( $F(1, 88) = 4, p < .05$ ) interactions. However, analysis of the simple main effects revealed a different pattern of responding to uncorrected mnHR. The former interaction revealed, firstly for Trial 5, that the UPNFb and PFb groups had significantly greater mnHR increases compared to both the PNFb and UPFb groups. During Trial 8, there were no significant differences between groups. However, both the PFb and UPFb groups had greater increases in mnHR compared to the PNFb condition which, in turn, had greater increases than the UPNFb group. The UPNFb and PNFb produced a significant increase in activity from Trial 5 to 8, compared to the UPNFb group which produced a non-significant decrease (see Figure 84). Further analyses of the control by feedback by trials interaction revealed, firstly for Trial 5, that the NCFb and NCNFb groups had greater mnHR increases compared to both the CFb and CNFb groups, although only comparisons involving the NCNFb condition were significant. During Trial 8, the NCFb group had significantly greater increases in mnHR than the three remaining groups. In addition, the NCNFb and CNFb groups produced non-significantly greater mnHR change scores compared to the CFb group. The NCFb group produced significantly greater increases in mnHR during Trial 8 compared to Trial 5, whereas, the NCNFb group showed a non-significant decrease between these two trials (see Figure 85).

3.3.2.2. Time Period 4. Corrected mxHR produced no predictability effect ( $F(1, 88) = 3.2$ ). Further inspection of means revealed that the results were in a similar direction as the raw HR scores; i.e. the UP groups produced a greater decrease in HR levels from the baseline period compared with the P groups (-9.77 and -6.65 change in bpm respectively).

3.3.2.3. Time Period 5. There were no feedback effects involving any of the corrected HR measures: avHR ( $F(1, 88) = 1.8$ ), mnHR ( $F(1, 88) = 1.6$ ) and mxHR ( $F(1,$

Figure 84. Mean change in minimum HR as a function of predictability, feedback and trials during Time Period 2.

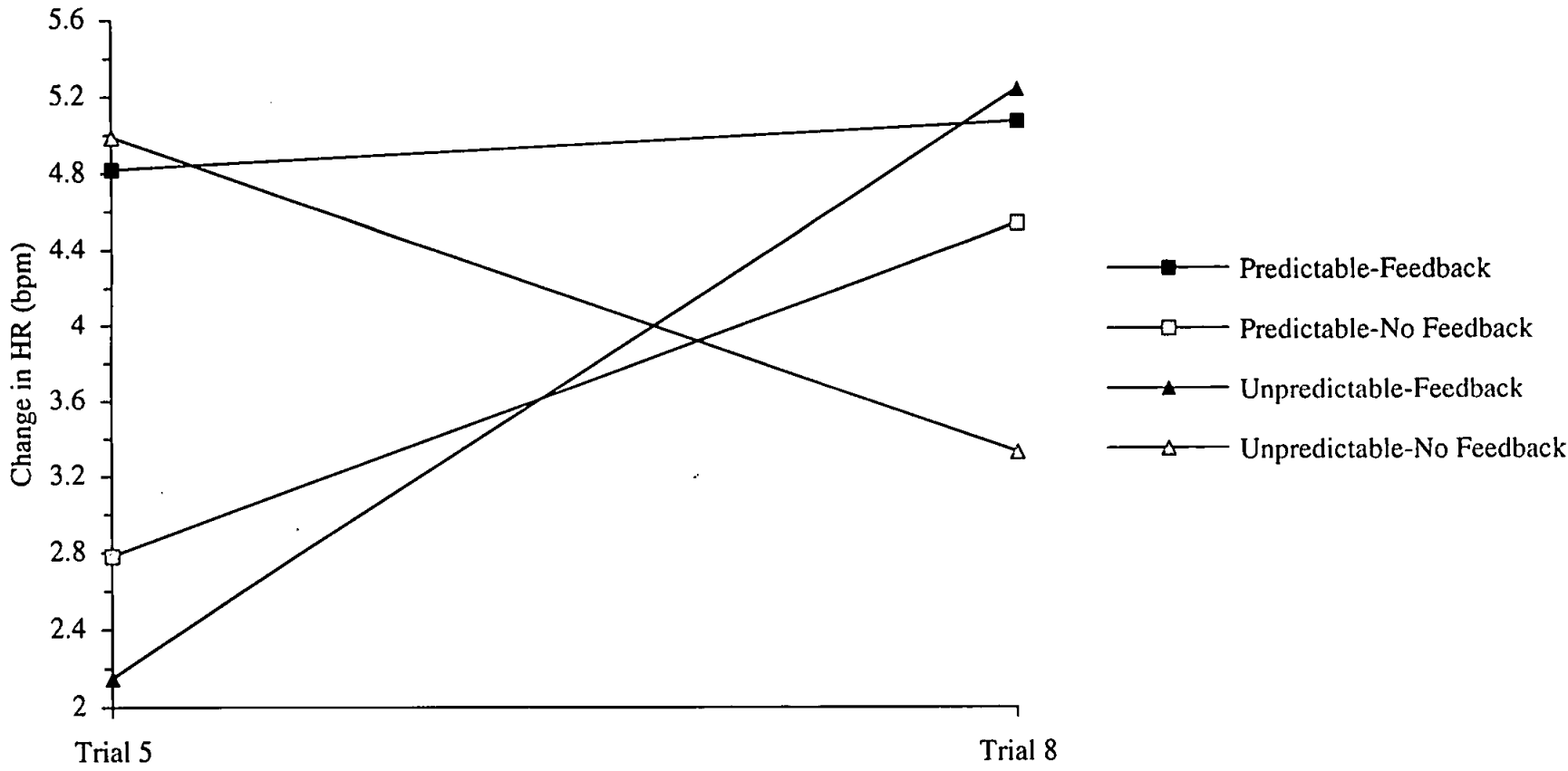
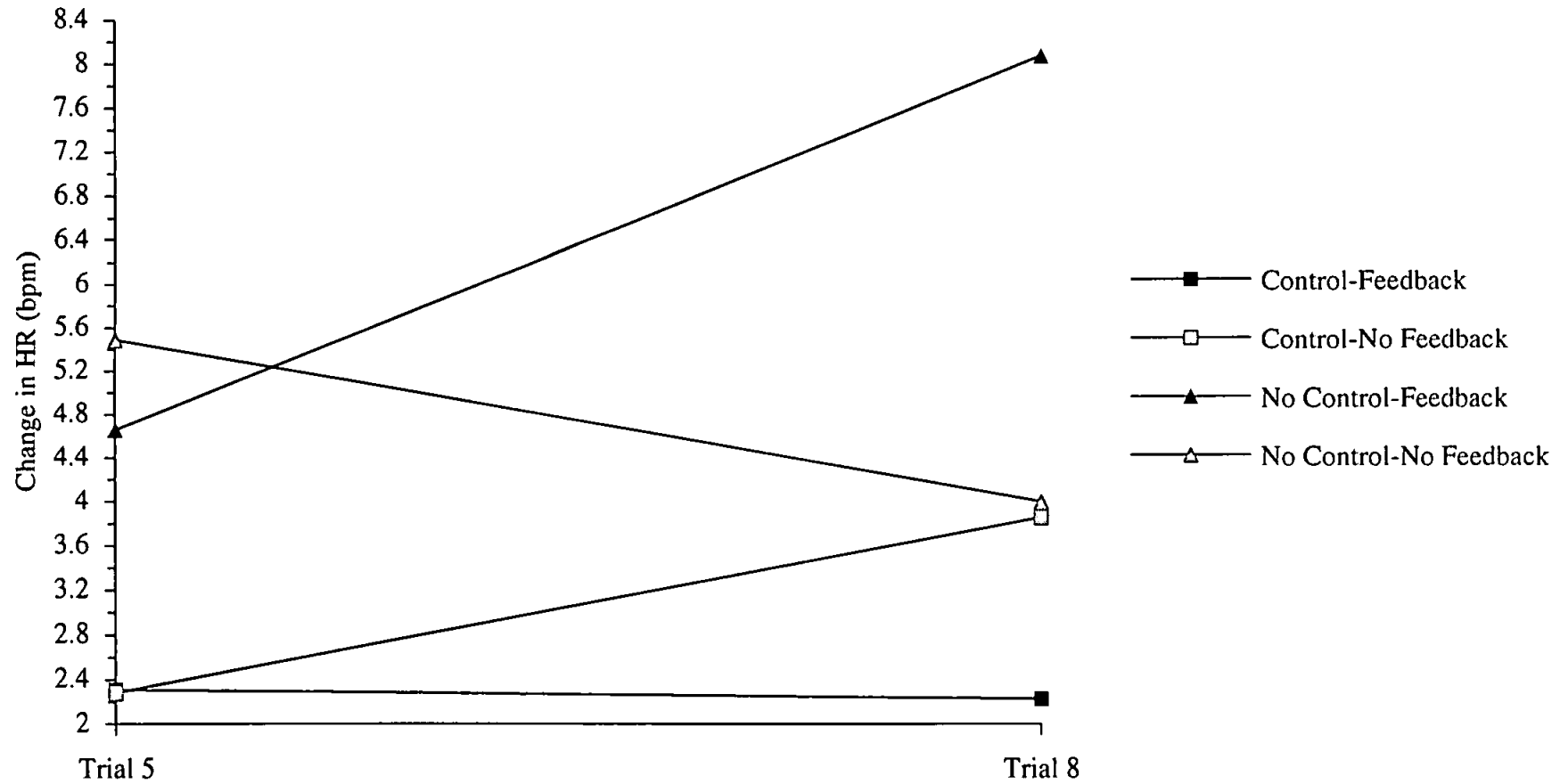


Figure 85. Mean change in minimum HR as a function of control, feedback and trials during Time Period 2.



88) = 2.1). However, inspection of the means revealed a similar pattern of responding as uncorrected HR, with the NFb groups producing either an increase in mnHR or a smaller decrease in avHR and mxHR from the baseline period compared with the Fb groups (avHR = -1.04 and -2.95; mnHR = 4.02 and 1.89; mxHR = -6.71 and -9.11 change in bpm respectively). In addition, all three measures produced a significant control by trials interaction: avHR ( $F(1, 88) = 7.5, p < .01$ ), mnHR ( $F(1, 88) = 4.6, p < .05$ ) and mxHR ( $F(1, 88) = 8.2, p < .01$ ). Further analysis of the simple main effects revealed a different pattern of responding to that of uncorrected HR. For example, during Trial 5, the NC groups had non-significantly greater increases in mnHR and smaller decreases in avHR and mxHR compared to the two C conditions. In Trial 8, the pattern of responding was reversed with significantly higher change scores in the C groups involving all three HR measures. In avHR and mxHR this difference was due to the NC groups producing a significant decrease in change scores between Trials 5 and 8, compared to the C groups which produced similar levels of activity during both trials. In mnHR the two C groups produced a significant increase in activity from Trial 5 to 8, whereas, the NC groups showed a slight decrease in HR change scores. Group means are plotted in Figures 86 to 88 for avHR, mnHR and mxHR respectively.

3.3.2.4. Time Period 6. Corrected avHR ( $F(1, 88) = 4.0, p < .05$ ) and mxHR ( $F(1, 88) = 4.1, p < .05$ ) revealed a two-way predictability by feedback interaction. Simple main effects, involving both avHR and mxHR, revealed that the PFb and UPNFb groups had smaller HR decreases from the pre-task baseline compared with both the PNFB and UPFB groups. However, none of these comparisons reached significance (see Figures 89 and 90 for avHR and mxHR respectively).

3.3.2.5. Time Period 7. Corrected avHR ( $F(1, 88) = 3.2$ ) and mxHR ( $F(1, 88) = 2.9$ ) produced no predictability effect. Further inspection of means revealed a similar pattern as raw HR scores for mxHR i.e. the P groups produced smaller decreases in HR from the baseline compared with the UP groups (-6.91 and -9.6 change in bpm respectively). However, avHR revealed that the P and UP groups had similar levels of activity (5.63 and 6.11 change in bpm for the P and UP groups respectively). AvHR also produced a significant feedback by time interaction ( $F(3, 236) = 2.7, p < .05$ ). As seen previously, however, further analysis of the simple main effects revealed a different pattern



Figure 86. Mean change in average HR as a function of control and trials during Time Period 5.

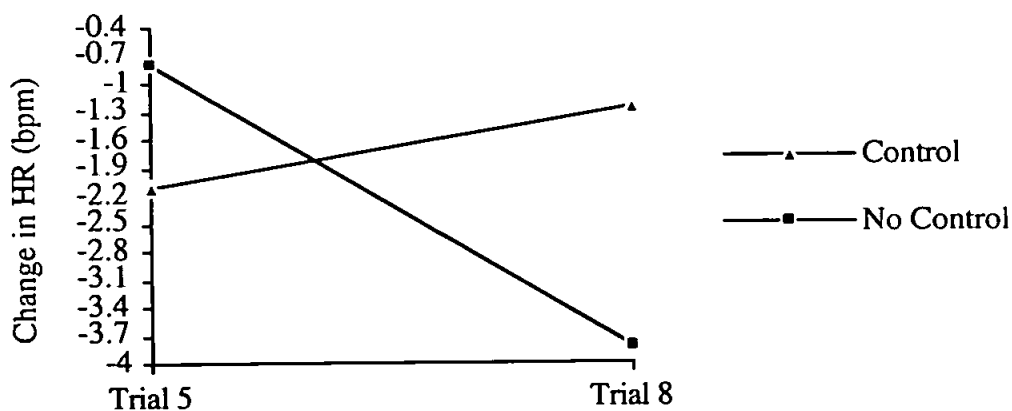


Figure 87. Mean change in minimum HR as a function of control and trials during Time Period 5.

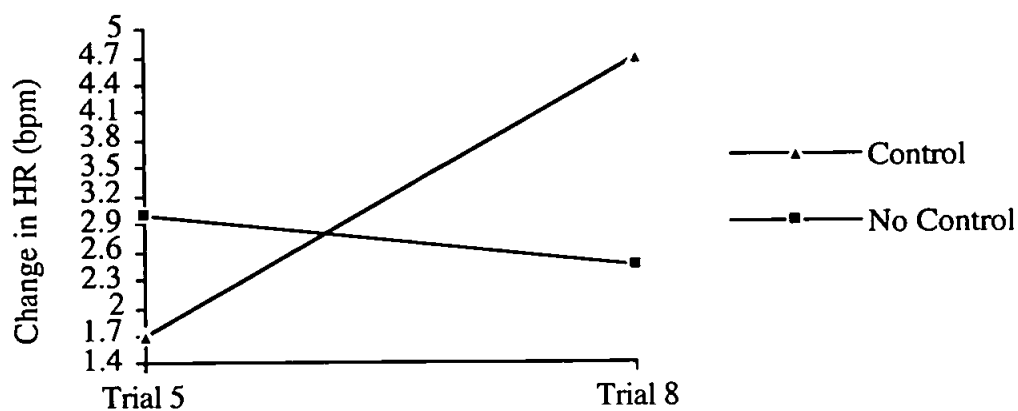


Figure 88. Mean change in maximum HR as a function of control and trials during Time Period 5.

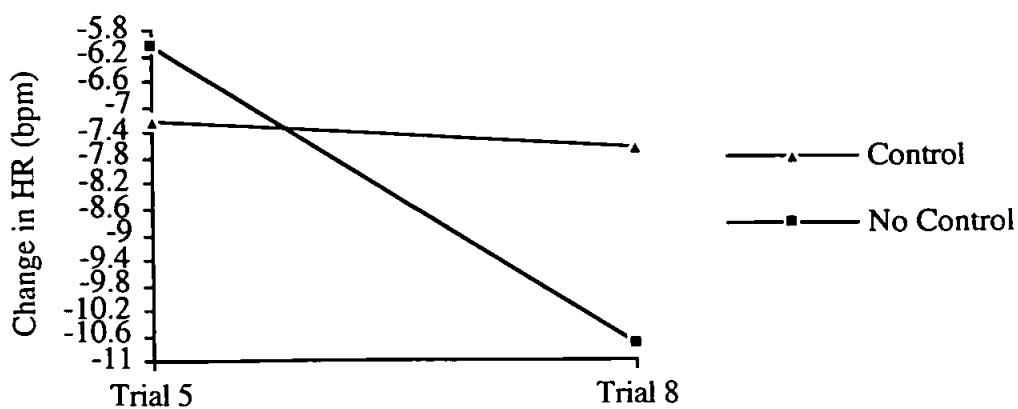


Figure 89. Mean change in average HR as a function of predictability and feedback during Time Period 6.

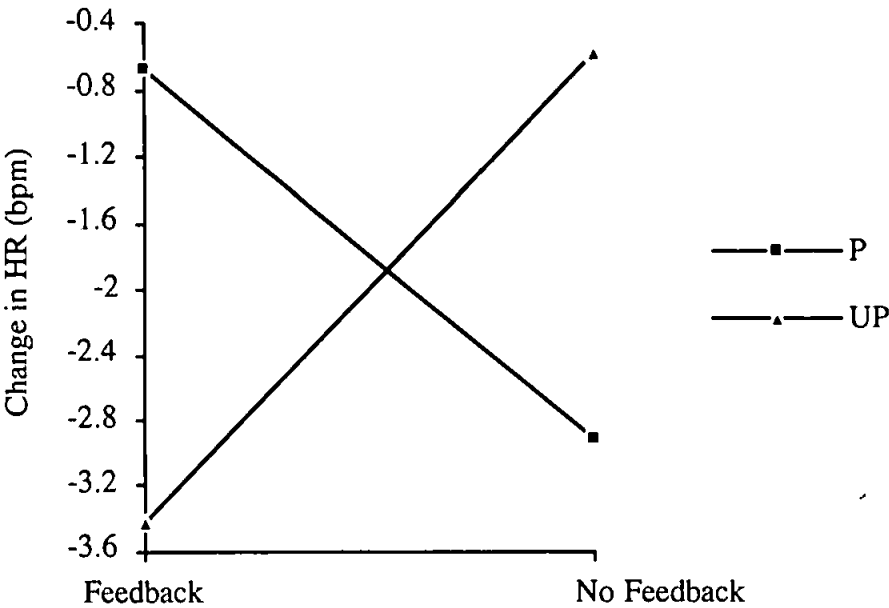
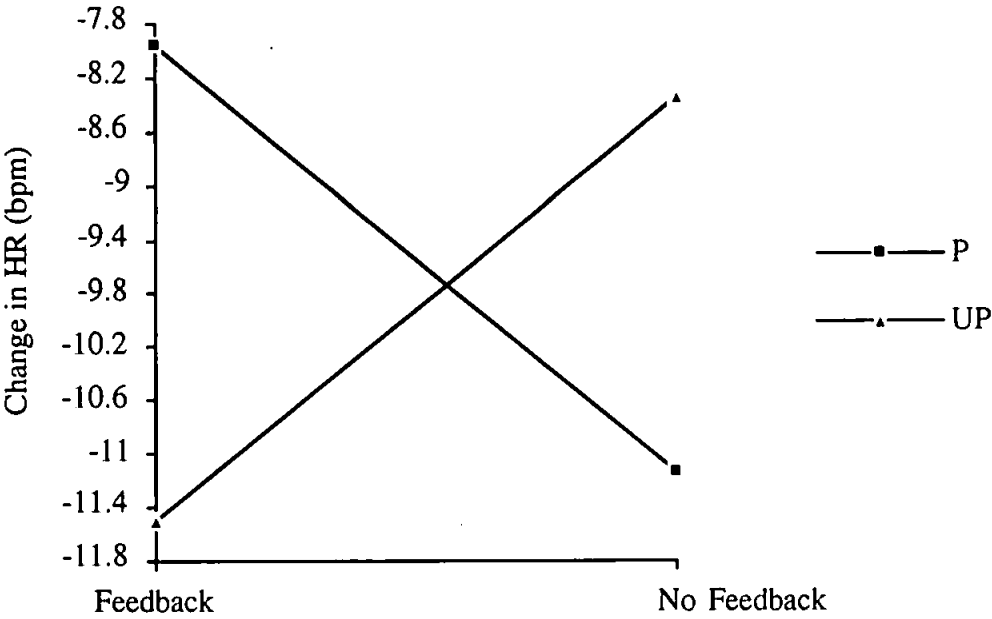


Figure 90. Mean change in maximum HR as a function of predictability and feedback during Time Period 6.



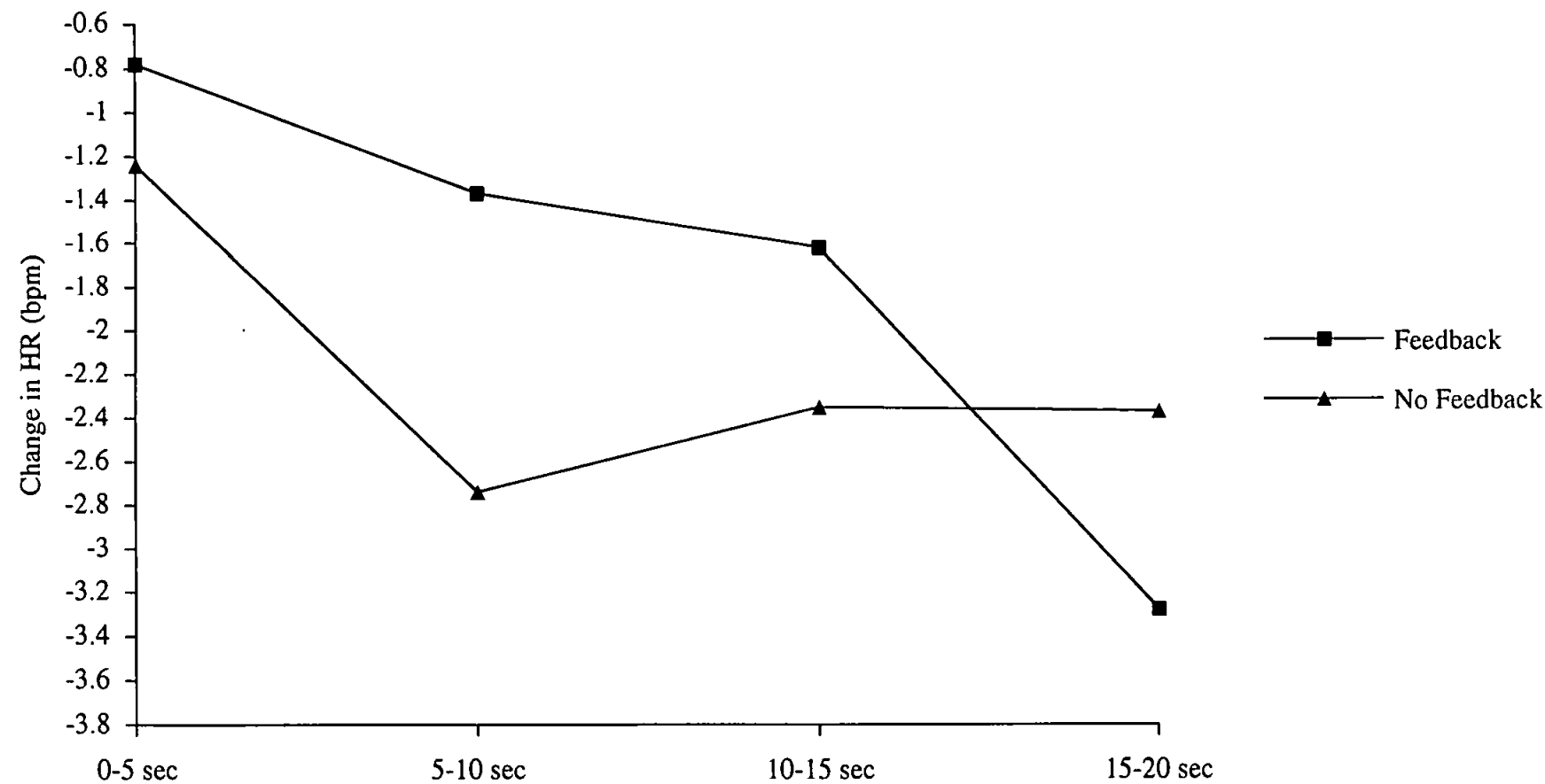
of responding to that of uncorrected avHR. That is, the Fb groups had smaller decreases in avHR from the pre-task baseline compared to the NFb groups, during the 0-5, 5-10 and 10-15 sec periods. However, the comparison between groups reached significance during the 5-10 sec period only. During the 15-20 sec period, the pattern of responding was reversed with non-significantly higher change scores in the NFb groups. This was due to the Fb groups showing a significant decrease in activity between the 10-15 and 15-20 sec periods, whereas, the NFb groups produced similar HR activity in both periods (Figure 91).

**3.3.3. Corrected HR Summary.** During Time Periods 1 and 3, the results involving corrected HR were similar to those of uncorrected HR across all cardiovascular measures. In Time Period 2, corrected mnHR produced significant predictability by feedback by trials and control by feedback by trials interactions. The former interaction was due to the UPNFb and PFb groups showing significantly higher change scores than the PNFb and UPFb groups during Trial 5. There were no significant differences between groups in Trial 8. The control by feedback by trials interaction was due to the NCNFb condition showing higher change scores than the remaining three groups during Trial 5, whereas, in Trial 8 the NCFb group had greater increases in mnHR compared to the other conditions. The difference between trials was mainly due to the NCFb condition producing a significant increase in HR activity between Trials 5 and 8.

In Time Periods 4 (mxHR) and 7 (mxHR and avHR), there were no significant predictability effects. Further inspection of the means revealed that the results were in a similar direction as uncorrected HR scores.

During Time Period 5, all three measures produced no feedback effect. Again, inspection of the means revealed similar results raw HR scores, with the NFb groups producing either a greater increase in mnHR or a smaller decrease in avHR and mxHR compared with the Fb groups. All three measures also produced a significant control by trials interaction during this period. However, the pattern of responding was different to that of uncorrected HR. The NC groups had greater increases in HR compared to the C groups in Trial 5, whereas, during Trial 8 the pattern was reversed with significantly higher change scores in the C conditions.

Figure 91. Mean change in average HR as a function of feedback and time during Time Period 7.



The results of Time Period 6 revealed a two-way predictability by feedback interaction involving avHR and mxHR. This was due to the PFb and UPNFb groups producing a smaller decrease in HR from the baseline compared with the PNFB and UPFB groups. In addition, corrected avHR produced a feedback by time interaction. However, as seen previously, the pattern of responding was different to that of uncorrected avHR with the Fb groups producing higher change scores compared to the NFb groups during the 0-15 sec after noise onset. The difference between groups only reached significance during the 5-10 sec period. Furthermore, during the 15-20 sec period the NFb groups had higher HR activity than the Fb conditions.

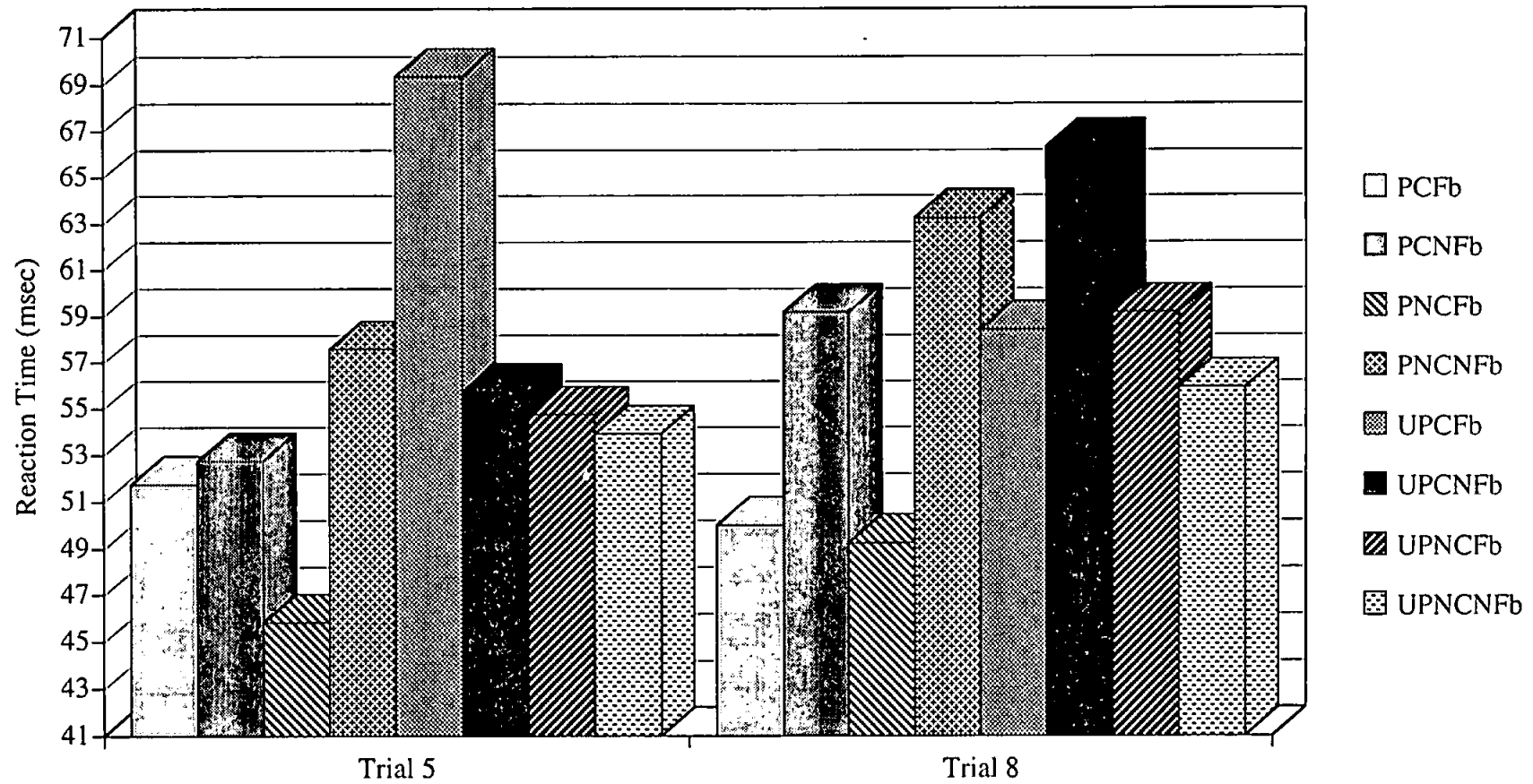
### 3.4. Reaction Time Data.

Reaction time data was assessed in a four-way MANOVA with predictability, control and feedback as between subject factors and trials as a repeated measure. There were no significant differences between groups (all  $F_s < 1$ ) (see Figure 92).

## 4. DISCUSSION

In keeping with predictions made in Section 1, the present study revealed a significant relationship between feedback, predictability and control during the cardiac response profile. According to a behavioural uncertainty interpretation, this relationship should be due to the feedback groups in both predictable and unpredictable conditions showing lower cardiac activity compared to no feedback conditions, during situations in which opportunity to control was available. These predictions were not substantiated. Instead, the provision of feedback led to the opposite effect. That is, there were greater increases in average HR from the pre-stimulus period compared with no feedback conditions which produced either smaller increases or greater decreases in HR. During predictable situations, this high level of responding in the feedback group was sustained across Trials 5 and 8, whereas, when feedback was not available, subjects produced a marked decrease in average HR change across trials. Consequently, in Trial 8 subjects in the feedback condition had significantly higher average HR activity compared to those in

Figure 92. Mean response time as a function of groups and trials.



the no feedback group. However, when the situation was one of unpredictability this level of sustained activity was not observed in the feedback group. Subjects in this group produced a slight decrease in average HR activity across trials, to similar levels as those in the no feedback condition.

These results appear to suggest, therefore, that it is the presence of a warning signal which in some way enhances the effect of feedback in situations with aversive consequences. When a situation is not perfectly predictable, the potential effects of feedback appeared to be reduced over time. It might be argued, that the combination of predictability and feedback enhances task motivation or engagement by aiding an individual's perception of control. Equally, it could be that the effects of predictability and feedback do not depend on having a perception of control. Indeed, such a suggestion would be supported by the pattern of responding in the no control groups in which differences emerged between feedback and no feedback groups with the former resulting in greater average HR change scores during predictable events. As in the feedback-control condition, this high level of activity was sustained across trials. However, subjects in the no feedback group produced an increase in HR activity over the course of the task, unlike those in the no feedback-control condition. In terms of the unpredictable groups, manipulation of feedback had no significant effects. Findings from the unpredictable groups were, therefore, in accordance with a behavioural uncertainty explanation which predicts no effects of feedback where opportunity to control is not available. Since information informing subjects of noise duration does not reduce response or response outcome uncertainty. However, the pattern of responding in the predictable groups was opposite to that which would be predicted by an uncertainty interpretation; namely, that the presence of performance feedback should ameliorate the negative effects of aversive stimulation.

The proposition that predictability and feedback have effects above and beyond those of control, would also be supported by findings from the 0-2 sec period after noise onset. That is, irrespective of control, when performance feedback was available, subjects in the predictable condition displayed markedly smaller decreases in average and maximum HR from the pre-task baseline compared to those in the unpredictable condition. When feedback was absent, the pattern was reversed with smaller average and maximum change

scores in unpredictable groups. Essentially, predictability only appears to have beneficial effects in terms of a reduction in cardiac function when feedback is absent. When feedback is made available, predictability appears to be stress-inducing. Similar results were found during the 0-2 sec period after feedback. Moreover, these effects were obtained over the course of the task. Subjects in the predictable, feedback group sustained greater increases in minimum HR from the pre-task baseline across Trials 5 and 8 compared to those in the no feedback condition. Initially, data from the unpredictable groups was similar to that which occurred during the impact period with significantly greater minimum HR change scores during no feedback compared to feedback conditions. However, over the course of the task subjects in the feedback condition produced a significant increase in levels of responding to similar levels as the predictable-feedback group. In addition, those subjects in the no feedback condition showed a marked decrease in HR activity. Consequently, during Trial 8 the feedback groups had greater minimum HR increases compared to the no feedback groups. These findings are difficult to incorporate within a behavioural uncertainty explanation since feedback would be expected to facilitate stress-reduction during both predictable and unpredictable situations. It could be that an explanation in terms of differential effort may be more appropriate.

According to a differential effort interpretation, the provision of performance feedback should increase and then sustain cardiovascular activity compared to situations in which feedback is absent. These differential effects should be more pronounced in control compared to no control conditions, since individuals in the former group have a stronger incentive to be trying hard; namely, the ability to shorten event duration. The results of the present study appear to only partially support such predictions. That is, during situations in which no controlling response was available, feedback augmented average HR activity compared to no feedback conditions, but only when the aversive event was temporally predictable. When the situation was unpredictable, feedback had no differential effects. Similarly, when subjects had the opportunity to control, feedback led to an increase in cardiac function to similar levels as the feedback-no control group. Again, these differential effects were most pronounced during situations in which a warning signal preceded the aversive event.



It would appear, therefore, that the incentive or motivational properties of feedback are most important with respect to changes in autonomic function when the aversive event is perfectly predictable, regardless of whether or not individuals have control. The finding that changes in the degree of predictability had a greater impact on feedback effects compared to the control manipulation was surprising. For instance, in Chapter 6 it was suggested that the degree of perceived control was fundamental in promoting the beneficial effects of feedback. Indeed, this supposition was based on literature reviewed in Chapter 3, in particular the work of Foushee, Davis, Stephan and Bernstein (1980) which suggested that the availability of feedback altered subject's perception of the possibility of control and *vice versa*.

In the present study, where any differences did arise as a result of the control manipulation these were dependent on what part (i.e. time period) of the trial they occurred in. For instance, during the cardiac response profile the presence of feedback augmented cardiovascular activity to a greater extent during control as compared to no control conditions. This may suggest that performance feedback provided an additional incentive above that already available from having a controlling response; i.e. that the effects of these two factors are additive. However, these effects do not appear to be *beneficially* additive.

Such findings should be treated with caution, however, due to the pattern of responding observed in the 0-2 sec period after feedback. During this period, subjects in the feedback and no feedback groups showed similar increases in minimum HR from the pre-task baseline, when opportunity to control was available. Where any non-significant differences did arise across the course of the task, these were due to the no feedback group producing greater minimum HR change scores compared to the feedback group. Those subjects in the latter condition showed similar levels of responding during both Trials 5 and 8. It could be argued that these results partially support an explanation in terms of behavioural uncertainty. That is, the absence of information informing subjects as to the success or otherwise of their controlling response augmented task uncertainty over the course of the experiment which, in turn, led to an increase in minimum HR activity.

However, this type of interpretation cannot easily incorporate the findings from the no control conditions in which the presence of feedback led to significant increases in minimum HR change scores across Trials 5 and 8, compared to the no feedback condition

which showed a decrease in level of responding over the course of the task. Instead, these results can be more easily incorporated into a differential effort explanation. That is, the motivational properties of information feedback augments cardiac activity through enhanced active coping mechanisms, even in situations where no physical control is present. The pattern of responding observed in the inter-trial interval would tentatively support such an interpretation. That is, the feedback groups, irrespective of predictability or control manipulations, produced a sustained increase in average HR activity from the pre-task baseline across the first 15 sec of the inter-trial interval, compared with the no feedback groups which produced a marked decrease followed by, a slight increase in average HR change scores.

In summary, data from the present study indicate that temporal predictability, opportunity to control and performance feedback have independent effects on the cardiovascular response during aversive situations. The relationship between these three factors also appears to be important particularly during the feedback and noise impact periods, and in the inter-trial interval. In terms of impact and the eight seconds after noise offset, the provision of feedback, irrespective of the opportunity to control, both increased and sustained cardiac activity compared to no feedback conditions but only in situations which were perfectly predictable. It was suggested that feedback, in the form of information about the duration of the aversive event, may have provided additional predictability in that subjects knew exactly when the event would occur *and* how long it would last for, whereas, those with no feedback received only temporal information. The motivational effects resulting from this extra predictability may have enhanced active coping strategies which, in turn, increased cardiac activity even in situations where no physical control was available. It was suggested that these results may indicate the additive effects of temporal predictability and performance feedback. Indeed, this was confirmed by further results from the impact period during which subjects who received either predictability *or* feedback produced markedly lower HR than those who experienced either predictability and feedback, or neither of these factors.

In terms of the control-feedback relationship the pattern of responding was less clear-cut. For instance, during the impact period and the following eight seconds, the manipulation of control appeared to have little differential effect upon cardiovascular

activity in response to feedback. As mentioned previously, any differential effects during this period were mainly due to changes in predictability. However, during the period after feedback had been given, differences did emerge between control and no control conditions across the course of the task. These were primarily due to feedback increasing cardiac activity, when opportunity to control was not available. Where control was present, feedback had little differential effect. The pattern of responding in both control and no control conditions is in accordance with work by Light and Obrist (1980), and partially supports a differential effort explanation. The implications of these results and those concerning the predictability-feedback relationship will be discussed further in Chapter 11.

An additional aim of the present study was to compare the prediction-control relationship to that observed in Chapter 9, following changes in the manipulation of the controlling response. These changes were instigated after suggestions that the warning signal in the PC condition may have had additional benefits since it predicted not only the onset of the noxious event but more importantly, the controlling response. Thus, the meaning of control in the unpredictable and predictable conditions may have been significantly different. These differences could, in turn, have been responsible for the high level of cardiovascular activity observed in the PC condition.

Due to the problems associated with uncorrected HR in the preceding study, the findings presented below will be concerned only with results involving corrected HR. Indeed, these problems were re-confirmed by data from the present experiment in which significant differences were found between uncorrected and corrected HR results. Further inspection of the pre-task baseline means, indicated that these differences may have been due to the UPNC group producing markedly lower activity levels compared to the remaining groups in all three HR measures.

Analyses of corrected HR revealed that any significant differences arising as a result of the predictability-control manipulation were primarily due to levels of responding during Trial 1. In previous studies (see Chapters 6, 7 and 8), it had been suggested that any differences observed during the first trial were an anomaly since subjects in all groups had received identical experimental stimuli up to that point. In the present study, although this was still the case, subjects had received significantly different instructions prior to the

beginning of the task. Therefore, in the following discussion the results of Trial 1 will be treated as equal to those observed in later trials.

During Trial 1, the 0-2 sec period after feedback revealed that the UPNC and PC groups produced greater increases in average HR and smaller decreases in maximum HR compared to the UPC condition which, in turn, showed greater increases and smaller decreases than did the PNC group. Essentially, these results were similar to those reported for corrected minimum HR during Trials 5, 8 and 13 of the preceding study, with the exception of activity in the two intermediate conditions - PNC and UPC. That is, during the previous experiment subjects in the former group generally had greater increases in HR activity compared to those in the UPC condition, whereas, in the present study the pattern of responding was reversed.

Furthermore, during the previous study any changes across the various time periods of the trial were primarily due to subjects in the PC condition showing a decrease in minimum HR, or less sustained activity compared to the remaining three groups. In contrast, during the present experiment where any differences did arise over the course of the task it was generally due to the UPNC group producing a marked reduction in HR activity. For instance, during the warning signal the UPNC group produced a decrease in maximum HR to similar levels as the UPC and PNC groups. Consequently, the PC condition had significantly greater maximum HR change scores than the remaining groups.

The high level of responding observed in the PC condition during the present study would appear to indicate, therefore, that the differential effects of control in predictable and unpredictable conditions during the preceding experiment were not a result of any possible confounding effects. Instead, these results substantiate the finding that control without temporal predictability is stress-reducing, whereas, the same control with a warning signal is stress-inducing. In terms of theoretical perspectives, these results can be interpreted in much the same way as those from the previous experiment (see Chapter 9, Section 4). Namely, that the effects of temporal predictability and opportunity to control are independent. A finding predicted only by Miller's (1979) minimax hypothesis, since the three predictability theories all propose that control has no differential effects over and above those associated with additional predictability. However, the Minimax Theory cannot adequately explain why these effects should be observed in unpredictable but not

predictable conditions and, more importantly, why effects in the predictable condition are in the opposite direction to those predicted.

In Chapter 9, it was suggested that uncertainty concerning the nature and outcome of the controlling response was responsible for this increased cardiac activity in the PC condition. However, it was also noted that an explanation in terms of uncertainty could not explain why activity should be enhanced in the predictable compared to the unpredictable condition, since levels of uncertainty would be expected to be greater in the latter situation due to the absence of a warning signal. Furthermore, when task uncertainty was minimised by the provision of feedback, as in the present experiment, cardiac activity in the PC condition was not reduced to levels occurring in the PNC group. It could be argued, that the controlling response in the present study still entailed a certain amount of uncertainty. For instance, during the previous experiment it had been suggested that uncertainty was increased due to subjects not being able to differentiate between long and short duration events. In the present study, this was believed to have been rectified by employing long duration noise during some of the trials. However, by providing noise which was associated with unsuccessful controlling independently of task performance, this may have increased response ambiguity and, therefore, ambiguity of the task in general. For example, subjects may have believed that their reaction time was within specified limits but they still received the long duration noise; i.e. they had been punished. In addition, the timing of the controlling response may have evoked a certain degree of ambiguity. In the previous study, for example, task performance and noise duration were associated both in the pre-task instructions and during the experimental trials themselves. However, during the present experiment subjects were simply told that their response to the word "START" at the beginning of the trial determined the duration of the white noise, presented at the end of the trial. None of the subjects, however, had any way of knowing whether or not this actually happened. Thus, the manipulation of control in the present study may have led to an increase in behavioural uncertainty.

Nevertheless, none of these suggestions can explain why responding was greater in the control compared to no control condition only when the event was temporally predictable. Furthermore, findings reported at the beginning of this section in terms of the prediction-control-feedback relationship, would lend greater support to a differential effort

rather than a behavioural uncertainty explanation. Indeed, such an interpretation would be partially supported by a number of other findings both during Trial 1 and in Trials 5 and 8. For example, during the inter-trial interval of Trial 1 subjects in the predictable groups showed more sustained minimum HR increases from the pre-task baseline across the 20 sec of the inter-trial interval, compared to those in the unpredictable groups. Those subjects in the latter condition, produced a marked reduction in minimum HR change scores across the course of the inter-trial interval. Consequently, during the last five seconds of this period the predictable groups had significantly greater minimum HR increases from the pre-task baseline compared to the unpredictable groups. Furthermore, during the impact periods of later trials opportunity to control led to sustained average and maximum HR from the pre-task baseline, and markedly increased minimum HR change scores across Trials 5 and 8. In contrast, during situations in which control was not available subjects showed marked decreases in average and maximum HR change scores across the course of the task. Consequently, during Trial 8 the control groups had significantly smaller decreases in average and maximum HR and greater increases in minimum HR from the pre-task baseline, compared to the no control groups.

Finally, in terms of the pattern of responding observed in the three HR measures during the present study, this was not in accordance with that which was reported in Chapter 9. For instance, during the previous study it had been found that the additive effects of predictability and control were particularly prominent in minimum HR and that this, in turn, may suggest that the ability to lower cardiac activity in aversive situations may be more important in terms of long-term haemodynamic dysfunction than the sympathetically-mediated increases detailed by Obrist and colleagues (e.g. Obrist, 1981). However, during the present experiment the effects of predictability and control, and that of feedback, were seen equally across the three HR components. Thus, the special significance of changes in peak deceleration or minimum HR during situations eliciting active coping mechanisms would appear at the present time, to be in doubt. These issues will be discussed further in Chapter 11.

## CHAPTER 11

### DISCUSSION

#### 1. INTRODUCTION

The purpose of this final chapter is to summarise the experimental findings reported in earlier chapters and to integrate them into a framework relevant to a biopsychosocial model. In particular, Steptoe's (1991) stress-diathesis model which was identified in Chapter 1 will be reviewed in the context of the current findings.

In addition, this chapter will also discuss the general question concerning the conceptualisation and measurement of various autonomic response components associated with the psychobiological stress response. Furthermore, an attempt will be made to describe the inter-relationships between the different components. This will include an evaluation of the extent to which autonomic measures can be used to measure such generalised constructs as 'stress' and 'arousal'.

#### 2. SUMMARY OF EXPERIMENTAL FINDINGS

##### 2.1. Feedback

The psychophysiological effects of performance feedback were examined during two experiments reported in this thesis. Experiment One was concerned with the effects of RF on patterns of cardiovascular and electrodermal responding during a complex, information-handling, visual-spatial task. The prediction being that the receipt of feedback would augment autonomic activity compared to a no feedback condition. Additionally, it was predicted that the provision of feedback would increase task performance compared to situations in which no feedback was available.

Feedback did lead to an increase in performance compared with a condition where no feedback was available. Moreover, this increased level of performance was maintained across the course of the task, whereas, the absence of feedback led to a performance decrement over trials. Nevertheless, there was only very limited support for the prediction

made concerning the effects of feedback on physiological measures. Levels of HR responding were marginally higher in feedback compared with no feedback subjects but only at the beginning of later trials. Furthermore, support from the other measures was weak. The absence of a clear physiological discrimination between the two groups may imply that electrodermal and, to a lesser extent, cardiovascular responding are insensitive to changes in the availability of performance feedback. However, the experiment included a number of factors which may have precluded any significant effects of feedback.

The first relates to the extremely high level of difficulty employed during the task which may have fostered a sense of "giving up" or, at least, discouraged subjects from continuing to exert their maximum effort. Indeed, task difficulty interacted with feedback, although not significantly, in predicting task performance with subjects in receipt of feedback producing lower failure rates compared to no feedback subjects during easy trials. There was little difference between the two groups in difficult trials. Thus, feedback may have elicited greater changes in physiological function if the task had employed more 'realistic' levels of task difficulty. The second issue relates to subjects lack of perceived control which occurred as a direct result of the level of task difficulty employed. It was suggested that if perceived or actual instrumental control had been available during this experiment individual's perceptions of the likelihood of success might have been significantly altered. This, in turn, may have augmented the effects of performance feedback in terms of physiological function. Finally, the type of feedback employed could have been insufficient for this particular task. The literature suggests that RF which provides straightforward 'success-failure' or 'right-wrong' information may be of little use in complex, problem-solving tasks since it provides no directive information which can be used to control subsequent responding by helping individuals acquire knowledge about the properties of that task. Instead, feedback which alters physiological function should consist of 'cognitive material' which enables subjects to perceive not only that their response was incorrect, but *why* it was incorrect. These and related issues were examined during Experiment Five.

During Experiment Five the nature of RF effects were examined using a simple reaction-time task with high intensity white noise employed as the aversive stimuli. It was found that although feedback alone did increase cardiovascular responses relative to a no



feedback condition particularly during the inter-trial interval, these changes were largely dependent on the degree of temporal predictability available. Feedback increased and sustained cardiac activity in the impact period and after feedback had been given to a greater extent when a warning signal preceded the aversive event than when the same event was unpredictable. Indeed, additional findings during the impact period suggested that in unpredictable situations the effects of feedback may even have been beneficial as was shown by a lowering of cardiovascular reactivity.

While the finding that the absence of both temporal predictability and feedback led to higher HR than situations in which only one of the factors was available is partially consistent with predictions, the finding that the presence of both predictability and feedback also led to higher HR was largely unexpected. This may have occurred because feedback, in the form of information about noise duration, provided additional predictability in that subjects knew exactly when the event would occur *and* how long it would last for. The motivational effects resulting from this extra predictability (and perhaps reduced uncertainty) might have enhanced active coping strategies leading to increased HR activity, even in situations where no controlling response was available.

Thus, Experiment Five provided some evidence that the provision of RF can significantly alter physiological reactivity during simple reaction-time tasks employing 'realistic' levels of task difficulty. Furthermore, in accordance with suggestions made during Experiment One, the availability of perceived instrumental control also led to changes in the effects of feedback. Subjects in receipt of feedback generally produced increased cardiovascular activity compared to a no feedback group where opportunity to control was available. These additive effects are believed to have been mediated by the additional incentives provided by performance feedback. Feedback also had similar augmentative effects in situations where no controlling response was available perhaps indicating, as suggested above and by Light and Obrist (1980), that feedback enhances active coping mechanisms which are independent of the actual ability to control.

Taken together, the results of Experiments One and Five suggest that there is sufficient support for the motivational or incentive effects of RF on cardiovascular and performance measures particularly during situations involving aversive consequences,

where the noxious event is perfectly predictable, and in situations where the task employed is relatively easy and the feedback is predominately that of success.

## 2.2. Predictability

The primary aim of those experiments in this thesis which included a manipulation of predictability was to find out whether or not the warning of an aversive event would be beneficial in terms of a reduction in autonomic function. Experiment Two provided little support for this prediction with subjects in both predictable and unpredictable conditions showing similar levels of cardiovascular and electrodermal responding during a simple, non-avoidance paradigm employing mutilation slides as the aversive stimuli. Where any significant differences did emerge, however, these were in line with predictions. Levels of HR responding were higher in those subjects receiving unpredictable aversive stimuli than in those receiving predictable ones during the cardiac response profile.

Experiment Two also involved a manipulation of warning signal duration in order to assess its role in augmenting or attenuating these beneficial effects of predictability. Warning signal duration alone was not found to influence physiological activity. It did, however, interact with predictability in predicting the cardiovascular response during the inter-trial interval. Those subjects in the predictable condition who received a long warning signal showed higher HR levels than subjects in either the predictable-short, or unpredictable groups. There were no differential effects involving the electrodermal system. Despite some suggestive effects, these data do not provide adequate support for the predictions made concerning the mediating influence of warning signal duration.

The largely unconfirmed effects of temporal predictability and the lack of any changes in these as a result of warning signal duration was believed to be due to a number of stimulus and design factors the most salient of which appeared to be the employment of stimulus intensities which were too mild. Hence, Experiment Three employed a more aversive stimulus (high intensity white noise) in a similar paradigm to that of the previous study. Consequently, this experiment did provide some support for the predictions made concerning the effects of predictability on the physiological measures. Levels of HR activity were higher in those subjects receiving unpredictable as opposed to predictable

aversive stimuli during both anticipatory and impact periods. These data provide some evidence for a preparatory type interpretation for the beneficial effects of temporal predictability. Support for the predictions from electrodermal measures was weak. Response amplitudes were higher in the unpredictable compared to the predictable condition but only during the warning signal.

The differential effects of predictability were further extended in Experiment Four using a reaction-time task with high intensity white noise as the aversive stimulus. The analysis of change scores revealed higher HR during anticipatory and impact periods in the group receiving unpredictable compared to predictable aversive events, but only in situations where subjects had no opportunity to control the duration of those events. Where a controlling response was available, the effects of temporal predictability produced higher HR change scores in predictable compared to unpredictable conditions. This pattern of responding was unexpected since the only theoretical explanation which allows for independent effects of predictability and control - Miller's (1979) minimax hypothesis - indicates that the effects of these two factors should be beneficially additive.

Nevertheless, similar findings were also reported in Experiment Five which employed essentially the same reaction time task and high intensity white noise as the aversive consequence. Conditions where both predictability and control were present and those in which neither factor was available led to greater HR change scores during the feedback period than situations including predictability *or* control. During Experiment Four, these heightened HR responses were believed to be due to enhanced levels of effort or task involvement perhaps mediated by a certain amount of behavioural uncertainty resulting from response/response outcome ambiguity, and the lack of any trial-by-trial performance feedback. However, when this uncertainty was reduced as in Experiment Five by the provision of feedback, the prediction-control group still maintained greater HR responses than the two unpredictable groups. In contrast to the preceding study, however, subjects in the predictable conditions showed similar patterns of responding regardless of whether or not they had control. Indeed, the presence of control appeared relatively unimportant in terms of cardiovascular reactivity compared to the relationship between predictability and feedback. As mentioned in Section 2.1, performance feedback both increased and sustained HR change scores during event impact and after feedback had been

given but only in situations where the aversive event was perfectly predictable. Where no warning signal was provided, feedback had little differential effect.

Experiment Five included no measure of electrodermal activity. However, electrodermal measures recorded in Experiment Four provided little support for the differential effects of predictability observed in cardiovascular parameters. Where any differences did emerge as a result of the experimental manipulation these were due to subjects in the predictable groups showing greater response amplitudes to the warning signal, and event onset and offset compared to those in the unpredictable conditions. Predictability did not interact with opportunity to control. This desynchronisation between autonomic indices was similar to that observed during Experiments Two and Three, in which the predictability manipulation had little effect on the three parameters chosen to represent electrodermal activity compared to significant effects in the cardiovascular system. This type of dissociation has been observed many times in the psychophysiological literature (see Steptoe, 1981), where it has been shown that electrodermal and cardiovascular measures respond very differently to the type of threat or situation encountered.

To recapitulate the main findings: Experiments Two to Five indicated that temporal predictability can have ameliorative effects when the event or situation is sufficiently aversive. These beneficial effects are, in turn, dependent on an individual's perception of control. Adding control to a signalled noxious event appears to be one of the factors which makes predictable events aversive. Predictability alone, without control, appears to reduce psychophysiological stress. These additive effects are mirrored in the relationship between predictability and feedback. That is, irrespective of control opportunities, predictability has beneficial effects only in those situations which do not provide feedback. When combined with information feedback, predictability augments the cardiovascular response.

### 2.3. Control

Three main issues were addressed by the experiments in this thesis which included a manipulation of control. The first was whether the availability of control does indeed reduce the effect of a noxious event or situation. The second was whether or not the effects

of control were independent to those of temporal predictability. The third concerned the possible relationship between control and feedback.

The results of Experiment Four partially support the beneficial effects of control. The analysis of change scores revealed lower HR during both anticipatory and impact periods in the group given the opportunity to control the duration of the aversive event, compared to those subjects who had no controlling response available. These data provide some support for extended versions of both the preparatory response and minimax explanations for the beneficial effects of control. However, control alone was not found to influence electrodermal activity. Moreover, the differential effects observed in the cardiovascular system were not replicated to any great extent in Experiment Five. Where any significant differences did arise as a function of control *per se*, these were due to heightened HR responses in the control group during the inter-stimulus interval. The discrepancy between findings reported in Experiments Four and Five were probably a result of design differences; notably, the manipulation of control. During the former study, for example, the nature of the controlling response was such that the beneficial effects could have been due to the confounding of predictability and control rather than a result of control manipulation *per se*. In contrast, during Experiment Five this was not the case since the manipulation of predictability and that of control were separated. On this basis, it might appear that where control is manipulated independently of temporal predictability, the former variable has augmentative effects on the cardiovascular system in aversive situations involving active coping responses.

Experiments Four and Five also provided some support for the predictions concerning the independent, additive effects of predictability and control. During the former study, the analysis of change scores revealed lower HR during anticipatory and impact periods in the group who had a controlling response, but only in unpredictable situations. When the event was temporally predictable, the opportunity to control led to heightened HR responses. An unexpected finding given Miller's (1979) suggestion that the provision of both predictability and control should lead to the greatest reduction in psychophysiological stress compared to situations in which only one factor was available or, where both predictability and control were absent.

Nevertheless, similar results were found in Trial 1 of Experiment Five where the combination of both predictability and control increased and maintained cardiac activity during the feedback period and to the warning signal. During later trials, however, these effects were weaker. The prediction-control group had greater HR change scores compared to the remaining groups during the cardiac response profile, but only in the last two seconds of Trial 5. During Trial 8, it was the two no control groups who produced the greatest cardiovascular reactivity. Moreover, contrary to the pattern of responding observed in Experiment Four, the relationship between predictability and control did not reach significance during the warning signal or anticipatory intervals. It could be that changes in the way control was manipulated during this final study reduced the effectiveness of the controlling response, and that for control to have any differential effect in this type of aversive situation, both the response and the response outcome needs to be less ambiguous (i.e. the contingency between reaction time and noise duration should be strengthened), and the controlling response needs to be made perfectly predictable by the provision of a warning signal before event onset.

These factors may also be partially responsible for the lack of any clear relationship between control and feedback during Experiment Five. Indeed, this relationship was highly variable depending primarily in which part of the trial (i.e. feedback versus impact period) the effects occurred. For instance, during the feedback period the provision of control led to a greater reduction in HR change scores across the course of the task when feedback was available, compared to situations in which feedback was absent. In contrast, where there was no opportunity to control, the analysis of change scores revealed higher HR in feedback compared to no feedback conditions. However, during later periods of the trial (i.e. the cardiac response profile), the provision of information feedback had augmentative effects regardless of the level of control available. Instead, the factor which appeared to be most important in terms of cardiovascular function during this period was whether or not the aversive event was temporally predictable (see Sections 2.1 & 2.2). These findings will be discussed further in Section 4.

Taken together, the results of Experiments Four and Five would appear to indicate that the effects of perceived control in simple, reaction time (active coping) tasks employing aversive consequences are largely dependent on the presence or otherwise of temporal

predictability. Control alone, without predictability, appears to ameliorate psychophysiological stress. Adding predictive information to a situation involving control appears to eliminate or, at least, reduce these beneficial effects. No firm conclusions can be made concerning the nature of the feedback-control relationship based on the findings from these two studies, except that in aversive situations the effects of these two factors appear to be independent.

### 3. AUTONOMIC RESPONSE COMPONENTS OF THE STRESS RESPONSE

#### 3.1. Cardiovascular Activity

During this thesis, cardiac activity was measured using three HR parameters; maximum HR acceleration, minimum HR deceleration and average HR. There were three main reasons for choosing these particular response parameters (see Chapter 5, Section 2.2.1). Firstly, it was hoped that including minimum and maximum HR would provide additional relevant information in that experimental manipulation may affect the pattern of fluctuations (from a mean value) but not consistently change mean level. At the same time, however, both of these measures produce substantial correspondence with average HR. Thus, by taking multiple dependent measures (with comparable content) it was hoped that both the reliability and generalisability of the results would be increased. Secondly, it was believed that the inclusion of these measures may have psychological significance in that each of the parameters may respond differently to the type and/or degree of threat imposed. For instance, it has been suggested (e.g. Opton, Rankin & Lazarus, 1966; Seraganian, Hanley, Hollander, Roskies, Smilga, Martin, Collu & Oseasohn, 1985) that peak HR may yield response curves which are markedly elevated at the most psychological stressful points; i.e. where the degree of effort and task-involvement is at its greatest. Finally, it was hoped, based on the experimental work of Mizejeski (1978), that these non-invasive measures may have some significance in determining the specific neural-humoral influences that modify HR. That is, any increases in minimum HR as a result of behavioural arousal may indicate the incremental removal of parasympathetic inhibition whereas, increases in maximum HR during high behavioural arousal could indicate a

sympathetic response. Thus, any changes in the pattern and extent of these response parameters may contribute to the understanding of specific neural influences in the initiation, maintenance and/or development of certain haemodynamic dysfunctions, such as, hypertension.

In terms of the additional information provided by measuring three response parameters this was largely supported. That is, average, maximum and minimum HR did contribute independently to the results reported in all five of the experiments contained in this thesis. This, in turn, enhanced both the validity and generalisability of these results when discussing the effects of environmental factors, such as, predictability, control and feedback. However, both the psychological and physiological meaning of these response measures were inconclusive. In terms of the former, the pattern of responding in all three HR parameters was inconsistent both within and across experiments which meant that any suggestions concerning, for example, the link between peak HR and maximal effort/task engagement were largely unsupportable.

During the first study, for instance, the experimental task consisted of periods promoting both high and low levels of effort which, based on predictions made in Chapter 5 Section 2.2.1, should have enhanced any potential differences between average-maximum HR and minimum HR. Indeed, it had been suggested that average and maximum HR should be most elevated during periods of high effort (i.e. high behavioural arousal) whereas, minimum HR should show a less clear-cut pattern of responding with similar activity in periods of high and low behavioural arousal. Although the results of Experiment One showed slight differences between the three cardiac measures in their response patterning during the task, these were insignificant and certainly not as great as had been anticipated by the literature. A similar pattern emerged in Experiments Two and Three with all three HR measures showing a high level of communality. However, when the experimental situation was changed from one of passive exposure (i.e. Experiments Two and Three) to that of active coping, as in Experiment Four, a different pattern emerged with average and maximum HR producing only slight changes in responding as a result of the experimental manipulation. In contrast, minimum HR showed highly significant changes as a function of predictability and control both within and across trials. It was suggested (see Chapter 9, Section 4), that peak deceleration or the ability to lower cardiac activity (i.e.



'relax') may be more responsive to the degree of effort and/or behavioural uncertainty engendered by the task, particularly in situations involving aversive consequences and active coping mechanisms. However, this argument could not be sustained in the light of the findings from Experiment Five in which a similar active coping task was employed. That is, where any significant differences did emerge as a result of the experimental manipulation these were due to changes in the pattern of average and maximum HR responding and were not, as previously suggested, due to minimum HR. Thus, the significance of employing these three HR response parameters in terms of the differential psychological meaning they might contain, appears questionable.

Consequently, it might be argued that these findings do not create a strong position from which to propose that the three HR components should be used as an index of neural innervation, nor that these parameters should be part of the picture in models of haemodynamic dysfunction. Nevertheless, I would still like to propose that by employing such non-invasive measures (in combination with pharmacological sympathetic and parasympathetic blocking agents) in future research the physiological mechanisms involved in mediating the stress-illness link may be clarified to a certain extent. At present, for example, most contemporary models examining physiological mediation in stress-illnesses processes are concerned solely with psychophysiological hyperreactivity. That is, individuals have a tendency to respond to aversive stimulation with overly large autonomic or neuroendocrine responses and a reduction in these responses after termination of that stimulation. The relationship between these heightened responses and ill-health is, therefore, that repeated or sustained exposure to the event may be pathogenic. Indeed, support for the relevance of this process to disease comes from the hypertension literature in which it has been shown that exaggerated responses to certain tasks resulting primarily from heightened sympathetic tone contributes to the development of essential hypertension (e.g. Matthews, Weiss, Dettre, Dembroski, Falkner, Manuck & Williams, 1986; Obrist, 1981; Steptoe, 1981).

However, this view has been challenged in a number of respects. Firstly, by those studies which have indicated that physiological processes other than cardiac hyperreactivity are responsible for the initiation, maintenance and/or development of hypertension in some individuals (Steptoe, 1981). Secondly, because support for the hyperreactivity process in

the mediation of other bodily dysfunctions remains elusive (Stephoe, 1991). Essentially, these difficulties might simply be a result of the limitations imposed by existing measurement techniques in which researchers have not been able to disentangle the complex network of physiological processes. Equally, however, it could be that the biological pathways mediating stress influences on health are more complex than is sometimes appreciated, and that these difficulties have occurred because other potentially important mediating processes have been ignored.

In this thesis, it was suggested that it may not be the degree of hyperreactivity *per se* which is important for all individuals, but in certain situations the converse; namely, an individual's ability to lower physiological function and for how long and to what extent. This is not meant to imply that the two processes are mutually exclusive. On the contrary, both may play an important role in the stress-illness link. For instance, it could be that cardiovascular hyperreactivity influences the aetiology of haemodynamic dysfunctions such as essential hypertension, whilst the maintenance and/or recurrence of this disorder in those with a pre-existing health problem may depend on the duration and magnitude of lowered cardiac function in that it weakens the individuals biological defences making him or her more vulnerable to the disease in the long-term. These differing influences may, in turn, be provoked by a number of other factors ranging from the type of aversive stimulation (e.g. active-passive, controllable-uncontrollable, predictable-unpredictable), the degree of threat encountered, the level of effort and/or distress, individual differences in coping resources and strategies, and the organisms biological vulnerability or predisposition.

If it is established that different mediating mechanisms may be important, it will then be necessary to focus greater attention on the specific neural pathways underlying these processes. For instance, cardiovascular hyperreactivity underlying hypertension has been associated with exaggerated sympathetic/adrenomedullary stimulation resulting from cardiac-somatic uncoupling (e.g. Obrist, 1976, 1981). On the other hand, the influences of lowered function may primarily be under parasympathetic control where excessive, metabolically inappropriate vagal drive masks sympathetic effects. Obviously, I do not mean to suggest that these sympathetic-parasympathetic influences necessarily act synergistically as during exercise (Robinson, Epstein, Beiser & Braunwald, 1966).

Indeed, such a position would be naive and largely unsupportable given recent work (e.g. Berntson, Cacioppo, Quigley & Fabro, 1994; Matthews *et al.*, 1986; ). Berntson *et al.* (1994) suggested, for instance, that autonomic control of dually innervated target organs cannot adequately be perceived of as a continuum ranging from parasympathetic to sympathetic dominance. Instead, autonomic control has multiple modes in which the two divisions may vary reciprocally, coactively, or independently. Thus, it is not simply a matter of sympathetic activation corresponding with parasympathetic inhibition, rather autonomic control ranges from co-activation to co-inhibition of sympathetic and parasympathetic branches and reciprocal and uncoupled activation in between (i.e. autonomic control is two-dimensional). Opposing or reciprocal influences will, therefore, result in mutually synergistic effects on the target organ whilst, non-reciprocal modes of co-activation/inhibition minimise target organ response since the two branches yield opposing effects.

On the basis of such propositions, which have received growing recognition in the psychophysiology literature, it is likely that even if a future research program did find that the influence of lowered cardiac function was indeed important in the long-term effects of haemodynamic dysfunction, then more refined methods for evaluating the relative contribution of sympathetic and parasympathetic tone will need to be developed. Only then will the study of links between physiological processes and ill-health be significantly advanced. The position taken in this thesis - the employment of maximum, minimum and average HR in order to delineate specific neural innervations - was probably naive. Nevertheless, the use of such measures should not be prematurely dismissed since the study of more than one cardiovascular parameter, although inherently time-consuming, may increase reliability in identifying consistent patterns of response. These response patterns may, in turn, help identify the facets of aversive stimulation (e.g. the level of behavioural effort and uncertainty, predictability and personal control) which are important in the initiation and development of stress processes and, consequently, health outcomes.

### 3.2. Electrodermal Activity

Electrodermal activity (EDA) will not be discussed in much detail largely because of the inconsistency of results reported during the experiments contained in this thesis. In terms of the environmental factors control and feedback, there were no differential effects on EDA. Temporal predictability had a significant impact on one EDA parameter; namely, response amplitude. However, the pattern of these effects was significantly different across experiments. In Experiments One and Three, the presence of temporal information warning of the occurrence of an aversive event had no differential effect on response amplitudes. In contrast, during Experiment Two the warning signal was found to attenuate responding compared to situations involving temporal unpredictability, whilst in Experiment Four predictability was found to have augmentative effects.

The differential effects of predictability should not have been unexpected given the vastly differing results reported in the literature. Indeed, the presence of temporal information has been associated with increases in EDA, decreases in EDA or, no differential effects (see Chapter 2, Section 3.3.2). Nonetheless, it was surprising that the only significant results obtained in the electrodermal system were from response amplitude measures, particularly since the vast majority of past studies have reported predictability effects only with tonic SCL and/or frequency of NSFs measures. It is unclear why this was the case. Indeed, although the various parameters of EDA are not interchangeable with regard to behavioural significance in that they may represent partially independent sources of information (e.g. Venables & Christie, 1980), they do have similar underlying physiological mechanisms. That is, they are all a product of the eccrine sweat glands which are predominantly under the control of the sympathetic nervous system (see Wallin, 1981). Thus, it might be expected that the differential effects observed in one measure would, over the course of four experiments employing 184 subjects and differing aversive stimulation, be mirrored in one of the other EDA parameters during at least one study. Accordingly, it was suggested (see Chapters 6 & 9, Sections 4) that the significant response amplitude effects may have been spurious perhaps a result of the measurement and/or quantification procedures employed in this thesis. Indeed, it has been shown repeatedly in the psychophysiological literature that EDA effects are highly sensitive to such factors.

Even if this were the case, however, two factors are still unclear. Firstly, why there were no effects as a function of the other environmental factors manipulated in this thesis; namely, control and feedback. Past studies (see Chapter 4, Section 3) have reported that various types of control (e.g. instrumental, self-administered, equated, perceived and potential) can alter the pattern and/or magnitude of EDA parameters (especially SCL and frequency of NSF) in a variety of situations involving a number of aversive stimuli (e.g. noise, electric shock, cognitive tasks). Secondly, why the electrodermal system in general appeared unresponsive to situational variables whereas, the cardiovascular system responded to the presence or otherwise of predictability, control and feedback and, to alterations in these factors (e.g. changes in the controlling response).

This type of reported disassociation between the various parameters of the autonomic nervous system has a long tradition in psychophysiological research (e.g. Mason, 1972, 1975). Thus, the findings reported here were not entirely unexpected. Indeed, it merely indicates that factors such as control which attenuate the response in one system may have the reverse or no effect in other systems (see also Steptoe & Appels, 1989). Essentially, this would appear to suggest that the concept of a single stress response must be called into doubt, and that generalisations originally proposed by Selye (1956) concerning stress as a single, unitary response produced by "diverse noxious agents" are, with the advent of current measurement and recording technologies, unwarranted and untenable. Indeed, current views, such as the stress-diathesis model, emphasise the role of the individual's perception of a noxious stimulus and the effectiveness of coping strategies as determinants of the response to the situation. Thus, the physiological response is determined by the characteristics of the event (as perceived by the individual), the homeostatic demands imposed by the event and by the individual's response to that event. The same event may, therefore, lead to different patterns of physiological change in different people or at different times in the same person. Nevertheless, individual physiological parameters may still be of use as indices of the response to a particular class of events but, for this approach to be valid, it needs to be specified what properties of the event, or what component of the response, the measure is presumed to reflect. For instance, recent research has indicated that HR changes are linked to active coping mechanisms involved in being attentive and in preparation for making an effortful response

(e.g. Light, 1981). On the other hand, EDA has generally been taken as an indicator of affective constructs, such as, anxiety (Kilpatrick, 1972). Thus, affect elicited and effortfulness of coping may or may not be linked on any particular occasion and the degree of association varied accordingly. Consequently, future research programs need to focus on determining what facets of the experimental situation correspond with changes in individual response systems and how these are related, in turn, to individual's appraisal and coping strategies.

#### 4. A RE-EXAMINATION OF THE STRESS-DIATHESIS MODEL

As reviewed in Chapter 1, one contemporary framework for understanding the stress-health relationship is the stress-diathesis model proposed by Steptoe (1991) in which he identifies the potential relationships between physiological, cognitive, behavioural and environmental factors that might influence health outcome. This model will be briefly reiterated so that its propositions can be re-examined in the light of present experimental findings.

The basis for the stress-diathesis model is that exposure to a noxious event or situation, whether it be electric shock, the loss of a job or the death of a spouse is not sufficient to produce disease or ill-health. Individuals differ in their response to 'the same' event. Instead, this response - the psychobiological stress response as Steptoe terms it - depends upon whether the stimulus is, for example, predictable rather than unpredictable or controllable rather than uncontrollable. Thus, variations in these environmental factors or 'psychosocial demands' will determine to some extent whether the stimulus will be linked to the stress process. However, the initiation and generation of this stress process is also said to be dependent on certain 'coping resources', particularly appraisal processes, such as, how the event is perceived and thought about and the responses taken to cope with the event (i.e. responses to change the situation or to deny or alter its interpretation). The extent, maintenance and outcome of these psychobiological stress responses is mediated, in turn, by two primary pathways; the cognitive-behavioural and the psychophysiological. Involvement of the cognitive-behavioural pathway implies that psychosocial stressors influence health through impairment of health-related behaviours or practices. That is, if it

is not possible to remove the source of stress or escape the situation, the individual may resort to potentially damaging behaviours, for instance, drinking alcohol, cigarette smoking or the intake of other drugs such as, tranquillisers or other benzodiazepines. Those associations between stress and illness which are not accounted for by alterations in health-related behaviours may be linked instead through psychophysiological pathways. The three main sources of mediation identified in the model are psychophysiological hyperreactivity, disease stability and host vulnerability processes. Each of these mediators has been found to influence the aetiology, maintenance and recurrence of a wide range of pathological responses from cardiovascular dysfunctions, gastrointestinal lesions to respiratory infections. The pattern and extent of cognitive-behavioural and psychophysiological effects on health-related outcomes is in turn expressed against a background of the individual's biological and constitutional vulnerability. Factors such as the person's genetic background, sex and age, physical fitness and (for women) menstrual cycle phase have all been shown to influence reactivity.

This type of stress-diathesis model therefore attempts to delineate the processes that intervene between noxious events and disease and to examine the social, psychological, and biological factors attributing to the stress process. As a result, it suggests certain mediating mechanisms to be tested, suggests potential linkages between factors at various levels (social, psychological and physiological) and attempts to specify temporal processes in which these possible linkages may work. However, at the same time, the multicomponent nature of stress emphasises that there will probably not be any direct or univariate link between environmental events, physiological responses and disease processes. One way of dealing with this complexity is to study single factors within this stress-diathesis framework. That is, if unpredictability is associated with gastrointestinal pathology, this variable should be studied in a given situation with a certain subject population whilst at the same time investigating the possible factors mediating the stress-illness link. By adopting this type of research strategy, detailed information can be obtained concerning the specific parameters and possible health outcomes of each psychosocial variable. Following this, the interrelationships between these variables can be examined in specific situations in order to allow a more precise prediction of who will respond, in what situation, and in terms of what biological, cognitive, behavioural and/or emotional

parameters. The aim of this thesis was to adopt such a strategy in the detailed examination of two psychosocial factors - control and predictability. By restricting the present investigation to this level it was hoped that propositions formulated within the stress-diathesis model in the past could be made explicit. Furthermore, an examination of the potential effects of feedback was also undertaken since it was believed, based primarily on past studies (see Chapter 3), that the stress-diathesis model could be extended in order to incorporate the potential effects of this factor.

The significance of control in health and disease has been studied extensively, particularly in recent years (see reviews by Anisman & Zacharko, 1982; Steptoe & Appels, 1989; Sklar & Anisman, 1981). Nevertheless, the role of controllability in health-related outcomes is far from clear due largely to the complexity of the control concept and the lack of terminological and methodological rigour. In terms of the latter, many studies have not systematically manipulated control parameters varying instead the type of control offered (i.e. instrumental, cognitive, retrospective, perceived), whether control is exerted over the initiation, termination, duration or intensity of stimulation, the type of controlling response available (e.g. active-passive, certain-uncertain, easy-difficult) and the degree of additional predictability offered by control. Thus, on the one hand it has been shown that the belief in control over sources of stress is protective, and that loss of control with the accompanying feelings of helplessness is deleterious. Indeed, such helplessness has been implicated in the aetiology and maintenance of such diverse dysfunctions as depression through the depletion of amine neurotransmitters in the brain (Anisman & Zacharko, 1982), and malignant tumour growth through the involvement of central neurochemical processes (Sklar & Anisman, 1981) and/or peripheral steroid and immunological responses (Riley, Fitzmaurice & Spackman, 1981). Alternatively, other studies appear to support the opposite contention; namely, that control, and particularly control that requires effort to exert, is associated with heightened sympathetic and catecholamine reactions which may predispose to hypertension and coronary heart disease, particularly in those at risk to develop such disorders (e.g. Light, 1987; Obrist, 1981; Steptoe, 1986).

Examination of the results summarised in Section 2.3 of this chapter would support the deleterious effects of both uncontrollability and controllability during situations involving active coping behaviour with aversive consequences. In this thesis, whether



control was beneficial or not was largely dependent on the presence of a second psychosocial factor; namely, stimulus predictability. Where no predictive information was available, uncontrollability provoked more substantial elevations in HR than a situation in which individuals had a degree of perceived control over the duration of the aversive event. In predictable situations, this pattern was reversed with heightened reactions to the task when control was present regardless of whether predictability could facilitate the coping response (Experiment 4) or not (Experiment 5). Thus, predictions made by Steptoe (1991) and Obrist and colleagues (e.g. Light & Obrist, 1980; Obrist, Gaebelin, Teller, Langer, Grignolo, Light & McCubbin, 1978), that the beneficial effects of control can be reversed in situations requiring alert, difficult behavioural responses and those in which task outcome is uncertain, should be extended to include the interactional effects of other environmental factors, such as, predictability.

Whilst the deleterious effects of uncontrollability and unpredictability were largely expected (see Miller, 1979), the negative additive effects of control and predictability were surprising. Indeed, it had been suggested both in Chapter 1 and during past studies (e.g. Weinberg & Levine, 1980), that the provision of temporal information might augment the beneficial effects of perceived control. This is primarily because past findings, particularly from the animal laboratory, have indicated that organisms show a distinct preference for temporal information concerning aversive stimulation and that this, in turn, leads to stress-reducing effects in terms of physiological indices. However, as shown in Chapter 2, matters involving human beings are slightly more complex. That is, for some people and in some circumstances, being informed of an aversive event is far from being either preferred or a 'stress-reducing' factor. It may well be that the opportunity to control augments the deleterious effects of such predictability in some situations, whilst in others availability of control ameliorates these effects. Unfortunately, however, there are no persuasive theories which can explain why this may be the case.

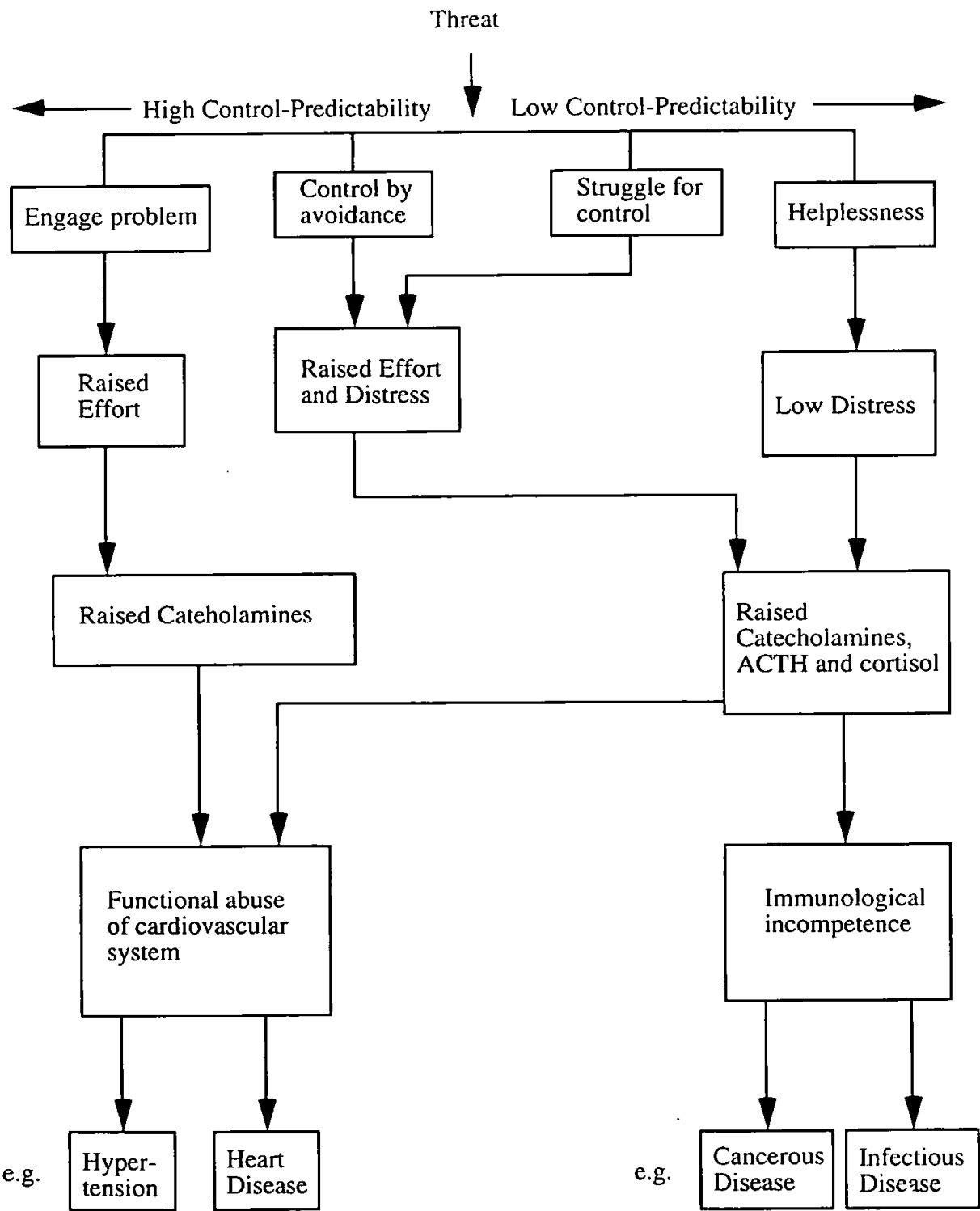
It could be argued that the differential effects of control may reflect individual differences in the preference for control, the capacity and resources available for control and strategies available for exercising that control, and that objectively similar environmental conditions may evoke different responses because of this 'person' denominator. Such formulations might be linked conceptually to Mason's (1975) theory

which emphasises the susceptibility of different neuroendocrine systems to the specific emotional component of different environmental settings. Equally, it might be that such differential responses are a result of specific experimental parameters which have been shown in the past to influence the direction and magnitude of psychophysiological stress. Those factors which have been detailed in this thesis (see Chapters 2 & 4) include; the intensity and chronicity of the aversive stimulation, the type and length of the warning signal, level of task difficulty, the type of controlling response (i.e. active-passive), whether the controlling response is over the initiation, termination or duration of the aversive stimuli, and the type of control (i.e. behavioural, perceived, cognitive, retrospective). In terms of the latter, for example, much of the original work employed an overt or 'instrumental' behavioural response involved in avoiding or escaping the aversive event. In contrast, the present experiments employed perceived instrumental control over stimulus duration. Although perceived and actual instrumental control have generally been treated as synonymous when employed in previous studies and during the experiments contained in this thesis, it may be that the latter manipulation is more salient in terms of eliciting autonomic responses (see Arntz & Schmidt, 1989). That is, the results of the studies reported here may have been significantly different if the individual's actions could have affected the occurrence of noxious events physically. Instead, in the present studies the adverse effects may have been due to the availability of control highlighting the generally helpless situation of the subject. It is not clear, however, why control should have had this type of effect in a predictable but not unpredictable situation, particularly since 'feelings of helplessness' would be expected to be greater in the latter condition. Indeed, one point to emerge as a consequence of the findings reported here is that a warning signal should not be regarded as incidental but rather as a key factor for the interpretation of the psychophysiological response in aversive situations. Furthermore, what may be of equal importance in terms of physiological correlates, is not whether control is possible or the degree of temporal information available but the consequences of these factors (see also Steptoe, 1983; Steptoe & Appels, 1989). For example, it might be that having responsibility for the exercise of control may create a different type of stress which may, in turn, have differing implications for health.

Indeed, such an interpretation would be given credence by the work of Frankenhaeuser (1983) in which she discusses the influence of task demands on health and efficiency through the sympathetic-adrenal medullary system with the secretion of the catecholamines adrenaline and noradrenaline, and the pituitary-adrenal cortical systems with the secretion of cortisol. Basically, Frankenhaeuser argues (e.g. Frankenhaeuser, Lundberg & Forsman, 1980; Lundberg & Frankenhaeuser, 1980) that *effort*, when control is possible, is more likely to be associated with raised catecholamine levels whilst *effort and distress*, when control is not possible, are more likely to be associated with raised catecholamine and raised corticosteroid levels. These results are consistent with the type of animal model proposed by Henry and Stephens (1977) in which the sympathetic-adrenal system is activated when the organism is challenged in its control of the situation whereas, the pituitary-adrenal system is associated with the conservation or withdrawal response.

Essentially, the model proposed by Frankenhaeuser provides an initial framework for understanding how factors, such as, control in stressful situations might influence the short or long term health of an organism. If the findings concerning temporal predictability are added to this, there is a possible basis for relating the effects of environmental factors in specific situations to a stress-diathesis framework (see Figure 93). For instance, Frankenhaeuser's model assumes that situations characterised by a response of raised effort (i.e. where a high degree of control and temporal predictability is available resulting in enhanced task engagement and the challenge to perform well) augments catecholamine levels. This could, in turn, increase the functional strain on the cardiovascular system. Indeed, such arguments would be supported firstly, by findings reported in Experiments Four and Five of this thesis, in which predictable-control conditions led to marked increases in cardiovascular reactivity to aversive stimulation. Secondly, by a large number of contemporary studies in which effortful coping behaviour has been found to result in heightened haemodynamic responses in mild hypertensives and subjects deemed to be at elevated risk for hypertension (e.g. Steptoe, Melville & Ross, 1984). In contrast, effort and distress together (i.e. where little or no control is available in a temporally unpredictable situation leading to a struggle for control and/or helplessness) might be expected to raise catecholamine levels together with an increase in ACTH and cortisol secretion. The former may lead to heightened cardiovascular activity as suggested by the

Figure 93. The possible links between threat, predictability, controllability, effort, distress and ill-health (adapted from Fisher, 1986; p. 170)



results of Experiments Four and Five, in which unpredictable-no control conditions resulted in increased HR reactivity. On the other hand, the latter has been implicated in the suppression of the immunological response (Cox & Mackay, 1982) which, in turn, could augment the likelihood of infectious diseases and in some situations the possibility of cancers (e.g. Sklar & Anisman, 1981; Stoll, 1988). Thus, the *consequences* of certain psychosocial factors in terms of effort or effort and distress may have different implications in terms of the stress-diathesis model (see also Bandura, 1977; Fisher, 1986).

This type of framework raises a number of important questions concerning the importance of environmental factors with regard to the stress-illness link. The first relates to the conceptual and methodological advantages of developing the demand-effort construct. Although such an explanation is attractive in that it seems to have great potential for generality and wide applicability within stress-illness research, it does tend to replace two difficult concepts (predictability & control) with another (effort). Of course, this may be quite legitimate, however, it does mean that the issue of effort needs to be addressed in more detail (see Kahneman, 1973; Schonpflug, 1983). The second question concerns the significance of 'person' factors within this framework. That is, although the model does not consider effort and demand as properties of the situation but rather as interactional concepts as much determined by people as by situations, the potential relationships between objective control levels and factors such as, differences or changes in subjective (i.e. cognitive) or perceived control over time, and individual differences in the preference for control and the type of behavioural strategies adopted, need to be examined further so that narrower, more precise hypotheses can be developed. This is particularly important as an individual's cognitive appraisal of the balance between the severity of the situational demands on the one hand and his or her personal coping resources on the other is crucial to a stress-diathesis type model.

The third issue raised by this framework is that it ignores, to any great extent, the possibility that conditions which evoke effort but not distress thereby activating the sympathetic-adrenal system, are potentially beneficial. That is, raised effort might alleviate the short-term effects of aversive stimulation particularly when that effort is rewarded (i.e. success is the outcome) whilst in the long-term, the cost may be raised arousal levels particularly when the outcome is negative. Thus, although raised effort levels may

generally be "depression resistant", there could be long-term penalties for struggle and failure. The notion that stress might not always have negative effects has a long tradition in psychophysiological research (e.g. Cannon, 1935; Selye, 1976). Selye (1976), for instance, suggested that the physiological stress reaction is not automatically deleterious since certain degrees of stress (what he termed 'eustress') can be a motivator to growth, development and adaption. Indeed, it cannot necessarily be inferred from the work presented in this thesis that the cardiovascular reactions associated with predictable-controllable aversive stimulation were maladaptive.

Finally, whether or not stress-reactions are maladaptive is, as indicated in the previous paragraph, dependent on the chronicity of aversive stimulation. That is, even though control-prediction conditions were found to elicit heightened HR responses during the experiments reported here, it could be that these effects might have been altered if measured over the long-term. Indeed, the development of interactive models of stress has served to highlight one of the inherent problems of stress research; namely, that stress needs to be understood in a dynamic, process-orientated model in which time plays a vital role. Thus, while assessment of psychosocial factors in acute studies is important in that it helps to identify potentially significant variables and allows parametric measurement of the independent influence of those factors, the findings of such studies should be treated with caution in applying them to stress-illness problems in the long-term. Since they examine only the initial reaction to the stimulus, where the novelty and unfamiliarity of the laboratory situation contribute to the general physiological response. If observations were continued into phases where the necessary skills had been obtained to successfully complete the tasks, and subject's general feelings of apprehension and uncertainty had been ameliorated, the conclusions concerning these psychosocial variables may be radically different. During Experiments Two to Five of this thesis, for example, levels of cardiovascular activity varied significantly both within trials (i.e. during anticipatory and impact periods) and across trials. In terms of the latter, subjects generally produced greater cardiovascular reactivity during later trials (perhaps indicating sustained motivation or task involvement). What must be included in future studies, therefore, are accurate estimates of the duration of both the psychological and biological stress processes in relation to the duration of the processes underlying disease. This exploration of the precise causal time

sequence will, in turn, enable researchers to eliminate the problem that the observed relationships between variables in past studies may have been a product of spurious effects rather than causal linkage. Additionally, knowledge of the duration of stress and ill-health may help our understanding of the variations in the stress-illness relationship, for instance, why certain stressors augment vulnerability in the long-term but reduce it in the short-term (e.g. Sklar & Anisman, 1981).

Although the relationships between predictability, control, demand, effort, distress and psychophysiological correlates have yet to be explored in a systematic program of research, certain relationships have emerged. It is hoped that more detailed examination of these issues will replace the overly simplified suggestions such as those made above with narrower, more precise hypotheses integrating individual psychosocial factors and mediating mechanisms such as, the degree of effort-distress and regulation of demands (e.g. changes in competence and aspiration level, the role of learning) to specific physiological correlates and, in turn, to certain types and degrees of physical risk.

The same need for specificity applies to the concept of feedback. For example, the framework outlined in Figure 93 explores the possible links between physical risk and decision-making about control; subjects' decision to deal with the problem raises effort and increases catecholamine secretion as long as success results. However, increased effort and distress result if the struggle to succeed produces failure. Thus, the provision of information feedback, which provides an individual with knowledge of specific event parameters, should lead to enhanced decision-making by making the controlling response and response outcome less uncertain. Lowered uncertainty should, in turn, promote increases in perceived capability and coping and behavioural efficiency in general (see Germana, 1972; Phillips, 1989). Consequently, the interactional effects of feedback with those of control and temporal predictability are likely to stimulate raised effort due to lowered situational ambiguity. Indeed, during Experiment Five the presence of feedback resulted in heightened HR responses compared to situations where no feedback was available. Furthermore, these augmentative effects were enhanced by the presence of temporal predictability and, to a lesser extent, the perception of control (see Chapter 11, Section 2).

However, if individuals receive no feedback to show that the response has been successful, effort and distress may result since the level of threat has been intensified by increasing behavioural uncertainty and limiting the individual's sense of control. Helplessness or failure to handle the problem associated with low effort may eventually result, particularly if the penalty of control is high (e.g. electric shock) and/or the situation offers little or no high valued incentives (e.g. money). Again, such an interpretation would be supported by findings from Experiment Five in which unpredictable or uncontrollable situations providing no feedback resulted in heightened HR responses to aversive stimulation.

As this analysis develops through future research, it may be possible to delineate the specific effects on effort-distress and corresponding physiological correlates produced by gradients of feedback; i.e. from high positive feedback through to high negative feedback. That is, high success rates resulting in high levels of positive feedback may promote increased effort and a corresponding shift to raised catecholamine levels, whilst a high degree of negative feedback resulting from low success rates may result in raised effort and distress. Consequently, tasks offering low positive or negative feedback, or those where no feedback is available could lead to intermediate levels of effort, distress and physiological activity. Obviously, this framework is highly tentative at present and its future success will depend not only on studies incorporating different types and degrees of feedback, but also on investigations into the potential interrelationships between predictability, control, feedback, effort-distress and psychophysiological reactivity. Nevertheless, this type of framework does have great scope for generality, as it can incorporate the differential effects of levels of task difficulty, the type of feedback employed (i.e. RF or KR) and the presence, type and magnitude of incentives. Such factors have been shown in past studies to influence the pattern and extent of physiological function during aversive stimulation (see Chapters 3 & 4).

Furthermore, this type of framework allows for the influence of personality dispositions or coping resources on the effects of feedback which is a fundamental element of the stress-diathesis model. For instance, information from the environment can be unambiguous (i.e. high positive feedback) and yet a person can experience uncertainty and distress. Such uncertainty can arise, for example, from the individual not knowing what to



do or being uncertain that they can successfully execute the behaviour required to produce the desired outcome. These efficiency expectations depend, in turn, on the type of task and the level of difficulty employed (see Chapter 6). On the other hand, even when there is ambiguity in the environment (i.e. low negative or no feedback), a person can feel confident about what to do and show little sign of distress. This can happen when a individual resolves the situational ambiguity by refusing to acknowledge or attend to the lack of clarity in the information provided. Moreover, some people may even seek ambiguity rather than clarity (Gibbons & Wright, 1981). Thus, the outcome of feedback in terms of effort-distress and the resulting physical effects depends on an individual's belief in the beneficial consequences of feedback which influences how the task will be appraised and, through appraisal, subsequent coping activity, behaviour and psychophysiological activity.

In conclusion, it is clear from the many facets of personal control, temporal predictability and feedback which have been considered here that no one explanation will be sufficient for the various independent and relational effects of this type of psychosocial factor. Future research will need to systematically examine each of these variables in the context of specific stressful encounters so that: (i) the meaning or significance of these variables can be assessed in differing types of situations; (ii) an understanding of which facets of the encounter are susceptible to the influence of these factors can be achieved; (iii) the relationship between characteristics of the situation and cognitive appraisals of these psychosocial variables can be evaluated. These steps are necessary if researchers are to understand how predictability, control and feedback influence psychophysiological processes and ultimately stress-related outcomes.

The outcome of such a systematic programme of research would benefit in the long-term such stress-diathesis models as that proposed by Steptoe (1991). Indeed, this type of model if it is to conceptualise and measure those psychological aspects of the stress process that impact on disease processes, needs to increase the specificity of predictions linking threats, stress processes and illness. More specifically, it should define: the parameters of psychosocial variables and coping responses that result in certain subjective emotional reactions, the psychophysiological changes resulting from these cognitive appraisals, and the expected effects on health and ill-health. Such a framework needs, as a

primary factor, to include temporal variables to specifically delineate the onset and duration of stressors, psychophysiological, social and cognitive mediating processes, and end-points, such as, ill-health and disease.

Nevertheless, the development of stress-diathesis models has already achieved a great deal. For instance, the majority of researchers within the psychophysiology field have now moved from the naive and largely insupportable position that the loss of such environmental factors as control is stressful and conversely that availability of control ameliorates psychophysiological stress. Instead, it has been recognised that the 'stress-reducing' or 'stress-inducing' effects of such variables are largely dependent on certain experimental parameters, and their *continuing* interaction with 'person' factors, such as, individual differences in coping resources and strategies. The independent and relational aspects of these variables are now being systematically examined. Equally, it has been recognised that there is a need to assess the specific neural innervations underlying such superficial measures as heart rate and the electrodermal response in order to examine the potential links between physiological activity and ill-health. This assessment will be furthered by the advance of contemporary methods and techniques encompassing a variety of approaches among humans, and in laboratory, clinic and population settings. Such advances will serve to unify the various disciplinary approaches involved in stress-illness research, thereby refining the predictive and explanatory power of stress-diathesis type models. Once this has been achieved, the implications of these models in terms of the prevention, management and treatment of stress-related illness and disease will be greatly advanced.

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APPENDIX  
PRE-TASK INSTRUCTIONS

EXPERIMENT 1

No Feedback Group

The Game

- (i) Direction and Speed. Planes A and C fly around the blue circuit in the direction of the arrows, and travel at a speed of 2 steps per move (e.g. 1 to 5 or 6 to 2 is one move). Planes B and D fly around the black circuit in the direction of the arrows, at a speed of 1 step per move (e.g. 1 to 2 or 3 to 4 is one move). All four planes move simultaneously.
- (ii) Object of the Game. The object of the game is to coordinate the continuous flight of all four planes around their respective circuits without crashing (namely at positions 1 and 2).
- (iii) Changing Speed Rules. To help you prevent crashing, you may change the speed of one plane at a time, for one move only, after which the plane will automatically revert back to its normal speed. Planes A and C may be slowed down to 1 step per move whilst planes B and D may be speeded up to 2 steps per move. You cannot change the speed of any one plane twice in succession on the same trial.

How to Play

At the beginning of each trial you will be presented with the planes in their starting positions (e.g. A1 B3 C5 D4). A delay will follow. When prompted with "what is your next move" you are required to voice your first positions for each plane immediately, each having moved one position at their respective speeds (e.g. A5 B4 C1 D2). Another delay will follow. When prompted with "what is your next move" you are required to voice your second positions for each plane immediately (e.g. A6 B1 C5 D3). Following another pause, you will be told that it is the end of the trial. Another delay will follow, you will then be asked to rate your amount of perceived control using the rating scale in front of you. This you do by speaking out loud the number on the rating scale which corresponds to the amount of control you are feeling at that moment in time. Following another pause you will be told that the next trial starts in 10 seconds.

You may terminate the experiment at any time. If you want to just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

### Feedback Group

#### The Game

(i) Direction and Speed. Planes A and C fly around the blue circuit in the direction of the arrows, and travel at a speed of 2 steps per move (e.g. 1 to 5 or 6 to 2 is one move). Planes B and D fly around the black circuit in the direction of the arrows, at a speed of 1 step per move (e.g. 1 to 2 or 3 to 4 is one move). All 4 planes move simultaneously.

(ii) Object of the Game. The object of the game is to coordinate the continuous flight of all four planes around their respective circuits without crashing (namely at positions 1 and 2).

(iii) Changing Speed Rules. To help you prevent crashing, you may change the speed of one plane at a time, for one move only, after which the plane will automatically revert back to its normal speed. Planes A and C may be slowed down to 1 step per move whilst planes B and D may be speeded up to 2 steps per move. You cannot change the speed of any one plane twice in succession on the same trial.

#### How to Play

At the beginning of each trial you will be presented with the planes in their starting positions (e.g. A1 B3 C5 D4). A delay will follow. When prompted with "what is your next move" you are required to voice your first positions for each plane immediately, each having moved one position at their respective speeds (e.g. A5 B4 C1 D2). Another delay will follow. When prompted with "what is your next move" you are required to voice your second positions for each plane immediately (e.g. A6 B1 C5 D3). Following another pause, you will be told whether your controlling has been successful. Another delay will follow, you will then be asked to rate your amount of perceived control using the rating scale in front of you. This you do by speaking out loud the number on the rating scale which corresponds to the amount of control you are feeling at that moment in time. Following another pause you will be told that the next trial starts in 10 seconds.

You may terminate the experiment at any time. If you want to just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

## EXPERIMENT 2

### Predictable Groups

The experiment you are about to participate in involves the use of injury and mutilation slides which will be presented on the screen directly in front of you. It is important that you do not avert your eyes from the screen during slide duration. During the experiment, I will be measuring various physiological responses including Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a five minute rest period which allows you to acclimatise to the room. As soon as these five minutes have passed, the first trial will begin. At the beginning of each trial the numbers 5, 4, 3, 2, 1 will appear on the monitor in front of you for a short period of time. After the last number has been presented there will be a short wait during which nothing will happen. After this, the first slide will be presented. After each slide has been presented, there will be a short wait during which nothing will happen after which, the next trial will begin.

After the trials have finished, I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

### Unpredictable Groups

The experiment you are about to participate in involves the use of injury and mutilation slides which will be presented on the screen directly in front of you. It is important that you do not avert your eyes from the screen during slide duration. During the experiment, I will be measuring various physiological responses including Heart Rate

and Skin Conductance. Because these measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a five minute rest period which allows you to acclimatise to the room. As soon as these five minutes have passed, the first trial will begin. During the trials, you will be presented with the numbers 5, 4, 3, 2, 1 on the monitor in front of you followed, on some trials, by the slide. On other trials, the numbers and slide will occur randomly to one another. After each trial, there will be a short wait during which nothing will happen after which, the next trial will begin.

After the trials have finished I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

### EXPERIMENT 3

#### Predictable Group

The experiment you are about to participate in involves the use of loud noise which will be presented via the headphones. During the experiment, I will be measuring various physiological responses including Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a one minute rest period which allows you to acclimatise to the room. After this one minute, you will be presented with an example of the noise that you will hear throughout the experiment. Following this, there will be a further five minute rest period after which, the first trial will begin. At the beginning of each trial the numbers 5, 4, 3, 2, 1 will appear on the monitor in front of you for a short period of time. After the last number has been presented there will be a short wait during which nothing will happen. After this, the first noise will be presented. After each noise has been presented, there will be a short wait during which nothing will happen after which, the next trial will begin.



After the trials have finished, I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

### Unpredictable Group

The experiment you are about to participate in involves the use of loud noise which will be presented via the headphones. During the experiment, I will be measuring various physiological responses including Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a one minute rest period which allows you to acclimatise to the room. After this one minute, you will be presented with an example of the tone that you will hear throughout the experiment. Following this, there will be a further five minute rest period after which, the first trial will begin. During the trials, the numbers 5, 4, 3, 2, 1 will appear on the monitor in front of you followed, on some trials, by the noise. On other trials, the numbers and noise will occur in a random order. After each trial, there will be a short wait during which nothing will happen after which, the next trial will begin.

After the trials have finished I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

## EXPERIMENT 4

### Predictable-Control Group

The experiment you are about to participate in involves the use of loud noise which will be presented via the headphones. During the experiment, I will be measuring various physiological responses including Heart Rate and Skin Conductance. Because these

measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a one minute rest period which allows you to acclimatise to the room. After this one minute, you will be presented with an example of the noise that you will hear throughout the experiment. Following this, there will be a further three minute rest period after which, the first trial will begin.

During the experimental trials I will be measuring your reaction time to the noise that you'll hear through the headphones. At the beginning of each trial the numbers 3, 2, 1 will appear on the monitor in front of you for a short period of time. After the last number has been presented there will be a short wait during which nothing will happen. After this, the first noise will be presented. The noise will initially be presented for a short period of time, and then the message "Press button now" will appear on the screen, immediately as this appears you should press this button as fast as possible, trying not to move your whole arm as this affects HR recording. Do not press the button before the message "Press button now" appears on the screen. Your reaction time will be monitored by the computer and if it is within a certain time limit the duration of the noise will be reduced from 5 to 2 sec, if it is not within this time limit the noise will stay on for 5 sec.

After the trials have finished, I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

#### Predictable-No Control Group

The experiment you are about to participate in involves the use of loud noise which will be presented via the headphones. During the experiment, I will be measuring various physiological responses including Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a one minute rest period which allows you to acclimatise to the room. After this one minute, you will be presented with an example of

the noise that you will hear throughout the experiment. Following this, there will be a further three minute rest period after which, the first trial will begin.

During the experimental trials I will be measuring your reaction time to the noise that you'll hear through the headphones. At the beginning of each trial the numbers 3, 2, 1 will appear on the monitor in front of you for a short period of time. After the last number has been presented there will be a short wait during which nothing will happen. After this, the first noise will be presented. The noise will initially be presented for a short period of time, and then the message "Press button now" will appear on the screen, immediately as this appears you should press this button as fast as possible, trying not to move your whole arm as this affects HR recording. Do not press the button before the message "Press button now" appears on the screen.

After the trials have finished, I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

#### Unpredictable-Control Group

The experiment you are about to participate in involves the use of loud noise which will be presented via the headphones. During the experiment, I will be measuring various physiological responses including Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a one minute rest period which allows you to acclimatise to the room. After this one minute, you will be presented with an example of the noise that you will hear throughout the experiment. Following this, there will be a further three minute rest period after which, the first trial will begin.

During the experimental trials I will be measuring your reaction time to the noise that you'll hear through the headphones. At the beginning of some trials the numbers 3, 2, 1 will appear on the monitor in front of you for a short period of time followed by, a short wait during which nothing will happen. The noise will then be presented. However, during some trials these numbers will not appear before the noise. The noise will initially

be presented for a short period of time, and then the message "Press button now" will appear on the screen, immediately as this appears you should press this button as fast as possible, trying not to move your whole arm as this affects HR recording. Do not press the button before the message "Press button now" appears on the screen. Your reaction time will be monitored by the computer and if it is within a certain time limit the duration of the noise will be reduced from 5 to 2 sec, if it is not within this time limit the noise will stay on for 5 sec.

After the trials have finished, I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

#### Unpredictable-No Control Group

The experiment you are about to participate in involves the use of loud noise which will be presented via the headphones. During the experiment, I will be measuring various physiological responses including Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind, it is important that you remain as still as possible throughout the experiment.

After I leave the room, there will be a one minute rest period which allows you to acclimatise to the room. After this one minute, you will be presented with an example of the noise that you will hear throughout the experiment. Following this, there will be a further three minute rest period after which, the first trial will begin.

During the experimental trials I will be measuring your reaction time to the noise that you'll hear through the headphones. At the beginning of some trials the numbers 3, 2, 1 will appear on the monitor in front of you for a short period of time followed by, a short wait during which nothing will happen. The noise will then be presented. However, during some trials these numbers will not appear before the noise. The noise will initially be presented for a short period of time, and then the message "Press button now" will appear on the screen, immediately as this appears you should press this button as fast as possible, trying not to move your whole arm as this affects HR recording. Do not press the button before the message "Press button now" appears on the screen.

After the trials have finished, I will come back into the room and debrief you. If you wish to terminate the experiment at any time you are free to do so. Just speak to me through the intercom which is open throughout the experiment. Do you understand the procedure?

## EXPERIMENT FIVE

### Feedback Groups

Predictable-Control Group. The experiment you are about to participate in involves measuring both Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind it is important that you remain as still as possible throughout the experiment. After I leave the room there will be a 1 minute rest period which allows you to acclimatise to the room. Following this there will be an example of the noise that you will hear throughout the experiment; firstly, the short duration noise which lasts 2 sec and then 15 sec after that the long duration noise which lasts 5 sec. Following this, there will be a further 3 minute rest period to enable you to settle back down before the beginning of the experiment. During this time nothing will happen. After these 3 minutes the first trial will begin.

During the trials I will be assessing the impact of aversive noise on reaction time. At the beginning of each trial the word "START" will appear on the monitor for a few seconds, as soon as this appears press this button as fast as possible, trying not to move your whole arm as this affects HR recording. The computer then stores your RT, if it is within certain time limits set by the computer (which are not necessarily the fastest RT possible) then the duration of the noise will be reduced from 5 sec to 2 sec. If your RT is not within these limits then the duration of the noise will be 5 sec. Shortly after you've responded you will be told what the duration of the noise will be.

There will then be a few seconds rest during which nothing will happen. Following this, you will receive a warning of 3, 2, 1 on the screen, 5 sec after that you will receive the noise.

After the trials are completed I will come back into the room, ask you a few questions and debrief you. If you wish to terminate the experiment at any time, just speak

to me through the intercom, which is open throughout the experiment. Do you understand the procedure?

Predictable-No Control Group. The experiment you are about to participate in involves measuring both Heart Rate and Skin Conductance. Because these measures are affected by movement of any kind it is important that you remain as still as possible throughout the experiment. After I leave the room there will be a 1 minute rest period which allows you to acclimatise to the room. Following this there will be an example of the noise that you will hear throughout the experiment; firstly, the short duration noise which lasts 2 sec and then 15 sec after that the long duration noise which lasts 5 sec. Following this, there will be a further 3 minute rest period to enable you to settle back down before the beginning of the experiment. During this time nothing will happen. After these 3 minutes the first trial will begin.

During the trials I will be assessing the impact of aversive noise on reaction time. Sometimes the noise is of 5 sec duration and sometimes 2 sec. At the beginning of each trial the word "START" will appear on the monitor for a few seconds, as soon as this appears press this button as fast as possible, trying not to move your whole arm as this affects HR recording. Shortly after you've responded you will be told what the duration of the noise will be.

There will then be a few seconds rest during which nothing will happen. Following this, you will receive a warning of 3, 2, 1 on the screen, 5 sec after that you will receive the noise.

After the trials are completed I will come back into the room, ask you a few questions and debrief you. If you wish to terminate the experiment at any time, just speak to me through the intercom, which is open throughout the experiment. Do you understand the procedure?

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acclimatise to the room. Following this there will be an example of the noise that you will hear throughout the experiment; firstly, the short duration noise which lasts 2 sec and then 15 sec after that the long duration noise which lasts 5 sec. Following this, there will be a further 3 minute rest period to enable you to settle back down before the beginning of the experiment. During this time nothing will happen. After these 3 minutes the first trial will begin.

During the trials I will be assessing the impact of aversive noise on reaction time. At the beginning of each trial the word "START" will appear on the screen for a few seconds, as soon as this appears press this button as fast as possible, trying not to move your whole arm as this affects HR recording. The computer then stores your RT, if it is within certain time limits set by the computer (which are not necessarily the fastest RT possible) then the duration of the noise will be reduced from 5 sec to 2 sec. If your RT is not within these limits then the duration of the noise will be 5 sec. Shortly after you've responded you will be told what the duration of the noise will be.

There will then be a few seconds rest during which nothing will happen. You will then receive the noise, on some trials you will receive a warning of 3, 2, 1 before the start of the noise, on others you will receive no warning.

After the trials are completed I will come back into the room, ask you a few questions and debrief you. If you wish to terminate the experiment at any time, just speak to me through the intercom, which is open throughout the experiment. Do you understand the procedure?

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After the trials are completed I will come back into the room, ask you a few questions and debrief you. If you wish to terminate the experiment at any time, just speak to me through the intercom, which is open throughout the experiment. Do you understand the procedure?

### No Feedback Groups

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within certain time limits set by the computer (which are not necessarily the fastest RT possible) then the duration of the noise will be reduced from 5 sec to 2 sec. If your RT is not within these limits then the duration of the noise will be 5 sec. Shortly after you've responded you will be told that the noise will follow.

There will then be a few seconds rest during which nothing will happen. Following this, you will receive a warning of 3, 2, 1 on the screen, 5 sec after that you will receive the noise.

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