INFORMATION TO USERS

This was produced from a copy of a document sent to us for microfilming. While the most advanced technological means to photograph and reproduce this document have been used, the quality is heavily dependent upon the quality of the material submitted.

The following explanation of techniques is provided to help you understand markings or notations which may appear on this reproduction.

1. The sign or "target" for pages apparently lacking from the document photographed is "Missing Page(s)". If it was possible to obtain the missing page(s) or section, they are spliced into the film along with adjacent pages. This may have necessitated cutting through an image and duplicating adjacent pages to assure you of complete continuity.
2. When an image on the film is obliterated with a round black mark it is an indication that the film inspector noticed either blurred copy because of movement during exposure, or duplicate copy. Unless we meant to delete copyrighted materials that should not have been filmed, you will find a good image of the page in the adjacent frame. If copyrighted materials were deleted you will find a target note listing the pages in the adjacent frame.
3. When a map, drawing or chart, etc., is part of the material being photographed the photographer has followed a definite method in "sectioning" the material. It is customary to begin filming at the upper left hand corner of a large sheet and to continue from left to right in equal sections with small overlaps. If necessary, sectioning is continued again—beginning below the first row and continuing on until complete.
4. For any illustrations that cannot be reproduced satisfactorily by xerography, photographic prints can be purchased at additional cost and tipped into your xerographic copy. Requests can be made to our Dissertations Customer Services Department.
5. Some pages in any document may have indistinct print. In all cases we have filmed the best available copy.

University
Microfilms
International

300 N. ZEEB RD., ANN ARBOR, MI 48106

8212217

STEINHAUER, STUART ROBERT

EMITTED AND EVOKED PUPILLARY RESPONSES AND EVENT-RELATED POTENTIALS AS A FUNCTION OF REWARD AND TASK INVOLVEMENT

City University of New York PH.D. 1982

Emitted and Evoked Pupillary Responses and Event-Related Potentials as a Function of Reward and Task Involvement

by

Stuart R. Steinhauer

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, the City University of New York.

1981

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

12-1-81
date

Gad Hakerem
Chairman of Examining Committee

December 21, 1981
date

Herbert D. Saltzstein
Executive Officer

Dr. Gad Hakerem

Dr. Doreen Berman

Dr. Samuel Sutton

Supervisory Committee

The City University of New York

For Ronnie, who shares my questions, and
Sheila, who shares my search for the answers

ACKNOWLEDGEMENTS

I wish to express my appreciation to those who have provided the knowledge, the enthusiasm, and the encouragement leading to the completion of this research: to Dr. Gad Hakerem, who introduced me to the fields of psychophysiology and experimental psychopathology, and who has constantly supported my efforts, to Dr. Samuel Sutton, who worked as closely as any advisor, and to Dr. Doreen Berman, who has been a wonderful teacher and thoughtful friend; to Drs. Walter Ritter and W. Scott Peavler for their helpful, incisive comments; to Steve Levine and Drs. David Friedman, Fran Bock and Leslie Prichep for sharing their experience; to Dr. Jack Orbach, who influenced my training as a psychologist and my well-being as a musician; to Drs. Mitchell Kietzman and Gerald Bruder for help in designing the time-training procedure; to Ray Simon for holding the lab together; to Jon Leong, who made the computer center comprehensible; to Drs. J. Richard Jennings and Joe Fleiss, and Rich Ulrich for statistical advice; and to Dr. Joseph Zubin, for continuing to expand my understanding of psychology.

I thank my family for the confidence they have had in me: my father, Benjamin, who, throughout his life, lovingly encouraged me to fulfill my own ambitions; my mother, Hortense, for her continuing affection and strength, and my brother, Ronald, for his friendship.

Whenever I needed help or just a shoulder to lean on, there were

sensitive friends nearby, especially Drs. Marjorie Goldman and Robert Lanson, Shari Thaler, Brian Heidorn, Lisa Morrow and Dale Sobotka. I am grateful to all of you.

I especially thank Sheila Pearlman, whose love and friendship have made the difficulties bearable, and the excitements memorable.

Abstract

Emitted and Evoked Pupillary Responses and Event-Related Potentials as a Function of Reward and Task Involvement

by

Stuart R. Steinhauer

Advisor: Dr. Gad Hakerem

This study was undertaken to explore the influence of motivational factors, as represented by differential rewards and task involvement, on psychophysiological responses that have been associated with information processing activities.

Pupil diameter, and event-related potentials (ERPs) at midline frontal, vertex, parietal and occipital locations were recorded from eight male subjects engaged in a betting task. Winning or losing on each trial was indicated by either a click or absence of a click. For any session, the same event always indicated winning or losing; the contingencies were reversed for separate sessions. In the Subject-Bet condition, the subject was permitted to place a bet of zero, 25 or 50 cents that he would win on that trial. In the Computer-Bet condition, a bet was selected by the computer and told to the subject, who still won or lost money depending on the outcome of the trial. Blocks of trials were also presented under conditions of event certainty, in which the subject was told which event would occur, and no betting was

involved.

Following the informational event (stimulus presence or absence), the pupil dilated for 1200 msec and then typically began to show a pattern of constriction termed the "recovery slope." The ERP was characterized by a contingent negative variation (CNV) which developed in the interval between a warning stimulus and the informational event, followed by a prominent P300 component recorded to the event which informed the subject whether money was won or lost. Emitted and evoked pupillary dilation, pupillary recovery slope and P300 responses were often double in amplitude during the Subject-Bet condition as compared to the Computer-Bet condition, demonstrating that the participation of the subject in determining trial value was a significant variable. Increasing bet value resulted in greater response amplitude both for the evoked P300 (as in earlier studies) and pupillary dilation, and for the emitted responses. The CNV differed only slightly between the betting conditions, and was not related to bet value. All responses were larger under conditions of event uncertainty (betting) than during the condition of event certainty. No differences in peak dilation were observed for winning vs. losing outcomes, but the recovery slope measure exhibited greater constriction for increasing loss of money, and less constriction as the amount of money won increased.

Emitted and evoked P300 components, which were largest in amplitude at the vertex location, were not significantly different in amplitude for the same conditions. Across sessions, however, P300 tended to be consistently larger for winning than losing conditions. Thus, for sessions during which the missing stimulus represented winning, emitted P300s tended to be larger in amplitude than the evoked

P300s, presumably as a result of the psychological constructs associated with the events. No previous studies have reported larger emitted P300s than evoked P300s. An evoked P450 component having a parietal maximum was also observed.

The results were interpreted as indicating that pupillary responses and event-related potentials reflect internally generated processes associated with motivational and information processing characteristics, and can be dissociated from the concept of the orienting response.

Table of Contents

APPENDIX

Analysis of Variance Summary Tables for:

List of Figures

Figure

Figure

List of Tables

Chapter I

Introduction and Literature Review

One of the enduring quests for psychology has been the identification of physiological correlates of complex cognitive and motivational states. Within the past two decades, it has become possible to monitor specific changes in psychophysiological responses that accompany patterns of information processing.

Perhaps the most precise electrophysiological reflection to date of mental activity is found in the event-related potential (ERP) recorded from the scalp. The most prominent characteristic of the ERP during active information processing is the P300 component reported by Sutton, Braren, Zubin and John in 1965. P300 is a surface-positive wave having a latency of approximately 250-400 msec, and is distinguished by its appearance and enhancement under conditions of psychological relevance (Donchin, 1979; Sutton, 1969, 1979; Sutton, Braren, Zubin & John, 1965; Sutton, Braren & Zubin, Note 1; Tueting, 1978). Changes in pupillary diameter represent another physiological response that similarly has been correlated with variations in information processing activity (Hakerem, 1974; Janisse, 1977) and with emotional reactions to events (Hess, 1965). The objective of the present study was to investigate the specificity with which pupillary motility and event-related potentials reflect interactions between motivational and information processing activity. Experiments were designed to manipulate the reward value associated with an event, the outcome of the event as a function of winning or losing the reward, and the degree to which the subject controls the outcome of the event.

In 1969, Levine reported an experiment in which pupillary diameter was measured during a guessing task. As information was presented to the subject regarding the correctness of his or her prediction, the pupil dilated, reaching a peak diameter after a latency of 1200 msec. When trials were segregated according to whether the subject had guessed correctly or incorrectly on each trial, it was observed that the pattern of pupillary motility following peak diameter was a process of constriction which differed between the correct and incorrect trials. The extent of this constriction was greater when the subject's guess proved to be incorrect than when it had been correct. Similar results have since been reported by Friedman (1972), Hakerem (1974) and by Steinhauer, Hakerem and Spring (1979).

While patterns of dilation have been examined in several pupillary experiments involving information processing (reviewed by Beatty, 1977; Friedman, 1972; Goldwater, 1972; Hess, 1972, 1975; Janisse, 1977), most studies have been concerned solely with the amplitude of dilation produced in such situations. One of the primary goals of the present study was to examine the factors that influence activity of the pupil past the initial peak dilation during performance of an information processing task.

An additional finding in the Levine (1969) experiment concerned the time at which uncertainty regarding each prediction was resolved. On some of the trials, the informational event which indicated the correctness of the subject's guess was the absence of a stimulus at a specific point in time. This meaningful absence of stimulation resulted in a "phantom" dilation, a response that was similar to the dilation evoked by stimulus presence. When the subject was actively guessing, the amplitude of pupillary dilation was increased relative to the small dilation observed when the stimulus carried little information. The importance of information delivery and utilization was, thus, isolated from the physical consequences of stimulation, indicating that endogenous processes were involved in the generation of pupillary changes. There have been no further reports of attempts to manipulate pupillary activity emitted as a consequence of the absence of a physical stimulus, and such an attempt was undertaken in the present experiment.

Sutton, Tueting, Zubin and John (1967) described an emitted positive wave having a latency of approximately 300 msec that appeared when uncertainty was resolved by a significant absence of a physical stimulus. It was, in fact, the initial explorations of P300 by Sutton and his colleagues that provided the framework and impetus for the study of pupillary activity and information processing performed by Levine (1969). Simultaneous recording of the ERP and pupil diameter during experimental procedures (Bock, 1976; Friedman, Hakerem, Sutton & Fleiss, 1973; Hakerem, 1974; Steinhauer et al., 1979; Steinhauer, Jennings, Zubin & Heidorn, Note 2) has indicated that P300 and pupillary dilation are similarly affected, lending empirical support to the notion that there is an association between the mechanisms underlying some forms of ERP and pupillary activity.

Within the year preceding the first published report of P300 (Sutton et al., 1965) there was a report (Walter, Cooper, Aldridge, McCallum & Winter, 1964) of a negative shift in the event-related potential that preceded the occurrence of an expected stimulus, the contingent negative variation (CNV). The CNV is responsive to many of the same variables that influence P300 (Donchin & Smith, 1970), and several authors suggested that P300 represented no more than a resolution of CNV activity (Karlin, 1970; Naatanen, 1967; Wastell, 1979). Although subsequent research has strongly suggested the relative independence of CNV from P300 through experimental manipulation (Donald & Goff, 1971; Donchin, Tueting, Ritter, Kutas & Heffley, 1975; Friedman et al., 1973; McAdam & Rubin, 1971; Ruchkin & Sutton, 1979b), the interrelationships between CNV and P300 are not fully resolved (Tueting & Sutton, 1973; Donchin & Heffley, 1979). Only in the work of Friedman et al. (1973) have CNV, P300 and pupillary dilation been recorded in the same experiment.

In reviewing the factors that seem to influence the post-peak dilation activity of the pupil, it is apparent that differential responses have been produced primarily by correct vs. incorrect outcomes during performance of guessing tasks (Friedman, 1972; Hakerem, 1974). It is not clear, however, whether it is possible to manipulate the amplitude of pupillary and ERP responses when individual trials represent information in addition to whether the subject performed correctly or incorrectly. Monetary rewards seem to offer an effective means for manipulating the relative importance of trial outcome, since there is less likelihood for satiation than would be expected, for example, with food rewards.

To provide a background for the experimental studies, the following sections include a review of studies of the pupillary response, the P300 component and contingent negative variation with respect to three major topics: effects of motivation and reward value, feedback associated with trial outcome, and generation of emitted responses. Although a large literature exists on experimental considerations in event-related potential research (e.g., Donchin, 1979; Donchin et al., 1978; Tueting, 1978), adequate discussions are more rare for research on pupillary measurement (e.g., Hakerem, 1974; Janisse, 1977; Tryon, 1975). An evaluation of significant factors in pupillary research is also presented.

Effects of Motivation and Value on ERPs and Pupillary Responses

P300 and motivation: Monetary reward tends to enhance P300 amplitude when value is associated with informational feedback. Sutton, Braren and Zubin (Note 1) reported a larger P300 for a win or loss of ten cents than for two cents. One value was paid for a correct response and the other deducted for an incorrect response on all trials of a given block. Larger differences between high and low rewards were observed when the reward values for separate blocks were shifted to five and 20 cents.

Wilkinson and Morlock (1967) recorded reaction times and evoked responses to clicks using high and low incentive conditions. For the high incentive condition, subjects were told their average reaction time after every 50 trials and were rewarded two cents for every msec less than 250 msec. The low incentive condition consisted of payment of a flat fee (unspecified) and no feedback. Although P200 and N250 were not affected by incentive, N100 and especially P300 clearly were greater under the high incentive condition.

Because no P300 was observed in a control condition, in which subjects merely listened to all clicks without responding, Wilkinson and Morlock interpreted the enhancement of P300 amplitude as reflecting a change in the motor potential associated with the response. It is more likely, however, that P300 was responsive to the differential outcome associated with each of the clicks and to the attention paid to the stimuli, and was relatively independent of the motor component. As demonstrated by the data of Sutton et al. (1967), P300 generation is highly dependent upon the associated informational characteristics of stimuli. In the control condition of Wilkinson and Morlock (1967), little relevant information was provided by the stimulus.

Campbell (1976) failed to find significant changes in P300 as payoff matrices were manipulated in a discrimination task. This may have been due to the small incentives employed, or to the emphasis of the task on performance rather than on monetary outcome of each trial.

An increase in P300 amplitude and concomitant pupillary changes were reported during a betting task by Steinhauer and Hakerem (Note 3; also reported in Hakerem, 1974). These findings are presented as part of the preliminary studies in Chapter II.

Johnston (1979) reported increased P300 amplitude when increasing value was associated with trial outcome in a guessing task similar to the procedure described by Hakerem (1974). In a separate experiment, subjects learned to use presentation of different stimuli to predict which of two responses was more likely to be reinforced (Johnston & Holcomb, 1980). One of three monetary levels (zero, $1 or $2) was made available on each trial and was rewarded for correct performance, or penalized for an incorrect response. After subjects learned the relationships between stimuli and reinforced responses, P300 amplitude was increased for trials on which larger reward values were available. Similarly, Jenness (1972a) observed a larger amplitude response for a "hot" stimulus associated with a reward or penalty than for a stimulus that resulted in a neutral outcome.

In a study of visual evoked responses in 11 year old boys (Homberg, Grunewald & Grunewald-Zuberbier, 1981), numerical figures were presented that carried no significance during some blocks, but in other blocks indicated the number of German pennies (zero, two, ten or 50) that would be paid to the children. Although no specific task was involved, P300 was observed to increase with increasing payoff, especially for the ten and 50 cent rewards, but no differential response was obtained to the same stimuli in the no-reward condition. Maximum P300 amplitude was observed over midline parietal scalp in this experiment.

The preceding studies indicate that P300 is increased in amplitude when a stimulus is associated with increasing reward value. This relationship apparently is maintained even when subjects are not required to engage in performance of a task associated with obtaining a reward. However, the presence of P300 in the Gomberg et al. study in the reward condition indicates that the imposition of rewards made all stimuli more relevant for the subjects.

<u>CNV and other slow potentials, and motivation</u>: This section will deal only with those studies that directly involved the manipulation of monetary value or motivational state. In studies of the CNV, it is often difficult to characterize the manipulation of motivation itself. As an example of such studies, Irwin, Knott, McAdam and Rebert (1966) presented a warning tone from either the left or right of the subject

to indicate whether a low or high intensity shock would be presented. When subjects were required to press a key following presentation of the shock, vertex CNV amplitude was appreciably increased as compared to not pressing. The anticipation of a higher intensity shock resulted in larger CNVs and significantly faster reaction times than following weak shocks.

While the authors claimed that motivational factors accounted for the changes in CNV amplitude, it is more likely that arousal was the variable being manipulated. Irwin et al. suggested that there was probably differential conditioning of emotional responses to the two warning stimuli. However, since there were no differential contingencies associated with subjects' responses, the faster RTs found for the high level of shock more likely indicate increased arousal following the warning signal than a greater tendency to respond for which the subject was "motivated" by the experimental conditions. Specifically, the paradigm lacked any differential incentives that might be involved in the manipulation of motivation. Although one example in the literature reports greater CNV negativity associated with large monetary rewards and with losing compared to winning (Milstein, Small, Gans and Moore, 1973), a more thorough analysis of the data failed to confirm the initial effects reported (communication from V. Milstein to S. Sutton).

Subjectively generated motivation <u>can</u> directly influence CNV amplitude, as succinctly demonstrated by McAdam, Irwin, Rebert and Knott (1966). Three of the researchers served as subjects, and on alternate blocks, attempted to voluntarily produce either high or low amplitude vertex CNVs between warning and imperative stimuli. All three subjects were successful in modifying CNV amplitude. They reported relaxed vigilance during generation of low amplitude CNVs, but used several strategies to enhance amplitude, such as timing each response carefully, or pressing as hard and fast as possible. Since personal motivation appeared to be so strong a factor, the authors suggested that naive subjects be employed in such experiments when possible.

The Bereitschaftspotential, a potential which precedes a voluntary motor response and often accompanies the CNV, can be enhanced by providing a monetary reward. McAdam and Seales (1969) recorded the CNV as subjects pressed a button every few seconds, with instructions to neither count nor "clock" (evenly space) their responses. In the experimental condition, subjects were told that "correct" responses would be rewarded with ten cents, and would be indicated by tone presentation following the response, but no tone would occur if incorrect. An enhancement of the Bereitschaftspotential was observed under the reward condition. Examination of responses following the presence or absence of the feedback tone was not attempted.

It has, thus, been demonstrated that the addition of rewards to performance of a task can be used to increase the amplitude of CNV. In contrast to the findings for P300, however, there has been no clear evidence that CNV amplitude can be quantitatively modified as a function of differential reward levels.

There have been several demonstrations of slow potential shifts in animals which accompanied the manipulation of rewards or contingencies. During conditioning of jaw movement in cats, larger conditioned responses were obtained from animals receiving a large compared to a small food reward (Irwin & Rebert, 1970). Slow potential changes (SPC) in motor cortex, lateral hypothalamus, medial amygdala and mesencephalic reticular formation were also larger for the high reward group. After conditioning, larger SPCs were observed for high reward at motor cortex than for low reward, and were observed only for high reward animals in the lateral hypothalamic and medial amygdaloid electrode placements.

For rhesus monkeys reinforced with food, larger CNVs were recorded during task performance when food deprived than when on a normal diet (Borda, 1970). A frontal CNV appeared early during training, and continued to be present whenever the animals performed appropriately, indicating high motivation. A central CNV tended to appear after periods of food deprivation, but decreased with overtraining.

Boyd, Boyd and Brown (1977a; 1977b; 1977c) examined the development of a negative wave appearing maximally in the post-arcuate and post-central regions of squirrel monkey cortex, but not appearing in non-cortical regions. This "M-wave" appeared after the animals learned to press a bar in response to a tone, decreased during behavioral extinction, and was not related to pre-motor or post-motor activity (Boyd, Boyd & Brown, 1977a). The amplitude of the M-wave decreased as the animals became satiated, and a higher rate of reinforcement was associated with a faster decrease in M-wave amplitude (Boyd, Boyd & Brown, 1977b).

Boyd et al. (1977c) manipulated the type of reinforcer, using either banana, sucrose, or non-flavored pellets in any single session. The number of pellets consumed correlated well with M-wave amplitude. Both the number of pellets eaten and the M-wave amplitudes were lower for animals on a non-contingent schedule, who showed faster reduction of the M-wave during the sesssion than for animals whose reinforcement was contingent upon a response. The M-wave was dependent on the animal's degree of hunger, the type of reward available, and the opportunity to perform some manipulative behavior. The M-wave was differentiated from a CNV which was also recorded (Boyd, Boyd & Brown, 1979; 1980). The widely distributed CNV, like the M-wave, decreased in amplitude as the animals became satiated.

Pupillary Responses and Motivation: Few attempts have been made to observe the effects of monetary rewards on pupillary motility. Kahneman, Peavler and Onuska (1968) presented one group of subjects with either a digit-span task (repeat all numbers in a string) or a digit transformation task (add one to each of the numbers before repeating), with instructions to either verbalize their responses aloud or to think the response silently. The largest dilation was produced by the digit transformation task when spoken aloud. A second group of subjects was then instructed to verbalize on all trials, but was told at the beginning of each trial that correct responses would be rewarded by either two cents or ten cents. Larger dilations for the ten than for the two cent reward were observed only for the digit-span task. The transformation task produced larger dilations than the digit-span task, but no differences related to the amount of the reward.

Kahneman, Beatty and Pollack (1967) had demonstrated earlier that a difficult transformation task (as described above) could result in a maximum dilation response, with no additional dilation when a detection task was added to the transformation task. Maximum pupillary dilation was interpreted to indicate that the subject was utilizing maximum information processing capacity. Consequently, the failure to observe differential dilation associated with differential rewards during the transformation task (Kahneman et al., 1968) was interpreted as reflecting maximum available processing capacity due to the transformation task, so that any effects of differential reward would not be apparent. Performance under both levels of reward was similar, with few errors, indicating that the differential rewards were not effective in changing the subjects' performance. Verbalization requirements, which resulted in larger dilations, were presumbaly associated with the response becoming a public, rather than private and covert, action. It is reasonable to assume that the active motor responses involved in verbalization contributed, to some extent, to the larger dilations (Bernick & Oberlander, 1968).

Kahneman and Peavler (1969) utilized differential incentives in a paired-associate learning task. After subjects had learned a list of eight nouns, separate trials were presented on which each of the nouns was paired with a different number from two through nine. Subjects were required to respond with the appropriate noun after a digit was presented on the test block. Correct responses were rewarded with five cents for nouns paired with odd digits, and a reward of one cent for even digits. During training, a brief dilation was observed after presentation of each noun. After pairing with digits, subjects correctly recalled 55% of the nouns paired with a high reward but only 18% of the nouns paired with a low reward. The authors reported no differential response to incentive immediately following presentation of the digit indicating the value for the trial, but a larger dilation for the higher incentive that appeared four to six sec later, at the start of the verbal response. Larger dilations were recorded during high reward trials, intrusions and blanks (no response) as compared to low reward blanks, presumably because less effort was applied by the subjects under the low reward conditions.

While little of the pupillary literature has been concerned with quantitative variations in value, a substantial literature has been concerned with the differential reaction of the pupil when subjects are presented with "emotionally meaningful" stimuli. The tendency of the pupil to dilate under conditions of emotional arousal or pleasantness has been consistently documented (Nunnally, Knott, Duchnowski & Parker, 1967; Peavler & McLaughlin, 1967; Stelmack & Mandelzys, 1975; White & Maltzman, 1978). In contrast, the contention that the pupil exhibits constriction when one is presented with unpleasant stimuli has not been substantiated (Janisse, 1973; Nunnally et al., 1967; Paivio & Simpson, 1966; Peavler & McLaughlin, 1967; Stelmack & Mandelzys, 1975; Vacchiano, Strauss, Ryan & Hochman, 1968; White & Maltzman, 1978).

Popular interest in pupillary measures of covert processes was aroused by the study of Hess and Polt (1960) in which pictures considered to vary in their affective content were presented to four men and two women. The male subjects exhibited pupillary dilation when presented with a female pinup, while for the female subjects, pupillary dilation was reported after presentation of pictures of either a male pinup, or mother with baby, were shown.

Hess, Seltzer and Shlien (1965) presented 15 pictures which included male and female nudes to five heterosexual and five homosexual male subjects. Heterosexual males tended to show a greater percentage increase in pupil diameter (compared to control slides) to pictures of females, while subjects in the homosexual group exhibited a greater percentage of pupillary dilation to pictures of males, presumably demonstrating a positive response to the preferred stimulus.

Hess (1965) asserted that pupillary dilation is produced by stimuli (including pictures) which are considered positive in affect, while stimuli having negative (aversive) content result in active constriction. These conclusions led to considerable debate (see, for example, Dooley & Lehr, 1967; Hess & Polt, 1967; Janisse, 1977; Loewenfeld, 1966; Woodmansee, 1966). In agreement with Hess, J. D. Barlow (1969) reported pupillary dilation to pictures of preferred political candidates and constriction to non-preferred candidates. In a subsequent study, pupillary diameter was correlated with subjective rankings of five stimuli ranging from pleasant to aversive (J. D. Barlow, 1970). Both pupillary dilation to pleasant stimuli and constriction to unpleasant stimuli were reported. Additional support for Hess' hypothesis was reported by Bergum and Lehr (1966; cited in Hess, 1972) for pictures and by Hutt and Anderson (1967) for emotional and neutral words, although Goldwater (1972) has pointed out that no evidence for constriction was actually presented in either study.

Serious concern has been raised regarding subtle manipulations of experimental procedures that may affect pupillary activity. In a widely cited study, Chapman, Chapman and Brelje (1969) recorded pupillary dilation in male subjects shown pictures of nude or semi-nude males and females, similar to the slides employed by Hess. Two different experimenters were used: one formal and stern, the other more relaxed. The friendly experimenter added to the instructions the statement that the subject would find some of the pictures interesting. Although pupillary dilation was obtained to both the male and female pictures, larger pupillary dilations were observed among subjects tested by the friendly experimenter. The authors emphasized that it was the personality difference between the two experimenters which enhanced pupillary dilation. They failed to note, however, that the additional instruction to the subjects of the friendly experimenter changed the informational value of the stimuli, so that the task of observing pictures was now more relevant.

No analogous effect of experimenter influence was observed in a well controlled study by Clark and Johnson (1970) in which a digit span task was employed. Subjects were given either information or misinformation regarding the expected outcome of the task, which neither enhanced nor inhibited the normal course of dilation while stimuli were presented, or the constriction as the digits were recalled.

The indirect influence of motivational factors may have been involved in an experiment conducted by Polt (1970), in which subjects performed multiplication problems either under a baseline condition, or with the threat of shock for incorrect responses. Pupillary dilation was greater for problems solved during threat of shock. Polt argued that the threat itself was not likely to have produced greater individual dilations. Instead, he suggested that the subjects were more motivated to perform in order to avoid shock, so that the dilations reflected greater effort at the task.

Informational Effects of Feedback

Event-Related Potentials and Feedback: Feedback stimuli may indicate that a response or discrimination is correct or incorrect, or may directly inform the subject as to how behavior should be modified. In guessing tasks, the subject is usually required to make some prediction which is either confirmed or disconfirmed by stimuli to which the evoked potential is recorded. Differences in the relative amplitudes of right and wrong responses have not been consistent (Sutton, 1971; Sutton & Tueting, 1975), with larger P300 amplitudes for wrong than right responses in some studies, and the opposite pattern in others.

In the initial reports of Sutton and co-workers (Note 1; Sutton et al., 1965), P300 was observed to be larger following wrong than right guesses. Sutton et al. (Note 1) reported that payoff as well as other factors such as stimulus probability appeared to modify the differences between right and wrong responses. Larger P300s were observed for wrong guesses by Tueting et al. (1971) and by Friedman et al. (1973), but only when the probability of a specific stimulus was high - that is, larger wrong than right P300s appeared only when the subject was unlikely to make an incorrect guess. For the latter two studies, P300 amplitude was inversely related to the probability of being correct, but was more complexly related to wrong guesses. Larger responses for incorrect predictions have been reported by Johnston (1979).

Levit, Sutton and Zubin (1973) reported the opposite effect for the normal subjects in their study: correct predictions resulted in a larger P300 to the feedback stimuli. Verleger and Cohen (1978) observed no differences between correct and incorrect outcomes during a partial replication of the Levit et al. study.

The sequence of events in an experiment has been noted as a possible factor influencing amplitude of right and wrong responses. Sutton et al. (Note 1) reported that when two guesses in succession were both correct or both incorrect, the second right or wrong guess produced a larger response than the first similar outcome. Chesney and Donchin (1979) observed that P300 was always larger, regardless of guess outcome, when the stimulus differed from the previous trial, but not if the same stimulus was repeated. They interpreted this finding with respect to the "expectancy" hypothesis presented by K. Squires, Wickens, N. Squires and Donchin (1976), which posits that a change in stimuli between trials is a more "surprising" event, resulting in a larger P300 response amplitude.

A paradigm that employed guessing the next of two possible stimuli, and then placing a "bet" of zero to five cents on the prediction, was employed by Poon, Thompson, Williams and Marsh (1974; discussed further in Marsh, Poon & Thompson, 1976). A specific sequence of stimuli was repeated until the subject learned the sequence. P300 was largest during the acquisition phase, and stimuli guessed incorrectly resulted in larger P300s than those correctly guessed. It seems possible to interpret these data as reflecting greater uncertainty during the acquisition phase as compared to relative certainty during the overlearning phase. Moreover, as subjects learned the sequence, an incorrect guess became more unlikely, so that both the rareness of incorrect guesses and the uncertainty resolved by presentation of the stimulus were probably two major factors involved in the generation of P300 amplitude. CNV amplitude did not differ in the learning and overlearning conditions. No information was presented regarding the effects of the betting procedure.

Sutton et al. (1978) separated the effects of winning or losing from the correctness of the subject's guess. Although the subject placed a guess before each trial, the contingency for winning was that two successive stimuli were both guessed correctly or both guessed incorrectly, but if the guess outcomes were not the same, the subject lost money. The resulting waveforms indicated no differences for guessing correctly or incorrectly, but losing resulted in a larger P300 than winning. These data indicate that the reward contingencies probably exert a major influence on the relative amplitudes of right and wrong responses.

P300 has been recorded to feedback events that indicate success or failure during performance of motor or discrimination tasks (Adams & Benson, 1973; Campbell et al., 1979; Jenness, 1972a, 1972b; Johnson & Donchin, 1980; Leifer, Otto, Hart & Thompson, 1976; Ruchkin, Sutton, Munson, Silver & Macar, 1981; K. Squires, Hillyard & Lindsay, 1973a; Stuss & Picton, 1978). In most of the discrimination tasks, a stimulus was presented which was then followed by the subject's response, indicating the subject's classification of the stimulus. In these studies, feedback followed the subject's response.

Jenness (1972a) reported that feedback indicating correct discrimination elicited a smaller response than disconfirming feedback. Leifer et al. (1976) devised a complex design in which subjects learned to predict the likelihood of certain relationships. P300 was inversely related to the probability for correct guesses, but was only large for incorrect predictions that seemed to be based on incorrect strategies. In general, P300 was larger for correct outcomes.

The amplitude of P300 may be related to the information conveyed by the stimulus. Stuss and Picton (1978) reported that P300 was larger for disconfirming feedback only until the subjects learned the appropriate associations in their task. Jenness (1972a) similarly observed a reduction to feedback as subjects' performances improved during a discrimination task. Thus, as a subject becomes more sure of his/her response, feedback becomes less informative. If this is so, then feedback responses should be influenced by the subject's confidence that a correct response has been made. K. Squires et al. (1973a) and Campbell et al. (1979) required subjects to rate their confidence that a threshold stimulus had been presented. P300 to feedback was largest when a high confidence report of stimulus absence was disconfirmed (that is, when subjects had missed a stimulus, but were sure that they had not). This is precisely the condition in which feedback provides the greatest information. In contrast, a confirmation of a confidently identified event is of little "informational value " (Sutton, 1979) nor is it a particularly "surprising" (Donchin, 1979) event. The informational value is also reflected in the amplitude of responses to the discriminative stimuli, which carry questionable information, compared to feedback stimuli which clearly indicate the correctness of the subject's response, an event high in information. P300 is larger for unambiguous feedback stimuli than for stimuli being discriminated (e.g., Campbell et al., 1979; Jenness, 1972a; Stuss & Picton, 1978). Similarly, P300 is larger in guessing tasks when the stimuli serve a feedback function than in counting tasks (Chesney & Donchin, 1979; Donchin, 1979), in which each stimulus provides no resolution of the total number of stimuli to be counted.

Just as an unlikely event results in a large P300 during guessing tasks (Friedman et al., 1973; Tueting et al., 1971), the probability of event outcome appears significant in detection tasks. Data from the studies of K. Squires et al. (1973a) and Campbell et al. (1979) indicate increased P300 amplitude to the feedback stimuli for low probability events. For example, when the subject was more confident of a correct decision, there were fewer instances of a wrong response, resulting in a relatively large P300 amplitude, but when the subject was less certain, the probability of being wrong increased, which was accompanied by a reduction in P300 amplitude.

Pupillary Responses and Feedback: The data examining pupillary motility as a response to feedback has emanated primarily from the laboratories of Hakerem and his colleagues (Bock, 1976; Friedman et al., 1973; Hakerem, 1973, 1974; Levine, 1969; Steinhauer et al., 1979), with a single exception (Haughney, 1976).

Just as relevant feedback enhances P300 (Sutton et al., 1965) and CNV amplitude preceding the feedback event (Friedman et al., 1973), pupillary dilation is increased when a stimulus resolves uncertainty. Little or no dilation was reported for subjects who were told, without guessing in advance, which auditory stimulus would be presented, but a large dilation was observed when subjects guessed which stimulus would be presented on each trial (Bock, 1976; Friedman et al., 1973;

Levine, 1969; Steinhauer et al., 1979).

Even without prior knowledge of the stimulus, the relevance of an event is a contributor to pupillary dilation. Little dilation was observed for a condition during which subjects made no pre-trial guess and received no pre-trial information as to the nature of the stimulus, but were merely required to report the stimulus that had been presented (Levine, 1969). The pupillary response evoked was only slightly larger than the response obtained under the condition of complete certainty.

No differences in pupillary dilation were observed for right vs. wrong guesses (Levine, 1969). When Friedman et al. (1973) presented the guessing task, the relative probabilities for stimuli were varied across blocks of trials. For correct guesses, the amplitude of pupillary dilation was inversely related to stimulus probability, but a more complex interaction was observed for incorrect guesses. For extremely low or high probability stimuli, the amplitude of dilation was largest, decreasing as stimulus probability approached chance. An analysis of outcome probabilities indicated that rarely occurring events resulted in the greatest dilation. For example, guessing incorrectly was an unlikely event for a high probability stimulus. These findings paralleled the P300 data, mentioned previously, which were a partial replication of the Tueting et al. (1971) study. Similar patterns were apparent in the data collected by Bock (1976), although no specific correlation between dilation amplitude and trial outcome was attempted in her data.

Friedman's (1972) primary data, collected under conditions of unequal stimulus probabilities, suggested that no differences in the amplitude of pupillary dilation should be observed between correct and incorrect responses if alternative stimuli were presented with equal probability. Friedman presented additional data which verified this prediction.

Auditory and visual stimuli were equiprobable in the guessing task presented by Steinhauer et al. (1979). Pupillary dilations to right and wrong responses, evoked by the auditory stimuli, did not differ in amplitude, nor were any differences observed for the contractions resulting from presentation of visual stimuli.

A different result was obtained only by Haughney (1976), who provided feedback as a tone that either was presented or omitted with equal probability (a clock indicated the time that the tone could occur). Wrong guesses resulted in larger dilations than correct guesses, a finding in contrast to the above studies.

For the pupillary dilation response, event probability is apparently a more critical event than the characteristics of the specific outcome. In contrast, the effects of event outcome are most readily observed in the changes in pupil diameter which follow the ocurrence of peak dilation. Each of the stimuli in the Levine (1969) study, when averaged according to guess outcome, exhibited a similar differentiation between correct and incorrect responses. The incorrect responses consistently produced a smaller pupillary diameter than the correct responses, with the difference between conditions becoming greater during the one second following peak dilation for which data was collected.

Friedman (1972) briefly mentioned the same finding in his data, although no attempt was made to relate the extent of constriction to stimulus probability. Similar findings appear in the unpublished data collected by Bock (1976). Clear differences between right and wrong responses in the post-dilation pupillary diameter were observed by Steinhauer et al. (1979) for normal subjects, but not among schizophrenic patients. Stimuli and outcomes were equiprobable, further demonstrating that the difference between right and wrong outcomes was not a result of differential probability of stimuli.

Pupillary Responses During Cognitive Activity: A critical consideration in the conduct of pupillary research involves the relative processing demands of the tasks selected. Hess & Polt (1964) observed that dilation was evoked during the solving of arithmetic tasks, with greater dilation for more difficult tasks. Kahneman and Beatty (1966) described a gradual increase in pupillary diameter during a digit-span task as the numbers were told to the subjects, and constriction as the subjects repeated the numbers.

A larger pupillary dilation was evoked when subjects were required to transform numbers by adding the number one to each digit before repeating them aloud (Kahneman, Beatty & Pollack, 1967). Smaller dilations were observed for a task in which subjects searched for a specific letter in a visual array. Kahneman et al. interpreted these findings as indicating that pupillary dilation indexed the amount of mental effort involved in the performance of a task. The smaller dilation to the detection task was believed to indicate that spare processing capacity was available to the subject.

Additional experiments performed by Kahneman and his colleagues (Kahneman, Onuska & Wolman, 1968; Kahneman, Peavler & Onuska, 1968; Kahneman & Peavler, 1969; Kahneman, Tursky, Shapiro & Crider, 1969), in exploration of information processing demands, provided a major component of his formulation on mental effort (Kahneman, 1973). According to this model, the pupil was described as dilating under conditions requiring either the expenditure of mental effort (e.g., during a mathematical transformation or other problem solving), or during the storage of information (as in the digit-span task). The dilation ceased once the problem was solved, returning rapidly towards an initial level, and a slower constriction process was observed during recall of information. In the digit-span task, the decrease in diameter was related to the rate at which numbers were recalled. Kahneman described this effect as "dumping," the immediate diameter of the pupil serving as an approximate index of the amount of material currently stored. This formulation is particularly interesting being the first attempt to describe a functional process related to the constriction that follows pupillary dilation during the processing of information.

The magnitude of pupillary dilation may be increased by demands on mental activity (Beatty & Wagoner, 1978; Hess & Polt, 1964; Kahneman et al., 1967; Peavler, 1974). The Beatty and Wagoner study demonstrated quite clearly that more complex classification of stimuli was accompanied by both greater dilation and increased latency to peak dilation.

Responses Emitted Following Stimulus Absence

Emitted Positive Potentials: In the Sutton et al. (1967) paper first describing the emitted P300 response in detail, three different demonstrations of the emitted positive wave were provided:

1) When the subject guessed whether a single or double click would occur, an emitted potential was present following the time that a second click did not occur.

2) An emitted potential appeared only when the subject was asked to guess whether single or doubles clicks were to be presented, but not if the task was to guess whether the clicks would be soft or loud. The emitted response was present only when stimulus absence provided feedback relevant to the task.

3) If the subject was required to guess whether a double click would be presented with a short, medium or long interclick interval, the appearance of a large positive wave was related to the earliest time at which the subject's uncertainty was resolved. The subject had to wait no longer than the end of the medium interval to determine the outcome: if an additional click occurred at that time, a large evoked response was recorded, but since the absence of a click at that time indicated that the long interval would occur, a large emitted potential was recorded prior to the second click in relation to the point in time at which informational absence occurred.

Sutton et al. (1967) discussed the endogenous nature of the P300 component - that is, the characteristic that P300 is a reflection of some internally generated mechanism rather than a result of a sequence of processes initiated by some external (exogenous) event, such as the physical presentation of a stimulus. This classification of a component as endogenous has been extended to the P300 that is evoked by a physical stimulus because it can be demonstrated that P300 is related to the significance of the stimulus at a particular time; the same stimulus produces little or no such response under conditions in which the stimulus carries little information (Donchin, Ritter & McCallum, 1978; Donchin, 1979). The terms "endogenous" and "exogenous" are often used interchangeably with the terms "emitted" and "evoked" (e.g., Donchin et al., 1978). This review follows the terminology according to Weinberg et al. (1970): an evoked potential is one which is generated after presentation of an external stimulus, while an emitted potential is one that is produced by the absence of a stimulus.

Event-related potentials that are emitted following the absence of a stimulus have been recorded using different experimental manipulations, but may be summarized under three (often overlapping) rubrics.

1. An emitted potential may be observed when the absence of a stimulus at a specific time provides significant information to the subject (Prichep, Sutton & Hakerem, 1976; Ruchkin & Sutton, 1973, 1978a, 1978b, 1979a, 1979b; Ruchkin, Sutton & Stega, 1980; Ruchkin, Sutton & Tueting, 1975; Ruchkin et al., 1981; Sutton et al., 1967; Weinberg, Walter, Cooper & Aldridge, 1974; Weinberg, Walter & Crow, 1970).

2. In signal detection tasks, an emitted potential can be recorded following a correct rejection (Kerkhof, 1978; Ruchkin, Sutton, Kietzman & Silver, 1980; Ruchkin, Sutton & Stega, 1980; K. Squires, N. Squires & Hillyard, 1975b; N. Squires, K. Squires & Hillyard, 1978).

3. In a regularly presented series of stimuli, if some proportion of stimuli are omitted, an emitted positive wave appears (J. Barlow, 1969; Ford, Roth & Kopell, 1976; Friedman, Erlenmeyer-Kimling & Vaughan, in press; Halgren, N. Squires, Wilson, Rohrbaugh, Babb &

Crandall, 1980; Hillyard, Courchesne, Krausz & Picton, 1976; Klinke, Fruhstorfer & Finkenzeller, 1968; Picton & Hillyard, 1974; Picton, Hillyard & Galambos, 1974; Purves & Low, 1979; Renault & Lesevre, 1978, 1979; Ritter, Simson & Vaughan, 1979; Rohrbaugh, Syndulko & Lindsley, 1978; Rusinov, 1959; Simson, Vaughan & Ritter, 1976; N. Squires et al., 1978).

The commonality across these studies is that the time at which a stimulus failed to occur was always evident, due either to learning (conditioning) of the time between stimuli, particularly for the third group of studies, or because some independent cue was provided to the subject that no stimulus had occurred (Hammond, Silva, Klein & Teas, 1979; K. Squires et al., 1975b; Weinberg et al., 1970). It may of course be considered that time-locking with an external cue provides an external event, raising doubt as to the emitted characteristics of responses recorded in these experiments.

Of primary interest for the present study are those emitted responses which are contingent on a stimulus omission that provides meaningful feedback to the subject, in the manner of Sutton et al. (1967). Neither the evoked not the emitted P300 is modality specific, i.e., they have the same scalp distribution regardless of modality or whether the stimulus is present or absent (Sutton et al., 1967; Ruchkin & Sutton, 1973). In a guessing paradigm, varying the probability of stimuli will affect both evoked (Tueting et al., 1971; Friedman et al,. 1973) and emitted (Ruchkin, Sutton & Tueting, 1975) response amplitudes, with larger P300s normally observed for more rare outcomes. For the guessing task, the emitted response, like the evoked P300, tends to be largest at vertex (Ruchkin & Sutton, 1973). Ruchkin et al. (1981) have reported separate early and late (P300E and P300L) components, as well as slow wave, that occur when an event provides feedback for performance. P300E was largest over central cortex, while P300L was largest at the parietal and occipital regions. Evoked and emitted versions of both components were obtained.

In general, an emitted P300 response has been readily obtained during guessing tasks. In contrast, emitted responses have been difficult to obtain during signal detection tasks where the subject has been required to discriminate a signal from background noise. In the earliest studies, an evoked response was observed for those trials on which the signal was correctly detected, but not for correct rejections, false alarms or misses (e.g., Hillyard et al., 1971; Paul & Sutton, 1972; K. Squires, Hillyard & Lindsay, 1973; K. Squires, N. Squires & Hillyard, 1975a). The failure to observe an emitted response when subjects had correctly determined that no signal had been presented (correct rejections) or even when they incorrectly reported presence of a signal (false alarms) cast doubt on the relationship between internal decision processes and the P300 component.

K. Squires, N. Squires and Hillyard (1975b) suggested that uncertainty regarding the time that the signal had been omitted might be responsible for the difficulty in deciding exactly whether or not a signal had occurred. Therefore, they used a light cue to indicate the time that an auditory stimulus might be presented. In addition, they required subjects to use an eight point scale for reporting confidence in the presence of a signal. Under this condition, P300 was observed when subjects reported false alarms with high confidence. In a second task, Squires et al. manipulated the probability of presentation of the signal, and increased stimulus intensity to provide between 95 and 100% correct performance. An emitted response to correct rejections was observed on blocks for which stimulus probability was .9, so that stimulus absence was rare and unexpected, in addition to being more discriminable than in the earlier task.

N. Squires et al. (1978) varied both the probability of signal presentation and the intensity of stimuli, which were presented at 44 dB (threshold), 65 dB and 90 dB across blocks. Both the evoked and emitted components increased in amplitude for increasing stimulus intensity and for decreasing stimulus probability, even for emitted responses to stimulus absence in blocks with threshold signals. Emitted and evoked responses were maximal at the midline parietal location. Using stimuli at only an 80% detection level, Kerkhof (1978) was able to demonstrate P300s for correct rejections and false alarms, but only after the data was enhanced through use of a Woody filter (Woody, 1967).

Ruchkin, Sutton and Stega (1980) noted that the emitted responses obtained in the guessing studies tended to be considerably larger than those found during detection tasks. They compared guessing and detection procedures in an experiment where, following an initial, easily detectable click, a second click occurred with a probability of 50%, and was adjusted so that the subject discriminated at about 80% accuracy. On some blocks, the subject was required to guess whether the single or double click would occur, while on others, no guess was required, but the subject was asked to report whether a single or double click had occurred. Correct guesses and detections were rewarded. For both types of tasks, emitted and evoked P300s were recorded, with no significant amplitude differences between tasks. However, considerable overlap was observed with the long-latency positive slow wave component (N. Squires, K. Squires & Hillyard, 1975). Using principal components-varimax analysis, Ruchkin et al. discriminated a separate slow wave factor from P300. The slow wave (designated by them as "Slow Wave") was relatively equal over vertex and parietal scalp during the guessing task, but was largest at parietal during the detection (report) task, while P300 was largest at parietal for both of the tasks.

Evidence for the complicated relationship between P300 and slow wave was further provided by Ruchkin, Sutton, Kietzman and Silver (1980), who examined P300 and slow wave for hits and correct rejections as a function of auditory signal discriminability. P300 amplitude was increased with increased accuracy, but slow wave decreased as accuracy improved. Apparently, slow wave was related to further processing activity invoked only when a difficult decision was required of the subject.

The potential produced by a stimulus omitted from a regular series of stimuli was first mentioned by Rusinov (1959), describing data collected by Kali Kats. The subject was instructed to press a rubber bulb to the third of three tones, but if the third tone was delayed, an emitted potential was observed in the raw EEG over central cortex at the time the tone should have been presented; conditioned motor activity could also be recorded from the muscles involved in the normal response. Rusinov emphasized the reflex component involved, in that the same EEG response was not apparent for trials on which the tone was not delayed and the subject responded normally.

Subsequent research has demonstrated, in the averaged ERP, an emitted P300 component that tends to be larger than in a passive condition when the subject is required to count the number of omissions or respond to them, is maximal over midline parietal cortex, and can be elicited by omissions among stimulus trains consisting of either vibratory (Klinke et al., 1968), visual (Barlow, 1969; Purves & Low, 1979; Renault & Lesevre, 1978; 1979; Ritter et al., 1979; Simson et al., 1976) or auditory stimuli (Ford et al., 1976; Friedman et al., in press; Halgren et al., 1980; Hillyard et al., 1976; Picton & Hillyard, 1974; Picton et al., 1974; Ritter et al., 1979; Rohrbaugh et al., 1978; Simson et al., 1976).

The emitted P300 obtained when a stimulus is omitted from a train of stimuli tends to be accompanied by the appearance of an earlier negativity (Ford et al., 1976; Klinke et al., 1968; Naatanen & Michie, 1979; Ritter et al., 1979; Simson et al., 1976) having a scalp distribution closely related to the modality in which stimuli are being presented. The emitted P300 response, on the other hand, seems to be independent of modality, similar to the P300 evoked by the target stimulus in counting tasks, both of which seem to have a parietal maximum.

Emitted Pupillary Responses: Pupillary dilation may be elicited in a variety of situations where no evoking stimulus exists, if a contingency has been established between an initial event and a future event which is either emotionally or informationally significant. For example, Nunnally, Knott, Dunchowski and Parker (1967) elicited pupillary dilations by presenting a number indicating the possible firing of a loud, blank gun, although the gun was fired once only before measurement began. No dilations were obtained to "neutral" numbers.

During a study of reaction time and pupillary dilation, Bradshaw (1968) included the possibility of either a long or short foreperiod between a warning stimulus and the imperative stimulus. For three of eight subjects, a small, early dilation occurred during presentations of the long foreperiod, at the latency at which a response would have been obtained had a short foreperiod been presented. Bradshaw interpreted these responses as evidence that the subjects had processed the information that the earlier foreperiod had not occurred. Alternatively, it might be argued that the early dilations were a consequence of temporal conditioning during the experiment.

A more controlled elicitation of emitted pupillary activity was provided by Levine (1969). When the subject was instructed to guess whether initial stimuli would be followed by a final click one second later, pupillary dilation was observed either after the occurrence of a click, or, with the same latency, following the absence of the click. In addition, when the emitted dilation was averaged according to response outcome, the same difference appeared between right and wrong responses that has been described for the evoked pupillary responses: after peak dilation, a greater constriction occurred for incorrect responses.

The dilation emitted after an absent click was less sharply peaked and usually smaller in amplitude than the evoked dilation. There appears to be a parallel effect in the reduction of the emitted pupillary dilation with the reduction in amplitude and flattening reported for the emitted P300 component (Ruchkin & Sutton, 1973; 1979a). The sources of this difference between the emitted and evoked responses are discussed below. In the Levine study, as in Sutton et al. (1967), the contrast between diminished responses under conditions of stimulus certainty vs. the presence of the response under conditions of uncertainty emphasizes the crucial role attributable to the significance of the stimulus in relation to task demands, rather than the role of conditioning.

Enhancement of Emitted Responses

Typically, the emitted P300 is of a lower amplitude and has a broader wave envelope (Ruchkin & Sutton, 1973) than the evoked P300 recorded under similar experimental conditions. Ruchkin and Sutton (1973, 1978a, 1979a) suggested that the variability of P300 latency for individual trials of the emitted response was greater than for the evoked response. While the evoked P300 is time-locked to the occurrence of the stimulus, the emitted response presumably follows the subject's decision that no stimulus has been presented on that particular trial. The time at which the subject makes this decision will vary, and there is no mechanism for recording the precise time of the subject's decision. While motor responses are often employed, there is still some minimum, variable latency following the decision process, and potentials associated with the motor response are likely to be generated as a further source of confounding. By averaging according to the time that the critical stimulus would have occurred, the variability due to the time of the subject's decision is introduced into the averaging process. This produces a distribution of individual waveforms whose P300s would be expected to occur at different points in time, having the effects of decreasing the amplitude of P300 and reflected in the shape of the averaged response, which would be expected to become more broadly distributed.

Ruchkin and Sutton (1979a) attempted to minimize these effects by using the occurence of P300 rather than expected stimulus onset for time-locking their averages. The emitted responses produced by this method of "latency compensation" were larger in amplitude and had a "tighter" distribution than the original averaged response. In general, the amplitude and shape of the newly derived emitted P300 resembled the evoked P300. However, when the method of latency compensation was applied to the evoked responses, they also showed an enhanced amplitude, but the average emitted responses were still smaller in every subject than the average evoked responses.

An alternative approach employed by Ruchkin and Sutton (1978b) was to enhance the emitted P300 by use of a Woody filter (Woody, 1967). In this procedure, the average across trials serves as a template against which each unaveraged trial is compared. By shifting the latency of the raw trials, the optimal correlation between each trial and the template can be determined. A new average response is then derived from these latency adjusted trials to form a second template. The procedure continues iteratively until some criterion point at which no improvement occurs with succeeding templates. Thus, the final average represents the best estimate of points correlated across trials, rather than only relying on adjustment according to a single maximum peak. When the interval between an initial click and the second possible click was 1500 msec, P300 amplitude for the emitted response was smaller than for an interval of 700 msec (Ruchkin & Sutton, 1978a). Utilization of conventional averages and averages derived from a Woody filter both indicated a broader distribution and greater intertrial variability for the 1500 msec period. The evoked P300 was larger than the emitted response, even when the Woody filter was applied.

While averaging methods are related to the assumption that the neural activity following some critical event (stimulus delivery or subject's decision) occurs with a similar latency across trials, the method of latency compensation utilized by Ruchkin and Sutton involves the further assumption that the largest positive deflection within the selected time window actually represents P300. It is possible that some other source of activity or noise could be producing the maximum deflection, which would be identified erroneously as P300, and obviously results in the largest possible waveform obtainable from the raw data even if some of the individual components do not really represent P300.

The Woody technique may represent an incomplete solution for evaluating emitted responses. Kerkhof (1978) obtained emitted responses in a signal detection task only after applying a Woody filter to the data. Ruchkin, Sutton and Stega (1980) have remarked that the long interstimulus interval (1.9 sec) used by Kerkhof would result in greater uncertainty and variability in his subjects' decisions of stimulus absence: the emitted potentials obtained by Kerkhof did not appear to be different from similar averages obtained from background EEG under control conditions.

Another approach towards enhancing the emitted response was sought in the design of the present study by examining the specific origins of variability between trials. One source of variability is the time at which the subject makes a covert decision that no stimulus has actually been presented. The latency of this decision will change during the course of the experiment. At first, the subject may be indefinite as to when the second stimulus would have occurred. After repeated presentations of the second stimulus the subject can learn to discriminate the interval between a warning stimulus and the absent informational event more accurately. Therefore, the variability of the latency of the subject's decision will probably be greatest at the beginning of the experiment.

A second source of variability is the interval preceding the informational event. The variability of estimation of such an interval will be greater for longer intervals, so a relatively short interval is best (Picton, Hillyard & Galambos, 1974; Ruchkin & Sutton, 1978b). A short interval, however, may result in difficulty of identification of the initial components of the evoked response (<250 msec) which may not have recovered full amplitude (Picton, Hillyard, Krausz & Galambos, 1974; Roth, Krainz, Ford, Tinklenberg, Rothbart & Kopell, 1976). In addition, if the CNV is to be studied, there must be a sufficient interval between the warning stimulus and informational event for it to occur (Hillyard, 1974; Rebert & Knott, 1970).

The preceding discussion of variability in the generation of the emitted P300 is equally applicable to generation of emitted pupillary activity, although there is no parallel literature for the pupil. It is clear that reducing temporal uncertainty is likely to increase the amplitude of emitted responses. The use of external time-marking cues in a different modality from that of the critical stimulus (e.g, Beatty & Wagoner, Note 5; Weinberg et al., 1970) produces its own responses which may be difficult to separate from the emitted response to the stimulus which is absent.

It is possible to present a warning stimulus and train the subject to perform an overt response at the end of the desired interval using feedback to sharpen the temporal discrimination, a method used by McAdam (1966) to elicit the CNV and by Ruchkin (Ruchkin, Sutton & Stega, 1980; Ruchkin et al., 1981) in studying the emitted P300. Because such training involves the development of preparatory motor and readiness potentials (e.g., Gilden, Vaughan & Costa, 1966), it is reasonable to expect that there would be some conditioning of these overt reactions. Such activities can confound the emitted or evoked responses that are measured.

Alternatively, it should be possible to condition primarily covert activities that are associated with the recoginition of a specific time interval. Titration schedules, which are normally used to assess thresholds in some stimulus dimension, can be used to teach a discrimination (Rosenberger, 1970). By employing a yes-no response using a forced choice procedure, the experimenter can gain control over the subject's criterion, and can provide feedback regarding the correctness of the subject's response (Rose, Teller, & Rendleman, 1970), which the subject may use to modify future responses.

Methodological Considerations in Pupillary Research

Arguments involving the validity of much pupillary research, particularly with respect to emotionally laden stimuli, involve both the specific experimental manipulations as well as techniques of data analysis. Studies in which pictorial stimuli were employed to manipulate affect have been questioned repeatedly for a lack of technical controls (Loewenfeld, 1966; Peavler & McLaughlin, 1967; Tryon, 1975; Woodmansee, 1966). The greatest problem has been one of separating the effects of stimulus content from changes due to shifts in illumination when visual stimuli were presented. The pupil readily exhibits constriction to small increases in illumination, even following an extremely brief period of decreased illumination (such as the dark interval between slides that occurs when a slide projector is used for stimulus presentation). A contraction can also be recorded when a change in hue occurs, even if the stimuli are of equal brightness (Kohn & Clynes, 1969; Young & Alpern, Note 4). Such difficulties were avoided in the present study through the use of both "missing" stimuli and auditory stimuli.

Another source of confusion has been the determination of average pupil diameter from 1 or 2 measurements/sec averaged over as long a period as 10 seconds following exposure to a stimulus. More refined recording indicates that pupillary activity changes during fractions of a second while processing cognitive stimuli (Friedman et al., 1973; Hakerem, 1974; Kahneman & Beatty, 1966; Levine, 1969; Peavler, 1974), and the most informative reflections of mental activity may occur within one to three seconds following presentation of significant information or an event (Beatty & Wagoner, 1978; Hakerem, 1974). It is possible that the most relevant changes evoked in the pupil were not even detected in many of the earlier studies.

The introduction of signal averaging techniques to the study of pupillary activity (Hakerem, 1967, 1974) has permitted resolution of small, consistent changes in pupillary diameter following the presentation of relevant information, as demonstrated primarily in the work of Hakerem and his colleagues (Bock, 1976; Friedman, 1972; Hakerem, 1974; Levine, 1969) and of Beatty (Beatty, 1977; Beatty & Wagoner, Note 5; 1978). Computerized techniques have reduced any inadvertent methodological errors that were possible during the initial period of interest in pupillary reflections of psychological functioning (Bell, 1971).

Objectives and Hypotheses of the Present Study

The present study was implemented to investigate the magnitude of monetary reward and the subject's degree of engagement in the task, as motivating factors, on emitted and evoked activity of the pupil and event-related potential. The experimental procedures were designed to maximize the relevant psychophysiological responses while minimizing the influence of artifactual processes due to motor involvement, which may contaminate both the ERP (Gilden, Vaughan & Costa, 1966) and pupillary measurements (e.g., Simpson & Climan, 1971). A procedure developed by Friedman (1972) was used. In this, the subject initiates each trial by a key closure, which is followed after a delay by the feedback event. This technique permits recording of relatively artifact free responses to the events, and has the further benefit that subjects are better able to judge periods during which they can suppress blinking. Although inclusion of a motor response to the imperative stimulus enhances CNV amplitude (Peters, Knott, Miller, van Veen & Cohen, 1970), a motor response is not necessary for the development of CNV (Donchin, Gerbrandt, Leifer & Tucker, 1972), and CNVs were successfully generated in Friedman's paradigm following the initial press.

Before conducting the main study, preliminary studies were performed to establish the basic parameters to be employed. In these preliminary experiments, a physical stimulus always provided feedback, and no CNV was evaluated.

Movements of the eye may produce potentials that are detectable at the scalp, reflecting such factors as changes in the biopotential of the eyeball or muscular contractions produced by blinks (Hillyard, (1974). Since these potentials may be large and often follow stimulation, they must be excluded from the averaged response recorded at the scalp. Blinks were identified from changes seen in the pupillary recording during preliminary studies, but an additional amplifier was added to record eye movement directly during the main experiment, even for trials that did not contain blinks.

Modifications to the main experiment included the possibility of an absent stimulus as feedback to elicit emitted psychophysiological activity. An interval of one second between a warning stimulus and feedback events was selected both for enhancement of CNV and to permit recovery of initial components of the feedback stimuli.

The experimental manipulations were designed to investigate the following hypotheses:

1) Increasing monetary value is expected to result in increased amplitude of pupillary dilation, P300, and contingent negative

variation.

2) If the probability of winning or losing money is equal, then losing responses should result in increased P300 amplitude and pupillary constrictions following peak dilation as compared to winning responses.

3) It is hypothesized that the level of subjective motivation can alter the amplitude of ERP and pupillary responses. The factor of subjective involvement is varied experimentally by increasing or reducing the subject's ability to control the value of the reward or penalty. At the same time, the informational significance of each event is not altered across conditions, so that the informational event always provides consistent information regarding the winning or losing of money.

4) The inclusion of a "missing stimulus" paradigm should indicate which components of responses to feedback events are related to stimulus evoked activity, and which are primarily reflections of endogenous processes.

Chapter II

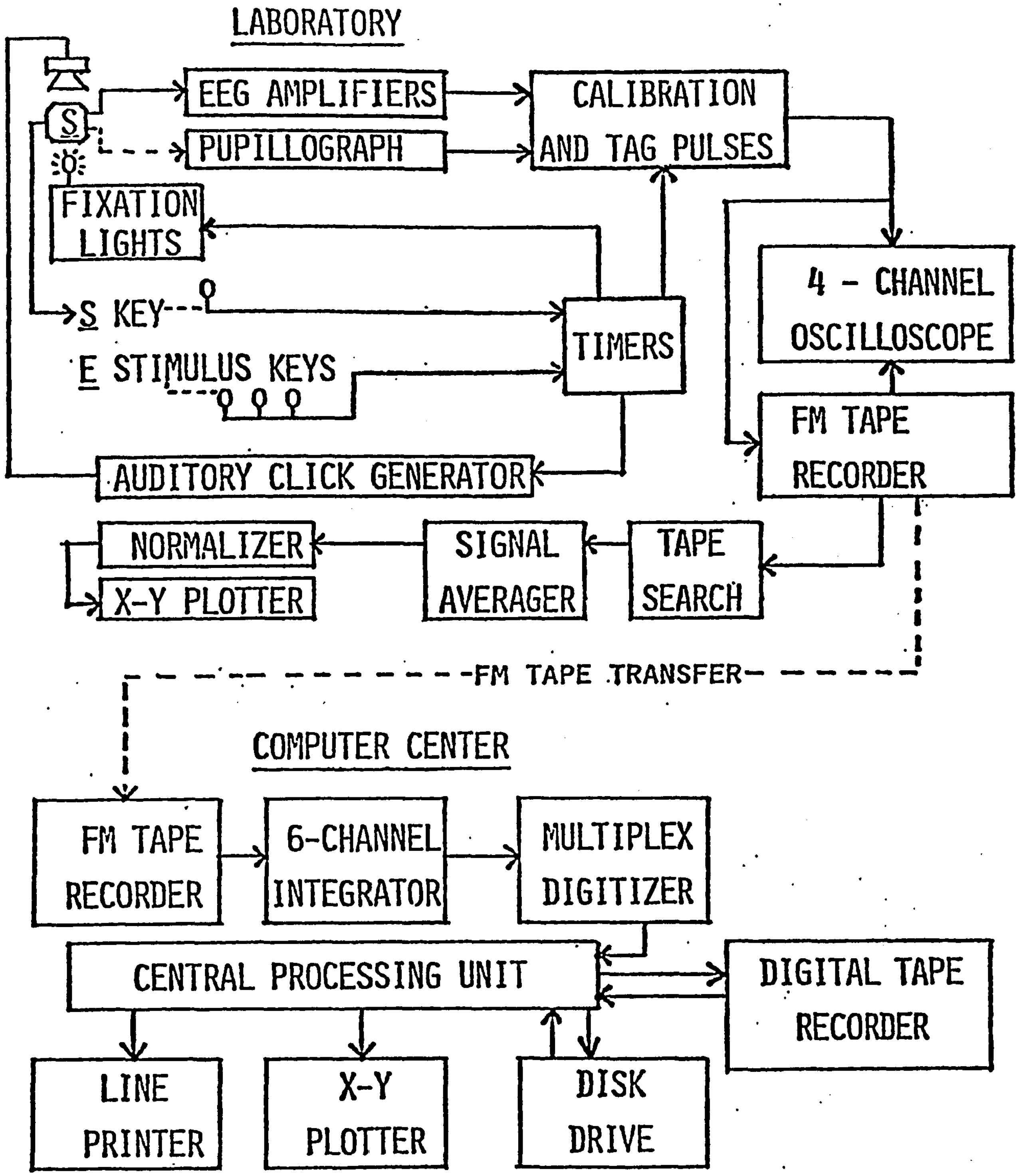
Preliminary Studies

Methods

The purpose of these preliminary studies was to measure pupillary activity and event-related brain potentials in subjects who were engaged in a betting task. The equipment was designed to record pupil diameter and EEG simultaneously. All data were examined on-line and stored on FM magnetic tape for subsequent analysis by digital computer.

Subjects: Two subjects were employed: a male, aged 25 (M1), and a female aged 18 (F1). They were paid $3/hour for participation, plus an additional amount determined by the outcome of the betting procedures.

Equipment: A schematic diagram of the apparatus appears in Figure 1. The subject sat in a darkened, sound-attenuating chamber that was electrostatically shielded with copper mesh. The experimenter communicated with the subject through an intercom system. Pupil and EEG data were monitored on a four-channel Tektronix 561 oscilloscope during real time as the data were fed to a 7-channel FM tape recorder (Sangamo Model 3560); one channel of the oscilloscope was used to monitor playback from the tape recorder. On-line averaging for observational purposes was performed with a TMC 400A Computer of Average Transients. For final data analysis, off-line computer facilities were employed (see Data Retrieval and Analysis). The sequence of events, including stimulus presentation, was controlled by



BLOCK DIAGRAM OF APPARATUS

Figure 1.

a locally-designed system of interconnected timers (accuracy = ± 0.5%) which were programmed through a control panel. A second control panel enabled the experimenter to select different stimulus configurations for each trial.

The subject's right arm rested on a plywood board on which a microswitch (Honeywell Model 7A1HL) was mounted. After a ready signal, the subject could initiate a trial by pressing the switch; the force necessary to activate this switch was approximately 1 oz. through 1 cm.

Pupillary recording: Pupil diameter was measured with a Smith-Kline General Precision Laboratories Pupillograph, developed by Lowenstein and Loewenfeld (1958) and described by Hakerem (1967).

The subject sat in a modified dental chair, facing the pupillograph. To minimize movements during recording, a bite-bar head-rest combination was used. The bite-bar was formed for each subject from Kerr dental compound. During the trial, the subject viewed a small red fixation light presented to the left eye through a series of mirrors. The diameter of the pupil of the right eye was then measured. Since the pupillary response is consensual in normal subjects, (Lowenstein & Loewenfeld, 1962), either pupil may be measured for determination of the pupillary response.

The pupillograph directed 12 horizontal scans of infra-red light at different vertical heights across an area of the eye 1-1/2 inches wide and 3/4 inches high. The light was reflected from the eye through a lens system to a photomultiplier which produced a voltage output when the light scan was reflected from the sclera or iris. The voltage dropped to zero as the scan reached the pupil and the light was absorbed. Since the scan moved at a constant rate, a square wave resulted which was proportional to the width of the pupil. The largest of each group of 12 scans was electronically selected as the diameter of the pupil. The scanning process was repeated 60 times per second. The output from this device was a continuous voltage which corresponded to the diameter of the pupil. The data were recorded on FM magnetic tape, with a voltage of zero equivalent to a pupil diameter of zero, and 720 mV equivalent to a pupil diameter of 8.0 mm. At the beginning of each trial, a calibration pulse was recorded onto the data channel, giving voltage equivalents for zero and 8.0 mm. By comparing the output of the pupillograph to the calibration pulse, it was possible to express pupil diameter directly in mm during off-line analysis. Using this system, a reliable resolution of .01 mm has been established for averages consisting of approximately 25 trials (Friedman, 1972).

EEG recording: Electrodes were affixed to the subject in a preparation room near the laboratory. A large silver-silver chloride electrode (Beckman #330421) was placed at the active midline vertex recording site (Cz according to the International 10-20 system; Jasper, 1958). The electrode site was cleansed with a 50/50 mixture of acetone and ether, and collodion was applied around the border of the electrode, which was filled with Beckman electrode paste. The collodion was dried with a hair blower. Another large electrode serving as ground was fastened to the back of the neck with an adhesive collar after cleaning the site with alcohol. A small electrode (Beckman #650399) was attached to the right earlobe with an adhesive collar, and served as the reference electrode. Any electrode showing a resistance greater than 5K ohms was removed and reapplied. All electrodes were then inserted into an electrode harness, which could be connected directly with the amplifier in the laboratory. At the termination of testing each day, the electrodes were removed and all skin areas were cleaned with alcohol. The electrodes were stored in saline solution.

The EEG was recorded with a Cyber J1 amplifier using a bandpass of .015 to 100 Hz. The amplifier was set to a gain of ten thousand, and was calibrated biweekly using a sine wave of known voltage. The EEG data were recorded on one channel of the tape recorder. As with the pupillary data, a calibration pulse was recorded on the EEG data channel at trial onset. The voltage levels of this calibration pulse were zero and 300 mV, representing an equivalent change of 30 uV. At the same time that a calibration pulse was deposited onto each of the data channels a special control pulse was recorded onto channel seven of the FM tape recorder. The control pulse consisted of a 220 Hz positive-going sine wave burst for 220 msec followed by a 200 msec negative going square wave. Detection of the negative pulse following the positive burst was used both to trigger the CAT 400A Signal Averager for on-line analysis and to generate an interrupt pulse to initiate analog-to-digital conversion for analysis employing a digital computer system.

Eye movement artifact: For these preliminary studies, eye artifacts were identified by blinks or movements seen on the pupillograph video display, and by oscilloscope output of analog pupil diameter and the vertex EEG. In preparation for the main experiment, two methods for recording eye artifacts were examined. An additional Cyber J1 amplifier was obtained. One set of eye movements was derived from an active electrode placed under the right eye and referred to linked earlobes. For a separate channel, an active electrode was placed in the center of the head just above the eyebrows, and the reference electrode was placed at the outer canthus of the right eye. One subject (F1) was asked to perform a series of voluntary eye movements in the horizontal and vertical planes, and both recording channels were monitored simultaneously.

Stimuli: Different click patterns were employed as informational events. The order of stimulus presentation was randomly generated by a computer program. Each click was produced by a one msec capacitor discharge, amplified (amplifier of local design) and attenuated to 60 dB sensation level (Hewlett-Packard attentuator, Model 350D) for one normal subject, and presented through a speaker placed approximately 2-1/2 feet above the subject's head. Extraneous background sounds produced by the air circulation equipment generated a constant ambient noise level of 65 dB SPL on Scale A of a Bruel & Kjaer Sound Level Meter (Type 2203).

Stimulus events were two 1 msec clicks presented with an interclick interval of either one msec, which was heard and identified as a single click, or 25 msec, producing an easily discernible double click pattern of the same total energy. Single and double clicks were equiprobable, with 25 trials/block and 600 trials per subject during each daily testing. There were two sessions/day separated by a 20 minute break.

Data Retrieval and Analysis: Following the completion of recording, preliminary analysis of the data was performed in the laboratory. The data were read from tape off-line through a tape search circuit that could be programmed to allow only trials with a specified outcome code to be output to the Signal Averager. The summed digital data were then normalized by dividing the voltage at each point by the number of trials, and the averaged data were drawn on a Mosely X-Y plotter, scaled to the averaged calibration pulse.

For more detailed analysis, the data were analyzed off-line with a Xerox Sigma 7 digital computer system. The analog data tapes were read into the system from a Honeywell Model 3600 tape deck at a speed of 60 inches per second. A circuit detected the control pulse that had been placed at the beginning of each trial during recording, and delivered a pulse to the computer to begin digitization. The pupil diameter channel and vertex evoked potential channel were digitized separately. Following the control pulse, the computer sampled the analog tape signal at a real-time equivalent of ten msec for 400 consecutive points, representing a four second trial. The voltage at each point was digitized and stored on digital tape.

During the digitization procedures, the outputs of the control pulse recognition circuit and the analog data were observed on an oscilloscope, and an analog output of the digitized data was monitored to verify the success of the analog-to-digital conversion.

Since the digitization procedure resulted in a representation of each point as a positive integer that was based on the voltage sampled, it was necessary to scale the data so that it could be interpreted as millimeters of pupil diameter or microvolts of the raw EEG. The first twenty points (200 msec) of each trial represented the calibration pulse that had been recorded onto the data channel. Each of the other points in the same trial was scaled with respect to the calibration levels, and the transformed data for each trial were stored on a separate digital tape.

Two tests were then conducted on the calibration pulse. The first test checked to see that the difference in digital counts between the points used for scaling was within a known critical range. The second test verified that the calibration pulse was flat around each of the scaling points, representing the original calibration voltage pulses. If either test failed, a warning message appeared on the output of the scaling program, indicating either that spurious noise had triggered digitization in the absence of a real trial, or noise had been detected in the digitization procedure. A final test was employed to examine all points in the trial to see if the level of electrical activity exceeded acceptable recording limits. Eye movements, for example, often produced large voltages that would have contaminated the evoked potentials. Large voltages that exceeded the linearity of the recording or digitization system were detected. Most trials detected through this procedure had been identified as artifactual at the time of data collection and, therefore, were not included in the final data reduction procedures. Trials exceeding either experimentally acceptable or linear voltage levels were identified on the printed output of the scaling program.

The output of the scaling procedure provided certain information about each trial. In addition to listing the trial number, it recorded the internal machine clock time during digitization, and indicated the difference in time from the beginning of the previous trial. By comparing the relative clock times between trials on the scaling program output, it was possible to match each trial to the experimental protocol. Each trial was then assigned a code representing its experimental contingency, and those trials to be excluded from all analyses were given a separate code.

The scaled data were next reduced by combining all trials having the same experimental outcome. Several statistics were computed across all trials for the same condition at each of the 400 data points (4.0 sec of real time). For each point, the mean, standard deviation, skewness and kurtosis were printed. An additional statistic was calculated for the event-related potential trials. In the individual trial data, the amplitude of each point was compared to the next time point. If the voltage difference between points was less than .4 microvolts, the point was considered the "same" as the next point. A change of greater than .4 microvolts was scored as a "rise" if the next point was higher or as a "fall" if the next point was lower. The program printed the percentage of trials that was "rising", "falling" or the "same" for each data point. The difference between the percentage of trials in which the point was rising or falling was indicated, along with the sign of the difference. This provided information about sequences of points that were increasing or decreasing in amplitude, as indicated by the sign in the difference measure (Prichep, 1974). For example, a positive-going wave would produce the same sign in the difference measure for all points up to the peak, but at the peak, the sign would change for the following sequence of points. The change in sign at the peak of a component reflected the greater proportion of individual trials that were now changing in direction. For components of the ERPs that were represented by clear peaks in the data plots, the change in polarity of the difference measure was in agreement virtually all of the time. The rise-fall criterion for the determination of the point at which latency (and amplitude) of the peak of the component were to be measured was employed primarily for those components in which similar amplitudes for several consecutive points, or multiple component peaks, made judgment of the component peak more difficult.

Plots of the mean responses, standard deviation, skewness and kurtosis were provided for each analysis. The statistics for each trial outcome were stored as part of a disk file for future reference. An additional statistic provided in the computer output was the difference of each point from a calculated baseline. The baseline measure utilized was the median response of the 13 points (130 msec) preceding the stimulus in the ERP recordings. For the pupillary data, the baseline value was the diameter of the pupil for the 130 msec ending 600 msec after the onset of the stimulus; 600 msec is a typical latency observed for the initial reaction of the pupil (Friedman, 1972). The selection of the median, rather than the mean baseline value, was designed to reduce the effects of any large peaks that might occur in the baseline activity of the ERP; little variation in pupil diameter was observed over this interval.

Experimental Procedures

In the betting paradigm employed, one of the two click stimulus events was presented on each trial. One event was designated *a priori* as the winning event and the other as the losing event. Presentation of the stimulus event indicated whether the subject had "won" or "lost" the bet for a particular trial. Before the beginning of each trial, a bet was explicitly made regarding the stimulus to be presented on the forthcoming trial. In the "Subject-Bet" condition (Study A), the subject determined the amount of the bet from choices of zero, five or ten cents. The "Computer-Bet" condition (Study B) differed in that the amount of the bet was selected by a computer program that drew from among the three possible bet values at random. During each intertrial interval, two adjacent red lights (LEDs), presented to the left eye at optical infinity, alternated at a frequency of two Hz. At the beginning of the trial (after the bet had been placed), the lights ceased to alternate, and only a single, steady fixation light was visible. If the subject did not initiate a trial within a four second time window by placing his/her head into position and pressing the microswitch, the lights again began to alternate, and the experimenter manually reinitiated the time window after a pause.

Once the trial was initiated, sampling began after 1.220 sec (Figure 2). The stimulus pattern was presented after an additional .500 sec delay, and sampling continued for a total of four seconds. At the end of the sampling period, the fixation lights began to alternate once again, signalling that the trial was terminated. The subject then sat back and verbally reported the outcome of the trial. The subject's report was recorded by the experimenter.

The report was considered to be a crucial feature of the paradigm. Requiring the subject to indicate the outcome of the trial (win or loss) assured that the subject had noted and processed the outcome associated with the stimulus event, and repeating the value of the bet indicated that the subject remembered the value of the bet that had been placed. There was a minimum intertrial interval of eight seconds.

Subject M1 participated in Studies A and B. Subject F1 participated in Studies A, B and C.

Preliminary Study A (Subject-Bet condition): The subject was permitted to bet either five cents, ten cents or zero ("pass") that the winning stimulus would occur. The subject verbally stated the amount of the bet for each trial. On the first day of testing, presentation of a single click signified that the subject had won the amount of the bet. At the termination of each trial, the subject reported whether the bet had been won or lost, and the amount involved. For example, if the subject bet ten cents and a single click occurred, the subject reported "won ten cents". Similarly, the same bet resulted in a report of "lost ten cents" if a double click occurred. Even when the subject had bet nothing he/she was required to report "won zero" or "lost zero" to indicate that attention had been paid to the outcome of the trial even though in both cases, the outcome was neither gain nor loss.

Several blocks of trials were included which represented a condition of event "Certainty." Prior to each of these trials, the subject was told which stimulus was about to be presented. No betting occurred on these trials.

On the second day of testing, presentation of the double click indicated winning and single clicks indicated losing.

Preliminary Study B (Computer-Bet condition): An attempt was made to hold information delivery constant while another variable, the extent of the subject's control over trial outcome, was manipulated. The experimental procedure was the same as for Day 1 of Study A (single click = win, double click = lose) with one change: rather than have

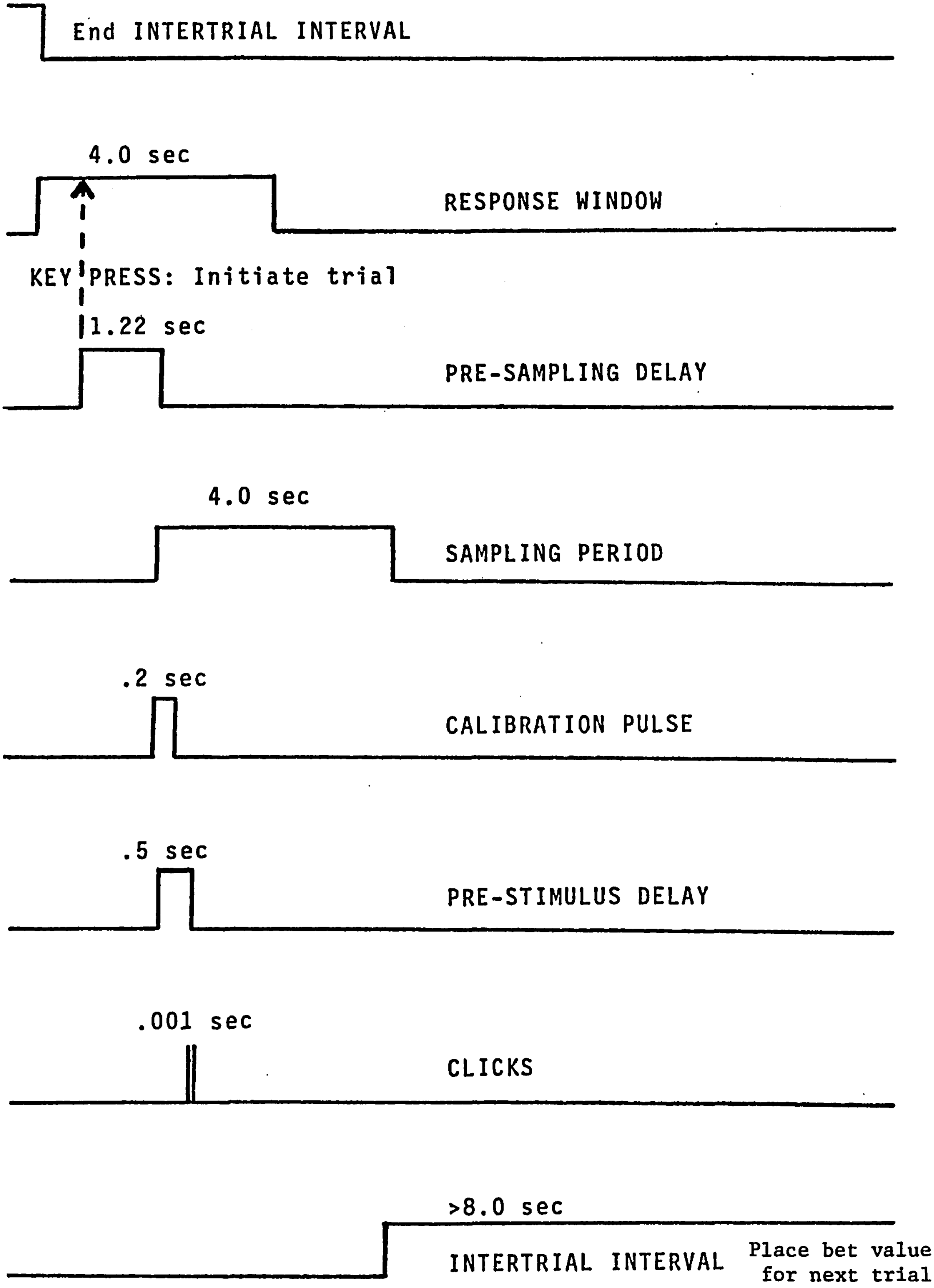


Figure 2. Sample Trial Sequence

the subject place the bet, the value of the bet was derived externally and imposed on the subject. Sequences of bets of zero, five or ten cents were computer generated, independently of the stimuli. The subject was told that the computer had randomly selected the bet value for each trial. Before each trial, the subject was told the amount of the bet for that trial. The sequence of events was, therefore, identical to study A: the bet was placed, stimulus presentation indicating winning or losing occurred during the trial, and the subject reported the amount that he/she had won or lost as a result of the trial.

In this procedure, it was the source responsible for determining the bet, the computer rather than the subject, that was changed from study A; the process of delivering information during the trial procedure did not differ.

Preliminary Study C: In studies A and B, the subject or computer controlled the amount of money wagered. In either situation, the subject had no control over winning and losing, as the computer program selected whether the winning or losing stimulus would be presented.

One subject (F1) was given the opportunity to either win or lose on any given trial by guessing which stimulus would occur. Before each trial began, a computer generated bet value of zero, five or ten cents was told to the subject. The subject then guessed whether a single or double click would be presented on that trial. Consequently, the correctness of the subject's guess was directly related to whether the subject won or lost money on the trial.

Results of Preliminary Studies

For each experimental condition, a separate pupillary and vertex event-related potential average was computed. Figure 3 illustrates the general characteristics of the pupillary response and evoked potential in the betting conditions, where the subject was uncertain as to the outcome, compared to the certain condition, in which no bet had been placed and the subject was informed of the stimulus to be presented. The pupillary response to a condition of uncertainty was a dilation that began approximately 600 msec after stimulus presentation. The amplitude of peak dilation occurred after a total latency of 1200 msec. These characteristics are identical with those reported by Friedman et al. (1973). Following peak dilation, the diameter of the pupil decreased, sometimes below the original baseline. After approximately 1600 msec, the pupil tended either to stabilize, or to return towards baseline diameter. The difference between pupillary diameter at peak dilation and 1600 msec later was measured and will be referred to as the recovery slope of the pupil.

In comparison, the pupillary response recorded during the "certain" condition showed either no dilation or minimal dilation following stimulus presentation, and it was difficult to observe any sign of a recovery slope. This difference between the certain vs. the uncertain conditions for the pupil is consistent with previous data (Levine, 1969; Friedman et al., 1973).

An analogous phenomenon occurred in the vertex event-related potential. Under the condition of stimulus uncertainty (betting), a prominent P300 response was consistently observed. However, when the

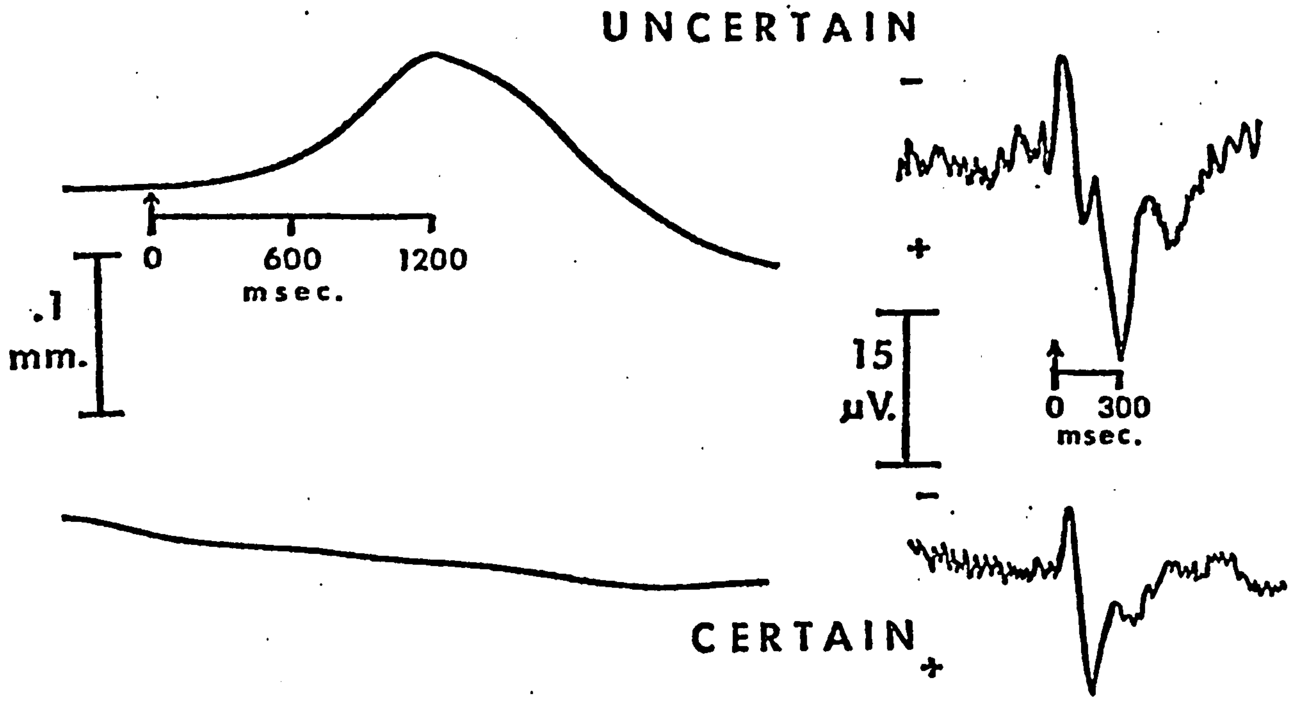


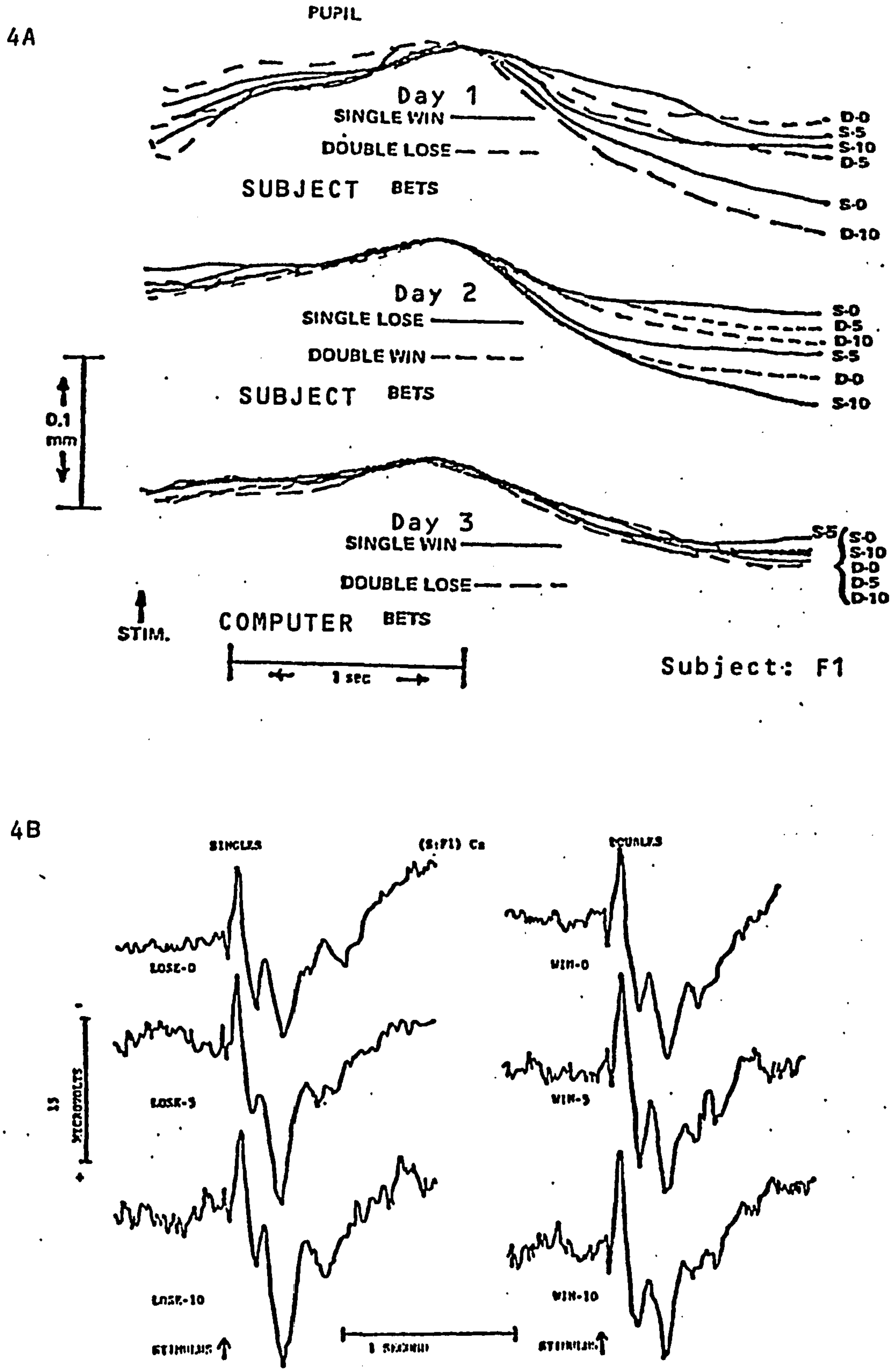
Figure 3. Averaged Pupillary Response (left) and Vertex Event-Related Potential (right) under conditions of UNCERTAINTY (during betting) and CERTAINTY.

subject was presented with stimuli under conditions of certainty, with no betting involved, P300 was greatly decreased (one subject) or not discernible (one subject). This parallel with the pupillary data has also been observed previously (Teuting et al., 1971; Friedman et al., 1973).

The description of evoked potential components in the present studies closely follows the recommendations of Donchin et al. (1977). In all ERP figures, positive deflections are drawn in the downward direction. A positive deflection, indicated by the letter "P" or a negative deflection indicated by the letter "N" is followed by the latency in msec at which the component was observed. In reporting across conditions, the average observed latency is used to represent the component. For the preliminary studies, a P334 and P332 component were measured. These components are apparently representative of the theoretical component referred to as "P300" (Donchin et al., 1977).

The theoretical term may be represented and recognized as a rounded number (or alternatively, by drawing a bar above the latency value). For clarity, the latency of the theoretical component will be presented along with the observed components measured. Thus, the positive component normally observed at 200 msec, P200, is observed in these preliminary data as a real P191. Only P200 and P300 will be discussed in the preliminary experiments. Because only two subjects participated in these preliminary studies, the data will be presented only descriptively.

Preliminary Study A: In Figure 4A, the averaged pupillographic curves of one subject for each of the individual outcomes have been grouped according to the day on which they were recorded (Days 1 and



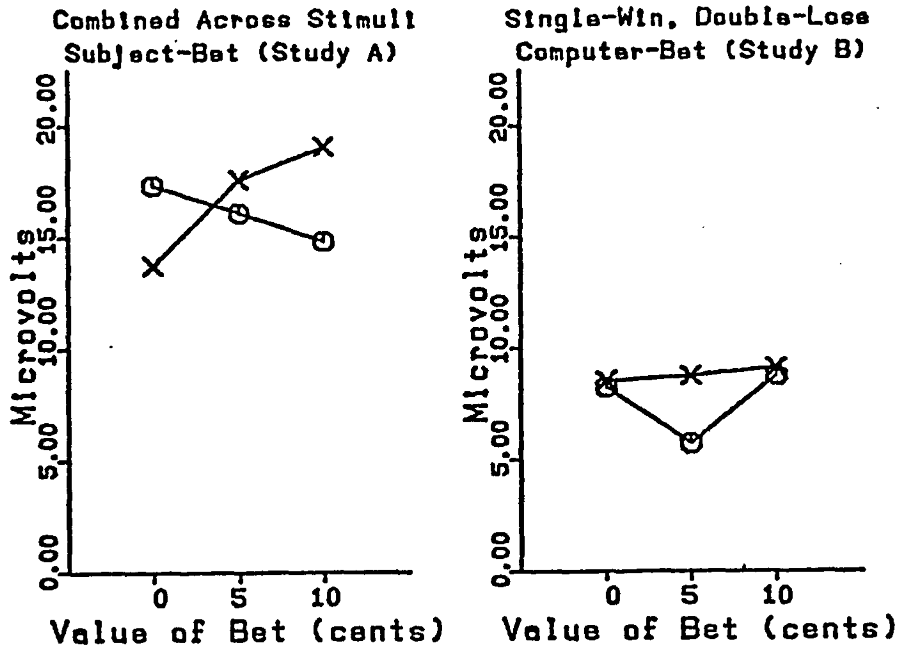
A) PUPILLARY RESPONSES FOR STUDIES A & B;
B) VERTEX EVOKED RESPONSE FOR STUDY A, DAY 2
(SINGLE=LOSE, DOUBLE=WIN)

Figure 4.

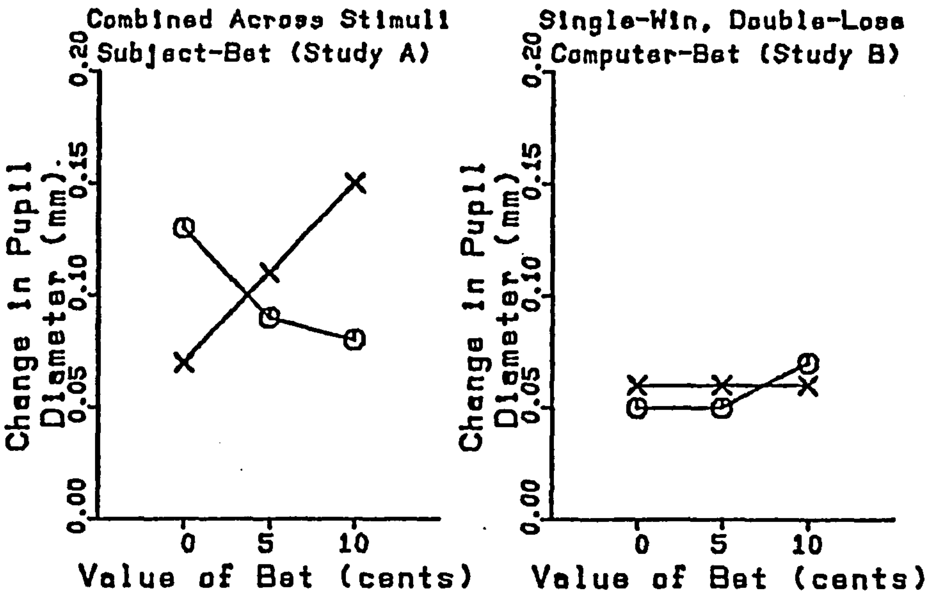
2). The curves are superimposed at the point of peak dilation, 1200 msec post-stimulus. At the top is the data for Day 1, during which the subject placed the bet values, and single clicks resulted in a win, while double clicks resulted in a loss. The middle group of curves represents the same paradigm for Day 2, except that double clicks represented winning and singles represented losing.

For these two days, several trends appeared for both subjects, and are represented in Figure 4A. Following the stimulus, there was a definite dilation of the pupil, but there was no differential dilation between the single and double click stimuli. The different bet values did not produce differential amounts of dilation, nor was there any effect of winning or losing apparent in the dilation response.

The recovery slope, following peak dilation, exhibited considerable variability. The amplitude of the recovery slope has been plotted in Figure 5 across both subjects according to outcome (win or lose) and value of the bet. A greater pupillary constriction is indicated by a larger value for the recovery slope. The recovery slope ranged from approximately .05 mm to over .10 mm in extent. There was a linear increase in the amplitude of losing responses as the amount of money lost increased. For both subjects, the condition representing a loss of ten cents produced the largest amplitude recovery slope. For values of five and ten cents, the recovery slope was largest in the losing condition, but when the subject had bet zero, a different pattern was observed. The responses to a winning stimulus following a bet of zero resembled the pupillary reaction to a loss of money (a large recovery slope), while a losing outcome following a bet of zero resembled the response to winning 5 or 10 cents (small recovery slope).



P334 at Vertex for Win (o) and Lose (×)



Pupil: Recovery Slope for Win (o) and Lose (×)

Figure 5.

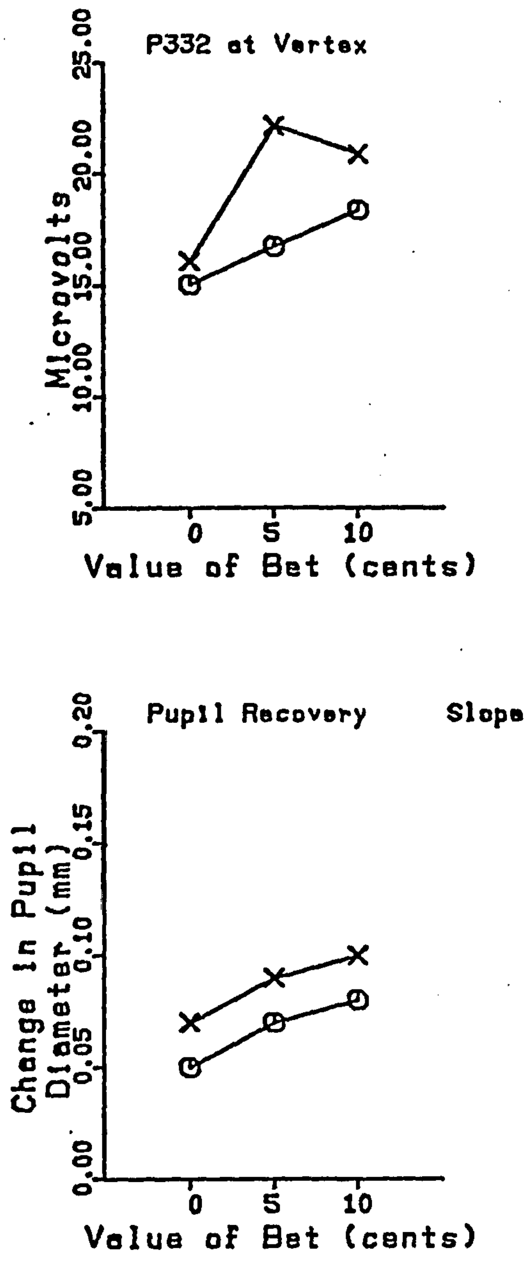
The event-related potentials corresponding to Day 2 for one subject are shown in Figure 4B. Although the "certain" condition evoked no P300 response, a clear P300 component, with an actual latency of 334 msec, was present when betting occurred. In this case, a stimulus effect was apparent in that the double click produced a larger P200 component (observed latency of 191 msec) than did a single click. The effects of winning vs. losing appeared only for the P334 component. The losing responses produced a larger amplitude P334 component than the winning responses (Figure 5). Furthermore, the effect of value was seen in an increasing amplitude of P334 with increasing bet value for the losing responses, but was less clear among the different values for winning responses. The reversal in the effects of betting zero was observed, as in the pupil recovery slope.

Preliminary Study B: To determine whether the specific outcome of each trial was the variable influencing pupillary and ERP activity, or if the participation of the subject in establishing the bet value was critical, testing for Study B (Day 3) was carried out by imposing a computer-generated bet for each trial. As can be seen in Figure 4A, in the Day 3 series of curves, stimulus presentation was still followed by a dilation, although the amplitude of the dilation tended to be smaller than when the subject had placed the bet. Furthermore, the recovery slope following peak dilation no longer seemed to vary according to either outcome or value. The recovery slope is plotted across the two subjects for the Computer-Bet condition in Figure 5. There was little difference among the conditions, but the recovery slopes were generally smaller than those recorded during the Subject-Bet condition.

Similarly, P300 amplitudes were not consistently different across values or between winning or losing outcomes, and the amplitudes were smaller than for the Subject-Bet condition. This is analogous to the decrease in pupillary dilation and smaller recovery slope recorded under the same conditions. For one of the subjects, P300 was barely discriminable under the Computer-Bet condition. Although the second positive component, P191, was slightly larger for the double clicks than for the single clicks, there was no reduction of P191 amplitude in Study B as compared to Study A.

Preliminary Study C: In Study C, subject F1 was told the amount of a computer generated bet, but was permitted to guess which stimulus would be presented on each trial. The subject's guess was, thus, directly related to whether she won (correct) or lost (incorrect) money. Data were pooled across stimuli and are plotted in Figure 6. There was a general tendency for both P332 and the pupil recovery slope to increase with value of the bet. In addition, losing (guessing incorrectly) produced larger amplitude responses than did winning responses, an effect most clearly observed in the pupillary data. The inversion of the effects of betting zero, seen in Study A, was not observed. Instead, the more consistent difference, other than the increase in amplitude with value, was that losing outcomes produced larger responses than winning outcomes.

Eye Movement Artifacts: A comparison was made of the eye movement artifacts produced by the two different recording systems. Examination of both channels simultaneously for the same voluntary eye movements revealed potential changes of similar magnitudes. The electrode placed under the eye proved more sensitive to vertical movements and blink artifacts, while the central forehead-outer canthus configuration was



Study C: Guess Stimulus for Win (∘) and Lose (×) across Stimuli: Subject F1

Figure 6.

slightly more responsive to horizontal movements. Changes in electrical potential recorded in this manner were opposite in polarity from deflections measured at the scalp electrodes. Based on these findings, eye movement artifacts were recorded in the main experiment from the electrode placed under the eye and referred to linked earlobes.

Discussion of Preliminary Studies

The data recorded from these two subjects suggest that both the pupillary response and P300 component of the event-related potential are sensitive to factors that may be loosely described as "motivational" in nature, at least for this preliminary discussion. P300 was enhanced when greater amounts of money were involved, and differences in value were most notable among the losing conditions.

The unexpected effects of winning or losing zero as compared to five or ten cents were apparent on the first day of testing with subject M1. Our initial reaction to the data was that something was wrong with the analysis. As a result, the procedure for Day 1 was repeated for subject M1, and the same results were obtained.

In Study C, the subject was asked to make an explicit guess as to the stimulus to be presented, but had no control over the value to be associated with a correct or incorrect prediction. In this instance, no reversal was observed for the zero value outcomes, but a definite tendency for larger responses following wrong predictions was apparent. Although only a single subject was evaluated in Study C, similar P300 findings have been reported for nine subjects by Johnston (1979).

The guessing requirement in study C is actually a variation of the basic guessing design used by Sutton et al. (1965, 1967), Levine (1969) Tueting et al. (1970) and Friedman et al. (1973), among others. While the explicitness of the guess is apparent in Study C, there is an implicit guess required even in Study A. By being asked to place a bet at all, the subject is enticed to make covert guesses. For example, if the subject has guessed that the losing stimulus is to be presented, a bet of zero would be consistent with that prediction. If, on the other hand, she believes that the winning stimulus is about to appear, the subject is likely to express this belief by placing a larger bet. In Study A, while any possible prediction remained covert, the subject could deal with the consequences of his or her bet as a money-earning strategy. In Study C, however, the subject was in the position of having her strategy examined directly by an external observer (the experimenter). The importance of making a correct guess, which was now openly under the subject's control, was apparently a more significant factor than the interaction of value and outcome. Physiologically, a correct guess of zero was more indicative of a correct guess than a missed opportunity to have won money.

The similarities between the pupillary and event-related potential recordings in the Computer-Bet conditions to the responses observed under conditions of stimulus certainty suggest that a lack of attention, even boredom with the task, may have been responsible for the decrease in pupillary and ERP activity when bets were placed for the subject (Study B). It is not clear whether the subject became uninterested in the task, or whether the decrease in direct involvement with trial outcome was a significant factor. Since both subjects were able to report the value and outcome associated with each trial, however, the decreased responses could not be attributed to a failure to process the information presented.

One procedural difference between studies A and B was that the subject verbalized the bet value in Study A, but the experimenter told the bet value to the subject in Study B. It was conceivable that the activity of verbalization ensured a degree of attention to the task that was not present in Study B. To examine this possibility, subject F1 was retested in the Computer-Bet condition, with the addition that before stimulus presentation on each trial, the subject verbalized the bet value that had been told to her by the experimenter. The results were a replication of Study B, indicating that verbalization by the subject had not been an important factor. To minimize any reduction in responding that may have been due to an increasing lack of attention during the experimental session, the main experiment was designed to intersperse blocks of trials involving both Subject-generated Bets and Computer-generated Bets.

The question of whether the trends observed were truly reflections of endogenous processes associated with the psychological relevance of outcomes could not be separated, in the preliminary studies, from the influence of stimulus-bound activity. The main experiment was specifically designed to elicit endogenous activity associated with the information represented by absence of a stimulus at a significant point in time. Except for the double-click condition employed by Levine (1969), no previous data was available on the specificity of the emitted pupillary response as a monitor of internal processing.

Random bet values can be generated by computers, but subjects do not really bet on a random basis, particularly if the option of no bet ("zero") is available. Winning or losing stimuli occurred 50% of the time (Study A), and subjects tended to bet zero on one-half of the trials, presumably attempting to match the trials on which a loss was expected. This yielded a probability of 50% for zero bets and approximate probabilities of 25% for each of the two remaining bet values. Since different stimulus probabilities have been found to affect both the pupillary response and the evoked potential, (Friedman et al., 1973; Tueting et al., 1971), any differences observed between zero-value outcomes and five or ten cent-value outcomes could be confounded by the fact that a bet of zero was twice as likely to be made as a bet of either five or ten cents. It was decided to attempt to reduce the tendency to place bets having a zero value by reducing the probability of a losing outcome in the main experiment.

Chapter III

Main Experiment: Methods and Procedures

The main experiment was designed to evaluate both emitted and evoked pupillary responses and event-related potentials for the Subject-Bet and Computer-Bet conditions recorded during the same session. Event-related potentials from midline frontal, vertex, parietal and occipital locations, pupillary diameter and eye movement activity were recorded. Testing was typically carried out over seven days during a two-week period for each of the subjects. On Day 1, the subject was familiarized with the experimental procedures and environment. Collection of physiological data took place on test Days 2-4 and Days 5-7.

There were significant modifications in the procedure from the preliminary studies:

1) Bet values were increased to 25 and 50 cents, in addition to the zero option, which was retained.

2) The Subject-Bet and Computer-Bet conditions were presented in separate blocks during the same sessions.

3) To elicit emitted pupillary and ERP activity, a "missing stimulus" event was included as one form of feedback. This modification also permitted recording of the CNV.

4) An attempt was made to reduce the tendency to bet "zero" on one-half of the trials (Discussion, Chapter II). This was accomplished by providing a condition in which, regardless of the amount of the bet, a signal stimulus would indicate that all bets were cancelled. Since the

probability of losing would be reduced to one-third, subjects should theoretically place more non-zero bets.

Subjects: Eight males aged 19 to 28 years (median age = 24.5 years) served as subjects. They were paid at a rate of $3.00/hour in addition to an amount dependent on the outcome of the betting procedure. Since subjects were not rejected if they usually wore glasses, it was necessary to place a corrective lens in front of the left eye for three subjects for ease of viewing the fixation light.

Electrode Placement: To provide information on the topographic distribution of event-related potentials, EEG data were recorded from four locations. Large silver-silver chloride electrodes were applied to midline frontal, vertex, parietal and occipital locations (Fz, Cz, Pz and Oz) as well as the neck for ground, and small electrodes were applied to both the left and right ears. When the electrodes were inserted into the harness, the two ear electrodes were joined and served as a common reference for the active scalp leads, which were each amplified and recorded on separate channels of the tape recorder. An additional channel was used to record eye movement artifacts from a small electrode placed under the right eye, which was also referred to the linked ears. The characteristics of the amplifiers matched the description given in the previous chapter.

Eye Movement Artifacts: Eye movement artifacts were recorded from a small electrode placed under the right eye, referred to linked ears. On each day of testing, the eye channel was calibrated by having the subject make purposeful up and down eye movements of 5, 10, 15 and 20 degrees. To accomplish this, subjects were asked to change their fixation between pairs of appropriately spaced dots on a wall six feet away, and the resulting voltage changes were recorded.

Seven of the eight subjects participated in a more detailed examination of the effects of eye movements on the potentials recorded across scalp. They were instructed to make large eye movements to the left, right, up and down. These movements exceeded 20 degrees. The ratio of the scalp potential at each active electrode to the amplitude of the eye artifact potential was calculated after at least three repetitions.

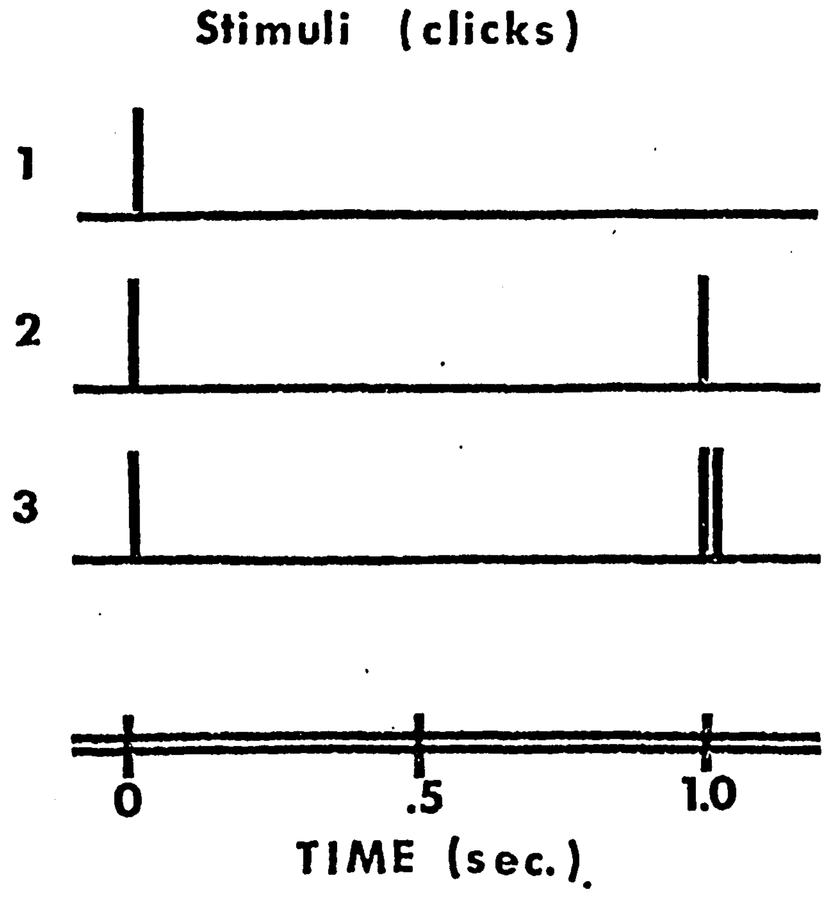
During the experiment, the eye channel was always monitored, as was one channel of EEG on-line and one channel from playback of the tape recorder. Using subjective criteria any large eye movements, as well as smaller eye movements that coincided with shifts in EEG activity, were labelled in the experimental protocol so that these trials could be eliminated from the analysis. These criteria were supplemented by the video display of the pupil, which showed movement of the right eye, and the analog output of the pupillograph, which indicated sudden decreases in pupil size due to either blinks or to eye movements. Most of the artifacts observed on the eye channel corresponded to blinks; there were relatively few trials where identifiable eye movements were not accompanied by blinking.

As an objective method for identifying large eye artifacts in the digitized data, amplitudes exceeding preestablished limits were automatically identified and warning messages were output on the initial computer analysis.

Stimuli: The stimulus configurations for the main experiment are depicted in Figure 7. For all three patterns, a one msec warning click (S1) was presented. One second later, one of three different events (S2) occurred: 1) in the missing stimulus condition, there was no additional stimulus after the warning click, so that the relevant information for the subject was the absence of a further stimulus; 2) in the single click condition, an additional one msec click was presented as the informational event; 3) for the double click condition, two 1 msec clicks were presented, ten msec apart.

Procedures

The general format for trial presentation was the same as in the preliminary studies, with an increase in sampling time to record five seconds of data. On each trial, a warning click was presented which provided no information to the subject regarding the outcome of the trial, although it served as a time marker. Differential information regarding winning or losing was provided one second after the warning click by either the presentation of a click ("single") or by the absence of a click. For a given three-day period, the absent stimulus always represented winning while the single click represented losing. For a separate three day-period, the single click indicated a win and the absent stimulus indicated a loss. The double click in the informational position always indicated that the bet was cancelled, i.e., that no money was won or lost on that trial. Thus, for all outcomes (win, lose or cancel), the time at which uncertainty was resolved was the same. Subjects were randomly assigned to begin with



CLICK STIMULUS PATTERNS.

EACH VERTICAL BAR REPRESENTS A 1 MSEC. DISCHARGE.

Figure 7.

either the absent stimulus or the single click as the winning event for the first three days.

Because the generation of clear emitted responses was dependent on each subject's accurate discrimination of the time that the second click might be omitted, training with respect to the one second interval was given (see "Time Discrimination Training"). At the beginning of each test session, the subject received approximately ten minutes of such training. This was carried out while the subject dark-adapted.

Twenty trials comprised each test block. At the beginning of test days 2 and 5, three blocks of "Certain" trials were presented, in which the subject was told which stimulus would be presented on the subsequent trial. There was no betting involved in these trials. These data were later used for comparison to the betting data, serving as a replication for the uncertainty vs. certainty comparisons described by Sutton et al. (1965), Levine (1969) and Friedman et al. (1973).

The rest of each day's testing consisted of between 400 and 420 trials involving betting, for a total of 2600 trials per subject. There was a two minute rest between blocks, a four minute rest after every third block, and a one-half hour break outside of the experimental room after 240 trials were completed. On one-half of the blocks, the subject selected the value of the bet (Subject-Bet condition, as in Preliminary Study A). On the other blocks, the value of the bet was determined by a computer generated sequence (Computer-Bet condition, as in Preliminary Study B).

Randomization Procedures

Subject-Bet vs. Computer-Bet condition: The decision of whether a block consisted of bets placed by the subject or by the computer was determined for each three-day period by ten rows drawn at random for each subject from a 12 X 12 Latin square (Beyer, 1966, p. 64). Odd numbers represented blocks in the Subject-Bet condition, even numbers indicated the Computer-Bet condition. The same betting condition rarely occurred for more than three consecutive blocks.

Randomization of Stimuli: For both Subject-Bet and Computer-Bet conditions, the order of presentation of stimulus events was determined by a computer program designed for random assignment of the three stimulus configurations. Three consecutive blocks of stimuli were generated simultaneously (60 trials). The total number of stimuli for each three day period was then evaluated by Chi-square tests to verify that the distribution of the three stimulus configurations was indeed random, and that there was no significant difference in the distribution of stimuli between the Subject-Bet and the Computer-Bet conditions.

Generation of Bet Values for "Computer-Bet" Condition: For each block of trials within the Computer-Bet condition, a bet of either zero, 25 or 50 cents was generated at a probability of .33. This was expected to be a more representative estimate of the subjects' betting patterns than the 50% occurrence of zero bets in Study A, given the inclusion of the "cancelled" trial outcome. All trials in the Computer-Bet condition were then evaluated for random total distribution of bets, and random distribution of bet values among the three event outcomes (win, lose, cancel). Non-random distributions were corrected by generating new sets of trials until distributions were obtained for which Chi-square values were no longer significant.

Time Training Procedures: The use of the absent stimulus was designed to enhance the generation of an emitted response at the time that no click was presented in the informational position. It was imperative to try to exercise some control over the variability produced by the subject in recognizing the interval between the warning click and the informational event. This was attempted by teaching the subject to recognize the proper time interval. A procedure was designed with the objective of conditioning any covert activities that might be involved in the recognition of the one second interval following the warning click. A double-staircase method (Cornsweet, 1962) was adapted for this purpose. A two-alternative forced-choice paradigm was devised in which for each trial, the subject was first presented with a sample one second interval, and then was asked to select which one of two additional pairs of clicks was also separated by one second.

The subject was presented with blocks of three trials each. On each individual trial, three pairs of one msec clicks were presented. The interstimulus interval for the first pair of clicks was one second, and served as the standard. Of the additional two click pairs, one pair was separated by one second, while the other pair was separated by an interval different from one second. The subject was informed that the first interclick interval was always one second in duration. After the third click pair, the subject reported whether the second or third pair had the one second interclick interval. He received one cent if he was correct, but did not lose anything if he was wrong. When the subject responded, he was given immediate verbal feedback as to whether he was right or wrong.

The placement of the same and different click pairs in the second or third position on each trial was randomized. For each three-trial block, the "different" interval was kept constant. Subsequent blocks were randomly assigned to contain a shorter or longer "different" interval than the one second standard. All randomizations were performed in advance by computer. The subject was never informed that the "different" interval was constant for any three trials, nor was he informed as to whether the "different" interval was shorter or longer in duration than the standard. Therefore, the subject could not reasonably try to judge whether he should be detecting a shorter or longer interval on any block of trials; he was forced to direct his attention towards finding the correct one second interval.

At the beginning of the time training procedure each day, delta t was set equal to ± 100 msec. That is, if the block indicated that the "different" interval was longer than one second, it was set to 1100 msec, and if it was the shorter interval, it was set to 900 msec. Two different step sizes were used. Initially, delta t was modified in 20 msec steps. After the first reversal (when the subject was either no longer correctly identifying all three trials of a block or no longer incorrectly identifying all three trials) the step size was reduced to ten msec.

A series of rules based on the subject's performance determined whether delta t was changed. If all three trials of a block were correct, then delta t was decreased on the next block in which the different interval was similar in direction from one second. If two out of three trials were correct, delta t was not changed. If only one or no trials were correct, delta t was increased. Changes in delta t for the shorter intervals were therefore independent of changes for the longer intervals.

On Day 1, which was used for training and familiarization with the equipment, time discrimination training continued until the subject reached a criterion of either no change in performance after five or more blocks, or until the subject was discriminating within ± 50 msec. The door to the experimental chamber was left open on this day and the subject was not dark adapted.

One of the problems of the double-staircase method appeared when the subjects began to report fatigue and boredom after even a few minutes of testing. The subjects continued to perform until they reached criterion on Day 1, so that the duration of the initial time training period ranged from approximately 30 minutes to 90 minutes, with rest periods at intervals of 20 minutes. The primary experimental procedure, the betting paradigm, does not lead so quickly to fatigue and adaptation, but it was feared that extensive time training on the experimental days would produce too great an initial fatigue. Therefore, it was decided that the period of time training would be limited on subsequent days. On Days 2 through 7, on which pupillary and evoked potential data were collected, the time training period was usually limited to about 10 minutes, allowing only 10 to 15 complete blocks. Time training on these days was carried out during the period of dark adaptation in the experimental chamber before the beginning of each session.

Instructions to Subjects: On the first day, the experimental procedures were explained to the subject, and informed consent was obtained. The subject was then brought into the laboratory (without electrodes) and positioned in the chair in front of the pupillograph.

For ease in reporting the outcome of each trial, the missing stimulus, single click and double click informational events were referred to, respectively, as single clicks, double clicks and triple clicks in the instructions to the subject. Thus, the subject reported the total number of clicks presented on each trial, including the warning stimulus. The following instructions were read to the subject, accompanied by demonstrations of the click stimuli:

"The test is designed to measure the activity of the brain and the pupil of the eye under several different conditions. Today, you will be trained for the tasks involved, and will be made familiar with the equipment. On the following six days of the test there will be two sessions each, separated by a 20 to 30 minute rest period.

You will be presented with three click stimulus configurations: A single click (demonstrate), a double click (demonstrate), and a triple click (demonstrate). Each trial will involve the presentation of one of these three configurations, and there will be 20 trials in each block, or group.

Before each trial begins, a bet will be placed of 25 cents, 50 cents or nothing, which can be called either zero or a 'pass.' For the first three days after today, a single (double) click will indicate that you have won the amount bet, while a loss of the amount bet will be indicated by a double (single) click. On all days, the triple click will indicate that the bet has been cancelled - there is neither a win nor a loss.

The stimuli will be presented in a computer-derived random order. This means that on every trial there is an equal chance that a single, double or triple can occur. Therefore, the frequencies of the stimulus configurations will be equal in the long run, but may not be equal within a single block.

On half of all the blocks, you will bet to win zero, 25 or 50 cents before the beginning of each trial of the block. Just say your bet out loud. On the other blocks, I will tell you a bet of zero, 25 or 50 cents that was randomly selected by the computer. The bets were generated independently of the stimulus outcomes, so that the actual outcomes of these trials are not fixed in any way. However, you will win or lose money whether you place the bet or the computer places the bet.

Before the beginning of each block, I will tell you whether the bet is to be placed by you or by the computer. At the end of the block, I will tell you the total of the winning and losing outcomes. There will be a pause between blocks.

Place your mouth near the bite board and look straight ahead. You will see two alternating red fixation lights. When the lights stop alternating, you must go into the bite board, lean your head against the head rest, and fixate the light with your eyes wide open. You will have 4 seconds in which to do this and to press the key with your right index finger. The press should be as light as possible.

About two seconds following your press, one of the click presentations will occur. It is extremely important that you do not move for a 6 second period after your press. It is during this period that your pupillary and brain activity are being recorded. Moving includes blinking, moving your eyes, moving the body, gasping, clenching your teeth, and coming out of the bite board too soon. If you must move, do not press the key. The lights will begin to alternate again, and will then stop, and you will have another opportunity to start the trial. The fixation light will remain stationary during the trial, but will then begin to alternate. This is your signal that the trial is over, and you may come out of position. There will be an interval of at last 8 seconds between trials within a block. When the trial is over, I want you to tell me whether you won, lost, and how much, or the bet was cancelled. For example, if the bet was 50 cents and the stimulus was a single (double), say 'won 50.' If a double (single) occurred and the bet was 25 cents, say 'lost 25.' Even if the bet was zero, say 'won zero' or 'lost zero.' When a triple is presented, use the word 'cancel' as in 'cancel 25' or 'cancel zero'. After you have reported, the bet will be placed for the next trial. We will now practice this procedure."

The procedure was then practiced for at least 20 trials until the subject clearly understood the task. The following section regarding the time training procedure was then read:

"For the double and triple stimuli, there is an interval of one second between the first and second clicks (demonstrate). It is important for you to learn this interval, and so a training procedure will be used each day before the beginning of the betting trials. It will not be necessary for you to go into the bite board during this procedure; just sit back in the chair.

On each training trial, you will first hear a pair of clicks which are separated by one second. Two more pairs of clicks will then be presented. One of these two additional pairs also has a one second separation, while the other pair is different. Your task is to tell whether the first or second of the additioinal pairs had the one second separation. I will immediately tell you if you are correct or incorrect. You will win 1 cent each time you are correct, but you will not lose anything if you are wrong. Since you will be learning to identify the one second interval more accurately as we proceed, the difference in the 'other' pair of clicks will be decreased, and you will find that it becomes more difficult to select the correct pair."

The first time training period then followed. On all subsequent days, an abbreviated time training procedure was included. Following the end of the first day of physiological data collection (Day 2), the subject was asked to describe any subjective reactions to winning, losing, or cancellation of the bets. Subjects were paid only after the completion of all seven days of participation in the study; typical overall duration was two weeks per subject.

Data Analysis: In general, the overall data analysis procedures were similar to those of the preliminary studies. A different procedure was used for digitization of data from the main experiment, however. One of the disadvantages of sampling analog data at a specific time interval is that the data extracted do not reflect activity that has occurred between sampling points. A hardware system was designed to integrate the total energy occurring between successive points. Following the control pulse detected during the digitization process, each of the six data channels was fed into a separate capacitor network until the next clock pulse representing a real time equivalent of ten msec was generated by the computer. The voltages stored in these accumulating capacitor networks were transferred to a second bank of capacitors for temporary storage. The accumulator network was then cleared and began to integrate voltages for the next ten msec period, while a multiplexing system digitized each of the voltages in the secondary storage unit, one at a time, representing the previous point. In this manner, 500 points representing a real-time of five seconds were digitized for each trial from each of the six data channels. As the initial integration occurred simultaneously on all channels, there was effectively no time lag between digitization of the separate channels.

Although the digitized data in the main experiment represented the integration of energy during each ten msec period, the same integration had been performed on the critical levels of the calibration pulse, so that as a result of scaling, all of the data were expressed in terms of the originally recorded voltage levels.

The scaled data of each subject for three consecutive sessions for which the contingencies were constant (e.g., emitted-win, evoked-lose) were stored on a single tape, while a separate tape contained the same subject's data for the other three day period, as well as all of the trials for the "Certain" condition. For each three-day period, nine outcomes (win, lose, or cancel X zero, 25 or 50 cents) were generated under the Subject-Bet condition and under the Computer-Bet condition. To evaluate the effects of value and the source of the bet on the warning click and on the contingent negative variation, both of which repesented data obtained before trial outcome was known to the subject, additional sets of data were obtained by averaging across stimuli for an additional six combinations (zero, 25 and 50 cents X Subject-Bet or Computer-Bet), providing averages with a much larger N (300-400) for these evaluations. In addition, the missing stimulus, single click and double click presentations were evaluated under the condition of complete certainty. Thus, a total of 51 sets of averaged data was obtained for each of the six data channels (pupil, event-related potentials, and eye movement) for each of the eight subjects.

Pupillary Analysis: A typical averaged pupillary response to a single click stimulus is drawn in Figure 8A. The warning and informational clicks are indicated by arrows numbered 1 and 2, and the calibration denotes a change in amplitude of 0.1 mm. Absolute initial diameter is not indicated on the plot. Following the warning click, there was little immediate response of the pupil, although an increase in diameter can be detected beginning with a latency of approximately 600 msec. The median baseline occuring in the period 470-590 msec after the warning click was measured as the baseline or initial diameter of the pupil. Following the occurence of the informational click, at an approximate latency of 600 msec, there was a greater amount of dilation, the peak of which was reached approximately 1200 msec after the informational click. For measurement of peak dilation, the average of the ten highest continuous points beginning within 1200 msec after the informational click was selected. Usually, the individual adjacent points varied by no more than 0.01 mm. The value recorded for peak dilation reflects the difference from the value of the initial diameter of the pupil. A negative value for peak dilation, therefore, indicates that the pupil actually decreased in diameter.

Following attainment of peak diameter, the pupil began to constrict. Based on the preliminary studies and after evaluation of the waveforms for the main experiment, the latency of 1600 msec following peak dilation (2800 msec following the informational event) was selected as the best estimate of cessation of constriction. The diameter of the pupil was averaged over ten points at this latency, and the difference in amplitude between peak dilation and the diameter 1600 msec later was recorded as the recovery slope. In several of the averages recorded, the pupil continued to dilate past the typical

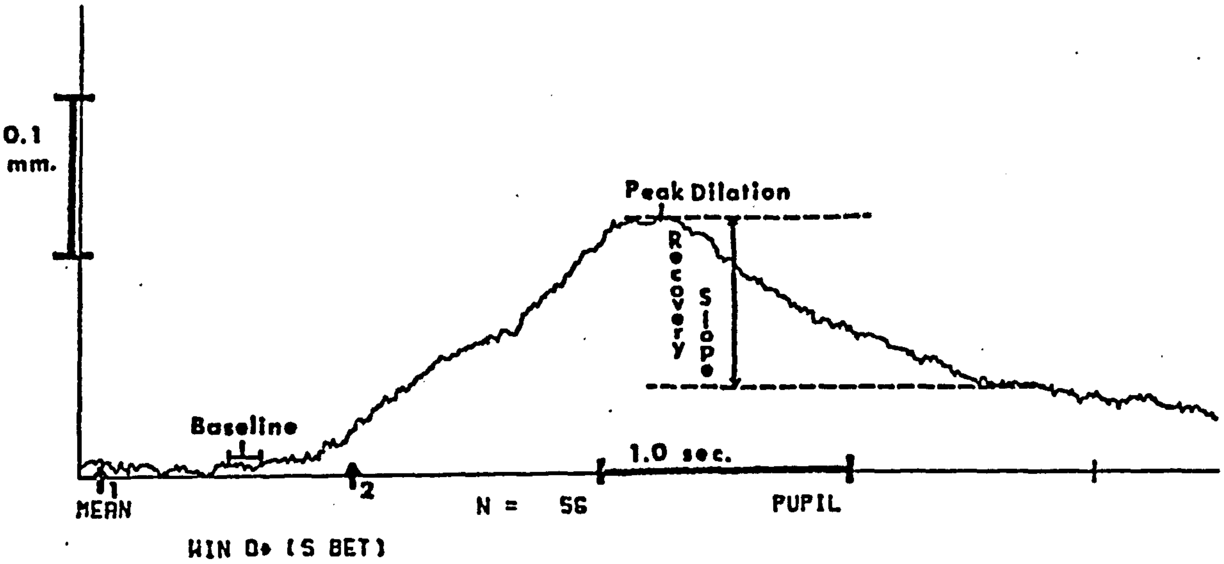


Figure 8A. AVERAGED PUPILLARY RESPONSE TO A DOUBLE CLICK PRESENTATION

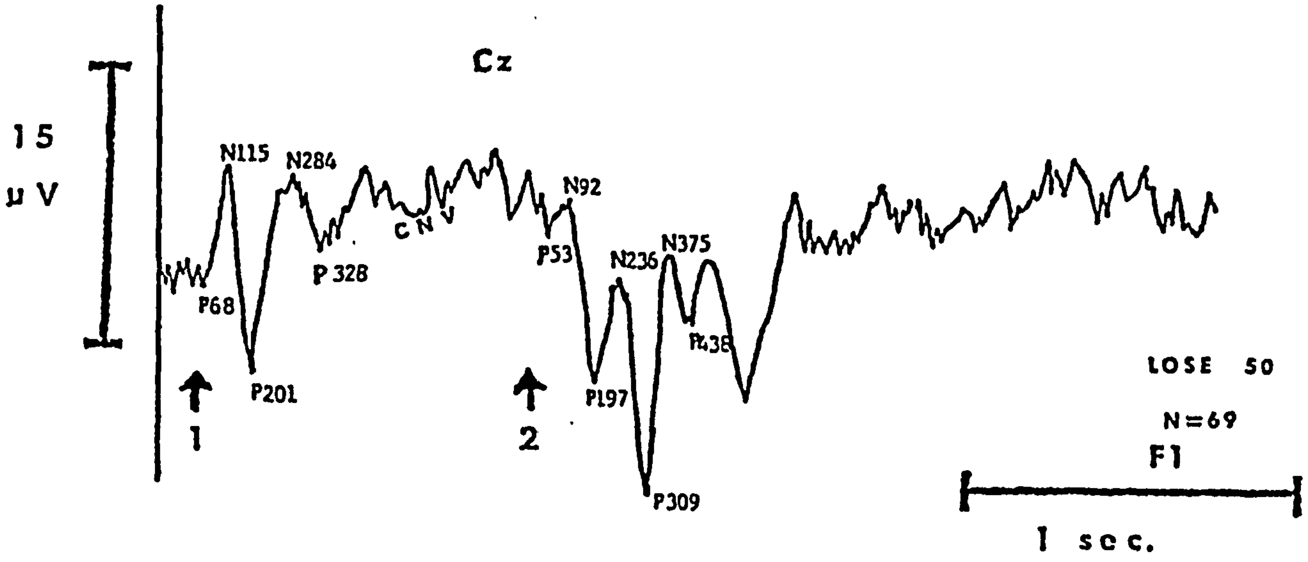


Figure 8B. AVERAGED EVENT-RELATED POTENTIAL AT VERTEX TO A DOUBLE CLICK PRESENTATION latency of peak dilation. In such cases, the diameter of the pupil was still evaluated in terms of the recovery slope, but the measurement yielded a negative value.

When a missing stimulus event was presented, the shape of the pupillographic response was similar to the evoked pupillary response. That is, the dilation following the time at which a click did not occur was larger than the dilation following the warning click. Therefore, the evaluation of the emitted pupillary response was similar to the evaluation of the evoked pupillary response in Figure 8A, although differences in the latency to peak dilation were noted and recorded.

Event-Related Potential Analysis: Averaged event-related potentials were plotted separately for each subject and each condition, and included the response to the warning click and the response to either additional clicks or the absence of a click. A response showing clear components to each click of a single click event is shown in Figure 8B. This is a recording taken from the vertex electrode, which generally produces the largest and clearest components.

Following the warning click, only five components were normally identifiable. Of these, P300 was often small and difficult to identify. In comparison, there were more distinct components following the presentation of the informational click. Although as many as ten distinct peaks were sometimes observed, no attempt was made to evaluate activity past the fourth positive component (P438 in Figure 8B). The P309 component following the second click is greatly enlarged compared to the P328 following the warning click, although the P53 and N93 components of the second response are attenuated. This is due to a failure of complete recovery of these components in the one second interval following the warning click.

Component Identification: The initial identification of components was based on the sequence, polarity, and latency of observed components (Donchin et al., 1977). To assist in the accurate identification of peak components, the following information was utilized:

1) P300 is usually largest at vertex or parietal locations (Tueting, 1978);

2) P300 tends to increase with monetary value (Sutton, 1979);

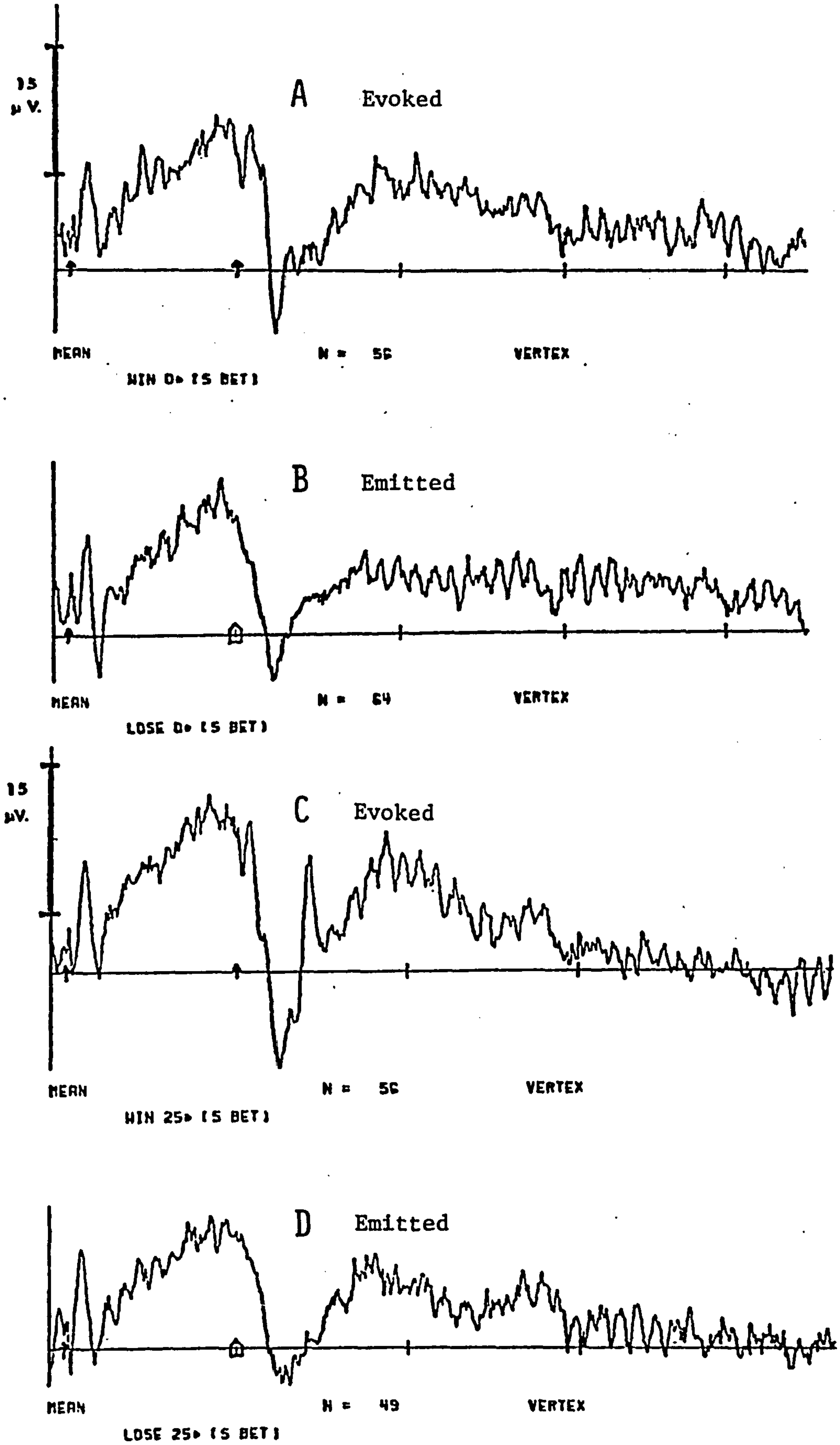
3) P400 is larger than P300 at the parietal location but is generally similar in distribution to P300 (Stuss & Picton, 1978; Tueting, 1978).

During the identification of components, all electrode placements were examined simultaneously. Within a given subject, components clearly delineated at one location were used to determine the location of components at less distinct locations. Finally, the rise-fall criterion, discussed earlier, was used to identify the most unclear components.

After determining the latency of each component, the amplitude of the difference of the peak from a pre-stimulus baseline was recorded. Although both baseline-to-peak and peak-to-peak amplitudes have been utilized in event-related potential analysis, the finding that even early peaks may be affected by experimental variables (Hillyard & Picton, 1974; Morrell & Salamy, 1971; Wilkinson & Morlock, 1967) provided the rational for utilization of the pre-stimulus baseline. For all points preceding the informational event, the baseline preceding the warning stimulus was employed. A second baseline was calculated for the period immediately preceding the informational event, to which all subsequent components were compared. It should be noted that shifts in baseline activity between successive stimuli may affect the components evaluated (Donchin, 1979).

In the missing stimulus condition, several components were evoked by the warning click, but the conveying of information by the absence of a stimulus resulted in a positive-going wave having an approximate latency of 300 msec following the point in time that a click was presented when presence of a click delivered information. This is termed the emitted P300 wave.

Examples of evoked and emitted potentials recorded during the same session are shown in Figure 9. This figure depicts the averaged response curves during 4.5 sec of the entire trial, providing samples of late poststimulus activity for observational purposes only. Figure 9A shows the response to a single click following a bet of zero. The evoked P300 response is clearly delineated, as are the early components of the response, although P200 is almost obscured because of the large amplitude of P300. Figure 9B represents the emitted response following a bet of the same value. The stimulus would have occurred at the location indicated by the unfilled arrow. A sharp, clear emitted P300 is evident, reminiscent of the emitted responses produced by the method of latency compensation employed by Ruchkin (Ruchkin & Sutton, 1979a). Figure 9C shows an evoked response to a bet of 25 cents. The emitted response following a bet of 25 cents (Figure 9D) does not exhibit a clearly defined peak. The rise-fall criterion was especially useful in determining the latency of such relatively broadly appearing components.



EVOKED (A & C) AND EMITTED (B & D)
EVENT-RELATED POTENTIALS

Figure 9.

In addition to the evaluation of ERP components, the amplitude of the contingent negative variation was measured. In Figure 8B, the CNV is labelled as a negative-going slow wave occuring between the warning and second stimulus. For evaluation of the CNV, the average amplitude of the ten most negative, continuous points preceding the second click was subtracted from the baseline calculation for the warning click. Clear CNVs can be seen in all of the waveforms in Figure 9.

Statistical Evaluation: For each experimental condition, the following measurements were recorded for each subject: initial pupillary diameter, amplitude of peak dilation, and recovery slope of the pupil; the amplitude and latency of the P68, N115, P201, N284, P328 components and CNV following the warning click; emitted late positivity (P366) following an absence of a click; P53, N93, P197, N236, P309, N375 and P438 components following each single click; and P40, N86, P211, N270, P336, N404 and P466 components following a double click. These actual components are considered analogous to the theoretical components named P70, N100, P200, N200 (or N250), P300, N400 and P450, which will be used in discussion. The data for each subject were based on an average of ·between 35 and 90 trials per condition, except for four sets of averages in the Certain condition for which the N was at least 22. Each measurement, as well as the latencies of evoked potential components, was entered into a mixed-model analysis of variance utilizing the BMDO8V program (Dixon, 1968).

The basic factorial design employed for pupillary and eye artifact analysis was: Source of Bet (2 levels: Subject-Bet and Computer-Bet) X Trial Outcome (2 levels: win or lose) X Value of Bet (3 levels:

zero, 25 cents, or 50 cents) X Subjects (8 subjects treated as a random factor). For evaluation of main effects or interactions due to the placement of electrodes, an additional factor, Electrode Location (4 levels: frontal, vertex, parietal and occipital) was included.

For evaluation of initial pupillary diameter, CNV, and components of the evoked response to the warning click, the factor of trial outcome (win vs. lose) was not appropriate. Instead, a comparison of changes over the first three-day period was made with response levels for the second three-day period (2 levels) as an evaluation of the stability of these responses. Similarly, the win/lose dimension did not apply to responses evoked by the double click, which represented cancellation. Instead, response amplitudes were compared between sessions for which the missing stimulus indicated winning and the single click signified losing vs. sessions in which the opposite relationship was true.

Additional levels were included in the analyses of variance for comparison of "Certain" responses to the betting conditions (3 levels: Certain, Computer-Bet, and Subject-Bet) and for comparisons among stimulus events (e.g., emitted response vs. single click evoked response; single click vs. double click evoked response).

Given the large number of individual tests in the analysis of the event-related potential data, only those results reaching a significance level of .01 or better were accepted for the ERP data. Results for the analysis of pupillary data were accepted if a .05 level of significance was obtained. No interactions above the first order were considered for either the ERP or pupillary data.

The repeated-measures design employed assumes that population variances and covariances are homogeneous for all levels of a factor. Violations of these assumptions result in an increased probability of Type I error. Jennings and Wood (1976) have discussed the use of the epsilon adjustment procedure of Geisser and Greenhouse (see Winer, 1980), in which the degrees of freedom employed for evaluation of each F-ratio are reduced by the value, epsilon, calculated from the sample variance-covariance matrix. Psychophysiological data typically violate the homogeneity of covariance assumption - frequently resulting in epsilon values of .5 and below (J. R. Jennings, personal communication). The most conservative test will result from setting epsilon to a minimum such that all F-ratios are evaluated with respect to F(1,N-1), which for this study is equivalent to F(1,7).

The first two steps of the procedure suggested by Geisser and Greenhouse (1958) were followed: the repeated-measures factor was evaluated with full degrees of freedom. If significant (at the .01 level of confidence for ERP data and .05 level for pupillary data, as noted above), the conservative test (df=1,7) was then performed, and if this was also significant, the null hypothesis was rejected.

For the finding of significant main effects or important interactions involving factors having more than two levels, Scheffé tests were characteristically employed, evaluated only at the .05 level of confidence for the pupillary data and at the .01 level for the ERP data. The Scheffé test is extremely conservative, even at the .05 level.

Chi-square tests were employed for the evaluation of each subject's betting behavior, and t-tests were utilized to evaluate changes in time judgment resulting from the time-training procedure.

Chapter IV

Results of Studies of Pupillary Responses

Analysis of pupillary data included examination of initial pupillary diameter, amplitude of dilation, and amplitude of recovery slope following peak dilation for each condition. Pupillary dilation and recovery slope measures were the most sensitive to the experimental manipulations. Dilation was greater when the subject placed the bets, and the amplitude of dilation increased as bet value increased.

The difference between winning and losing outcomes was most apparent in the recovery slope, with greater constriction for losing than for winning responses. For losing responses, the amplitude of the recovery slope increased as the amount of money lost increased. An opposite trend appeared for winning responses: the amplitude of the recovery slope decreased as the amount of money won increased.

Significance levels for the analyses of variance across all pupillary measurements are summarized in Table 1. Complete ANOVA summary tables, raw pupillary data, and means and standard deviations across subjects for each condition are presented in the Appendix.

Analysis of Baseline Pupil Diameter

The latency of the pupillary dilation response under conditions of uncertainty is approximately 600 msec (Hakerem, 1974). Pupillary baseline was measured as the median amplitude from 470 to 590 msec after the warning click stimulus. Analysis of baseline effects

	Bet Source	Out-come	Value	Outcome x Value	Bet x Outcome
Initial Diameter					
Across Stimuli:	.05				
Peak Dilation					
Across Stimuli:	.01		.05		
Emitted Response:	.01				
Single Click:	.01		.05		.05
Double Click:	.01		.05		
Recovery Slope					
Emitted Response:				.05	
Single Click:				.05	
Double Click:			.01		

Table 1. Summary of significant *p*-values for pupil analysis (complete ANOVA tables in Appendix)

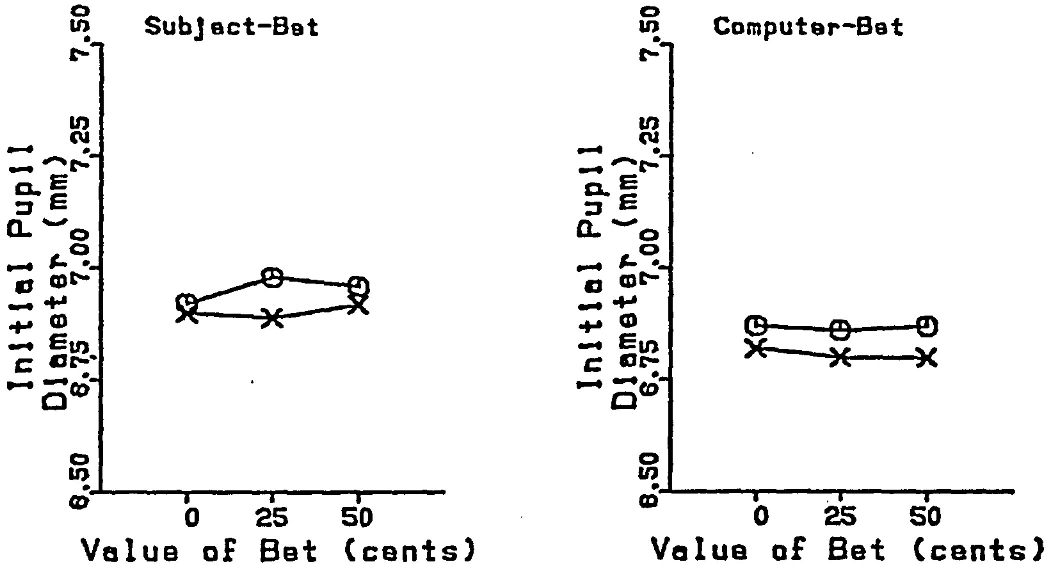
utilized trials combined across outcome and informational event. The only relevant factors were Source of Bet and Value of Bet, for which initial diameters are plotted in Figure 10. A main effect was observed for Source of Bet ($p<.05$), with the Subject-Bet condition producing a slightly larger initial diameter. No main effects for value were observed, and there was no significant difference between initial diameters during the first three test days as compared to the second three test days.

Analysis of Pupillary Peak Dilation

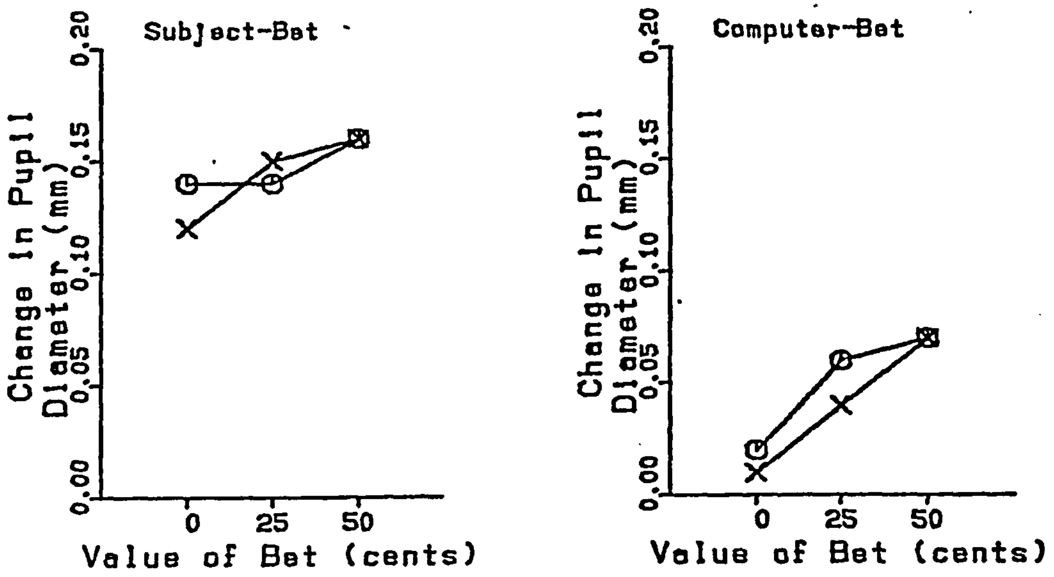
Little dilation was observed following the warning click, but a large dilation was typically observed following the informational event. The onset of the larger dilation at approximately 600 msec and attainment of peak diameter at approximately 1200 msec was observed for both the evoked and the emitted pupillary responses. Grand mean pupillary responses across trial outcome are shown for the Subject-Bet and Computer-Bet conditions as a function of Value of Bet, and for the Certain condition, in Figure 11. To demonstrate the effects of dilation, the curves are superimposed at initial diameter.

A significant main effect for Source of Bet was found for all stimulus conditions, with significantly larger dilations in the Subject-Bet than in the Computer-Bet condition (emitted response, $p<.01$; single click evoked response, $p<.01$; double click evoked response, $p<.01$; combined across stimuli, $p<.01$); mean amplitudes are plotted in Figures 10 and 12.

A main effect for Value revealed a somewhat linear increase in



Pupil: Initial Diameter for Days 1-3 (⊖)
and Days 4-6 (X) Across Stimuli



Pupil: Peak Dilation for Days 1-3 (⊖)
and Days 4-6 (X) Across Stimuli

Figure 10.

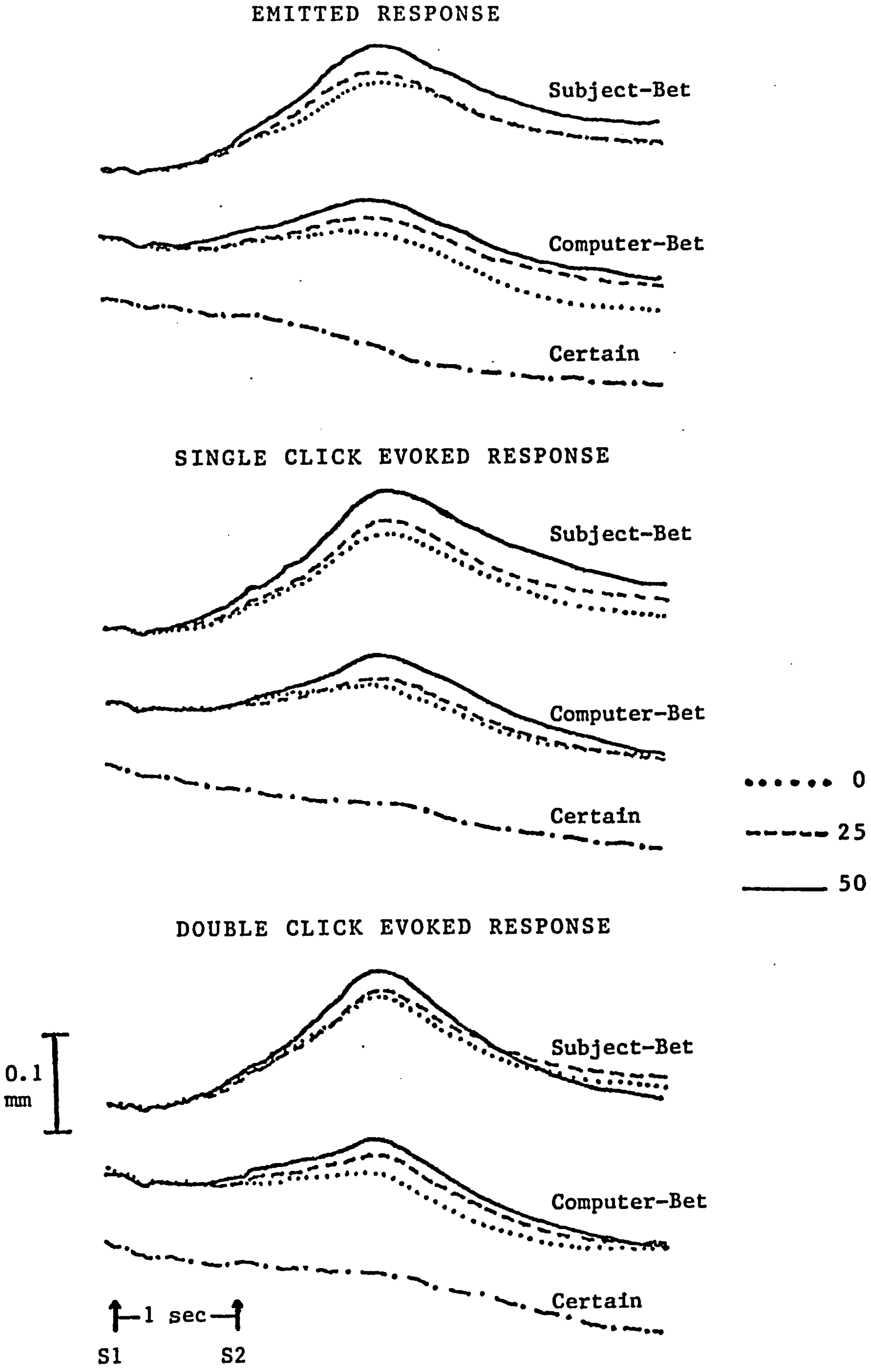
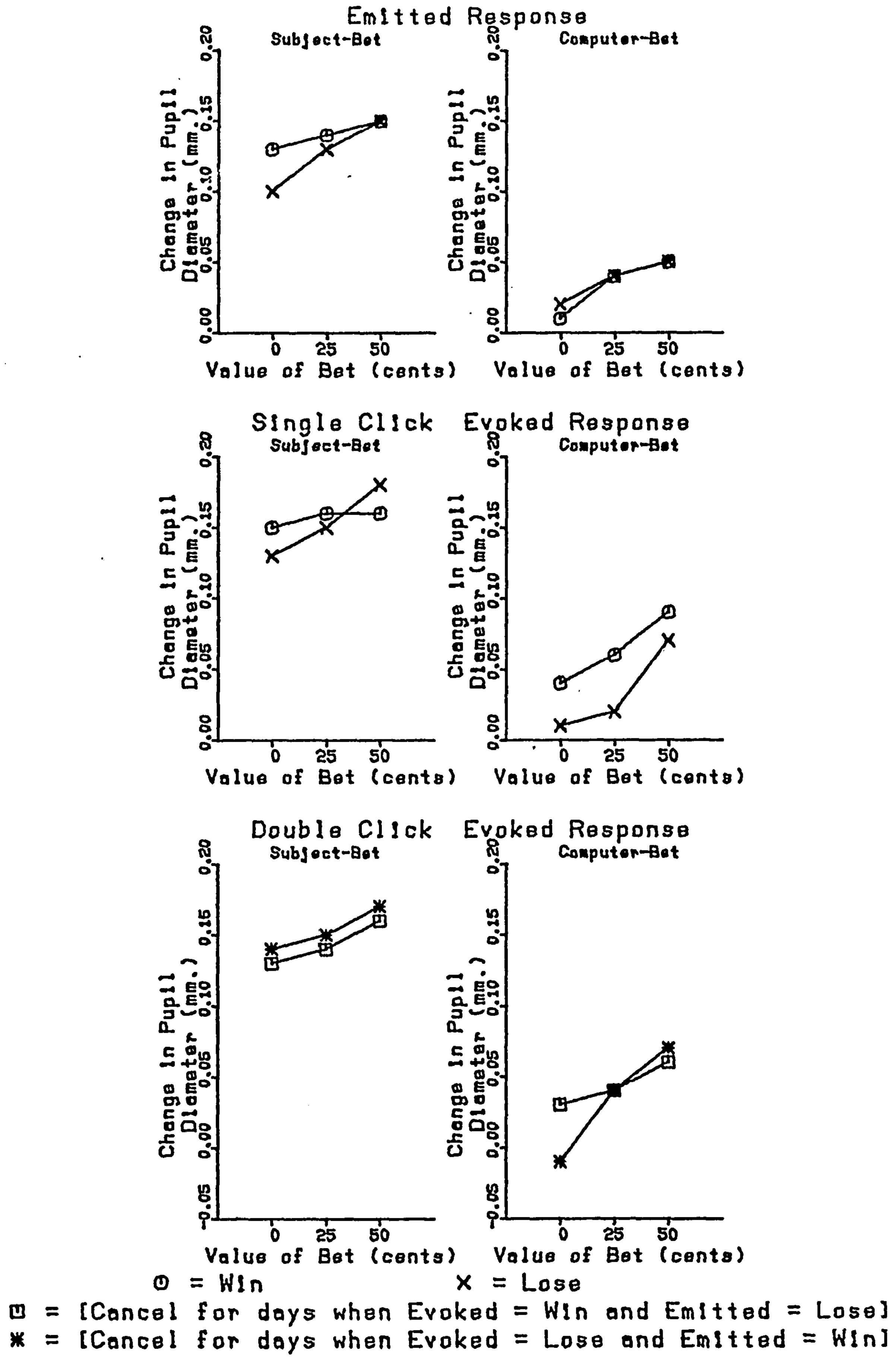


Figure 11. Pupillary Dilation as a Function of Value: Grand Means.



Pupil: Peak Dilation, Across Subjects

Figure 12.

pupillary dilation with increasing bet value (Figure 12) for the evoked single ($p < .05$) and double click responses($p < .05$), and was also observed in the response averaged across stimuli ($p < .05$). For each of these conditions, the amplitude of the dilation was significantly greater for a bet of 50 than for zero cents. The trend for the emitted dilation was in the same direction. Individual data indicated that only four of the eight subjects contributed to the differential increase in dilation for different bet values.

There was a significant Source of Bet X Outcome interaction for the single click evoked dilation ($p < .05$). Although there was little difference between winning and losing outcomes in the Subject-Bet condition, there was a larger winning than losing dilation in the Computer-Bet condition.

Evaluation of peak dilation among stimuli yielded a significant main effect ($F = 12.43$, $p < .01$). The mean dilation of .10 mm following a single click was significantly greater than the .08 mm dilation following a missing stimulus. The dilation of .09 mm following a double click was not significantly different from either an emitted or single click evoked response.

The latency to peak dilation was evaluated only for the Subject-Bet condition. The resolution of the dilation was not sufficiently clear in the Computer-Bet condition for an accurate determination to be performed and in several cases appeared to reach maximum diameter as early as 600 msec. The determination of latency was based on the initial data point at which the largest diameter was maintained. Since computation of peak dilation amplitude utilized a series of ten successive points, the procedure for evaluation of peak latency may have yielded a slight but consistent underestimation of latency.

The mean latencies to peak dilation under the Subject-Bet condition were 1090 msec for the emitted response, 1162 msec for the single click and 1099 msec for the double click evoked responses. Differences in latency were not significant. No significant effects of value, outcome, nor any interactions were observed for latency to peak dilation.

For any averaged response curve, the value of the standard deviation at each time point was relatively stable across the entire five second sample period. The amplitude of the standard deviation was evaluated at the latency of peak dilation for each condition. No significant differences among experimental conditions were found, indicating that temporal jitter between trials of different conditions was not significant. The only observable trend was a mean decrease of .04 mm in the amplitude of the standard deviation in the Subject-Bet condition compared to the Computer-Bet condition. The mean standard deviation ranged from .14 to .67 mm for individual subjects, with a modal value of approximately .30-.40 mm.

Analysis of Pupillary Recovery Slope

The region 1600 msec following peak dilation tended to show the greatest amplitude of differentiation in post-dilation diameter. There were several averages in which winning 25 or 50 cents produced a larger diameter of the pupil following the normal latency of peak dilation;

for these cases, the amplitudes were measured at the corresponding latencies of 1200 msec after the informational event, and 1600 msec subsequently. Averaged pupillary response curves for individual conditions are presented for the missing stimulus, single click and double click conditions in Figures 13, 14 and 15, respectively. Since no differences were observed in amplitude of dilation between winning and losing outcomes, the curves have been superimposed at peak dilation to emphasize the effects observed in the recovery slope. The amplitude of the recovery slope is plotted as a function of trial outcome in Figure 16.

No significant main effects were observed for the recovery slope of the pupil following an absent stimulus or a single click stimulus, but a main effect for Value was observed for the double click configuration ($p<.001$). The amplitude of the recovery slope increased as the value of the bet increased, with a significantly greater recovery slope for a bet of 50 cents than for either 25 or zero cents (Figure 15).

A significant interaction was observed between Outcome X Value of Bet for both the emitted ($p<.05$) and single click ($p<.05$) responses (Figure 16). For the emitted response, the effects of winning and losing zero were significantly different from winning or losing either 25 or 50 cents. Losing 25 or 50 cents produced a larger recovery slope than winning 25 or 50 cents. For the zero bets, in contrast, the response to winning zero was larger than the response to winning either 25 or 50 cents, and looked like a typical response to losing money. Conversely, losing zero produced a smaller recovery slope than losing either 25 or 50 cents.

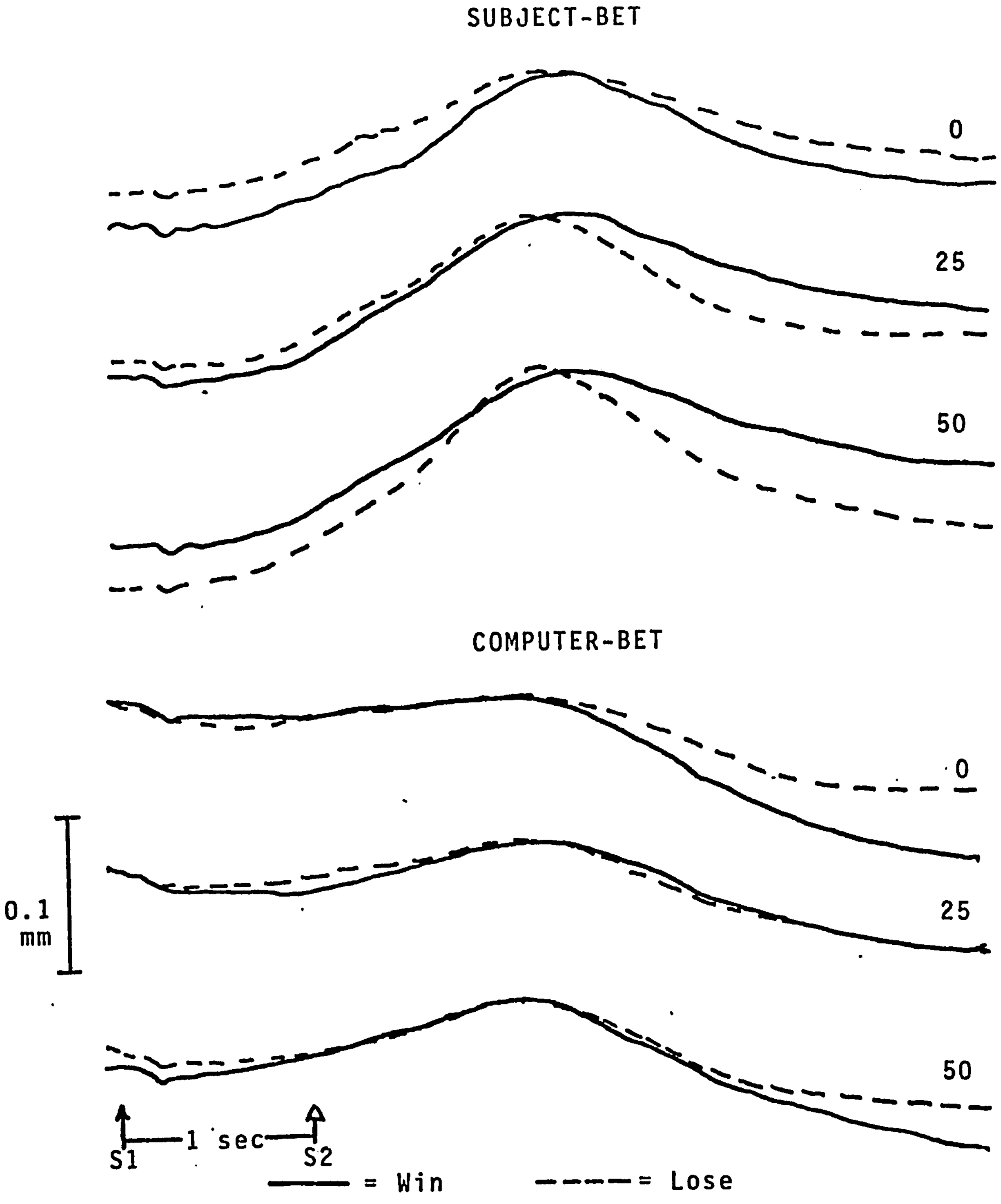


Figure 13. Emitted Pupillary Responses for Individual Conditions: Grand Means.

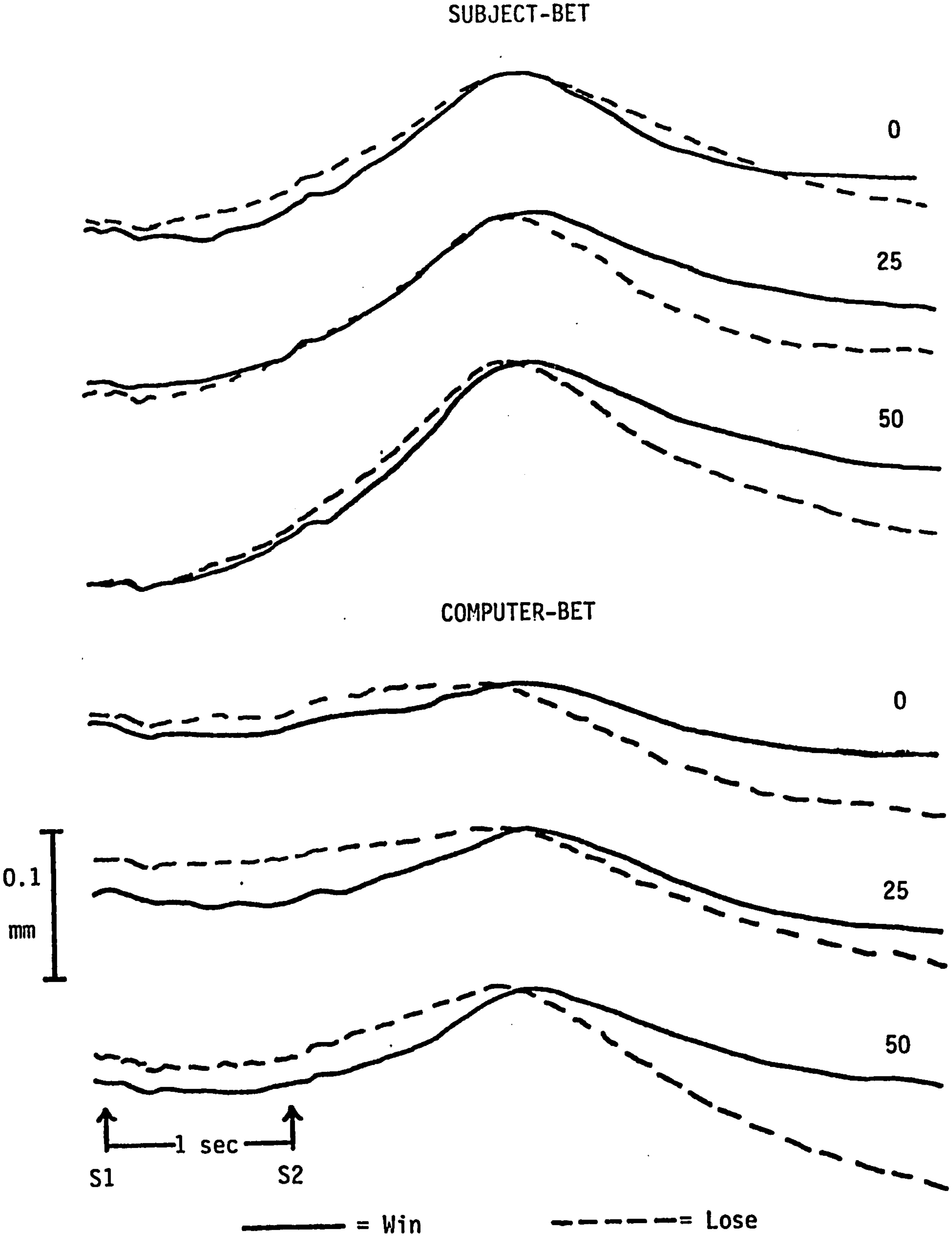


Figure 14. Single Click Evoked Pupillary Responses for Individual Conditions: Grand Means.

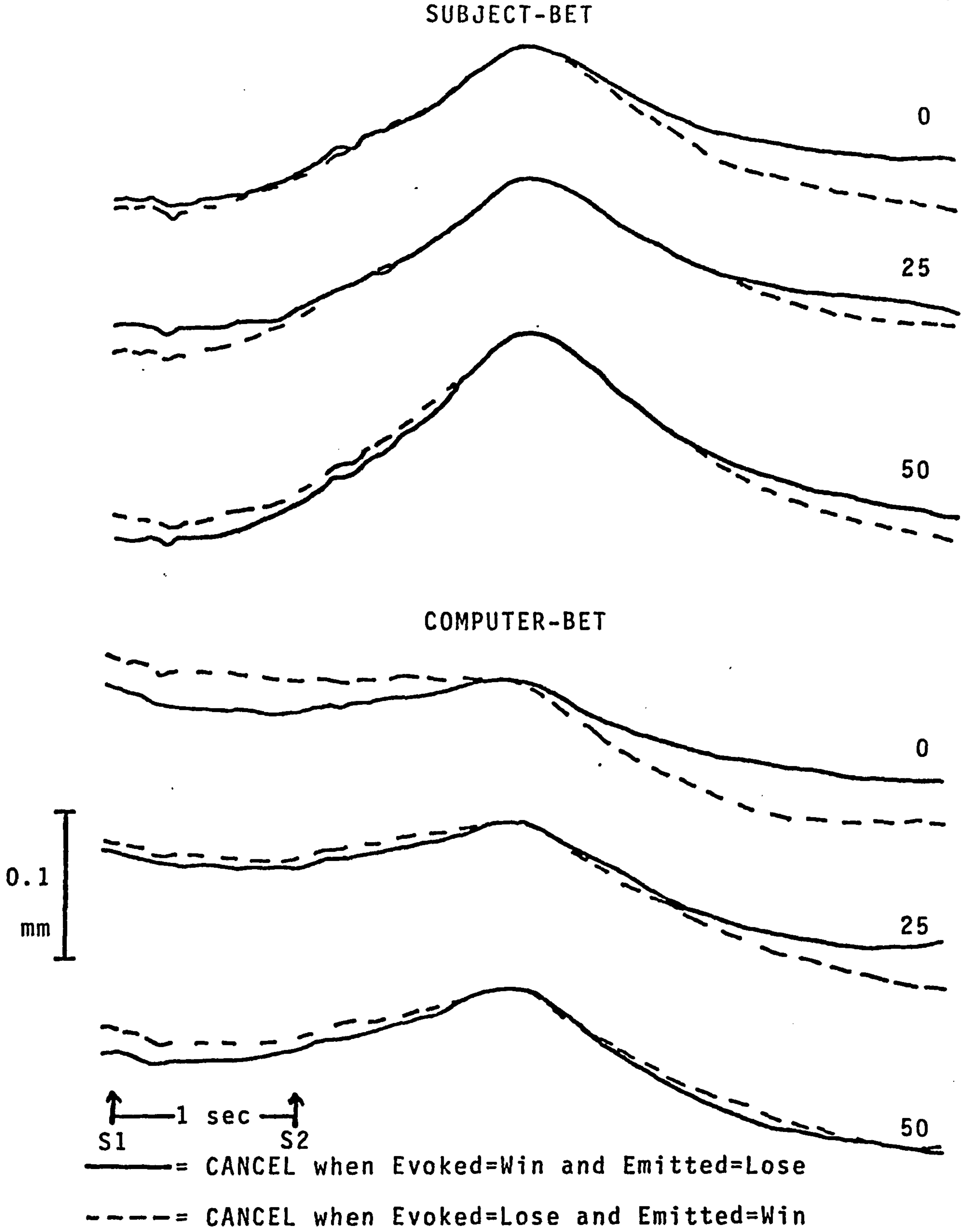


Figure 15. Double Click Evoked Pupillary Responses for Individual Conditions: Grand Means.

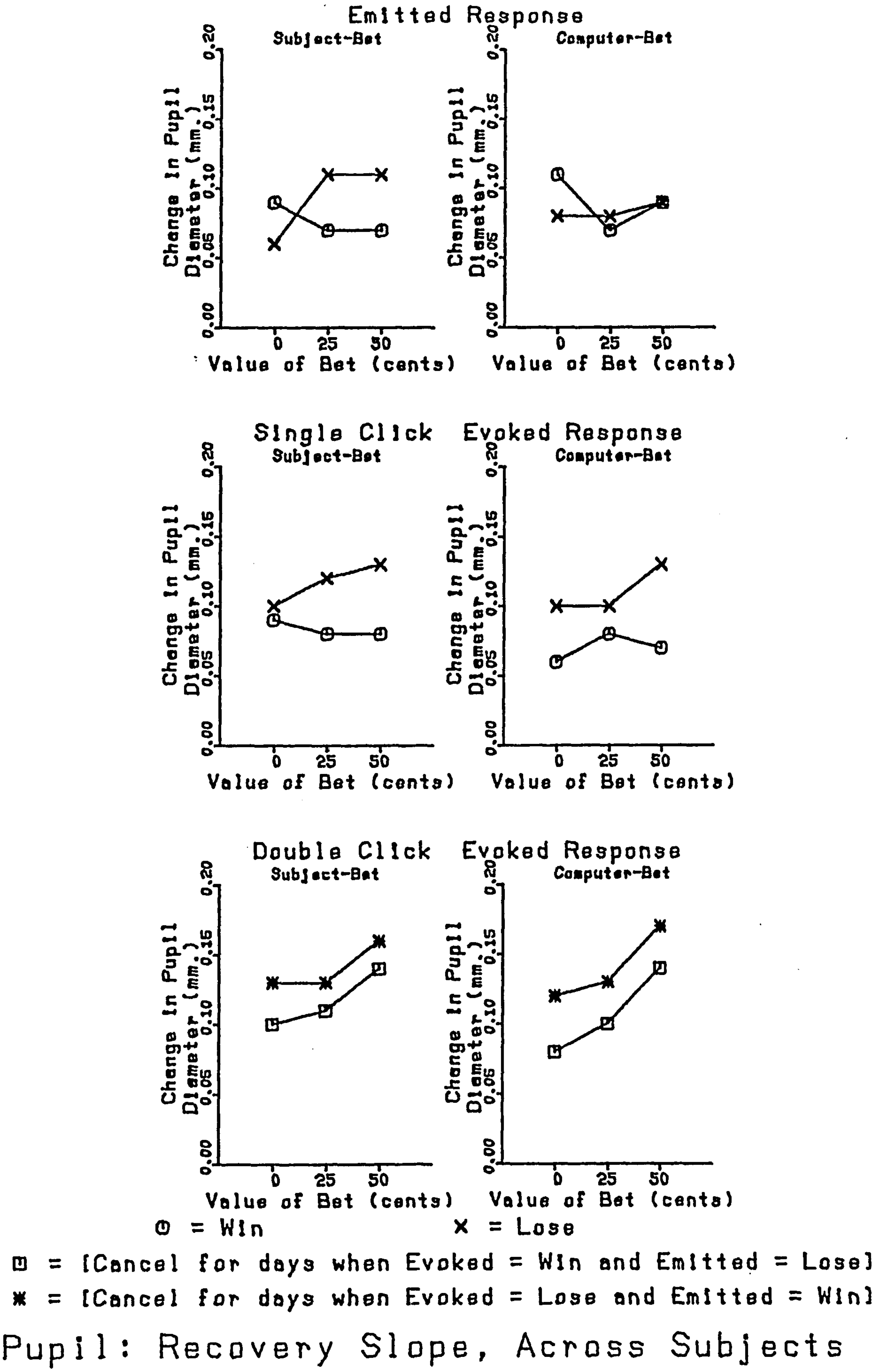

Emitted Response
Subject-Bet
Computer-Bet
Single Click Evoked Response
Subject-Bet
Computer-Bet
Double Click Evoked Response
Subject-Bet
Computer-Bet
Change in Pupil Diameter (mm.)
0.00
0.05
0.10
0.15
0.20
Value of Bet (cents)
0
25
50
⊙ = Win
× = Lose
⊡ = [Cancel for days when Evoked = Win and Emitted = Lose]
✱ = [Cancel for days when Evoked = Lose and Emitted = Win]
Pupil: Recovery Slope, Across Subjects

Figure 16.

The nature of the interaction between Outcome X Value of Bet for the single click was somewhat different from the emitted response, with a significantly greater difference between winning and losing 50 cents than for zero or 25 cents. Losing produced a larger recovery slope than did winning, and for the losing response, the recovery slope was larger as the amount of money lost increased. However, winning 50 cents produced a smaller recovery slope than did winning 25 cents.

In summary, the recovery slope for emitted pupillary responses in the Subject-Bet condition was characterized by a larger recovery slope for losing compared to winning except when zero was bet, in which case winning produced the larger response. A similar pattern was observed for the single click evoked pupillary response with the exception that there was little difference between winning and losing zero. The double click evoked response was best characterized as an increase in the amplitude of the recovery slope with increasing bet value during all sessions.

Comparison of Initial Diameter, Pupillary Dilation and Recovery Slope

Although the manipulation of value was apparent in pupillary dilation and recovery slope, no effect was observed on the initial diameter of the pupil. The average amplitudes of initial diameter, peak dilation, and recovery slope were pooled across subjects. Correlations were obtained for initial diameter with peak dilation and with recovery slope, paired by the six experimental outcomes for each stimulus presentation. Separate correlations were obtained in the

Subject-Bet and Computer-Bet conditions.

No significant relationship was observed between initial diameter and subsequent pupillary activity. Only a consistent, but non-significant negative correlation with initial diameter could be observed for the dilation and recovery slope responses, ranging from -.24 to -.36 in the Subject-Bet condition and from -.23 to -.55 in the Computer-Bet condition.

Comparison of Pupillary Responses in Certain vs. Uncertain Conditions

The unweighted average response amplitudes across outcome and value for each subject in the Subject-Bet condition and the Computer-Bet condition were compared to the amplitudes observed in the Certain condition. No significant effects were observed among baseline diameters for the Certain condition compared to the betting conditions. A slight trend for larger initial diameters in the Certain condition was observed.

The amplitude of peak dilation was significantly different among conditions for all stimulus events (emitted response, $\underline{F} = 16.83$, $\underline{p} < .01$; single click evoked response, $\underline{F} = 15.45$, $\underline{p} < .01$; double click evoked response, $\underline{F} = 13.32$, $\underline{p} < .01$), with larger dilations in the two betting conditions than under the Certain condition (Figure 11). The recovery slope of the pupil was significantly larger during the betting conditions than for the Certain condition for the emitted response ($\underline{F} = 16.15$, $\underline{p} < .01$) and the double click evoked response ($\underline{F} = 10.00$, $\underline{p} < .05$). The same trend was observed for the single click, though not significant.

Chapter V

Discussion of Pupillary Results

Pupillary motility following informational feedback consisted of a complex response that was sensitive to all three experimental manipulations: value of bet, trial outcome and source of bet (Subject-Bet vs. Computer-Bet). Increasing the subject's attention to stimuli by requiring the placing of a bet resulted in greater dilation than when the value of the bet was selected for the subject. The consequences of trial outcome were also demonstrated to reflect subjective qualities associated with winning or losing money. Nearly identical responses were either evoked by presentation of feedback stimuli or emitted in the absence of stimuli which provided sufficient feedback. This discussion is limited to factors dealing primarily with the pupillary data; a more general discussion and overview is provided in Chapter VIII.

Initial Pupillary Diameter

Initial pupillary diameter in the Certain condition was characterized by a weak trend for a larger diameter than in the betting conditions, although not significantly different. This finding is contrary to the report of Friedman (1972) that initial diameters are larger under conditions of uncertainty as compared to certainty.

The present findings are probably related to the fact that the Certain condition was presented at the beginning of test days two and five. It has been repeatedly observed that pupil dimaeter is largest at the beginning of an experiment and tends to decrease slightly during the session (Peavler, 1974; Simpson, 1969). The possibility that initial pupillary diameter in the Certain condition would be larger than in the betting conditions, because of its temporal location in the experimental session, had been anticipated. Since the objective of including the Certain condition in this study was to provide a partial replication for Friedman's (1972) report of reduced pupillary dilation and P300 amplitude during conditions of certainty as compared to uncertainty (represented by betting in the current study), the amplitude of initial diameter was not considered to be of immediate concern. Friedman had noted that the amplitude of pupillary dilation was not related to small variations in initial pupil diameter.

Pupillary Dilation Response

A pattern of increased dilation was observed when larger bet values were at stake. Since no such effect was found for the initial diameter, it may be suggested that any arousal due to increasing bet value was associated with the generation of the dilation following the presentation of the informational event, rather than a general enhancement of tonic sympathetic activity which would be implied if initial diameters had also been increased.

Kahneman & Peavler (1969) observed greater pupillary dilation during a learning task for trials associated with high rewards, and the behavioral data indicated that subjects performed better when higher rewards were involved. This suggested to the investigators that the differences in dilation were probably associated with the effort employed in learning the associations, so that the effect of increased reward in their task was to increase motivation to learn. Dilation was not considered to be increased as a direct result of value.

No differential task requirement was involved in the current studies. It may reasonably be assumed that the differential dilations observed were a direct reaction to the manipulation of value, although this was true for only four of the subjects. It is possible that some characteristic personality differences were being reflected by such intersubject differences, but the nature of such differences is purely conjectural in the absence of additional assessment.

The latency of 1162 msec for the single click evoked dilation is comparable to the 1200 msec latency reported previously (Friedman et al., 1973; Hakerem, 1974). Failure to find differential latencies for different values and outcomes, as well as the lack of the differential dilation to winning and losing outcomes, supports the findings of Friedman (1972) indicating that presentation of equiprobable stimulus outcomes does not produce differential pupillary dilation. The overall difficulty of processing seems to have been similar for all trials. In comparison, the latency and amplitude differences obtained by Beatty and Wagoner (1978) were directly related to differential processing requirements in the evaluation of stimuli, a characteristic that was purposely held constant in the present study.

Only the data of Haughney (1976) indicate differences in the amplitude of pupillary dilation between winning and losing outcomes. In his experiment, the subject was asked to describe his confidence in his prediction of stimulus presentation. Haughney (personal communication) has indicated that an anticipatory dilation appears in his data which is larger when a high confidence guess is wrong than right. One interpretation of this finding is that Haughney's differences in dilation to winning and losing outcomes are functions of the outcome probability, and are consistent with the report of Friedman (1972) that the amplitude of dilation is larger when outcome probability is decreased.

Recovery Slope of the Pupil

The findings for the recovery slope may be summarized by noting that unfavorable outcomes were characterized by greater constriction, and favorable outcomes were characterized by reduced constriction. The recovery slope measure was most sensitive to factors related to the outcome and monetary value associated with each trial. The most distinctive characteristic was the increase in the amount of pupillary constriction (after an initial dilation) given the loss of money compared to smaller cosntrictions when the subject won money. The value of the bet associated with each trial was also a significant factor, with a trend for greater constriction as a greater amount of money was lost, and an opposite though less pronounced trend for less constriction as greater amounts of money were won.

The trend for greater constriction associated with losing and reduced constriction for winning conditions was reversed for the emitted response when a bet of zero had been placed: that is, the response emitted after winning zero appeared as if it was a loss of money, while losing zero resulted in a response which resembled the waveform elicited by winning money. Apparently, at least for the emitted responses, the significance of the outcome was reflected in the recovery slope measure.

Post-session interviews with the subjects indicated that the occurrence of the winning stimulus was experienced, after a bet of zero, as missing an opportunity to have won, or in the words of one subject, "money down the drain." It really was an unfavorable (or aversive) outcome, much like a loss of real money. On the other hand, a losing outcome following a zero bet meant that, had the subject actually bet money, there would have been a loss, but since nothing was bet, there was no loss. Conceptually, it was an advantageous outcome. Therefore, even though the individual outcomes following a bet of zero always established *a priori* that the subject would neither gain nor lose money, the subjective importance of the actual outcome to the subject was still actively processed, and this processing activity was reflected in the pupillary data.

Although the presentation of the event signifying cancellation of all bets effectively resulted in a zero outcome for all such trials,there was greater constriction of the pupil as the value of the bet was increased, which is the same pattern that was observed for increasing loss of money. In comparison to the actual winning or losing of money, the data for the cancelled conditions suggest that trials associated with cancellation were aversive, and more aversive the greater the value of the cancelled bet. The implications of this conclusion are discussed in Chapter VIII.

The present findings reinforce and extend the specificity of observations made for correct and incorrect post-dilation activity by

Levine (1969), Bock (1976), Friedman (1972) and Steinhauer et al. (1979). The data collected by Haughney (1976) have also been re-evaluated using the same definition of recovery slope, and indicate a larger constriction for trials on which the subject lost money (Haughney, personal communication). Possible evidence for similar activity can be found in the data presented by Kahneman and Peavler (1969) in their Figure 2 (p. 314). Although they report no differences to high and low reward trials immediately following presentation of the stimulus digit, their graphic data indicate a separation between high and low reward stimuli which corresponds to the time of recovery slope activity in the present data. Whether this is a consistent difference is difficult to evaluate, since it is based on only one measurement/sec, but their data are suggestive of the recovery slope.

This report of differential response of the pupil to winning and losing outcomes by larger diameters following a favorable outcome, compared to smaller pupillary diameter in the event of an unfavorable outcome, appears supportive of Hess' (1965) conceptualization that the pupil dilates to positive stimuli and constricts to negative stimuli. It is not clear that the process reported here is identical to the characteristics of pupillary motility claimed by Hess. In none of the studies of Hess or his proponents was a sufficiently large number of trials employed to average out the effects of pupillary noise due to other afferent activity. No report is available that uses a high sampling rate to indicate the time course of pupillary activity following stimulation in Hess-type experiments. Instead, the average diameter over some lengthy sampling period has usually been presented, so that there has been no definitive demonstration that constriction is observed as a function of psychological response.

Few laboratories have examined the pupillary response in detail during the first two or three seconds following a significant psychological event (e.g., Beatty & Wagoner, 1975, 1978; Hakerem, 1974; Shiga & Ohkubo, 1978), as performed also in the present study. Juris and Velden (1978) have pointed out that "No psychological stimulation or situation can be described to date that consistently leads to pupillary constrictions that are not reconstrictions after a dilation" (p. 423). It may be that Hess' (1965) formulation must be modified in that constriction as a reflection of aversiveness is valid only following an initial dilation response.

The constriction process of the pupil observed in this study has arbitrarily been termed "recovery slope," but rather than merely denote recovery of the pupil following dilation, it is more likely to be an active physiological process. An immediate separation between winning and losing waveforms following peak dilation appeared only for some subjects, while for others a dual-stage constriction was noted: the pupil first constricted equally in the winning and losing conditions for up to one sec following the dilation, at which time winning and losing waveforms began to separate. The observation that constriction was often greater in extent than the initial diameter of the pupil further distinguishes this process from a mere recovery after dilation.

It is unlikely that neurohumoral mechanisms contribute significantly to the present findings, since a minimum latency for the sympathetic contribution of adrenergic substances is two to three sec (Loewenfeld, 1958; Lowenstein & Loewenfeld, 1961), and the dilation process has essentially terminated before then. The long term effects of adrenergic and cholinergic agents would be reflected as slow changes on subsequent trials, rather than as the relatively quick alterations in pupil diameter that were observed following stimulus presentation. The present observations are indicative of neural activity.

Central nervous system influences on pupillary activity are ultimately mediated by autonomic pathways. Pupillary dilation involves activity of posterior and lateral regions of the hypothalamus, which give off primarily ipsilateral fibers which descend within the spinal cord (Lowenstein & Loewenfeld, 1962; Zinn, 1972). Secondary fibers exit from the spinal cord at lower cervical and upper thoracic regions, joining the paravertebral sympathetic chain and synapsing in the large superior cervical ganglion. Tertiary fibers take a number of alternate routes to the pupillary dilator muscle (dilator pupillae). Control over constriction originates in the Edinger-Westphal nucleus of the oculomotor complex, whose fibers synapse in the ciliary ganglion, which projects short fibers to the sphincter muscle of the iris (sphincter pupillae). The mechanisms for dilation include both increased sympathetic activity or decreased parasympathetic activity (Loewenfeld, 1958; Lowenstein & Loewenfeld, 1950, 1962), with constriction of the pupil reciprocally evoked by increased activity of the Edinger-Westphal nucleus and decreased sympathetic activity.

It has been demonstrated that the contribution of supranuclear inhibition of the Edinger-Westphal nucleus to pupillary dilation can account for no more than 20% of observed dilation (Loewenfeld, 1958). Sources of inhibition to the Edinger-Westphal nucleus include cortical, reticular, thalamic and hypothalamic sources (Zinn, 1972). Cortical influences contributing to dilation have also been described (Loewenfeld, 1958).

Mechanisms that may be responsible for generation of the differential recovery slopes observed in the present data have not been described in the literature. The time course of the recovery slope suggests active interactions involving parasympathetic components. Recent evidence indicates that in addition to the oculomotor fibers, other efferent projections from the Edinger-Westphal nucleus exist (Loewy & Saper, 1978), which seem primarily to consist of projections to dorsal column, spinal trigeminal and inferior olivary nuclei, and spinal cord. Influences on central processes, however, are still unknown. It is likely that the effect of active parasympathetic activity is mediated through a decrease of inhibitory influences on the Edinger-Westphal nucleus. The active role of parasympathetic activity in the elicitation of classically conditioned pupillary dilation is indicated by normal development of the reflex even in the sympathectomized cat iris (Ashe, Cooper & Weinberger, 1978).

Recording of the pupil in darkness, as performed in the current experiment, results in minimal activation of the parasympathetic component (Lowenstein & Loewenfeld, 1950), at least prior to stimulation. It is likely that the responses reported for pupillary dilation measured under light-adapted conditions in the work of some researchers include some component due to inhibition of tonic parasympathetic activity. A possible effect of such activity was observed by Shiga and Okhubo (1978) during recording of the pupil from subjects engaged in a shadowing task, and a similar effect was probably responsible for the initial dilatory response seen in the data of

Haughney (presented by Johnston, 1979). Shiga and Ohkubo (1979) examined the effect of light adaptation on a simple motor response, and found that a separate early dilation appeared whenever initial pupillary diameter was less than five mm. The apparent effect of the task-related stimulus was to increase inhibition of the Edinger-Westphal nucleus before the sympathetic component was fully expressed.

The active process reported here is also to be distinguished from the "dumping" of stored information that results during the recall stage in retrieval tasks (Kahneman, 1973; Kahneman & Beatty, 1966). The pupil is dilated and remains so during such tasks until the moment of recall, demonstrating the continuous maintenance of effort on the part of the subject. Decrease in pupillary diameter in such experiments seems related to the decrease in current storage requirements as each successive piece of information is discarded from memory. Whether either reception or recall of data is accompanied by small, immediate pupillary reactions can only be determined by sufficient repetitions at a high sampling rate allowing measurement of the averaged response to each event.

Emitted Pupillary Responses

The generation of both pupillary dilation and recovery slope activity that was related to trial outcome following the absence of a physical stimulus indicates that the mechanism for such responses is under endogenous control, reflecting the cognitive characteristics of the individual and/or the cognitive character of the task, rather than being an exogenously elicited process. The decreased pupillary dilation amplitude for the emitted response relative to the evoked pupillary response parallels the decrease in amplitude reported for emitted as compared to evoked P300 responses (e.g., Sutton et al., 1967).

In a study of pupillary response to visual stimuli at threshold, Hakerem and Sutton (1966) observed that for correctly detected stimuli, contraction to light was followed by a definite dilation. The dilation was most evident at intensities too weak to produce contraction, when the stimulus was reported as seen. No dilations were observed for false alarms, misses or correct rejections.

Beatty and Wagoner (Note 5) reported similar findings using an auditory signal detection paradigm in which subjects reported their level of confidence in the decision that a signal was presented. Dilations were produced only on high confidence detection trials. In both studies, resolving of uncertainty by detection of the stimulus resulted in dilation. Since resolving of uncertainty can also be implied by false alarms (an incorrect detection) or correct rejections, one would expect to see dilations occurring under such conditions. It is likely that such factors as low confidence or time jitter contributed to the difficulty in generating averaged responses in these conditions.

In a separate study, Clark and Eckenberger (Note 6) failed to find any correlation between detection and pupillary dilation in an auditory signal detection task. In their paradigm, the signal could occur on 50% of trials, at any time during a ten second period. Their failure to observe pupillary dilation may have been associated with the uncertainty involved when the signal was potentially distributed over so long a time period. In this case, the equivocation due to temporal uncertainty (Ruchkin & Sutton, 1978b) may have interfered with the resolving of informational uncertainty.

In contrast, both Hakerem and Sutton (1966) and Beatty and Wagoner (Note 5) employed a warning stimulus and fixed foreperiod before stimulus presntation. This may have increased confidence for signal detect trials, since the subject knew when to expect information. It is possible that increasing stimulus detectability would enhance generation of dilation in response to either false alarms or correct rejections, as has been demonstrated in similar experiments involving P300 (K. Squires et al., 1975; N. Squires et al., 1975; Ruchkin, Sutton & Stega, 1980).

Chapter VI

Results of Event-Related Potential Studies

All ERP components, including the CNV, tended to be larger in the betting conditions than in the condition of stimulus certainty, and responses tended to be larger during the Subject-Bet condition than during the Computer-Bet condition. The analysis of the ERP data indicated sensitivity of both emitted and evoked components to larger bet values, and to winning and losing outcomes. The CNV was not sensitive to these variables.

There seemed to be little differential effect of these manipulations on latency of components. An unexpected observation was the generation of emitted P300 responses that were as large as, and sometimes larger than, the evoked P300s recorded during the same blocks of trials.

Scalp Distribution of Components

Grand means across subjects are presented for the emitted response (Figure 17), for the single click evoked response (Figure 18), and for the double click evoked response (Figure 19) at all electrode locations, separately by Subject-Bet and Computer-Bet conditions. The data have been collapsed across outcomes to illustrate differences among bet values. The responses to the Certain condition are presented for all stimulus events in Figure 20.

Electrode distributions for all components of the event-related

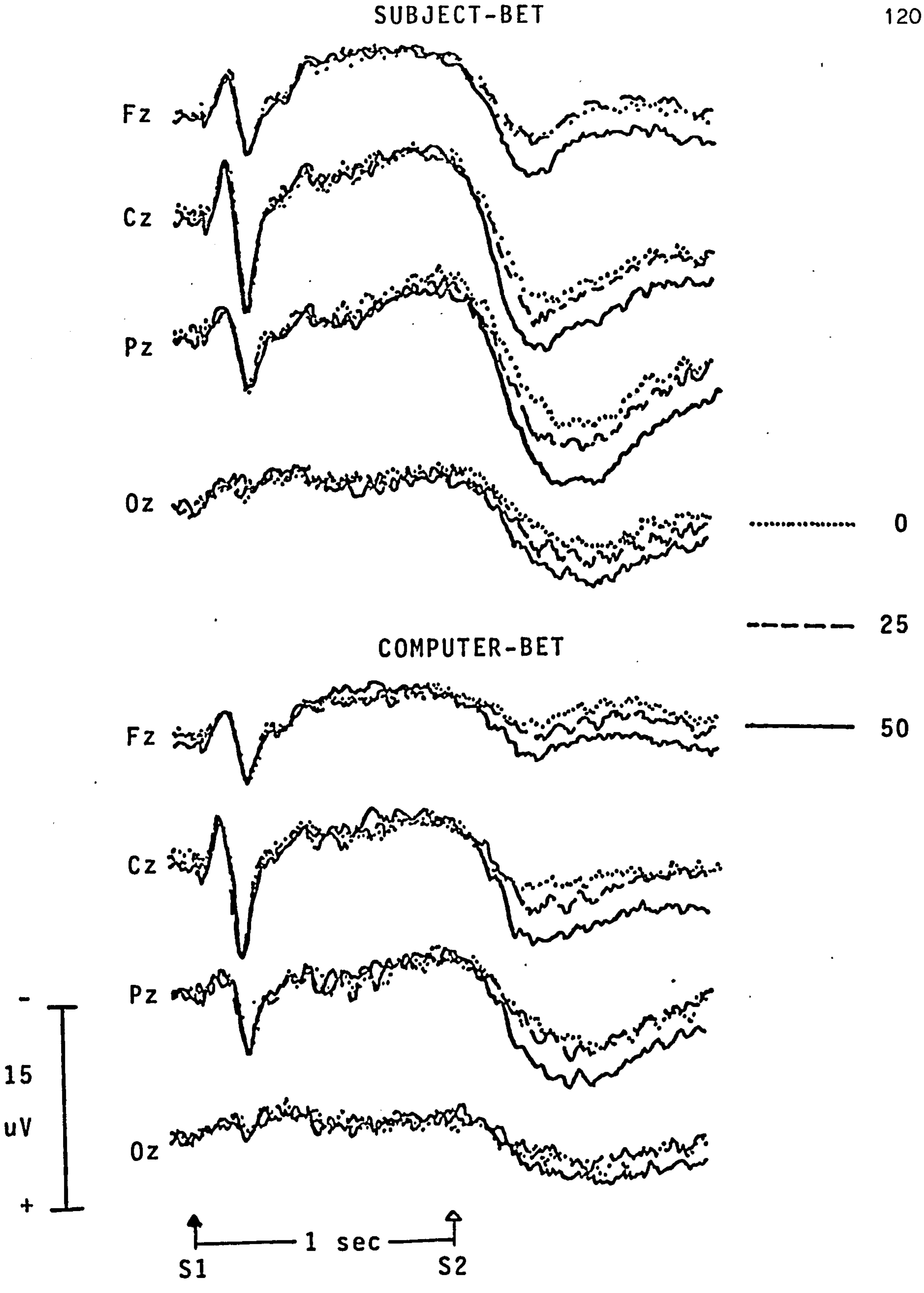


Figure 17. Emitted Potentials at all Electrode Locations for Individual Bet Values: Grand Means.

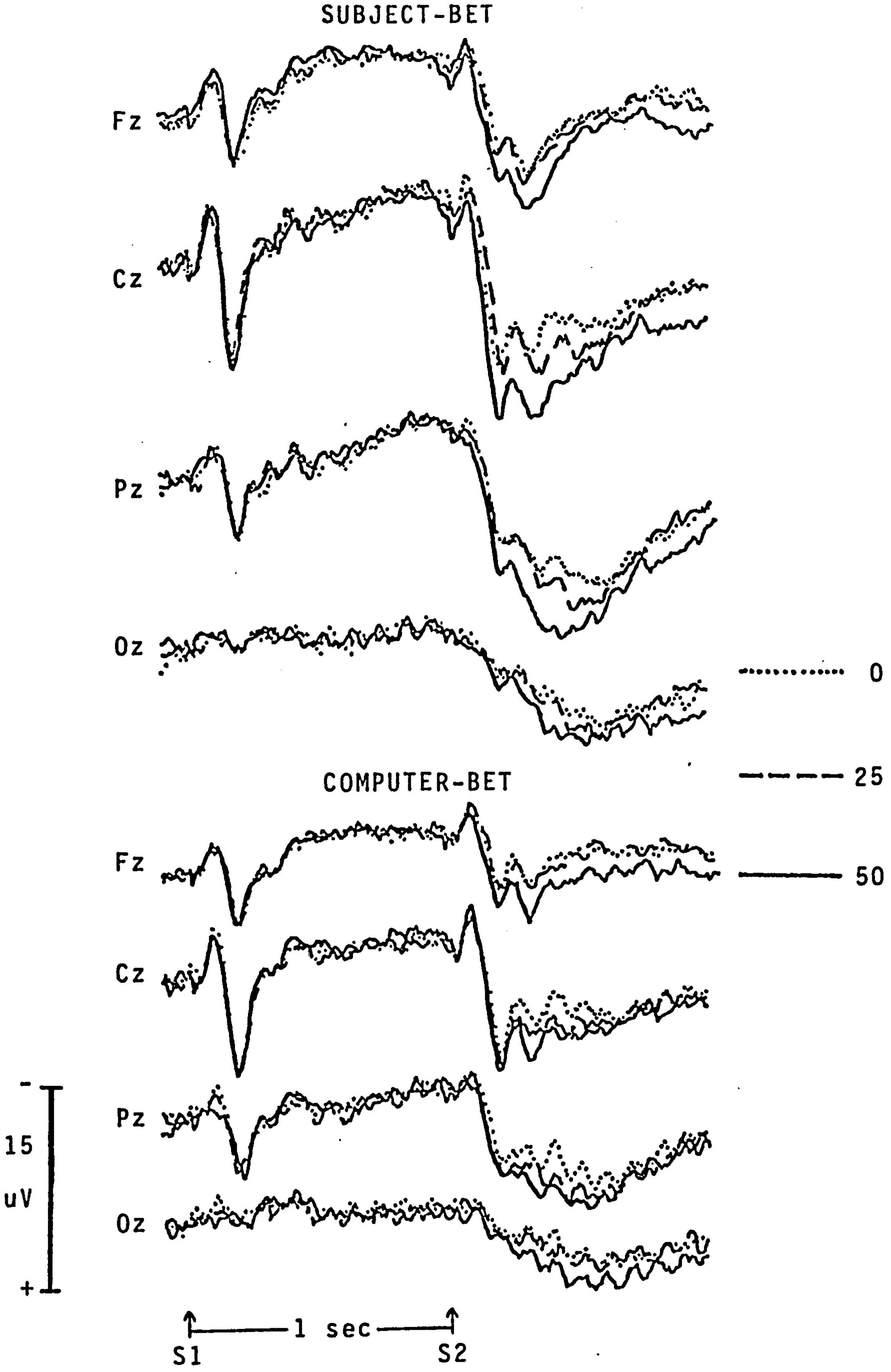


Figure 18. Single Click Evoked Potentials at all Electrode Locations for Individual Bet Values: Grand Means.

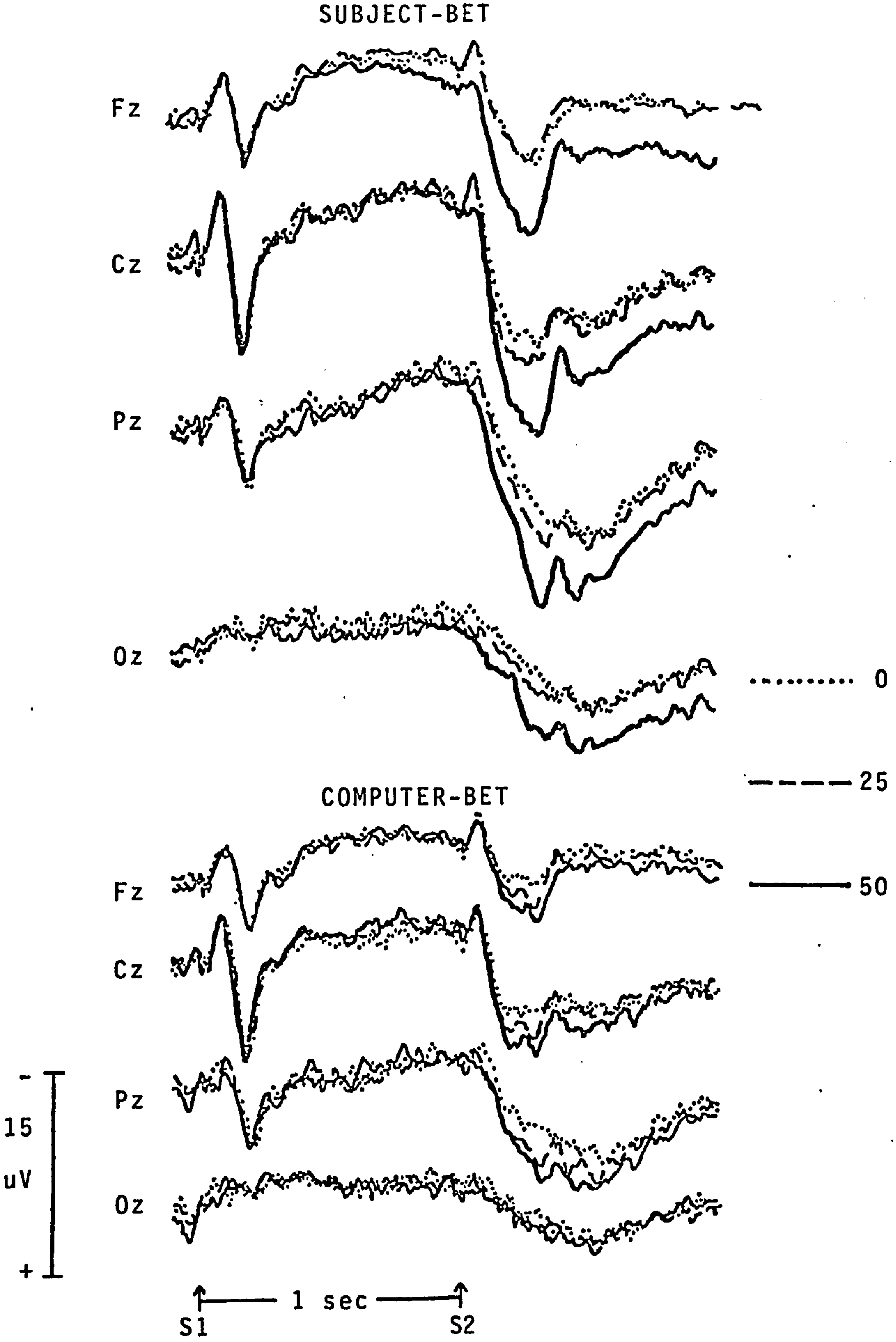


Figure 19. Double Click Evoked Potentials at all Electrode Locations for Individual Bet Values: Grand Means.

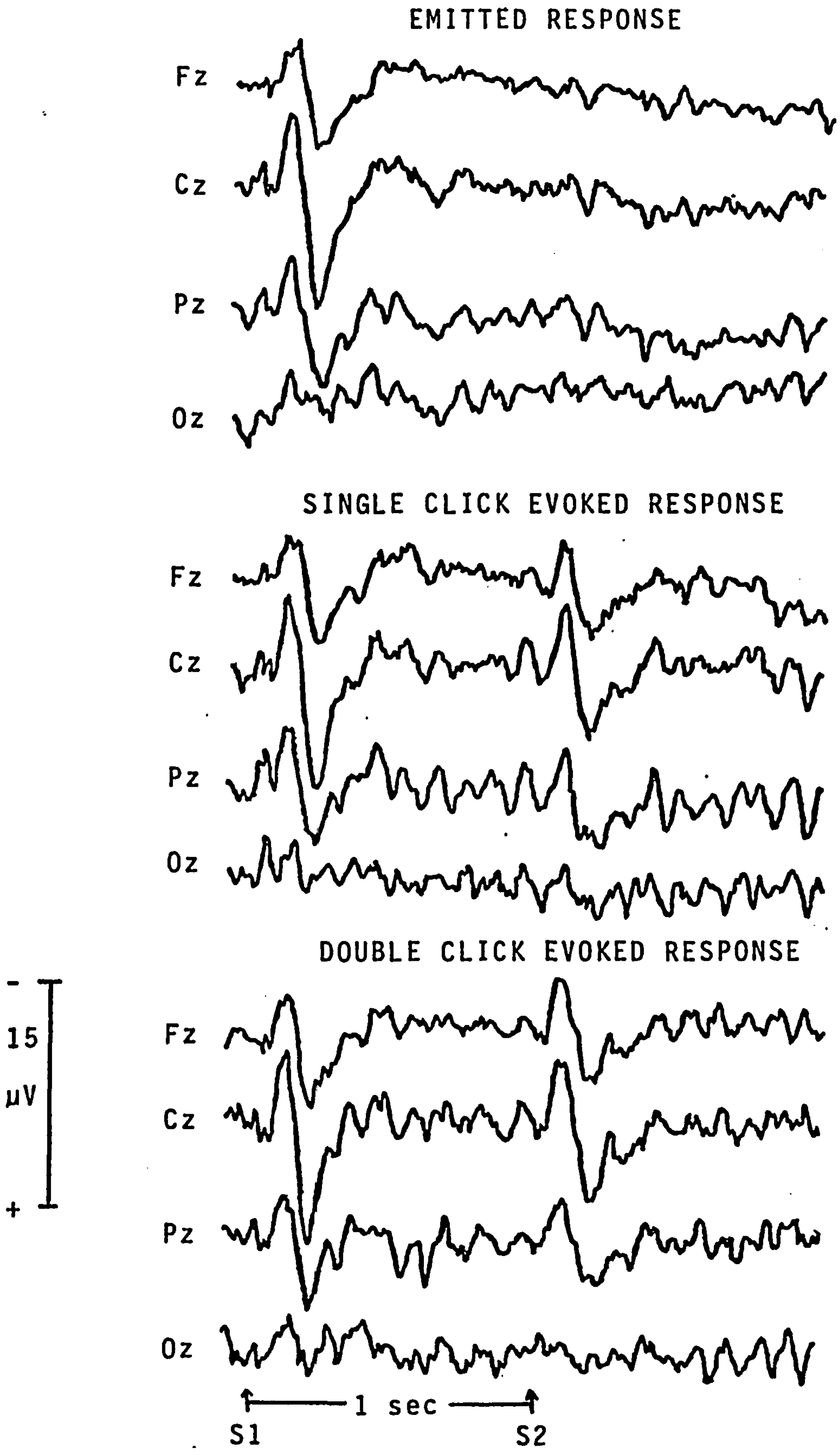


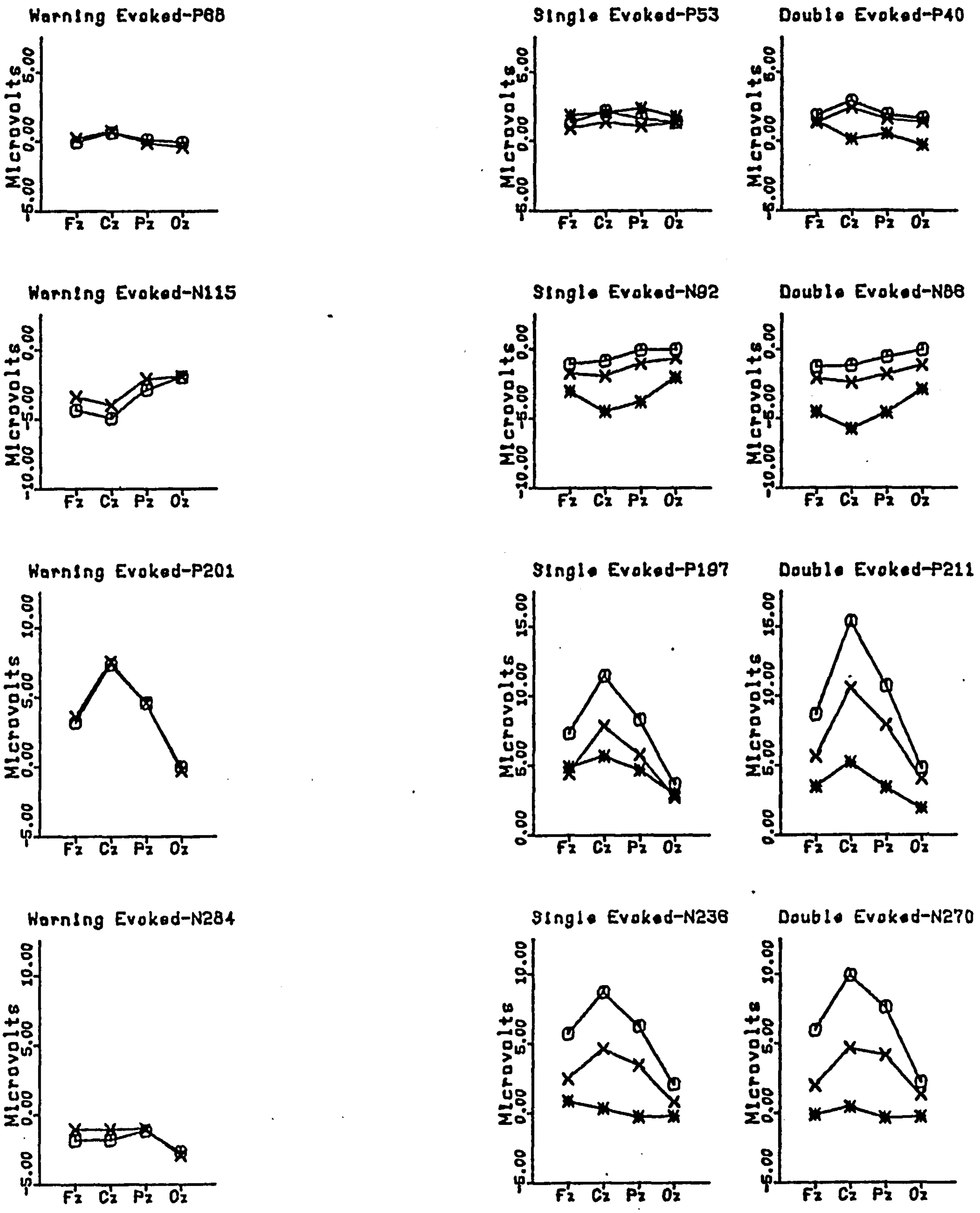
Figure 20. Event-Related Potentials Recorded During the "Certain" Condition at the Vertex Electrode: Grand Means.

potentials are plotted in Figures 21A and 21B. Each point represents the mean across bet value, trial outcome, and across subjects. For the warning click and for the CNV, mean amplitude at each scalp location is plotted separately for components measured under the Subject-Bet condition and under the Computer-Bet condition. In similar plots for the emitted P366 component following the missing stimulus, and for the evoked components following either a single or double click, responses in the "Certain" condition have also been plotted.

Several trends are visible from inspection of Figures 21A and 21B. The vertex location showed maximal positivity for the P200, N250 and P300 components of the informational stimuli, while the N350 and P450 components were more prominent at the parietal location.

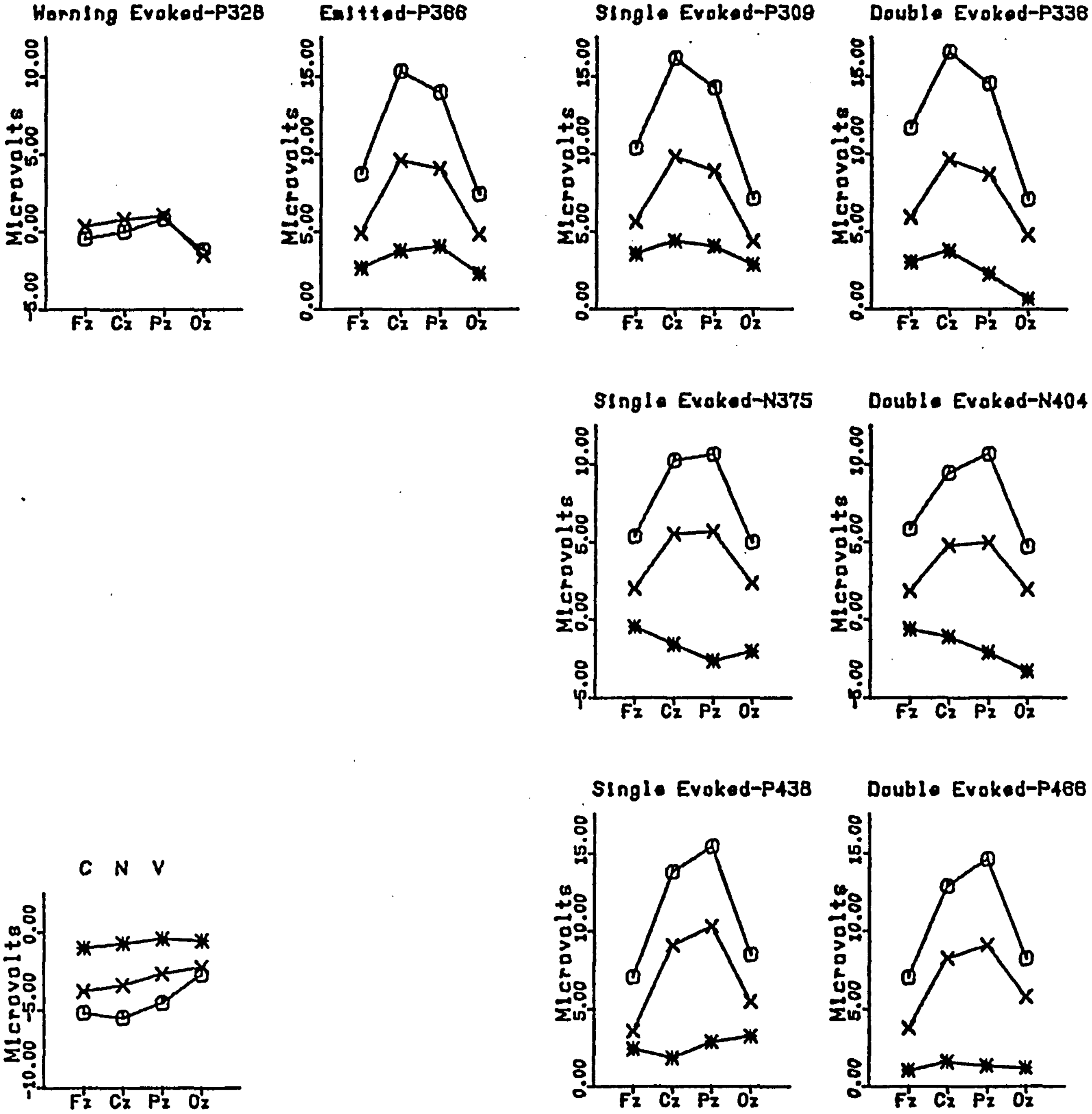
The distribution of components to the warning click was similar to the single and double clicks for the early components, but the later N284 and P328 components were most like the "Certain" responses to the single and double clicks. The emitted and evoked P300 responses had similar amplitudes and distribution.

Statistical evaluation of topographical differences and stimulus differences is included in the following sections. Given the large number of possible findings (22 components tested over five factors and interactions of factors, both for amplitude and latency effects), a .01 level of significance was adopted as criterion for reporting significant effects, using <u>df</u> = (1,7) because of possible correlations among components, since this is a within subject design. For the same



Mean ERP amplitude at all electrode locations for Subject-Bet (⊖), Computer-Bet (X) and Certain (✱) conditions

Figure 21A.



Mean ERP amplitude at all electrode locations for Subject-Bet (Θ), Computer-Bet (X) and Certain (✱) conditions

Figure 21B.

reason, no interactions above the first order were considered. Table 2 summarizes significant findings resulting from analyses of variance of the component amplitudes. Complete ANOVA summary tables, and means and standard deviations across subjects for all components, are presented in the Appendix.

Amplitude of Emitted Potentials

For the emitted potentials produced by a missing stimulus in the S2 position, only one component was evaluated: a prominent positive wave with an average latency of 366 msec. The early negativity reported by other authors (Ford et al., 1976; Klinke et al., 1968; Naatanen & Michie, 1978; Ruchkin & Sutton, 1979a; Simson et al., 1976) was not readily observed in these data. Grand means for individual conditions are plotted for the vertex location in Figure 22. The amplitude of responses to the informational event was measured relative to baseline previous to the time that a click would have occurred. The amplitudes for individual conditions are plotted for the vertex electrode in Figure 23.

Electrode location proved to be a significant main effect for P366 ($p<.01$). P366 was significantly larger at both the vertex and parietal electrodes as compared to both frontal and occipital electrodes (Figures 17 and 21B).

A second main effect was observed for Source of Bet ($p<.01$). The amplitude of the component was greatest when the subject had placed the bet.

	Missing Stimulus	Single Click						
	P366	P53	N93	P197	N236	P309	N375	P438
L=SCALP LOC	.01			.01	.01	.01		.01
B=BET SOURCE	.01			.01	.01	.01	.001	.01
O=OUTCOME				.01	.001	.01		
V=VALUE				.01		.01		
LxV						.01		

	Double Click						
	P40	N86	P211	N270	P336	N404	P466
L=SCALP LOC			.01	.01			
B=BET SOURCE			.01	.001	.01	.01	.001
O=OUTCOME							
V=VALUE							
LxB			.01	.01	.01		

	Warning Click					
	P68	N115	P201	N284	P328	CNV
L=SCALP LOC			.01			
B=BET SOURCE		.01				
D=3-DAY PERIODS						.01
V=VALUE						

Table 2. Summary of significant *p*-values for ERP amplitudes (complete ANOVA tables in Appendix)

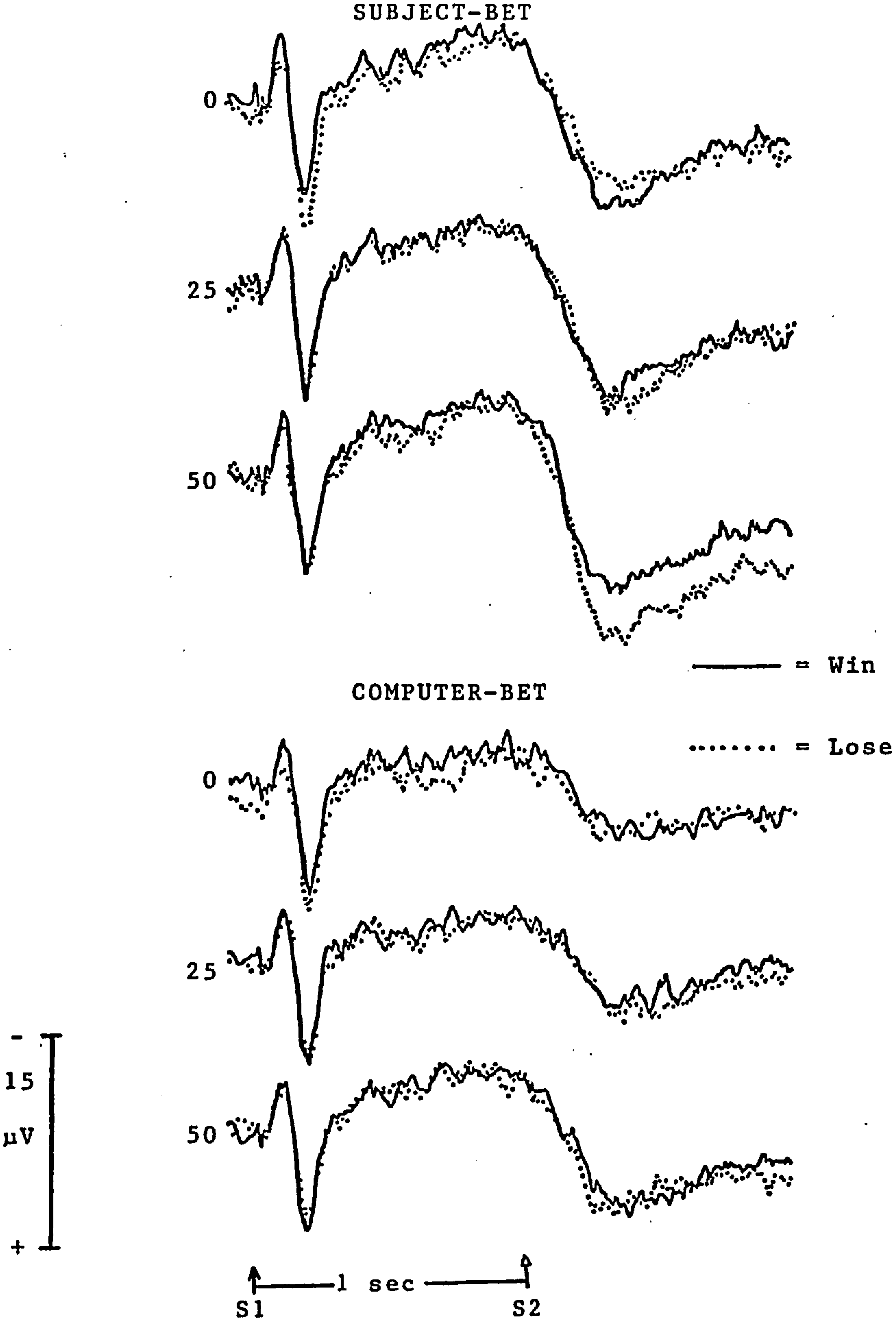
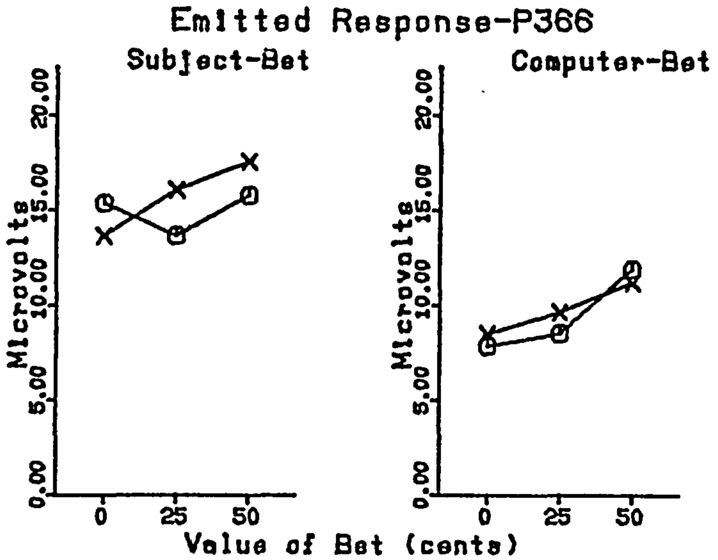


Figure 22. Emitted Potentials for Individual Conditions at the Vertex Electrode: Grand Means.



Cz Amplitude for Win (Ⓞ) and Lose (X) by Value, Across Subjects

Figure 23.

What appears to be an increasing amplitude of P366 as a function of value of bet did not reach the required level of significance (Figure 17). In the Subject-Bet condition, losing 25 or 50 cents tended to result in a larger response amplitude than winning the same amount, although this did not reach statistical significance (Figure 22). For the bet value of zero, the opposite trend was seen, with a larger response in the winning condition. Individually, five of the eight subjects exhibited a larger vertex response following a win of zero than following a win of 25 cents in the Subject-Bet condition. This pattern is similar to the pattern observed for the recovery slope of the pupil following the missing stimulus.

Amplitude of Single Click Evoked Potentials

The single click stimulus configuration, representing either winning or losing, was produced by presentation of one click in the informational position. Amplitudes of seven components were measured from the pre-stimulus baseline for the second click: P53, N93, P197, N236, P309, N375 and P438.

A main effect for Electrode Location (Figures 18, 21A and 21B) was observed for several of the later components. Inspection of the data shows that this is due to maximum positivity at the vertex site for P197 ($p<.01$), N236 ($p<.01$) and P309 ($p<.01$). In contrast, the P438 component ($p<.01$) was largest at the parietal location.

Another major influence appeared as the Source of Bet (Figures 21A and 21B), a main effect for all components except for P53 and N93

(P197, $p<.01$; N236, $p<.01$; P309, $p<.01$; N375, $p<.001$; P438, $p<.01$). Quite clearly, the effect of allowing the subject to place each bet caused a major shift toward positivity for all components (including negative components) as compared to imposing the value of the bet from the computer-generated list. Grand means for the vertex location appear in Figure 24. Observation of the P197 component is obscured by the large P309 component. Component amplitudes at vertex are plotted by condition in Figure 25.

Winning responses resulted in greater positivity than losing responses, a main effect observed for P197 ($p<.01$), for N236 ($p<.001$) and for P309 ($p<.01$) (Figure 24). A main effect for Value of the bet was present for the P197 ($p<.01$), P309 ($p<.01$) and P438 ($p<.01$) components, with larger amplitude components for the larger bet values. Significantly greater amplitudes were observed for both 25 and 50 cents over zero for P197, 50 cents significantly greater than both zero and 25 cents for P309, and 50 cents significantly greater than zero for P438. Differences in the effects of value were consistent under both the Subject-Bet and the Computer-Bet conditions.

A significant interaction between Location X Value for P309 ($p<.01$) reflected greater differences for different bet values at Cz and Pz than at Oz. No other interactions were significant.

Amplitude of Double Click Evoked Potentials

The double click stimulus pattern consisted of two clicks, separated by ten msec, in the informational position. The double click stimulus configuration was detected by all subjects as distinctly

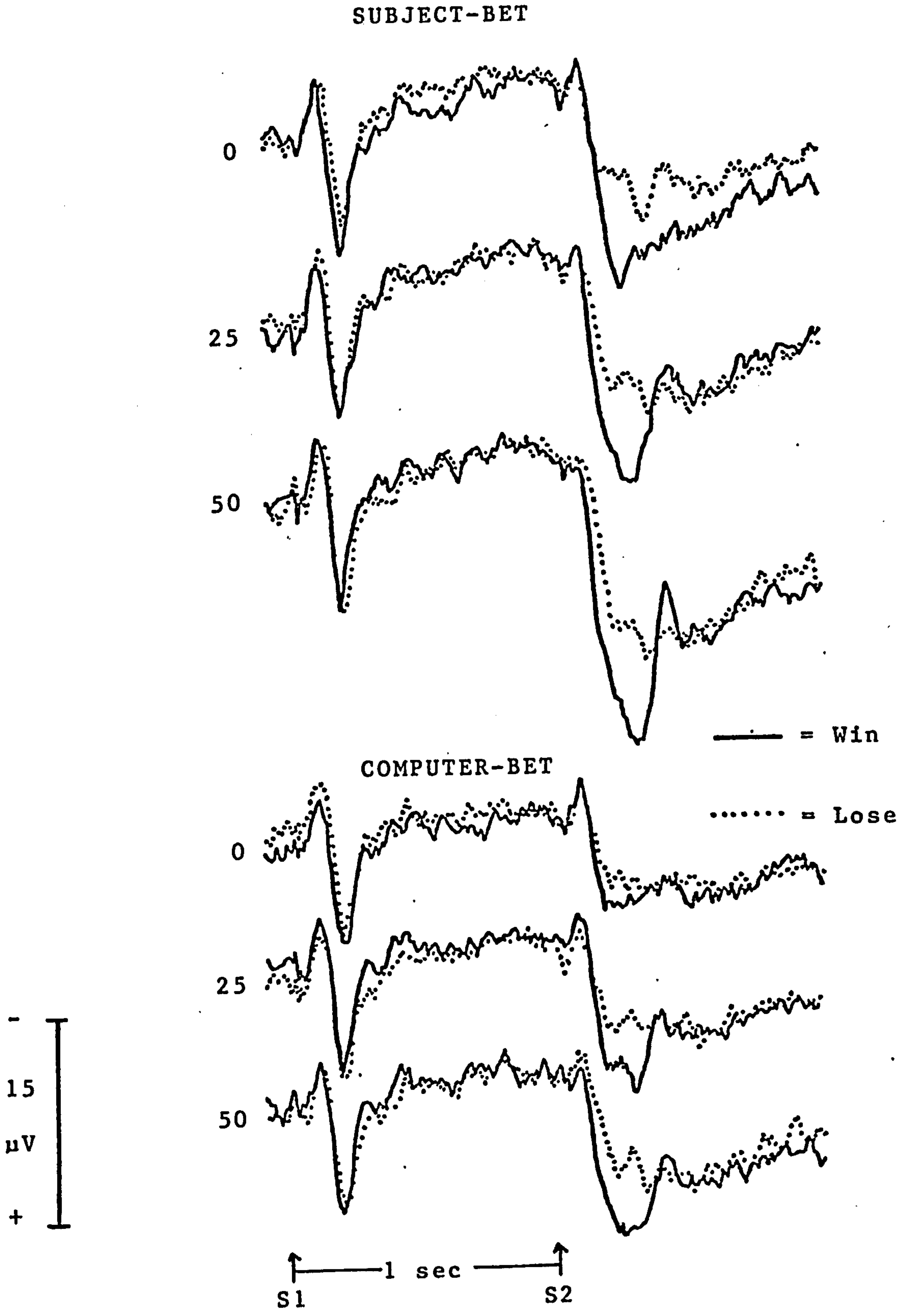
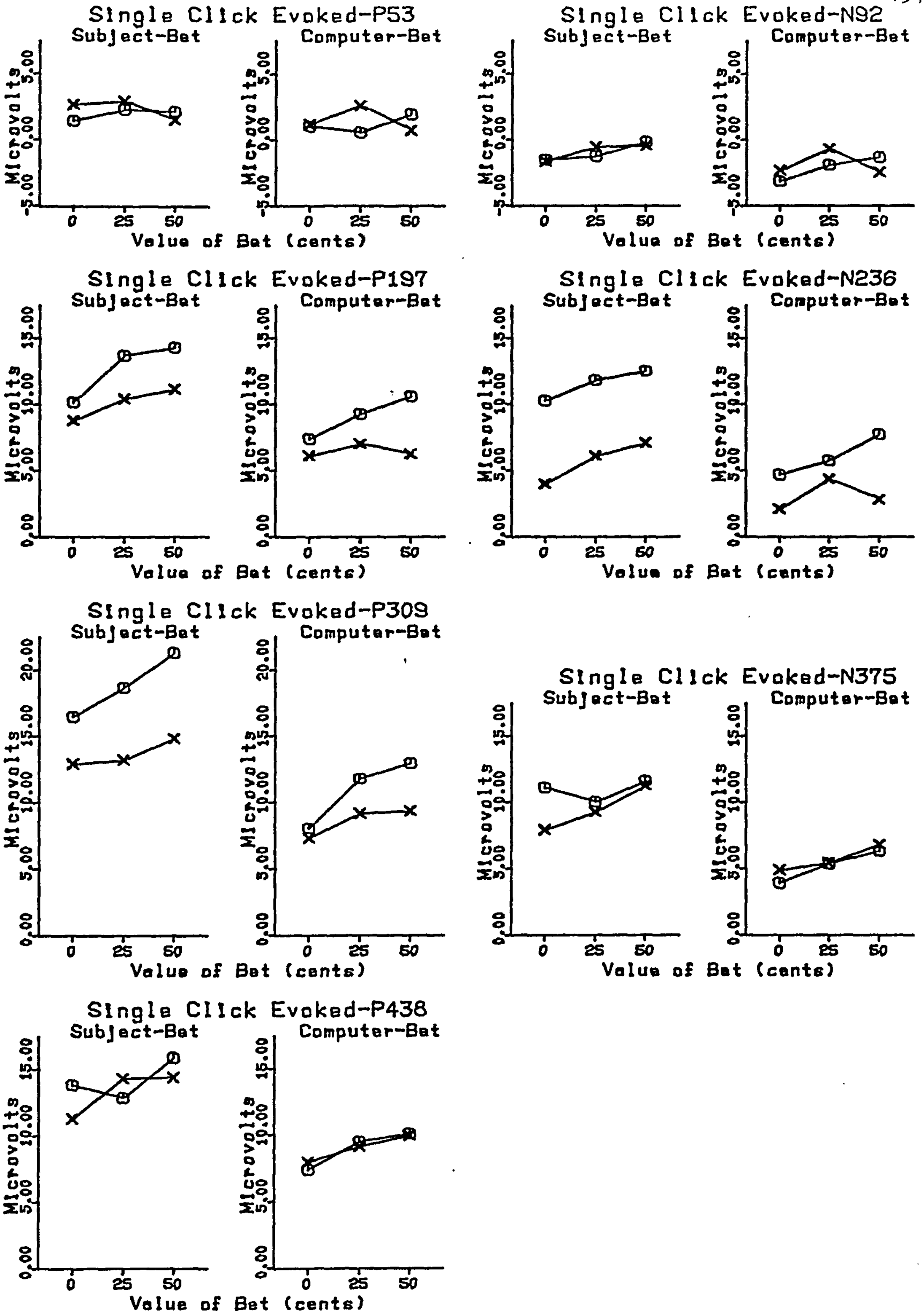


Figure 24. Single Click Evoked Potentials for Individual Conditions at the Vertex Electrode: Grand Means.



Cz Amplitude for Win (Θ) and Lose (X) by Value, Across Subjects

Figure 25.

different from the single click stimulus. Since the double click represented cancelling of the bet, trial outcome was zero whenever the double click was presented.

Electrode Location (Figures 19, 21A and 21B) was a main effect only for P211 ($p<.01$), with the largest amplitude response at vertex. Grand means at vertex are presented in Figure 26. Component amplitudes for all conditions are plotted across subjects for the vertex electrode in Figure 27, including a comparison of sessions for which either the missing stimulus or single click represented the winning outcome.

A main effect of Source of Bet was observed for P211 ($p<.01$), N270 (($p<.001$), P336 ($p<.01$), N404 ($p<.01$) and P466 ($p<.001$). As was true for the emitted and single click evoked responses representing winning and losing, the amplitude of the component in the "cancelled" condition was always more positive in the Subject-Bet condition than in the Computer-Bet condition.

Significant interactions between Location X Source of Bet were observed for P211 ($p<.01$), N270 ($p<.01$), and P336 ($p<.01$). The differences in amplitude between the Subject-Bet and Computer-Bet conditions at Fz, Cz, and Pz were all significantly greater than at Oz; for P211, the difference at Cz was also greater than at both Fz and Pz, and the difference at Cz was greater than Pz for N270 (Figures 19, 21A and 21b).

As value of the bet increased, greater positivity for all components with a latency longer than 200 msec was observed, although the trends were not significant. No significant differences were observed for the cancelled condition between sessions for which the

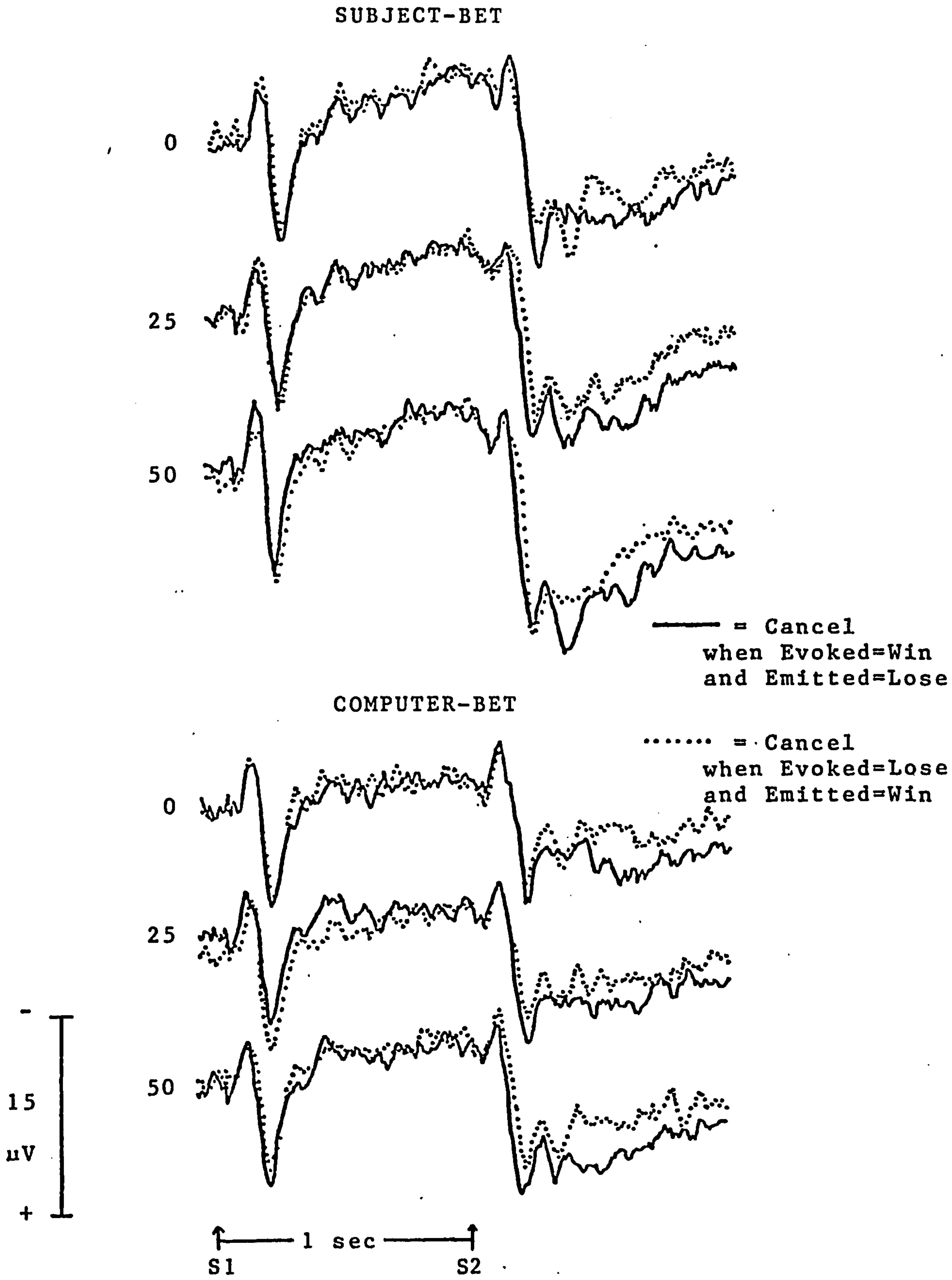
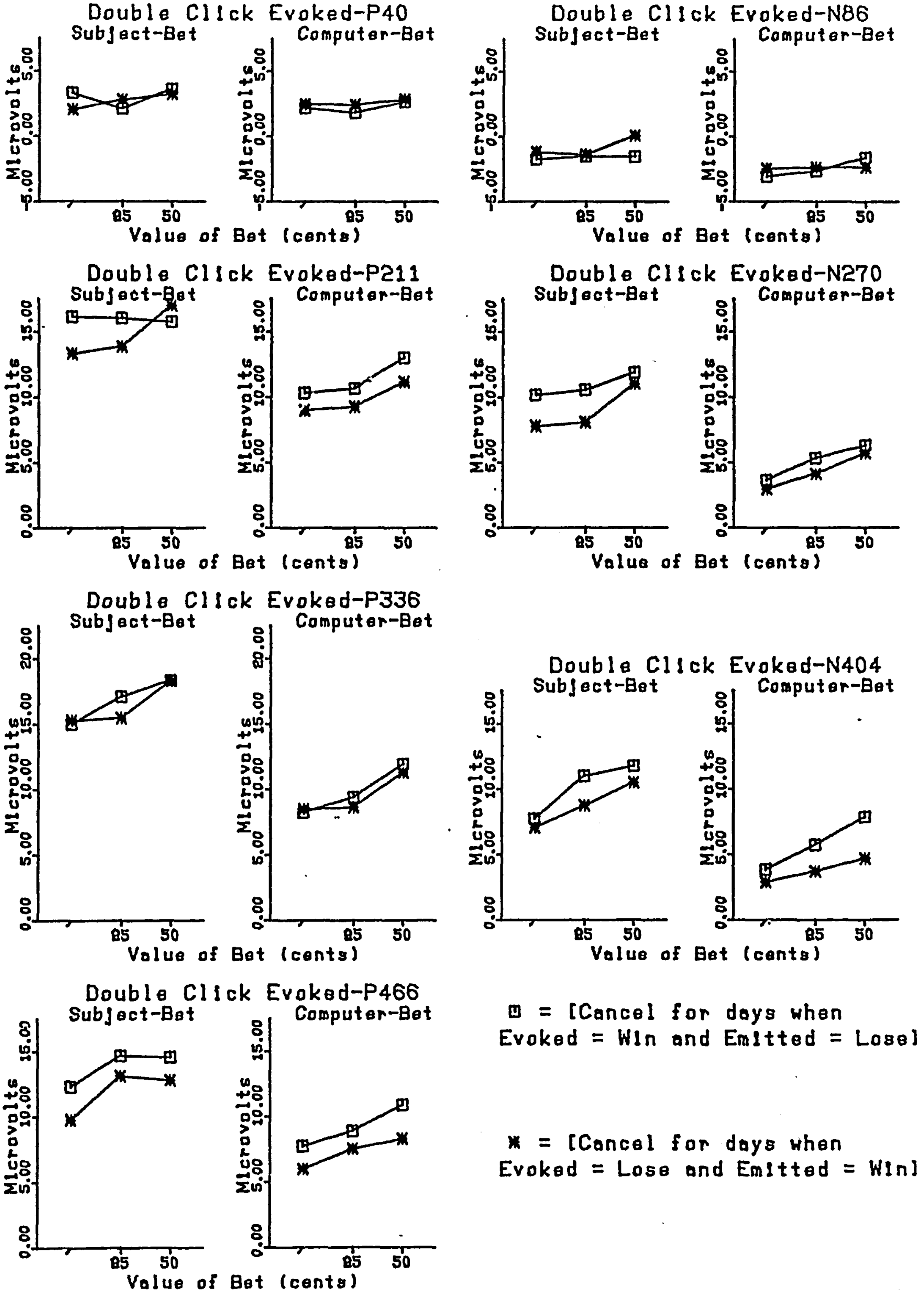


Figure 26. Double Click Evoked Potentials for Individual Conditions at the Vertex Electrode: Grand Means.



Cz Amplitude for Cancelled Trials, Across Subjects

Figure 27.

emitted response vs. the single click evoked response represented winning (Figure 27).

Comparison of Event-Related Potentials in the Certain vs. Uncertain Conditions

The amplitudes of the components of the emitted "Certain," single click "Certain " and double click "Certain" responses were compared with the unweighted average amplitude across each of the six conditions of the Subject-Bet condition and the Computer-Bet condition (Figures 20, 21A and 21B). Significant main effects across conditions were observed for the emitted P366 response following a missing stimulus ($F = 18.29$, $p<.01$), for N236 ($F = 15.78$, $p<.01$), P309 ($F = 19.13$, $p<.01$), N375 ($F = 32.45$, $p<.001$), and P438 ($F = 26.32$, $p<.01$) components evoked by a single click, and for the N86 ($F = 13.26$, $p<.01$), P211 ($F = 27.25$, $p<.01$), N270 ($F = 20.92$, $p<.01$), P336 ($F = 14.24$, p<.01), N404 ($F = 14.42$, $p<.01$) and P466 ($F = 19.06$, $p<.01$) components following a double click.

In all cases, the greatest positivity was observed in the Subject-Bet condition. In the Computer-Bet condition, all components were more positive than in the Certain condition.

Amplitude of Contingent Negative Variation

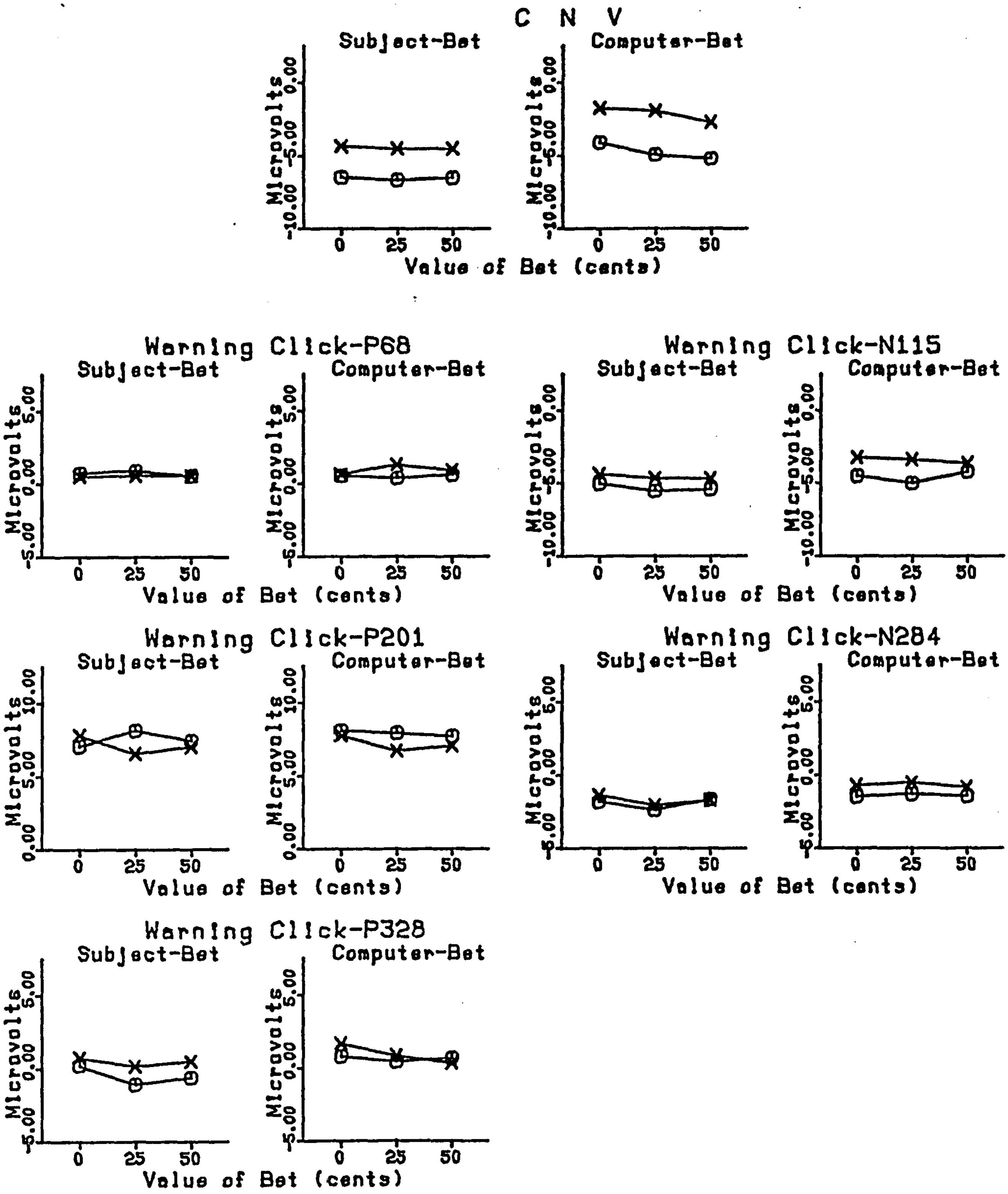
The amplitude of the contingent negative variation (CNV) was measured between the baseline preceding the warning stimulus to the average of a continuous set of ten most negative points that preceded the informational event. All trials having the same bet source (Subject-Bet or Computer-Bet) and same bet value were averaged together, regardless of the second stimulus, for determination of both CNV and components of the evoked response to the warning click. Separate sets of averages combined across stimuli were computed for the first three days of testing and contrasted with the second three days of testing for evaluation of possible change in CNV amplitude. CNV amplitude is plotted for the vertex response in Figure 28. The average latency of CNV to maximum amplitude was 830 msec.

A main effect was observed for reduction of CNV amplitude between the first three test days and the second three test days ($\underline{p}<.01$), with a smaller amplitude CNV recorded during the second three test days (Figure 28).

A non-significant trend was observed in which bets placed by the subject produced a more negative CNV than bets imposed by the computer. A comparison of CNV differences in the two betting conditions can be seen in Figures 17 through 19.

No other significant differences were found for the CNV. The cortical distribution showed similar large CNVs at both frontal and vertex electrodes, with a smaller amplitude at the parietal placement and the smallest CNV recorded at the occipital location (Figure 21B). While there were no significant differences in the distribution, the vertex CNV was the largest in amplitude for six of the subjects, while the other two subjects had a more prominent frontal CNV.

Although the possible effect of differential arousal to different bet amounts was originally anticipated, no effects of value appeared,



Cz Amplitude for Days 2-4 (⊖) and Days 5-7 (×) by Value, Across Subjects

Figure 28.

either in the statistical analysis or in the individual subjects' responses.

The CNV tended to be most negative in the Subject-Bet condition, and more negative in the Computer-Bet condition than in the Certain condition (Figures 20 and 21B), although these differences were not significant.

Amplitude of Warning Click Evoked Potential

As in the case of the CNV, the evoked response to the warning click was evaluated without regard to the informational event. Electrode location was a significant main effect only for the P201 component ($p<.01$). The vertex response for P201 was significantly more positive than at both frontal and occipital locations, and parietal was also significantly positive compared to the occipital location (Figure 21A).

For the N115 component, a significant main effect was indicated for Source of Bet ($p<.01$), with greater negativity under the Subject-Bet condition. No other significant main effects or interactions were observed for the warning click. The response to the warning click is visible in Figures 17 through 19.

Analysis of Latencies of ERP Components

The latencies of the ERP components were not affected in any consistent manner by the experimental variables. No significant findings were obtained for the latency of the emitted P366 component, although several subjects exhibited a tendency for a shorter latency for the winning responses than for the losing responses.

No pattern of latency effects was apparent in the components of the single click evoked response, although P309 for winning responses tended to be approximately 20 msec faster than for losing responses, similar to the trend for the emitted responses. No significant latency effects or noteworthy trends were observed for the double click or warning click components, nor for the point of resolution of the contingent negative variation.

Comparison of Single Click and Double Click Evoked Potentials

For comparison of amplitude and latency differences, type of stimulus (single or double click) was entered into the analysis of variance as an additional factor. The only component for which a main effect was found due to the difference between the stimuli was the second positive component: the P211 of the double click was significantly more positive than the P197 of the single click response ($F = 10.30$, $p < .05$) (Figure 21A). This difference is attributed to the difference in total stimulus energy and distribution (two clicks over 11 msec for the double clicks vs. one click for the single).

A significant interaction between Stimulus X "Outcome" was observed for the single-N236 and double-N270 ($F = 12.47$, $p < .01$) components (Figures 25 and 27). The source of this interaction was the clear differentiation between winning and losing outcomes observed for the single click component, but the lack of differential activity for the double click evoked response between sessions in which the single click represented winning from those in which the single click represented losing. No other amplitude differences were detected between the single and double click evoked potentials. No significant latency differences were observed between components of the single and double click evoked responses.

Comparison of Emitted vs. Single Click Evoked Potentials

The late positive P366 wave of the emitted potential was contrasted with the P309 component of the single click evoked potential. Overall, the amplitude of the emitted P366 component was not significantly different from the evoked P309 component ($\underline{F}$ = .23, n.s., $\underline{df}$ = 1,7). This was an unexpected finding, in that virtually all previous reports of P300 have consistently described a smaller amplitude for the emitted component as compared to the evoked component. It is especially notable that for the days during which the missing stimulus represented the winning condition and single clicks represented losing, emitted responses appeared that were larger than the evoked responses recorded at the same time for a given bet value under the same betting conditions (either Subject-Bet or Computer-Bet).

The number of instances for which a larger emitted P300 than evoked P300 response occurred was tabulated for all subjects. For this purpose, only the measurements obtained under the Subject-Bet condition were evaluated, since this condition seemed more indicative of maximum subject response. The comparison was performed separately for each bet value; 24 such comparisons were performed for each of the two, three-day sessions (three bet values X eight subjects). For sessions in which the single click represented winning, the evoked response was larger than the emitted response in 21 comparisons, equivalent in one, and smaller than the emitted response in two cases.

In the complementary sessions, when the missing stimulus represented winning, the emitted response was equal in amplitude to the evoked response for the same bet value in three cases, and smaller than the evoked response in eight instances. In the remaining thirteen cases, the emitted response accompanying a winning outcome was larger than the evoked response representing the same bet value (zero, 25 or 50 cents) recorded during the same blocks of trials.

The major difference between the emitted and evoked responses was that there was a reversal in P300 amplitude as a function of winning vs. losing when emitted and evoked P300s were considered separately (Figures 23 and 25). It seemed rational only to compare emitted with emitted potentials (win vs. lose) and evoked with evoked potentials (win vs. lose). However, the reversal in findings suggested that one might compare win vs. lose across evoked vs. emitted P300s. The justification for making such an unorthodox comparison is strengthened by the fact that no main effect of emitted vs. evoked P300s was found. The analysis yielded a significant main effect of winning vs. losing ($\underline{F} = 19.19$, $p < .01$). P300 was larger for winning than for losing responses. A confounding effect of test days was possible, since to compare "evoked win" with "evoked lose" (or "emitted win" with "emitted lose") one was always comparing trials recorded on different days.

The latency of P309 following the single click was significantly earlier ($\underline{F} = 24.57$, $\underline{p} < .01$) than the P366 component emitted in the missing stimulus event.

Analysis of Standard Deviation

It was considered possible that amplitude differences among the experimental conditions were produced by greater temporal variability for components of lower amplitude. The value of the standard deviation at the latency of P300 was entered into an analysis of variance, as represented by the intra-individual standard deviation across trials in each condition for each subject. No significant differences in variability were observed, nor were any trends apparent. Thus, the differences in amplitude of P300 across experimental conditions were not due to temporal jitter.

Correlations Between Pupillary Responses and Event-Related Potential Components

During inspection of individual waveforms, it appeared that subjects who were characterized by a large pupillary dilation tended to have large event-related potential amplitudes. To explore this apparent trend, the following measurements were made across the six outcome conditions, by subject: initial pupillary diameter, amplitude of dilation across stimuli and for individual stimuli, and amplitude of recovery slope of the pupil for individual stimuli, and the CNV, emitted P300, and evoked P200 and P300 components recorded at the vertex electrode.

Pupillary measurements were correlated across subjects with ERP measurements (Table 3). The amplitude of pupillary dilation was positively correlated with the amplitude of late components of the ERP.

	C N V	Emitted P197	Double P211	Emitted P366	Single P309	Double P336
Subject Bet						
Init. Diam.	.24	.52	-.74*	-.29	-.39	-.30
Peak Dilation						
All. Stim.	-.18	.73*	.68*	.80**	.66	.68*
Emitted	-.20	.70*	.67*	.81**	.67*	.69*
Single	-.16	.71*	.66	.76*	.63	.66
Double	-.17	.77*	.71*	.80**	.65	.67*
Recovery Slope						
Emitted	.06	.41	.38	.46	.20	.24
Single	.13	.37	.29	.43	.16	.19
Double	.16	.52	.36	.54	.26	.29
Computer Bet						
Init. Diam.	.47	-.45	-.71*	-.53	-.54	-.45
Peak Dilation						
All Stim.	-.60	.49	.76*	.86**	.74*	.76*
Emitted	-.60	.11	.39	.63	.52	.63
Single	-.70*	.50	.82**	.81**	.76*	.77*
Double	-.66	.57	.86**	.81**	.84**	.86**
Recovery Slope						
Emitted	.35	.32	-.12	.16	-.13	-.20
Single	.28	.18	-.14	.09	-.17	-.20
Double	.25	.27	-.10	.10	-.14	-.21

(* = $p < .05$; ** = $p < .01$; df = 7)

Table 3. Correlations among Mean Pupillary and ERP Amplitudes Across Subjects.

The values of the correlations were unusually high, both in the Subject-Bet and Computer-Bet condition. Pupillary dilation showed only a weak negative correlation with CNV in the Subject-Bet condition. Similar correlations were not obtained between the recovery slope measure and ERP components.

Although the overall amplitude correlations across individuals were significant, the correlations provided no information to indicate whether, for a single individual, the amplitude of one response in any condition provided a prediction of a response in the other physiological system during the same trials. For individual subjects, the amplitude of P300 at vertex was correlated separately with peak dilation and the recovery slope measure using paired data from each of the six outcome conditions (Table 4). This was performed separately for the two betting conditions and for each stimulus event. There was no consistent trend across subjects to indicate that an accurate prediction could be made from pupillary to ERP response amplitude, or from the amplitude of ERP components to pupillary amplitude, for any given condition.

	P300 and Peak Dilation			P300 and Recovery Slope		
Stimulus:	Emitted	Single	Double	Emitted	Single	Double
Subject Bet						
S1	-.76	.47	.85	.07	-.77	.08
S2	.16	.88	.36	.64	-.06	.19
S3	.68	.07	.68	-.21	-.34	.21
S4	.96	.64	.48	.70	.60	.07
S5	.45	-.18	.62	.44	-.17	.83
S6	.97	.93	.93	.91	.84	.79
S7	.53	-.14	-.43	-.20	-.10	.35
S8	.83	.12	-.38	.58	-.25	-.50
Computer Bet						
S1	.26	.83	.00	.62	-.93	.07
S2	.51	-.20	.30	.40	.12	.76
S3	.01	.31	-.03	-.19	-.33	-.12
S4	.95	.87	.65	.96	.86	.53
S5	.94	.78	.64	.58	.72	.54
S6	.78	.82	-.40	.29	.76	.41
S7	.08	.00	.17	.30	.29	.76
S8	.33	.42	-.51	.48	-.20	-.51

Table 4. Within-subject correlations for P300 with Pupillary Dilation and Recovery Slope Across Conditions.

Chapter VII

Results of Eye Artifact Analysis, Betting Strategies, and Time Training Procedure

Evaluation of Eye Movement Artifacts

The amplitude of the artifact recorded from the electrode placed under the right eye, referenced to the linked ears, was recorded each day from each subject for vertical movements of 5, 10, 15 and 20 degrees of excursion. All subjects exhibited a linear pattern of approximately eight uV/degree of eye movement.

For each experimental condition, the amplitude of the largest eye movement artifact occurring with a latency of ± 20 msec of the emitted or evoked P300 component for that condition was measured relative to a pre-stimulus baseline. These data were entered into an analysis of variance including experimental conditions and stimuli. The amplitude of this artifact, averaging from two to four uV, is plotted in Figure 29 for each of the stimuli according to experimental condition. No significant factors were revealed in the analysis of variance performed on these data. Furthermore, the *F*-ratios obtained were smaller than for any of the cortical electrodes, suggesting that any trends were more likely to be measurement of cortical activity at the eye electrode rather than potentials generated at the eye that were reflected as major factors in the cortical electrodes. The waveforms of the eye artifact, averaged across stimulus events, did indicate a trend for a slightly greater amplitude response with increasing value, and were

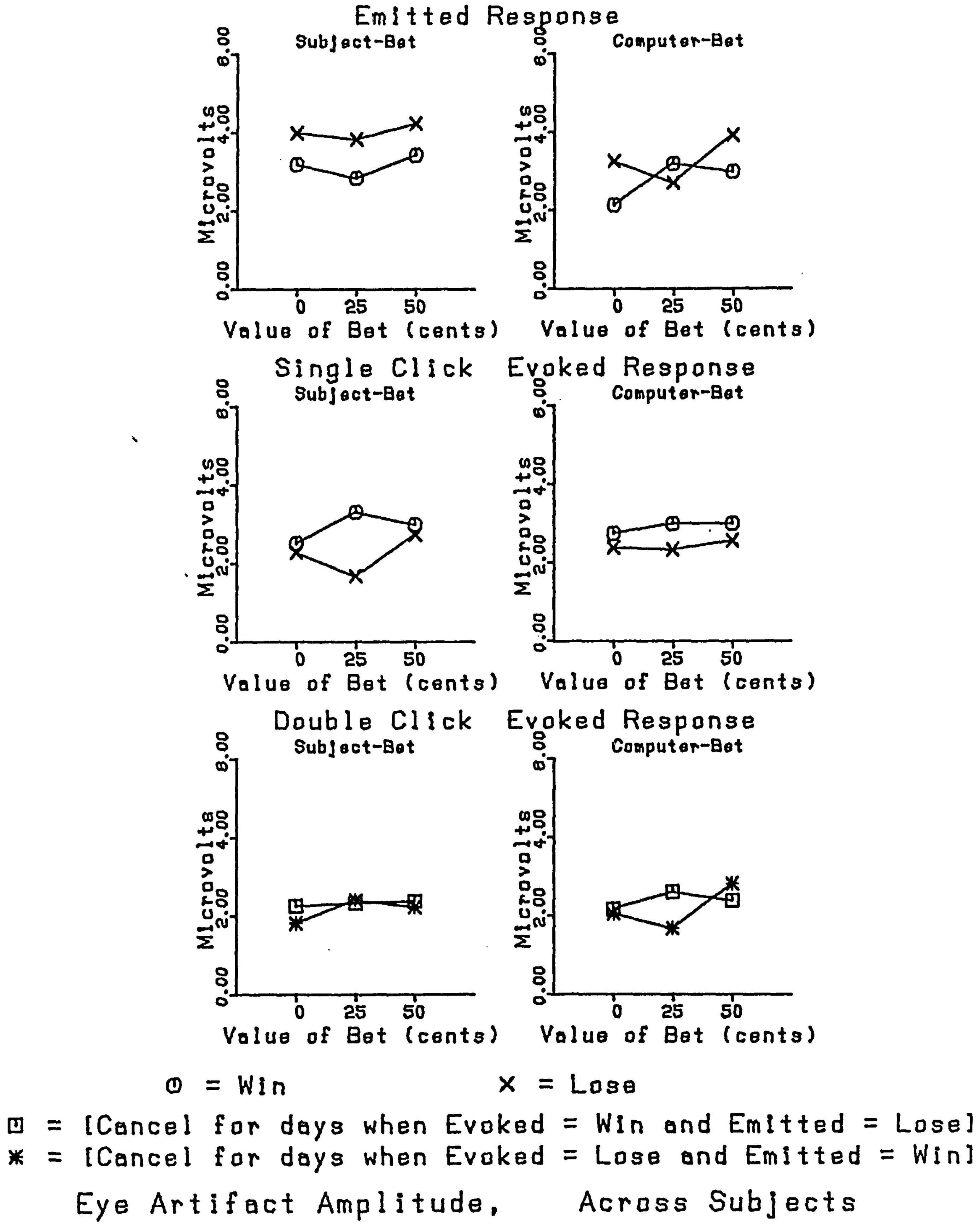

Emitted Response
Subject-Bet
Computer-Bet
Single Click Evoked Response
Subject-Bet
Computer-Bet
Double Click Evoked Response
Subject-Bet
Computer-Bet
Microvolts
0.00
2.00
4.00
6.00
0
25
50
Value of Bet (cents)
ⓞ = Win
X = Lose
□ = [Cancel for days when Evoked = Win and Emitted = Lose]
✱ = [Cancel for days when Evoked = Lose and Emitted = Win]
Eye Artifact Amplitude, Across Subjects

Figure 29.

similar in appearance to the slow components of the occipital recordings in this respect (although inverted in polarity).

To provide an evaluation of the possible contribution of undetected eye artifacts to the cortical responses, seven of the eight subjects performed large eye movements in the vertical and horizontal planes. The amplitude of the artifact at each cortical electrode was expressed as a percentage of the amplitude of the artifact recorded at the eye. Vertical eye movements consistently produced a larger response at the cortical electrodes.

The average per cent detectable artifact ± standard deviation for vertical eye movements was 37.1 ± 12.5% of total artifact as recorded at Fz, 22.9 ± 10.0% at Cz, 14.4 ± 10.8% at Pz and 5.4 ± 6.0% at Oz. For horizontal eye movements, the percentages detected were 15.4 ± 8.0% at Fz, 9.9 ± 8.8% at Cz, 7.1 ± 7.0% at Pz and 4.7 ± 6.6% at Oz. For even a one microvolt difference in amplitude of the artifact to have been detected at vertex, for example, a minimum potential difference between conditions of at least five uV would have been required at the eye.

Borda and Hablitz (1973) reported few vertical eye movements of greater than one degree using a task that required constant fixation. Such movements contributed no more than two to three uV to the vertex recordings in a CNV task, and virtually all of these artifacts were observed during blinks. In the present study, all trials on which blinks occurred, as detected by the eye channel and the pupillograph output, were eliminated. Blinks contribute a smaller percentage of artifact to the EEG than do ocular rotations which produce a similar EOG amplitude (Corby & Kopell, 1972) so that non-blink artifacts must also be ruled out. The average eye artifact obtained in the current study was equivalent to approximately 1/2 degree of rotation or less. In contrast, the criterion for automatic suppression of artifacts used by Papakostopoulos, Winter and Newton (1973) was up to three degrees, but they reported no significant effect of eye movements upon the vertex CNV.

The placement of the eye artifact electrode on the infra-orbital canthus, as employed here, resulted in a detection of 23% of the vertical artifact at vertex, higher than the values of 13.9% and 10-15% reported, respectively, by Corby and Kopell (1972) and by Girton and Kamiya (1973). Since the eye artifact averaged two to four uV, even in the worst case, the contribution of the eye artifact would be no more than approximately 0.9 uV (23% of four uV). Since significant differences between conditions ranged from three to eight uV at vertex, it is not possible that eye artifacts were responsible for these differences in P300 amplitude.

Analysis of Subjects' Betting Strategies

The inclusion of the double click stimulus, representing a cancelled bet, was designed to shift the strategy of the subjects' betting behaviors away from betting zero on 50% of the trials. Since only one-third of all trials were losses in these experiments, it was to the subject's advantage to bet 25 or 50 cents more often than in the preliminary studies. For each three day period, (Days 2-4 and Days 5-7) the actual number of bets placed is presented for each subject in Table 5 (for subjects S3 and S8, only 580 betting trials were completed

Bet Value:	Days 2 - 4			Days 5 - 7		
	0	25	50	0	25	50
Subject						
S1	162	289	169	187	224	209
S2	182	114	324	184	108	328
S3	157	239	224	165	193	222
S4	205	167	248	222	158	240
S5	175	174	271	170	146	304
S6	274	213	133	214	222	184
S7	225	220	175	228	180	212
S8	225	177	218	185	206	189
Average Percent:	32.35	32.12	35.52	31.84	29.53	38.63

	0	25	50
Average Percent, Across Sessions:	32.35	30.79	37.08

Table 5. Bet distribution for Days 2-4 and Days 5-7, Subject-Bet condition.

during the second three days). Although all subjects did bet zero less than 50% of the time, indicating that the effect of the "cancel" condition had been to change overall strategies, bets were generally not distributed equally among the three bet values.

Subject 1 placed more 25 cent bets (47%) during the first three days, then dropped closer to chance distribution for the remaining three days. Subjects 2 and 5 placed bets of 50 cents near or over 50% of the time for both the first and second sets of days. Subject 3 bet zero only 25% for the first and 28% for the second block of three days. On both sets of days, S4 tended to bet 25 cents less often than zero and 50 cents more often than zero. In comparison, S6 was conservative for the first three days, betting zero 44% of the time, but his bets were distributed closer to chance levels for the second three days. Subject 7 tended to bet 50 cents fewer times (28%) for the first three days only. Subject 8 tended to place bets of 25 cents less often (28%) only for the first three days.

In general, all subjects seemed to be employing some strategy other than chance distribution for at least the first three days. An analysis of variance of the proportions of bets across subjects did not indicate any significant differences among the three bet values ($\underline{F}$ = 1.14, n.s., $\underline{df}$=1,7), although there was a trend to bet 50 cents more than one-third and 25 cents less than one-third of the time. This trend was more pronounced during the second three days, resulting in a significant main effect between the two sessions ($\underline{F}$ = 5.65, $\underline{p}$ = .049, $\underline{df}$=1,7).

The interaction of the betting outcomes with stimulus presentation was evaluated by separate Chi-square tests comparing the expected distribution of the subjects' bets across the three different stimulus outcomes (based on the subjects' own bet distributions) with the observed distributions. Over all subjects, only a single significant distribution was observed: for the first three-day period, the distribution of bet values across stimuli was non-random for S8 (_Chi-square_ = 13.47, $p<.01$, _df_=4). An especially large number of zero bets coincided with presentation of the double click stimulus for this period. All other comparisons were non-significant. That is, regardless of each subject's overall betting strategy (with the exception noted), the bet values occurred randomly with respect to the stimuli, and, thus, with respect to trial outcome. Therefore, no explanation of differences between winning and losing outcomes can be attributed to differences in the objective probability of trial outcome, although this may not necessarily have coincided with each subject's subjective evaluation of trial outcome.

Results of Time Training Procedure

For individual days, each subject's performance during the time training procedure was assessed separately for discrimination of intervals either shorter or longer than one second. The delta t's for the last four blocks of each condition were averaged to yield a discrimination measure. Mean delta t's and standard deviations at the end of the training period were 83 ± 37 msec for the longer interval and 96 ± 22 msec for the shorter interval on the day of training (Day 1) for which the most complete data were available (i.e., subjects could discriminate either a 1083 msec or 904 msec interclick interval as different from 1000 msec). Absolute differences between the longer vs. shorter intervals were not significant ($\underline{t}$=1.24; all $\underline{t}$-tests reported in this section represent two-tailed tests with 7 degrees of freedom). The average discrimination intervals across Days 2 through 7 were 82 ± 21 msec for the longer interval and 93 ± 9 msec for the shorter interval, which also did not differ significantly in absolute value ($\underline{t}$=1.74). There was no significant difference between delta t's recorded on Day 1 compared to average delta t's for subsequent days ($\underline{t}$=2.62).

To evaluate the improvement in performance during the training procedure, the final delta t's recorded on Day 1 were compared to the poorest performance observed during the same session. Maximum delta t's on Day 1 averaged 116 ± 27 msec for the longer interval and 109 ± 32 msec for the shorter interval, which did not significantly differ ($\underline{t}$=.89). By the end of the training period, delta t's showed a significant improvement of 23 msec between poorest performance and final performance ($\underline{t}$=5.1, $\underline{p}$<.005).

The time training procedure resulted in an increase in accuracy for all but one subject (S6) in discriminating the interval at which the missing stimulus would be expected, as compared to what the performance would have been without the institution of the training procedure. It should be cautioned, though, that the implementation of the forced-choice procedure may have been no more effective than the accompanying repetition of between 60 and 90 interclick intervals that occurred during the actual time training procedure. It could be that mere repetition without decision-making would have been sufficient.

It is also tempting to look at the generally decreasing standard deviations after training as reflecting decreased variability in the distribution of delta t's, but as these data are averages across subjects, they represent only decreasing variability among the subjects. Although it would have been advantageous to obtain estimates of time discrimination at the end of each of the experimental sessions, it was not practical to do so.

CHAPTER VIII

Discussion

The results of this experiment demonstrated a major impact of motivational variables on the pupil and event-related potentials. Virtually every aspect of these responses that has been related to information processing was demonstrated to be sensitive to the experimental manipulations used. The pupillary recovery slope and P300 components of the event-related potential were the most reflective measures of the independent variables. The contingent negative variation showed a differentiation only between the betting conditions, and initial pupillary diameters were not related to the experimental manipulations.

Subject-Bet vs. Computer-Bet Conditions

Differences in response levels between the Subject-Bet and Computer-Bet conditions were evident even though there were no differences in the informational characteristics or payoff level between the two betting conditions. The increased amplitude of pupillary dilation and ERP components during the betting conditions as compared to the certain condition were similar to the findings of Friedman et al. (1973) in comparing uncertainty during a guessing task to a condition of certainty. The new finding was that the effect of trial outcome resulted in greater psychophysiological responsivity in the Subject-Bet condition compared to the Computer-Bet condition:

although the consequences of trials were the same, the participation of the subject differed in the two conditions. The opportunity to be involved in selection of each bet, even in the absence of direct control over winning vs. losing, provided substantial reinforcement. The findings are somewhat reminiscent of those of Boyd et al. (1977c), in which monkeys permitted to respond on a contingent schedule showed both increased behavioral performance and greater cortical responsiveness than those given food rewards without any response requirement.

The magnitude of the differences among the Subject-Bet, Computer-Bet and Certain conditions serves to re-emphasize the difficulties inherent in the interpretation of psychophysiological experiments in which the behavioral contribution of the subject is disregarded, and in the extreme case, when the subject is verbally instructed to remain passive. Sutton (1969) has reviewed the pitfalls of such an approach, pointing out that a lack of instructions leads to increased "subject option" with regard to interpreting the experimental situation. Thus, for example, the subjects of Johnston (1979) and of Homberg et al. (1981) may have had varying expectations or reactions to the amount of money involved on each trial, but in neither case was a behavioral component associated with the reward. In the present study, the subject had to report after each trial whether he had won or lost, and the amount involved.

Sutton (1969) also noted that ERP researchers have attempted to apply numerous psychological constructs to explain the psychological basis of P300, yet the psychological dimensions themselves (e.g., significance, task relevance, salience) may not yet have been adequately defined. It is thus difficult to identify the factor that has been changed between the two betting conditions in the current study. Certainly, the maintenance of differential responses as a function of win/lose and monetary payoff for P300 and pupillary dilation and recovery slope, though smaller in the Computer-Bet condition than in the Subject-Bet condition, indicate that trial outcomes still had an impact upon subjects in the Computer-Bet condition.

A possible explanation for the physiological differences found between the Subject-Bet and Computer-Bet conditions is that subjects had a greater feeling of personal involvement when they were allowed to place their own bets - the difference between personal choice and choice by fiat. Quite possibly, the level of "involvement" is a representation of greater arousal or interest which the subject brings to the Subject-Bet condition. One might be tempted to go so far as to consider these data as representing a vote for active decision making as being more stimulating, physiologically and psychologically, as compared to more passive situations.

Differences between the Subject-Bet and Computer-Bet conditions seem analogous to the conceptualization of locus of control (Rotter, 1966; Lefcourt, 1976). An individual is classified along the dimension of internal vs. external control: the "internal" is one who views events in life as being a result of self-generated choices, while the more "external" subject believes events occur more as a matter of uncontrollable factors such as chance or luck.

Rather than view each person as lying somewhere along the internal-external dimension, and classifying the personality of the individual accordingly, it is suggested by the present data that an individual's degree of internal or external reliance may change dynamically as a result of recent contingencies. Specifically, the Subject-Bet condition provided a condition in which the subject was able to interact in modifying the outcome of future events. In contrast, the contingencies imposed by the Computer-Bet condition negated the value of any decisions the subject may have generated internally, so that it was an externally controlled situation.

Subjects reported that they did not just sit passively during the Computer-Bet condition - they did, in fact, make covert guesses regarding the outcome, and several subjects expressed overt hostility towards the "strategy" used by the computer. At some times, of course, they were in agreement with the computer-generated "strategy." Therefore, it is possible that averaging according to the computer's decisions resulted in averaging across the subject's different expectations.

Two modifications of the experiment might serve to clarify the role of the subject's expectancies vis a vis computerized decision-making as a factor leading to reduction of response amplitude in the Computer-Bet condition:

1) Repeat the Computer-Bet condition with a single change: ask the subject, before trial onset but after the bet selection has been announced, whether there is agreement or disagreement with the computer's selection. It would then be possible to compare responses for trials on which the subject's strategy matched the computer's selections against trials on which there was disagreement. Of course, there is an inherent artifact in this procedure in that the procedure

of drawing the subject's attention to such decisions may itself modify a more typical strategy.

2) A more covert method for obtaining this assessment might be to perform the original experiment first in the Subject-Bet mode, and to record the subject's strategy. In a subsequent session, one may use the same stimulus and betting pattern in the Computer-Bet condition that was employed earlier for the Subject-Bet condition. If larger responses in the Computer-Bet condition were obtained using the subject's own strategy on identical blocks, as compared to a random betting pattern, then it would implicate the deviation from the subject's strategy as leading to reduced response amplitude. This second method seems somewhat unlikely to be effective, especially since it assumes that each subject will employ an identical strategy when presented with the same block of trials on different occasions. In this situation, a larger response under the Computer-Bet condition to blocks that were patterned after the subject's own responses would indicate that it was this deviation from the internally generated pattern, rather than loss of personal control or involvement, that resulted in the response decrements.

As noted above, the specification of the psychological dimension that differed between the Subject-Bet and Computer-Bet conditions is elusive. Nevertheless, the motivational effect of including the participation of the subject in the experimental design was a major one: this manipulation had the most powerful effect over the greatest number of measured physiological responses.

Effects of Monetary Value

Increasing bet value resulted in larger amplitudes of both emitted and evoked ERP components and pupillary dilation. The pupillary recovery slope was also related to bet value, although the direction of change was related to win vs. lose outcome. No enhancement of the CNV was observed with increasing bet value, and Campbell (1976) has also reported that CNV was not affected by monetary rewards. It is possible that the enhancement of the Bereitschaftspotential reported by McAdam and Seales (1969) when a reward was introduced was related more to increased interest in the task, which can affect pre-S2 activity (McAdam et al., 1966), than to the value of the reward itself.

The effect of differential rewards has been most clear in manipulating P300 amplitudes when the task was designed to emphasize the administration of rewards and penalties (Johnston, 1979; Sutton et al., Note 1) as in the present study. When differential information regarding performance characteristics is emphasized, P300 seems less likely to reflect additional changes in value. Campbell (1976) found no effects of differential rewards accompanying feedback, but his subjects were also engaged in using the feedback to modify their performance. Johnston and Holcomb (1980) observed no effect of differential rewards until after subjects had learned the contingencies for maximizing rewards, which was reflected in P300 during the final experimental session. In contrast, the value associated with each stimulus was the only relevant feature in the Homberg et al. (1981) study.

The relationship between absolute P300 amplitude and value appears to be related to the significance attached to the value manipulation. P300 increased from approximately 10 uV in a flat fee condition to 12 uV when a higher reward was possible (Wilkinson & Morlock, 1967). Sutton (Note 7) presented data for one subject in a guessing condition for whom losing two cents resulted in a P300 of approximately 15 uV and winning ten cents resulted in a P300 of over 25 uV in amplitude. The present data are comparable, with a vertex response ranging across subjects from nine to 27 uV for a bet of zero and from eight to 35 uV for a bet of 50 cents in the emitted P366, and from nearly two to 31 uV for a bet of zero and from nine to 39 uV for a bet of 50 cents in the evoked P309 component, as measured in the Subject-Bet condition.

Johnston (1979) employed bet values of zero, 20 cents and two dollars in his study, in which the bet value was presented to the subject, similar to the type of presentation used in the Computer-Bet condition of the present study. Unfortunately, no amplitude calibrations were provided for the Johnston data, so that amplitude comparisons are not possible. Values associated with performance in the Johnston and Holcomb (1980) study were zero, one and two dollars, yet the largest P300 amplitude presented (Figure 2, p. 398) appears to be less than ten uV in amplitude. One explanation for the small amplitudes for P300 in their data, relative to the present data, is that incentive values were presented to the subject arbitrarily, as in the Johnston (1979) study and Computer-Bet condition. Thus, the effects of value seem secondary to the primary tasks in the Johnston studies. In the data of Homberg et al. (1981), P300 amplitude was described in terms of an integrated area measure, but an estimation of the peak-to-baseline amplitude at Pz (at which the largest response was recorded) for the 50 German cent condition is nearly 30 uV, larger than the responses recorded by Johnston and Holcomb (1980) but similar to the amplitudes observed at Cz in the present study.

One of the differences between the preliminary and main experiments was a shift in the reward values employed (from zero, five and ten cents to zero, 25 and 50 cents), with the assumption that greater rewards would elicit responses of greater amplitudes. There was no great amplitude difference for P300, which ranged from approximately 14 to 23 uV in preliminary studies A and C. These amplitudes are well within the range of responses for the subjects in the main experiment. As part of the preparation for the main experiment, subject F1 participated in several single experiments, including two with similar reward values. On one day, bet values of zero, 25 and 50 cents, as in the main experiment, were employed. On a separate day, the bet values were zero, ten and 50 cents. For both days, the amplitudes evoked by zero and 50 cents were similar, and a smaller response was recorded to the ten cent bet for one day than to the 25 cent bet of the other. The data suggest that a self-scaling or anchoring process is invoked in these experiments. Once the subject is informed what the bet values are, it is possible that all responses tend to scale between certain physiological limits.

It should be possible to examine the hypothesis that there is a tendency to self-scale physiological responses between limits when money is used as a reward. The subject would first have to be told that all possible rewards fell between certain limits (e.g., zero to 50 cents), and the experimental procedure would be conducted for several blocks. At that time, the subject would be told that a new "limit" was imposed (e.g., two dollars). If the orginal values had resulted in scaling across the entire response range, then responses to the new maximum value should be no larger in amplitude than previous responses.

Effects of Winning vs. Losing

A clear difference between winning and losing was observed for the recovery slope of the pupil in both the emitted and evoked conditions, with a larger recovery slope exhibited for losing responses when money was actually lost. P300 was not consistent: evoked responses were significantly larger in the winning condition, but emitted responses tended to be larger for losing.

The discrepancy between the emitted and evoked potentials was resolved only when responses recorded during the same sessions were compared. In this case, winning responses were found to produce larger P300 amplitudes, regardless of whether winning was indicated by the missing stimulus or by the single click event. A similar comparison between emitted and evoked pupillary dilation responses would not have been justified, since evoked dilations were significantly larger in amplitude than emitted dilations. Furthermore, no differences between winning and losing outcomes were observed for the dilation response in either event, and the pattern for the recovery slope was similar across conditions for both events.

It is possible that the difference between winning and losing outcomes for P300 was related to characteristics specific to this experimental paradigm. The major procedural addition to the pilot experiment was the inclusion of the cancelling outcome. The double click provided no information that the subjects could utilize for developing a strategy in the Subject-Bet condition, but did serve to increase the proportion of non-zero bets. One subject (S6) reported that he tried to circumvent the cancelled outcome by repeating the bet that had been cancelled until the next winning or losing outcome; nevertheless, like the other subjects, he was influenced by the presence of the cancelled event to move his strategy closer to probability matching.

Although cancellation of the bet provided a neutral trial outcome, it could also be interpreted from two alternative perspectives: as a favorable outcome (no loss of money) or as an aversive outcome (no gain of money). The post-session reports indicated that for some subjects there was a neutral response to the double clicks ("They don't matter - a cancel is a 'safe'"), while for other subjects there was a more intense reaction, with the double clicks considered as definitely aversive ("I hate them").

The data for the pupillary recovery slope were more consistent with the notion that cancellation was an aversive outcome. For both the emitted and single click evoked response, losing resulted in a change in recovery slope that increased with increasing value. For all sessions, the data for the recovery slope following a double click matched the pattern observed for losing responses (Figure 16).

If the cancellation of a bet was viewed as an aversive situation, then the relative probabilities of favorable and aversive situations were not really equal. If the subject perceived both losing and cancellation as aversive, then two-thirds of all trial outcomes were not in the subject's favor, and only one-third of the trials presented a positive outcome (winning). Given this situation, winning was a low probability condition, and larger amplitude P300s are expected for low probability conditions (Tueting et al., 1970), as was observed. This explanation of the differences between winning and losing responses, however, fails to explain the equal amplitude of dilation observed in the pupillary response, since Friedman et al. (1973) observed that outcome probability was also reflected in the amplitude of pupillary dilation, co-varying with P300.

Accepting the "cancel" condition as an aversive situation implies that, at least for this paradigm, P300 was more sensitive than pupillary measurement to effects of the probability of event outcomes. The recovery slope measure did appear to provide a consistent indicator of the "meaningfulness" of trial outcome. "Meaningfulness" is used in this discussion to represent the notion that not only does winning differ from losing, but that different values produce different responses when associated with winning compared to losing outcomes.

The event-related potential findings do not suggest a consistent differentiation of winning vs. losing as reflected in P300. Sutton et al. (1978) have demonstrated that the issue is more complex: winning or losing could be separated from the correctness of a subject's prediction. In their paradigm, P300 was larger for losing and winning than in response to whether the subject's guess had been correct or incorrect; P300 was also increased in amplitude and occurred with a slightly longer latency for losing than for winning. The same latency trend was observed for emitted and evoked responses in the present study. The most reasonable assumption to date regarding the relative amplitudes of winning vs. losing P300s (and similarly, for correct vs.

incorrect prediction outcomes) is that P300 is related to the probability of the specific event outcome.

Strategies and Expectancies During Betting and Guessing

In the typical guessing paradigm, a guess is followed by presentation of some feedback stimulus that may be considered to provide:

1. Feedback regarding the correctness of the subject's guess (Sutton et al., 1965; 1967; Levine & Hakerem, 1969; Ruchkin & Sutton, 1979b). Where there is minimal confusion as to the nature of the feedback (confusion defined as equivocation by Ruchkin & Sutton (1978b)), the response of physiological systems may represent the recognition of the relationship between the subject's prediction as compared to the outcome.

2. The information provided by the feedback event may also be used in guiding behavior for the future. That is, detection of overall probabilities will shift the subject's guessing strategy (Tueting et al., 1971; Friedman et al., 1973), and human subjects attempt to match stimulus probabilities in these studies, rather than maximize by assumption of a strategy by which the predominant outcome is always selected. In other words, people choose to involve themselves in the game rather than stand by passively. Subjects in the present study claimed to understand that trial outcomes were determined on a random basis, yet also indicated that they actively sought to find underlying patterns at all times. Donchin (1979) has hypothesized that P300 represents the utilization of information to provide a guide for future

selections. Thus, P300 in response to an incorrect prediction may represent modification of the currently held strategy.

Expectancy and feedback were integral components of the Subject-Bet condition. The subject was asked to bet that he would win on any trial. To plan this bet, the subject was certainly required to make some covert prediction as to the event that would occur. We could speculate that the amount bet by the subject reflected his level of confidence in predicting the outcome of each trial. If the losing stimulus seemed likely, a bet of zero would probably have been placed. Conversely, betting 50 cents probably expressed a high degree of confidence that the winning stimulus was about to appear. Of course, an alternative reason for betting zero could be that the subject was just not sure at all, and was willing to pass up the possibility of winning on the next trial just to learn what the stimulus would be - to try to "get a feel" for the current pattern. The middle bet value may, in this sense, represent either a small degree of confidence, or indecision.

If a presumably high confidence bet (50 cents) was placed and the subject won, this was confirmation that high confidence was valid. For the evoked potential, it is somewhat analogous to Friedman et al.'s (1973) high probability condition in which the subject correctly guessed the frequent stimulus - the likely event in that experiment (the subject can have high certainty in the outcome), so that confirmation would be expected. A disconfirmation - losing - would be subjectively unlikely for the subject. Losing after having bet a great amount of money bet is subjectively a rare outcome, and the evoked potential is consequently increased in amplitude. Similarly, the zero bet may represent high confidence that the losing outcome would occur. If there was a loss, P300 would be small, since the prediction was confirmed. But a winning outcome, as a disconfirmation of the prediction, is an unlikely outcome (again, with respect to subjective probability), so that a win following a bet of zero results in a larger P300 than does a loss of zero cents.

An analogous process may be operating for determination of the pupillary recovery slope, beginning with the assumption that pupillary dilation does, indeed, represent a more favorable outcome. Thus, a high confidence, high value bet, when confirmed, reinforces the level of confidence, but when disconfirmed results in a larger recovery slope representing the fact that a high expectation was proven in error. Similar reasoning explains why a bet of zero should lead to a larger recovery slope when the subject has won: if the subject expected to lose (the reason for betting zero), then his expectation was disconfirmed.

The weakness of the reversal of the effects of a zero bet in the main experiment may be explained by the fact that the possibility of three different stimulus outcomes limited the ability of the subject to be confident in predicting the next event to occur. If the same event occurred for several trials in a row, the subject might increase his confidence that the next trial would be a different outcome, but he would still have to choose between two different possible outcomes. Perhaps the confidence levels were decreased in the main experiment compared to the preliminary studies.

Emitted and Evoked Potentials

The observed similarities between the emitted and evoked responses reinforce previous interpretations that both P300s represent similar central mechanisms (Sutton et al., 1965; Ruchkin, Sutton & Tueting, 1975). While apparent differences in the emitted and evoked P300s due to winning and losing have been pointed out, it was the similarities which characterized these data best. Emitted and evoked responses both tended to increase when associated with larger values. Similar amplitudes were seen over all conditions, with large increases in amplitude during the Computer-Bet condition as compared to "Certainty" and a further increased response in the Subject-Bet condition. The scalp distributions for emitted and evoked P300s were also identical, with largest responses at vertex, indicating a central-parietal distribution.

These results also emphasize one of the unique findings of the present study - the elicitation, at least for some subjects, of larger emitted than evoked P300 responses recorded during the same blocks (for the same bet value). Within any session, emitted and single click evoked responses represented opposing conditions (winning vs. losing), so that the psychological significance was not the same. The important aspect, however, was that winning or losing within a block could produce a larger emitted response than evoked response - the psychological effect of endogenous processes was not necessarily hidden by the effects of an evoking physical stimulus. In most attempts to enhance the amplitude of emitted potentials through the use of latency compensation (Ruchkin & Sutton, 1979a) or application of a Woody filter

(Ruchkin & Sutton, 1978b), the evoked potentials so treated were still larger than the emitted responses.

There have been few observations of equal amplitudes for emitted and evoked P300s. Purves and Low (1979) reported similar P300 amplitude for responses evoked by rhythmically presented visual stimuli compared to responses emitted after stimulus omission on 10% of the trials. It is not clear from their description whether emitted P300s were compared to the responses evoked by all stimuli, but this seems likely. In this event, the claim that the emitted responses are not different from the evoked P300s ignores the fact that stimulus omissions were a relatively rare event, which is a factor known to enhance the emitted P300 (Ruchkin & Sutton, 1973). Ruchkin et al. (1981) used presence or absence of a stimulus as feedback in a time estimation task. Differences in amplitude between the latency adjusted emitted and evoked responses were not significant, although the emitted P300s were smaller than the evoked P300s.

The present study provides the only instance in which larger emitted than evoked responses were recorded, during comparable conditions (same bet value, though different outcome, as noted above), for individual subjects. These data may not be entirely immune from the effects of probability. In a preceding discussion (Effects of Winning vs. Losing) the possibility was presented that winning outcomes could be considered a low probability event. A lower "effective" probability for a winning response may be reflected as an increase in P300, and emitted P300s tended to be larger than evoked P300s only for sessions in which a winning outcome elicited an emitted response.

The recording of exceptionally large amplitude emitted responses is probably due, in part, to the inclusion of the time judgement training procedure. The subjects participating in this experiment were able to discriminate better than a 100 msec difference in the duration of a one sec interclick interval. Ruchkin, Sutton and Stega (1980) trained subjects to recognize a shorter interval (700 msec) using the easier method of time reproduction, requiring an accuracy of ± 100 msec, which for the shorter S1-S2 interval of their paradigm represents a proportionally greater margin of variability than for the one second interval in this study. The subjects in this study were, therefore, at least as accurate in determining the time of information delivery as in any previous studies of emitted responses.

Ruchkin and Sutton (1978a) have demonstrated that the amount of latency variation in emitted P300 responses is increased for longer S1-S2 intervals, resulting in a lower amplitude for responses recorded after long S1-S2 intervals . In the present data, there was a tendency for the latency of the emitted P300 to be shorter in the winning condition than in the losing condition. If the amplitude differences between winning vs. losing were due solely to the latency of the subject's decision that winning or losing had occurred, then the emitted P300 should be larger for the earlier response (winning). This was not observed for the emitted responses. Therefore, the differences between winning and losing for the emitted responses were apparently not related to decision latency.

There are a variety of difficulties encountered in trying to localize the presumed cerebral generators of event-related potential components, and this is especially a problem for components such as

P300 which seem to have the same distribution independent of modality (Ritter et al., 1979; Simson et al., 1976). It has been suggested that the differences in skull thickness over different cortical regions result in larger potentials apparently recorded over some regions. Thus, the vertex maximum in some experiments may reflect the lesser thickness of the skull compared to mid-parietal (Deecke, comment in Donchin, 1978). Vaughan and Ritter (1970) demonstrated an inversion over the Sylvian fissure for P200 of the auditory evoked response, and suggested that components of the auditory response are generated near primary projection cortex. This view was challenged by Kooi, Tipton and Marshall (1971), who suggested that the nose reference used by Vaughan and Ritter was an active, rather than indifferent, reference. Using a non-cephalic reference, no inversion was found over any cephalic region. Vaughan has countered that on the basis of calculations using the dipole model (Vaughan, 1974), the non-cephalic reference used by Kooi et al. was actually an active reference. Additional data provided by Picton et al. (1974) appear to favor the view that late auditory components are widespread in source, with greater localization over fronto-central cortex up to 200 msec, but with components occurring after 300 msec localized more posteriorly.

It has also been reported that P300-like responses can be recorded from human hippocampus and amygdala (Halgren et al., 1980). Rare, task relevant tones or visual stimuli elicit larger responses than non-targets, and distraction reduces the amplitude of responses to rare stimuli. Halgren et al. also reported that emitted responses were observed to the omission of stimuli in a regular train. The authors indicated that scalp and limbic generation of late positive components may only be correlates of the same process, but posed the possibility that scalp activity is related to volume conduction of the limbic activity observed. An involvement of limbic cortex in the generation of P300 is consistent with the present findings that both emitted and evoked P300s increase in amplitude when associated with increasing bet value, since limbic activity has long been associated with affective components of behavior, and winning or losing 50 cents is likely to evoke more of an emotional response than winning or losing two cents or nothing.

Despite the numerous guessing and discrimination tasks which indicate a maximum response for P300 as recorded at vertex (Chapter I), it is still commonly asserted that evoked and especially emitted P300s are maximal over mid-parietal scalp (Donchin, 1979; Simson et al., 1976). Differences in the locus of maximum P300 scalp amplitude seem related to task differences that invoke different, though probably overlapping, functional cortical systems. A detailed discussion of task influences on the evoked P300 has appeared (Tueting, 1978; see also Roth, 1978).

Principal Components Analysis (PCA) of the positive wave complex that occurs following a task-relevant stimulus has established the independence of positive slow wave activity from P300 (K. Squires et al., 1977; N. Squires et al., 1975, 1977). Slow wave activity is similar in onset to P300 but tends to have maximum effect after the termination of P300. In the studies mentioned, P300 and slow wave were typically observed to be maximal over parietal cortex, but this was probably a function of the counting tasks that were used.

In experiments described earlier, Ruchkin, Sutton and Stega (1980) and Ruchkin, Sutton, Kietzman and Silver (1980) obtained maximal P300 amplitudes at Pz, but slow wave distributions represented both at Cz and Pz. The study of Ruchkin, Sutton and Stega (1980) is especially relevant in this respect. Threshold stimuli were employed for either a guessing or detection task, and in both cases, emitted and evoked P300s appeared largest at parietal. Slow wave was also maximal at parietal for the detection task, but showed a maximum at vertex instead during the guessing task. This study implicates slow wave as the possible determinant of the vertex P300 response, at least for the guessing task. However, the increased "equivocation" introduced by the difficulty of the detection/discrimination problem weights even the guessing task used by Ruchkin et al. to the domain of detection; a more parietal P300 ("P3b") tends to be observed for detection studies (Tueting, 1978), which may correspond to the P300L of Ruchkin et al. (1981).

Although the present results based on peak-baseline amplitudes shed no light on the relationship beween the observed P300 and slow wave, the data have been subjected to a principal components analysis by Dr. David Friedman. Preliminary results indicate the identification of three major factors: a CNV, slow wave, and P300 factor, observed in both the emitted and evoked response data. The preliminary results are parallel with the component analyses for CNV and P300: CNV was differentiated only by Source of Bet, while P300 showed a main effect for Trial Outcome and Electrode Location, and interactions of Electrode Location with Value of Bet, with Source of Bet, and with Stimuli. The slow wave factor was significant for Source of Bet and for Bet Value, but not for Trial Outcome. For the present discussion, the most interesting results concern the scalp distribution shown by the slow wave and P300 factors: slow wave was maximum at Pz and smallest at Fz, as observed in most previous studies. P300 was entirely different, with a maximum response at Cz and then at Fz, and smaller responses indicated at Pz and Oz. Although preliminary observations only, the PCA supports the contention that a true vertex maximum exists for the emitted and evoked P300 components in this paradigm.

One explanation for the vertex P300 is that it represents a component different from the parietal P300. Donchin (1979) employs differences in topographic distribution as one of the critical characteristics in distinguishing separate components. There is another alternative, though, which does not seem to have received much attention. This hypothesis is that P300 is related to a widespread cortical system which is distributed from frontal to at least parietal regions. The reason that different P300s are observed is that the region of this neuronal pool which is maximally accessed is shifted in relation to the task that is imposed. The existence of such a system would explain why, even with different categories of tasks that have different maximum P300 localizations, the latencies still tend to be in the 300-400 msec range: the same general pool is responding. Similarly, slow wave activity could be related either to the same or to a partially overlapping system.

Dissociation of CNV from P300

While P300 was sensitive to virtually all facets of experimental manipulation, the contingent negative variation was relatively unresponsive. The CNV responded primarily to differences among the Subject-Bet, Computer-Bet and Certain conditions, and even these differences were not statistically significant. P300 was approximately three times the amplitude of CNV, replicating previous observations (Donchin & Heffley, 1979; Tueting & Sutton, 1973).

Tueting and Sutton (1973) noted the difficulties in determining whether the relationship between pre- and post-stimulus activity (e.g., CNV and P300) involved independent reactivity in some experimental designs, with covariance occurring between responses by coincidence, rather than functional independence (their formulation number (3)), or whether pre- and post-stimulus processes could be related, but with additional, independent post-stimulus processing activity (their formulation number (4)). They provided some evidence supporting formulation (4), which they did not consider conclusive. An example of this formulation in the present data could be the fact that differences were observed for both CNV and P300 in the Subject-Bet vs. Computer-Bet conditions, but independence of additional processing was observed for winning vs. losing and bet value only for P300. Of course, information regarding trial outcome was not available prior to or during development of the CNV.

A more direct evaluation is provided by comparing the responses of CNV and P300 to information that was available to central mechanisms prior to the generation of either response. If parallel processing of CNV and P300 occurs then such variables should have similar effects, even allowing for differences in overall amplitude. Two factors met this constraint: the source of the bet, and the value of the bet.

Both CNV and P300 were affected by changes in the source of the bet, but only emitted and evoked P300s (and other post-stimulus components) showed any relationship to the value of the bet. Since the CNV, occurring first, did not respond differentially when different bet values were placed, but P300 did demonstrate such sensitivity, an additional, separate neurophysiological processor may be postulated for the post-stimulus activity. These data are generally supportive of Tueting and Sutton's formulation (3).

The lack of CNV responsiveness to all variables except manipulation of source of bet seems to correspond to the distinctions between models of attention and arousal as described by Tecce (1972). It seems most reasonable to attribute to manipulation of reward value an arousal function, for which changes in the intensity of responses may be expected (Tecce, 1972). While no such aspect was elicited in the CNV, an increase in general level was maintained by permitting the subject to take an active part in placing the bet value, and this seems consistent with the concept of attention. The shifting of attention was demonstrated by the selectivity of CNV to responses when they were made more relevant by instruction.

P450 Component of the Evoked Potential

A prominent observation in the data was the presence of a P450 component for the single and double click evoked responses. P450 increased with bet value, and was attenuated in the Computer-Bet condition, as was P300, but did not differentiate winning from losing. Previously, a similar late component (often referred to as "P4") has been observed during learning and/or performance of a pitch discrimination task (Gardiner & Walter, 1969; Jenness, 1972a; Karlin, Martz, Brauth & Mordkoff, 1971), for visual categorization (Stuss & Picton, 1978) or for time estimation by the method of production (Campbell, 1976; Ruchkin et al., 1981). In all cases, an earlier P300 component was also reported.

The component recorded by Gardiner and Walter (1969) was more prominent during the pitch discrimination task than during intensity discrimination using the same stimuli, and occurred with an actual latency of 600 msec. Karlin et al. (1971) observed an earlier component at 444 msec, which was sensitive to speed of reaction time. Only the vertex response was reported for these two studies. Jenness (1972a) obtained a component having a 500 msec latency during the learning of a difficult discrimination. Although attributed to the requirement for pitch discrimination, it also appeared prominently in the waveforms resulting from feedback stimuli. Several scalp locations were recorded, but only the vertex response was discussed.

Stuss and Picton (1978) reported P450 components both to visual signal and auditory feedback stimuli, with a maximum amplitude occurring within a 500-800 msec latency range. To the signal stimuli, both P300 and P450 were maximum in amplitude at Fz. These components were apparently superimposed on a positive sustained potential evoked by presentation of the slide (Picton & Stuss, 1978). For some subjects, P450 exhibited an increase in amplitude as learning progressed. A more posterior P300 and P450 were reported following the feedback stimuli. Mean peak latency was actually 647 msec, longer than reported in other studies. P450 was similar to P300 in responsiveness to the experimental variables. Stuss and Picton stated that P450 "was maximally recorded at the vertex but had almost equal amplitudes also in the parieto-occipital regions" (p. 149). P450 was also larger compared to P300 in the posterior regions, but both components were maximal at vertex. Roth (1978) mistakenly interpreted a separate discussion of this data (Picton, Woods, Stuss & Campbell, 1978) as portraying both Pz and Oz as larger than Cz in amplitude, but previous data indicate only a vertex maximum response for P450 where topographical data were available for discrimination feedback (Jenness, 1972a; Stuss & Picton, 1978).

A parietal P450 has been described for feedback stimuli used in time estimation tasks. Campbell (1976) identified a parietal "P4" component distinct from the central-parietal P3 also observed. The latency of the component was only specified as falling within a 450 to 650 msec range. Ruchkin et al. (1981) identified a P300L which occurred with a parietal maximum and a latency of 400 to 600 msec. P300L, which was assumed to represent the component referred to as P3b (see Tueting, 1978), was extracted as a separate factor from the more central P300E (early) and slow wave components through the use of principal components analysis.

The present study demonstrates a P450 which is maximum at Pz, with the next largest amplitude observed at Cz, followed in amplitude by Oz and finally, Fz. The prominence of the component at the parietal location is further emphasized by the decreased negativity of the N350 component at Pz, indicating a shift from a vertex maximum for components earlier than 350 msec to a parietal maximum for later activity. The P450 reported here was earlier than the component reported by Stuss and Picton, with a latency similar to the P450 components observed by Karlin et al. (1971) and by Jenness (1972a). Inspection of the grand means for both the emitted and evoked responses across scalp locations (Figures 17, 18 and 19) reveals a positivity, even in the emitted responses, that occurs later than P300 and which is most prominent at the parietal and occipital locations. It is not clear whether this corresponds more closely to P300L or to phasic slow wave activity (Sutton & Ruchkin, Note 8).

If P450 is a manifestation of a process independent of slow wave, its functional significance remains unclear, since in this study, no further characteristics were indicated that had not been discriminated in earlier components. It may represent an additional processing component involving re-evaluation of the information presented by trial outcome. An evaluative function was also suggested by Stuss and Picton. It is also not clear why P450 is usually reported in studies involving feedback for above-threshold discrimination or time-estimation performance.

Comparisons Between Pupillary and ERP Activity

If the P300 wave and pupillary response were reflections of an identical underlying mechanism, it would be redundant to study both. Current evidence, however, indicates differences in the information carried by these two measures. In the present study, the dilation of the pupil was sensitive to effects of value, but not to winning vs. losing. However, the recovery slope of the pupil differentiated those trials in which the outcome was in the subject's favor (winning money or losing nothing) from trials in which the outcome was less favorable (losing money or winning nothing). A greater constriction was observed for unfavorable responses. This is consistent with the larger constriction (following peak dilation) which has been reported for incorrect compared to correct guesses in uncertainty tasks (Friedman et al., 1973; Levine & Hakerem, 1969; Hakerem, 1974; Steinhauer et al., 1979). The event-related potential results were less consistent between informational events than the pupillary results, with larger P300s for losing responses for the emitted potential, but larger evoked potentials for winning responses. Only across sessions were the ERP data consistent, and no consistent association between winning vs. losing and amplitude of P300 could be determined between the preliminary and main studies.

Another difference between pupillary activity and P300 response can be found in the literature on discrimination. The concept of equivocation has been employed (Ruchkin & Sutton, 1978b) to explain the decreased amplitude of P300 as the discriminability between stimuli is decreased. Adams and Benson (1973) used a standard auditory stimulus to provide positive feedback, but used stimuli of different intensities as negative feedback across different blocks. P300 in response to the standard stimulus was decreased in amplitude when the difference in intensity between the alternative feedback stimuli was decreased. Ruchkin and Sutton (1978) attributed this to the "equivocation" (loss of information) involved when the discrimination became more difficult. A similar explanation has been applied to the results of signal detection experiments.

The pupil appears to behave differently from P300 during supra-threshold discrimination tasks. Larger dilations are obtained for comparison tones that are most similar to the standard tone in a pitch discrimination task (Kahneman and Beatty, 1967); the amplitude of dilation is positively correlated with the number of errors for a given discrimination. The increased amplitude of the dilation is explained as reflecting greater effort required in correctly performing the discrimination. In contrast with the positive relationship between P300 and pupillary dilation observed when probabilities are manipulated in a guessing task (Friedman et al., 1973), it is likely that an inverse relationship would appear between P300 at Pz or Cz and amplitude of pupillary dilation if a similar discrimination task were employed during simultaneous measurement. In the guessing task, pupillary dilation indicates the resolving of uncertainty (involving outcome probability), while in the discrimination task, dilation may reflect the effort involved in performing difficult discriminations.

The fact that pupillary dilation can be related both to the informational qualities of an event, e.g., outcome probability (Friedman et al., 1973) or value associated with an event (the present study) seems similar to observations for P300. It is also true that dilation is enhanced when greater "mental effort" is invoked (as summarized in Chapter I), and recently, slow wave activity in the ERP has been described in terms of the effort exerted in processing information (Ruchkin, Sutton, Kietzman & Silver, 1980; Sutton & Ruchkin, Note 8). Since the pupillary dilation response occurs with such a long latency (600 msec, peaking at 1200 msec), it is reasonable to assume that additional processing activity may be reflected in the dilation response. The functional relationship between ERP components and the recovery slope, which has an even longer latency than the dilation response, is not clear. Because of the prolonged latency, we might expect that further levels of processing also should be reflected in the recovery slope. At the present time, it is not clear what general functions are represented by the recovery slope of the pupil, nor which components of the ERP are most closely paralleled. It has been suggested that a PCA of the pupillary response might indicate considerable overlap between components representing primarily the dilation and the recovery slope regions, and both could be related to processing effort (Sutton, personal communication). Analyses to clarify these relationships are currently being planned. There is also little evidence, at the present time, that indicates whether parallel findings for the pupil and ERP are the result of common underlying processes, or whether they represent parallel but independent physiological processing systems. Parsimoniously, the former relationship seems likely, but it will be necessary to clarify the mechanisms involved in generating both the pupillary response and event-related potentials before any hypothesis can be substantiated.

The discrepancies between the information expressed in the pupillary and ERP measures should be viewed as an advantage of using multiple psychophysiological measures. It is also not reasonable to assume that only a single physiological mechanism is capable of reflecting information processing characteristics. For example, a type of "P300 chauvinism" is present in the assertion of Israel, Wickens, Chesney and Donchin (1980) that the ERP provides the only adequate measure of workload capacity, since other measures such as heart rate, GSR and especially pupil diameter are influenced by physical workload and emotional arousal. Shiga and Ohkubo (1978) had already demonstrated just such sensitivity in the averaged pupillary dilation response recorded during performance of a shadowing task.

Is There a Common Mechanism Underlying Pupillary and P300 Activity?

The correspondence between pupillary and P300 activity under different experimental conditions such as varying event probability (Friedman, 1972; Steinhauer et al., Note 3), differences between certainty vs. uncertainty (Friedman, 1972; present data) and effects of value (present data) could be explained by one or more of the following propositions:

1) Pupillary activity is reponsible for the generation of P300 responses;
2) P300 activity is responsible for generation of pupillary responses;
3) P300 and pupillary responses are generated by separate, but parallel, central mechanisms which react to events in a similar manner;
4) P300 and pupillary responses are generated by a common underlying mechanism.

The first proposition, that pupillary activity is responsible for P300, may be rejected on the basis of latency characteristics, since the pupillary response occurs much later than P300. The earlier latency of P300 does support proposition number two that P300 activity can influence the course of pupillary motility. As described earlier, many psychological variables affect the pupil and P300 amplitude in a parallel manner. However, there are some discrepancies between pupillary and P300 response trends, as illustrated by the consistency with which the pupil, after an initial dilation, tends to constrict more as greater amounts of money are lost (as in the present data) or when the subject guesses incorrectly as compared to correctly (Friedman, 1972), while P300 does not show as great a consistency for either losing vs. winning (or correct vs. incorrect guesses) to result in larger P300 amplitude (see Chapter I). There may be some significant component of P300 activity which influences the pupillary response, but P300 is apparently not the sole determinant of cognitively-based pupillary activity.

The differences between pupillary and P300 responses may reflect additional processing activity which occurs during the interval between the generation of P300 and the expression of the pupillary response, but this does not rule out the possiblility that the initiation of both responses has a common source. In considering the remaining propositions for either separate, but parallel, mechanisms, as contrasted with a common neurophysiological mechanism underlying both responses, the latter alternative (number four) is both more parsimonious and more appealing theoretically given the variety of psychological factors that result in relatively similar responses of the two systems.

With regard to the known neurophysiology of pupillary dilation, it does not seem likely that this response is under subcortical initiation and control. Although the hypothalamus provides a definitive nodal point for propagation of autonomic nervous system activity, the normal time course for psychosensory dilation is obtained only when cortical influences (either direct to the hypothalamus or mediated through thalamic pathways) remain intact; under early stages of anesthesia, when cortical activity is suppressed but no changes are detected in the electrical excitability of the hypothalamus, pupillary dilation is slower and does not achieve maximal amplitude (Loewenfeld, 1958). In addition, weak cortical stimulation provides strong sympathetic discharges in normal, awake animals, which suggests that the strong sympathetic activity of decorticate animals represents hypersensitivity of the thalamus and hypothalamus following cortical removal, rather than the localization of the pupillary dilation reflex to the thalamus or hypothalamus per se (Lowenstein & Loewenfeld, 1963).

In her discussion of the neurophysiology of dilation, Loewenfeld (1958) concludes only that the center for initiation of the normal dilation reflex must lie above the level of the midbrain, but does not invoke the activity of any specific cortical region, since stimulation of most cortical regions results in pupillary dilation. Although projections from frontal cortex to the hypothalamus have been suggested for the initiation of pupillary dilation, perhaps the best that can be claimed at the present time is that "the central pathway for pupillary dilatation is not completely known" (Truex & Carpenter, 1969, p. 389).

Control over the parasympathetic component of pupillary movements also can be influenced by cortical mechanisms. Direct cortical pathways, possibly originating in area 8, act to inhibit the Edinger-Westphal nucleus (Lowenstein, 1955). An indirect pathway, also inhibitory, is found via the cortico-thalamic-hypothalamic pathway, with hypothalamic fibers projecting to the Edinger-Westphal nucleus (Lowenstein, 1955).

Friedman (1972) summarized data suggesting that the diffuse thalamic activating system (DTAS) described by Lindsley (1960) is involved in the elicitation of localized orienting responses (Lynn, 1966). Friedman suggested that activity of the DTAS could be responsible for both pupillary dilation during information processing, as part of an immediate but limited orientation response, and for generation of P300 on stimulation of cortical cells in the association cortex. The conclusions of Lowenstein and Loewenfeld (1963), cited above, are not entirely consistent with the notion that the DTAS specifically initiates pupillary dilation. Stimulation of the DTAS could serve an alerting function, involving activation of cortical regions. Subsequent processing at the cortex would initiate pupillary dilation and other sympathetic activities.

Sokolov (1963) has described pupillary dilation as one of the prototypical components of the orienting reaction (whether pupillary dilation is always a sign of orienting is discussed in the following section). Indirect evidence points to the involvement of limbic regions in the registration of orienting responses. Disruption of orientation reactions, as measured by reduction in electrodermal responding, has been reported following bilateral amygdalectomy in rhesus monkeys, although not after removal of the hippocampus (Bagshaw, Kimble & Pribram, 1965). Electrophysiological recording indicates that attention to ambiguous stimuli is initially accompanied by the appearance of slow "theta" rhythms in the hippocampus, but this activity diminishes as training progresses (John, 1967a), indicating a relationship between electrical activity of the hippocampus and behavior associated with orienting.

With respect to the P300 response, the primary role of cortical association areas, especially parietal-temporal cortex, has been strongly implicated as the functional source for P300-like activity (Vaughan, 1974). However, the wide scalp distribution over which the P300 can be detected makes it difficult to establish specific sources for the P300 response, or even whether it is plausible to consider the P300 in conjunction with a single generator (Picton et al., 1978; Roth, 1978).

It may be hypothesized that components of the limbic system are involved both in the determination of the integrated pupillary dilation response and in the generation of P300. Direct connections among limbic regions (amygdala, hippocampus, cingulate cortex), thalamus and hypothalamus are well established (Truex & Carpenter, 1969). Reflex pupillary dilation has been demonstrated by stimulation of the amygdala in cats, dogs, and monkeys (Koikegama & Yoshida, 1953). Stimulation of the dorsomedial amygdala in cats results in pupillary dilation, but lesions in the central gray markedly reduce dilation produced by such stimulation (Fernandez de Molina & Hunsberger, 1962), suggesting that the central gray matter provides a contributory pathway but is not responsible for the initiation of pupillary dilation.

With the report (Halgren et al., 1980) that P300-like responses can be recorded in humans from electrode placements in the hippocampus, hippocampal gyrus, and amygdala, and that these responses show a reversal of polarity when electrodes are moved over small distances (indicating the localization of a generator of current), the possibility is raised that scalp-recorded P300 reflects volume conduction of such limbic activity. A reconceptualization of the P300 literature by Sutton (Sutton & Ruchkin, Note 8) suggests that the common factor running through experiments in which P300 ("P3b") is obtained is the evaluative, affective component of the experimental situation -- the drawing of attention to an unusual or rare event, or the occurrence of an event which conveys meaningfulness, for which both affective and cognitive components may exist. In the present study, the manipulation of monetary value resulted in clear changes in P300 amplitude, even across conditions in which the informational value did not differ.

The classic associations of limbic activity with emotionality (Papez, 1937; MacLean, 1949) are consistent with the hypothesis that P300 reflects an affective component, as suggested by Sutton (Note 8), and with the finding of limbic P300 activity (Halgren et al., 1980). (Note 8). A tentative hypothesis to explain the correspondence between P300 and pupillary activity as recorded during certain information processing tasks is that both types of responses reflect cognitive and affective components, and that the affective component is substantially mediated by activity in limbic regions.

The Orienting Model vs. Information Processing as Explanatory Mechanisms

The major theoretical framework that has been invoked to explain pupillary dilation during increased attentional demand is the model of the orienting response as described by Sokolov (1963; 1969). The orienting reaction is described as a diverse response to novelty, including both directional orientation of the body (head turning and eye movements) as well as autonomic components (pupillary dilation, GSR, alpha blocking) which are invariably invoked in some form when a novel stimulus or some characteristic of a previously habituated stimulus is modified (Sokolov, 1969). The mechanism for the orienting response is the establishment of a neuronal trace, based on previous stimulation, to which afferent stimulation is continuously compared. A discrepancy between the afferent stimulus and central model is coded as an orienting response, the amplitude of which is generally proportional to the amplitude of the discrepancy. The model assumes that new information modifies the template, so that the neural model "constantly undergoes revisions in order to account for the characteristics of the stimulus which is operating at a given moment" (Sokolov, 1969, p.677).

The possibility that P300 reflects a component of the orienting reaction was first put forth by Ritter et al. (1968) and further elaborated by Friedman et al. (1973). The role of attention and motivational variables in the development of the orientation response has been stressed (Bernstein, 1969), and such work provided a framework to which the P300 and pupillary dilation data of Friedman et al. (1973) could be compared. The model predicts the observed increase in response amplitude with the resolving of greater uncertainty (Friedman, 1972). The model can be similarly effective in explaining the generation of emitted responses to stimuli that are expected, but absent: the missing stimulus results in a large discrepancy with the template, resulting in generation of the orienting response.

Similar models have been invoked to explain P300 amplitude, under the assumption that all stimuli are matched to a previously established template (John, 1967a) resulting in a neural "readout" during comparison of incoming stimuli with memory-encoded information.

Hillyard et al. (1971) specifically related the evocation of P300 to the match between a sensory event and a neural "template" or "model" as described by Sokolov. Friedman (1978) has suggested that generation of P300 in the same paradigms that elicit pupillary dilation supports the notion that P300 is primarily a manifestation of the orienting response.

The orienting model does not seem sufficient to explain many of the pupillary and ERP responses that have been recorded. The basis for this argument is both theoretical and empirical. Modifications of the orienting concept have been presented according to which informational characteristics of a stimulus may be separated from the physical characteristics (Velden, 1978), reminiscent of the approach applied for the discrimination between endogenous and exogenous components of the ERP (Sutton et al., 1965). The relevance, or situational motivation of a stimulus, is a critical component of such a model.

A defense of traditional orienting theory was presented by O'Gorman (1979), who argued that stimulus-induced novelty was sufficient to account for many of the effects attributed to stimulus significance (Bernstein, 1969). Reactions to O'Gorman's view emphasized the influence of task relevance, including the selective attention to specific stimuli that could be provided by instructions (Maltzman, 1979) and the continuous monitoring for stimuli to which significance has been attached (Bernstein, 1979). Bernstein fortified his argument by referring to data collected by Peavler (1974), in which pupillary dilation in response to auditory presentation of digit strings was increased only after subjects were told that they would be required to recall the digits. No dilation was observed when no recall was required. Bernstein interpreted the pupillary dilation as an orientation response elicited when the stimuli were made relevant. No such interpretation was given by Peavler (1974), who attributed the increased dilation both to the attention process and to the effort involved in performing the task, but not to an orientation response to the task.

The statements of Bernstein (1979) and Friedman (1978) imply that if an experimental manipulation elicits pupillary dilation, then it must be an orienting paradigm, and any responses which accompany dilation must, therefore, be related to orienting reactions. There is a circularity in that the assumption is made that pupillary dilation is always a component of the orienting response. An alternative view is that pupillary dilation is not necessarily indicative of orienting, but is more likely to reflect non-orienting, complex, information processing characteristics in some situations. The discussions of Bernstein, Maltzman and Velden express dissatisfaction with the success of classical orienting theory because of its failure to encompass all possible forms of response variability. The difficulty, according to Naatanen, is that "the concept of orienting reflex (OR) has grown too broad to serve as a useful conceptual tool in our scientific efforts" (1979, p. 61).

The requirement for comparison to a template in the evocation of P300 (John, 1967b) was rejected by Picton et al. (1974) for three reasons: 1) similar emitted responses are obtained when stimulus modalities differ (visual and tactile), 2) the fronto-central maximum for the N1-P2 components of the auditory ERP differs from the missing stimulus negativity observed maximally at vertex, and 3) shorter interstimulus intervals increase the emitted response (due to reduction of temporal uncertainty), but decrease the amplitude recovery for evoked components. Ford et al. (1976) also rejected John's neural readout model because responses to omitted tones are longer in latency than evoked responses, indicating that, if a read-out process exists, it is not precisely time-conditioned.

It has been established that the introduction of novel stimuli, which provide the classical demonstrations of orienting, result in a P300 having a maximal response over the frontal scalp region (Courchesne, Hillyard & Galambos, 1975; Courchesne, 1977). The frontal P300 has not been observed to deviant stimuli that could be encoded within the same modality as other ongoing stimuli (Courchesne, Courchesne & Hillyard, 1978). Naatanen (1979) summarized data indicating that both voluntary and involuntary orienting resulted in selective negativity at frontal cortex. An instruction to attend to stimuli produces changes in P300 but no change in alpha suppression or skin conductance, classical measures of the orienting response (Becker & Shapiro, 1980). From these data, it can be surmised that a posteriorly distributed P300 component, though possibly representing a neuronal match or mismatch, does not reflect the classical orienting response to novelty as described by Sokolov, which instead is more likely to be associated with activity in the frontal region. Courchesne et al. (1978) specifically indicate that posteriorly distributed P300s are not consonant with the Sokolovian orienting model, and only non-targets that are easily classified elicit a parietal P300. Since Friedman et al. (1973) recorded the ERP at vertex only, it was not possible to determine if the the pupillary response obtained was associated with a more frontal or parietally distributed response.

The present data do not support the orienting model in that, during the betting procedure, pupillary dilation is associated with P300 activity which is maximal over the centro-parietal region. If one accepts the notion that it is the centro-frontal P300 that is the sign of orienting (Courchesne et al., 1975, 1978; N. Squires et al., 1975) then this would suggest that the P300 and pupillary dilation findings are not reflecting an orienting response, but instead, reflect more complex information processing. The waveshape characteristics of the pupillary response are also not congruent with a model relying on matching or mismatching to a template. While no differences in dilation were observed between winning and losing outcomes, responses to loss of money resulted in greater constriction than winning of money. The establishment of a template could not predict this difference, since it is differentially modifiable by the manipulation of value, and by arbitrary assignment of winning or losing to stimulus presence or absence.

During such tasks as guessing (Bock, 1976; Friedman et al., 1973; Levine, 1969), counting targets (Steinhauer et al., Note 2) and betting (the present data) it is probable that patterns of pupillary dilation and post-dilation activity are more closely related to the active processing of information than with orientation.

The typical differentiation between correct and incorrect responses has been observed in the pupillary data recorded from normal subjects during performance of a guessing task (Steinhauer et al., 1979), similar to the tasks employed by Friedman et al. (1973) and

Bock (1976). Unpublished ERP data collected in our laboratory during the guessing task indicate the same centro-parietal distribution for P300 as observed in the present study. Pupillary dilation has also been observed to be related to decreasing conditional probability of event outcome during counting of an infrequent target, which was accompanied by maximal P300 amplitude recorded over parietal cortex (Steinhauer et al., Note 2). Beatty and Wagoner (1978) have demonstrated increased amplitude of pupillary dilation when the same stimuli are categorized according to more complex rules, while maintaining the relative probability of the stimuli.

Multiple evidence exists that pupillary and ERP activity may be dissociated from the classical representation of the orienting response in specific paradigms. The pupillary response and event-related potential recorded in the present study seem most representative of internal mechanisms associated with complex aspects of information processing activity.

Concluding Remarks

This study has indicated that the manipulation of motivational factors directly influences the characteristics of pupillary motility and event-related potentials. The active participation of the subject in the Subject-Bet condition was sufficient to enhance all responses measured, even though the information conveyed on each trial, as well as the gain or loss of money conveyed by event information, was the same in the Computer-Bet condition. The use of increasing monetary values was also found to enhance the amplitude of the responses measured, further demonstrating the importance of motivational factors.

The sensitivity of the pupillary recovery slope to the winning and losing of money, paralleling previous reports for correct and incorrect guessing behavior, was also described. The complexities that distinguish winning and losing money from being right or wrong in one's guess, however, do not yet appear resolved for the event-related potential (e.g., Sutton et al., 1978).

Finally, this study has provided further evidence that psychophysiological responses correlated with either informational or motivational processes can be correlated with endogenous mechanisms of the central nervous system. This was demonstrated for the late positive activity of the event-related potential, and for both the dilation and recovery slope components of the pupillary response.

NOTES

Note 1. Sutton, S., Braren, M. & Zubin, J. Sensory, conceptual and emotional components of the evoked response to sound stimuli in man. Presented at the meeting of the Psychonomic Society, Chicago, October, 1965.

Note 2. Steinhauer, S. R. & Hakerem, G. Effects of betting and guessing on pupillary dynamics and vertex evoked potential. Presented at the meeting of the Eastern Psychological Association, New York City, April, 1975.

Note 3. Steinhauer, S. R., Jennings, J. R., Zubin, J. & Heidorn, P. B. Pupillary dilation, P300 and heart rate are influenced by conditional probability. Presented at the meeting of the Society for Psychophysiological Research, Vancouver, October, 1980.

Note 4. Young, R. S. L. & Alpern, M. The pupillary reflex to chromatic light exchanges. Paper presented at the 10th Symposium on the Pupil, New York City, June, 1977.

Note 5. Beatty, J. & Wagoner, B. L. Pupillary measurement of sensory and decision processes in an acoustic signal-detection task. Paper presented at the meeting of the Psychonomic Society, Denver, 1975.

Note 6. Clark, W. R. & Eckenberger, J. Pupillary size in an auditory signal detection task. Paper presented at the annual meeting of the Midwestern Psychological Association, Chicago, 1976 (cited in Janisse, 1977).

Note 7. Sutton, S. The sensitivity of the evoked potential to psychological variables. Paper presented at the meetings of the EEG Society, Washington, D. C., 1970.

Note 8. Sutton, S. & Ruchkin, D. S. The late positive complex - advances and new problems. Paper presented at the Sixth International Conference on Event-Related Slow Potentials of the Brain (EPIC VI), Lake Forest, Illinois, June, 1981.

References

Adams, J. C. & Benson, D. A. Task-contingent enhancement of the auditory evoked response. Electroencephalography and Clinical Neurophysiology, 1973, 35, 249-257.

Ashe, J. H., Cooper, C. L. & Weinberger, N. M. Role of the parasympathetic pupillomotor system in classically conditioned pupillary dilation of the cat. Behavioral Biology, 1978b, 23, 1-13.

Bagshaw, M. H, Kimble, D. P. & Pribram, K. H. The GSR of monkeys during orienting and habituation and after ablation of the amygdala, hippocampus and inferotemporal cortex. Neuropsychologia, 1965, 3, 111-119.

Barlow, J. Some observations on the electrophysiology of timing in the nervous system. Electroencephalography and Clinical Neurophysiology, 1969, 27, 545.

Barlow, J. D. Pupillary size as an index of preference in political candidates. Perceptual and Motor Skills, 1969, 28, 587-590.

Barlow, J. D. Pupillary size as an index of preference. Perceptual and Motor Skills, 1970, 31, 331-336.

Beatty, J. Activation and attention in the human brain. In M. C. Wittrick, J. E. Bogen, M. S. Gazzaniga,, H. J. Jerison, S. D. Krashen, R. B. Nebes & T. J. Teyler, The human brain. Englewood Cliffs: Prentice-Hall, 1977, 63-85.

Beatty, J. & Wagoner, B. L. Pupillometric signs of brain activation vary with level of cognitive processing. Science, 1978, 199, 1216-1218.

Becker, D. E. & Shapiro, D. Directing attention toward stimuli affects the P300 but not the orienting response. Psychophysiology, 1980, 17, 385-389.

Bell, R. R. Experimenter expectancy in pupillometric research. Perceptual and Motor Skills, 1971, 33, 174.

Bergum, O. & Lehr, J. Prediction of stimulus approach: Core measures experiment II. Research Report R66-36; Rochester, New York: Xerox Corp., 1966 (cited in Hess, 1972).

Bernick, N. & Oberlander, M. Effect of verbalization and two different modes of experiencing on pupil size. Perception & Psychophysics, 1968, 3, 327-330.

Bernstein, A. S. To what does the orienting response respond? Psychophysiology, 1969, 6, 338-350.

Bernstein, A. S. The orienting rsponse as novelty and significance detector: reply to O'Gorman. Psychophysiology, 1979, 16, 263-273.

Beyer, W. H. CRC handbook of tables for probability and statistics. Cleveland: Chemical Rubber Co., 1966.

Bock, F. A. Pupillary dilation and vertex evoked potential similarity in monozygotic and dizygotic twins and siblings. Doctoral dissertation, City University of New York, 1976. (Dissertation Abstracts International, 1976, 36, 6432B).

Borda, R. P. The effect of altered drive states on the contingent negative variation (CNV) in rhesus monkeys. Electroencephalography and Clinical Neurophysiology, 1970, 29, 173-180.

Borda, R. P. & Hablitz, J. J. Use of a simple visual display to reduce eye movement artifacts in CNV recordings. Electroencephalography and Clinical Neurophysiology, 1973, 34, 433-436.

Boyd, E. S., Boyd, E. H. & Brown, L. E. A cortical evoked potential that reflects the conditioned, positive incentive value of the stimulus. I. The evoked potential. Electroencephalography and Clinical Neurophysiology, 1977, 42, 341-354.

Boyd, E. S., Boyd, E. H. & Brown, L. E. A cortical evoked potential that reflects the conditioned, positive incentive value of the stimulus. II. Drive motivation (hunger). Electroencephalography and Clinical Neurophysiology, 1977, 42, 355-363.

Boyd, E. S., Boyd, E. H. & Brown, L. E. A cortical evoked potential that reflects the conditioned, positive incentive value of the stimulus. III. Manipulative motivation and the value of the reward. Electroencephalography and Clinical Neurophysiology, 1977, 42, 364-371.

Boyd, E. S., Boyd, E. H. & Brown, L. E. Observations on the M-wave and the CNV in the squirrel monkey. Electroencephalography and Clinical Neurophysiology, 1979, 46, 320-336.

Boyd, E. S., Boyd, E. H., & Brown, L. E. The M-wave and CNV in the squirrel monkey: generality of cue modality and reward. Electroencephalography and Clinical Neurophysiology, 1980, 49, 66-80.

Bradshaw, J. L. Pupillary changes and reaction time witn varied stimulus uncertainty. Psychonomic Science, 1968, 13, 69-70.

Campbell, K. B. The effects of outcome of decision, task relevant information, surprisal, incentive, and value on the human evoked potential. Thesis, University of Ottawa, 1976.

Campbell, K. B., Courchesne, E., Picton, T. W. & Squires, K. C. Evoked potential correlates of human information processing. Biological Psychology, 1979, 8, 45-68.

Chapman, L. J., Chapman, J. P. & Brelje, T. Influence of the experimenter on pupillary dilation to sexually provocative pictures.

Journal of Abnormal Psychology, 1969, *74*, 396-400.

Chesney, G. I. & Donchin, E. Predictions, their confirmation and the P300 component. *Psychophysiology*, 1979, *16*, 174.

Clark, W. R. & Johnson, D. A. Effects of instructional set on pupillary responses during a short-term memory task. *Journal of Experimental Psychology*, 1970, *85*, 315-317.

Corby, J. C. & Kopell, B. S. Differential contributions of blinks and vertical eye movements as artifacts in EEG recording. *Psychophysiology*, 1972, *9*, 640-644.

Cornsweet, T. N. The staircase-method in psychophysics. *American Journal of Psychology*, 1962, *75*, 485-491.

Courchesne, E. Event-related brain potentials: comparison between children and adults. *Science*, 1977, *197*, 589-592.

Courchesne, E., Courchesne, R. Y. & Hillyard, S. A. The effect of stimulus deviation on P3 waves to easily recognized stimuli. *Neuropsychologia*, 1978, *16*, 189-199.

Courchesne, E., Hillyard, S. A. & Galambos, R. Stimulus novelty and the visual evoked potential in man. *Electroencephalography and Clinical Neurophysiology*, 1975, *39*, 131-143.

Dixon, W. J. (Ed.). *BMD Biomedical computer programs*. Berkeley: Univ. of California Press, 1968, 586-600.

Donald, M. W. & Goff, W. R. Attention-related increases in cortical responsivity dissociated from the contingent negative variation. *Science*, 1971, *172*, 1163-1166.

Donchin, E. Use of scalp distribution as a dependent variable in event-related potential studies: excerpts of preconference correspondence. In D. A. Otto (Ed.), *Multidisciplinary perspectives in event-related potential research (EPIC IV)*. Washington: U. S. Environmental Protection Agency, 1978, 501-514.

Donchin, E. Event-related brain potentials: a tool in the study of human information processing. In H. Begleiter (Ed.), *Evoked brain potentials and behavior*. New York: Plenum Press, 1979, 13-88.

Donchin, E., Callaway, E., Cooper, R., Desmedt, J. E., Goff, W. R., Hillyard, S. A. & Sutton, S. Publication criteria for studies of evoked potentials (EP) in man. In J. E. Desmedt (Ed.), *Progress in clinical neurophysiology, Vol. 1. Attention, voluntary contraction and event-related cerebral potentials*. Basel: Karger, 1977, 1-11.

Donchin, E., Gerbrandt, L. A., Leifer, L. & Tucker, L. Is the contingent negative variation contingent on a motor response? *Psychophysiology*, 1972, 9, 179-188.

Donchin, E. & Heffley, E. F. The independence of the P300 and CNV reviewed: a reply to Wastell. Biological Psychology, 1979, 9, 177-188.

Donchin, E., Ritter, W. & McCallum, W. C. Cognitive psychophysiology: the endogenous components of the ERP. In E. Callaway, P. Tueting & S. Koslow (Eds.), Event-related brain potentials in man. New York: Academic Press, 1978, 349-411.

Donchin, E. & Smith, D. B. D. The contingent negative variation and the late positive wave of the average evoked potential. Electroencephalography and Clinical Neurophysiology, 1970, 29, 201-203.

Donchin, E., Tueting, P., Ritter, W., Kutas, M. & Heffley, E. On the independence of the CNV and the P300 components of the human averaged evoked potential. Electroencephalography and Clinical Neurophysiology, 1975, 38, 449-461.

Dooley, R. P. & Lehr, D. J. Critique of a pupillary response experiment. Perceptual and motor skills, 1967, 25, 603-604.

Fernandez de Molina, A. & Hunsberger, R. W. Organization of the subcortical system governing defence and flight reactions in the cat. Journal of Physiology, 1962, 160, 200-213.

Ford, J. M., Roth, W. T. & Kopell, B. S. Attention effects on auditory evoked potentials to infrequent events. Biological Psychology, 1976, 7, 65-77.

Friedman, D. The effects of stimulus uncertainty on the pupillary dilation response and the vertex evoked potential in man. Doctoral dissertation, City University of New York, 1972.

Friedman, D., Erlenmeyer-Kimling, L. & Vaughan, H. G., Jr. Event-realted potential (ERP) methodology in high-risk research. In N. Watt (Ed.), Proceedings of the plenary conference on children at risk for schizophrenia. in press.

Friedman, D., Hakerem, G., Sutton, S. & Fleiss, J. L. Effect of stimulus uncertainty on the pupillary dilation response and the vertex evoked potential. Electroencephalography and Clinical Neurophysiology, 1973, 34, 475-484.

Gardiner, M. F. & Walter, D. O. Differences between human evoked potentials elicited by the same acoustical stimuli during loudness discrimination tasks. In E. Donchin & D. B. Lindsley (Eds.), Average evoked potentials: Methods, results and evaluations. Washington, D. C.: U. S. Government Printing Office, 1969, 335-342.

Geisser, S. & Greenhouse, S. W. An extension of Box's results on the use of the F distribution in multivariate analysis. The Annals of Mathemematical Statistics, 1958, 24, 885-891.

Gilden, L., Vaughan, H. G., Jr. & Costa, L. D. Summated human EEG potentials with voluntary movement. *Electroencephalography and Clinical Neurophysiology*, 1966, *20*, 433-438.

Girton, D. G. & Kamiya, J. A simple on-line technique for removing eye movement artifacts from the EEG. *Electroencephalography and Clinical Neurophysiology*, 1973, *34*, 212-216.

Goldwater, B. Psychological significance of pupillary movements. *Psychological Bulletin*, 1972, *77*, 340-355.

Hakerem, G. Pupillography. In P. H. Venables & I. Martin (Eds.), *A manual of psychophysiological methods*. Amsterdam: North-Holland Pub. Co., 1967, 335-349.

Hakerem, G. The effect of cognitive manipulation on pupillary diameter and vertex evoked potentials. In E. Dodt & K. E. Schrader (Eds.), *Die normale und die gestorte Pupillenbewegung*. Munchen: Bergmann Verlag, 1973, 239-245.

Hakerem, G. Conceptual stimuli, pupillary dilation and evoked cortical potentials: a review of recent advances. In M. P. Janisse (Ed.), *Pupillary dynamics and behavior*. New York: Plenum Press, 1974, 135-158.

Hakerem, G. & Sutton, S. Pupillary response at visual threshold. *Nature*, 1966, *212*, 485-486.

Halgren, E., Squires, N. K., Wilson, C. L., Rohrbaugh, J. W., Babb, T. L. & Crandall, P. H. Endogenous potentials generated in the human hippocampal formation and amygdala by infrequent events. *Science*, 1980, *210*, 803-805.

Hammond, E. J., Silva, D. A., Klein, A. J. & Teas, D. C. A technique for separating endogenous from exogenous human cortical potentials. *Electroencephalography and Clinical Neurophysiology*, 1979, *46*, 482-485.

Haughney, G. V. *Pupillary responses as a function of prediction and outcome*. Unpublished Master's Thesis, New Mexico State University, 1975 (cited by Johnston, 1979).

Hess, E. H. Attitude and pupil size. *Scientific American*, 1965, *212*, 46-54.

Hess, E. H. Pupillometrics. In N. S. Greenfield & R. A. Sternbach (Eds.), *Handbook of psychophysiology*. New York: Holt, Rinehart & Winston, 1972, 491-531.

Hess, E. H. *The tell-tale eye*. New York: Van Nostrand Reinhold, 1975.

Hess, E. H. & Polt, J. M. Pupil size as related to interest value of visual stimuli. *Science*, 1960, *132*, 349-350.

Hess, E. H. & Polt, J. M. Pupil size in relation to mental activity during simple problem solving. Science, 1964, 143, 1190-1192.

Hess, E. H. & Polt, J. M. Reply to "critique of a pupillary response experiment" by Roger P. Dooley and Donald J. Lehr. Perceptual and Motor Skills, 1967, 25, 659-660.

Hess, E. H., Seltzer, A. L. & Shlien, J. M. Pupil responses of hetero- and homosexual males to pictures of men and women: a pilot study. Journal of Abnormal Psychology, 1965, 70, 165-168.

Hillyard, S. A. Methodological issues in CNV research. In R. F. Thompson & M. M. Patterson (Eds.), Bioelectric recording techniques. Part B: Electroencephalography and human brain potentials. New York: Academic Press, 1974, 281-304.

Hillyard, S. A., Courchesne, E., Krausz, H. I. & Picton, T. W. Scalp topography of the P3 wave in different auditory decision tasks. In W. C. McCallum & J. R. Knott (Eds.), The responsive brain. Bristol: Wright & Sons, 1976, 81-87.

Hillyard, S. A. & Galambos, R. Eye movement artifact in the CNV. Electroencephalography and Clinical Neurophysiology, 1970, 28, 173-182.

Hillyard, S. A. & Picton, T. W. Event-related brain potentials and selective information processing in man. In J. E. Desmedt (Ed.), Progress in clinical neurophysiology, Vol. 6. Cognitive components in cerebral event-related potentials and selective attention. Basel: Karger, 1979, 1-52.

Hillyard, S. A., Squires, K. C., Bauer, J. W. & Lindsay, P. H. Evoked potential correlates of auditory signal detection. Science, 1971, 172, 1357-1360.

Homberg, V., Grunewald, G. & Grunewald-Zuberbier, E. The variation of P300 amplitude in a money-winning paradigm in children. Psychophysiology, 1981, 18, 258-262.

Hutt, L. D. & Anderson, J. D. The relationship between pupil size and recognition threshold. Psychonomic Science, 1967, 9, 477-478.

Irwin, D. A., Knott, J. R., McAdam. D. W. & Rebert, C. S. Motivational determinants of the "Contingent Negative Variation." Electroencephalography and Clinical Neurophysiology, 1966, 21, 538-543.

Irwin, D. A. & Rebert, C. S. Slow potential changes in cat brain during classical appetitive conditioning of jaw movements using two levels of reward. Electroencephalography and Clinical Neurophysiology, 1970, 28, 119-126.

Israel, J. B., Wickens, C. D., Chesney, G. L. & Donchin, E. The event-related brain potential as an index of display-monitoring workload. Human Factors, 1980, 22, 211-224.

Janisse, M. P. Pupil size and affect: a critical review of the literature since 1960. Canadian Psychologist, 1973, 14, 311-329.

Janisse, M. P. Pupillometry: the psychology of the pupillary response. New York: Hemisphere Pub., 1977.

Jasper, H. H. Report of committee on methods of clinical examination in EEG. Appendix: The ten twenty electrode system of the International Federation. Electroencephalography and Clinical Neurophysiology, 1958, 10, 371-375.

Jenness, D. Auditory evoked-response differentiation with discrimination learning in humans. Journal of Comparative and Physiological Psychology, 1972a, 80, 75-90.

Jenness, D. Stimulus role and gross differences in the cortical evoked response. Physiology and Behavior, 1972b, 9, 141-146.

Jennings, J. R. & Wood, C. C. The epsilon-adjustment procedure for repeated-measures analyses of variance. Psychophysiology, 1976, 13, 277-278.

John, E. R. Electrophysiological studies of conditioning. In G. C. Quarton, T. Melnechuk and F. O. Schmitt (Eds.), The neurosciences: a study program. New York: Rockefeller University Press, 1967a, 690-704.

John, E. R. Mechanisms of memory. New York: Academic Press, 1967b.

Johnson, R., Jr. & Donchin, E. On how amplitude of P300 varies with the utility of eliciting stimuli. Electroencephalography and Clinical Neurophysiology, 1978, 44, 424-427.

Johnston, V. S. Stimuli with biological significance. In H. Begleiter (Ed.), Evoked brain potentials and behavior. New York: Plenum Press, 1979, 1-12.

Johnston, V. S. & Holcomb, P. J. Probability learning and the P3 component of the visual evoked potential in man. Psychophysiology, 1980, 17, 396-400.

Juris, M. & Velden, M. The pupillary response to mental overload. Physiological Psychology, 1977, 5, 421-424.

Kahneman, D. Attention and effort. New York: Prentice-Hall, 1973.

Kahneman, D. & Beatty, J. Pupil diameter and load on memory. Science, 1966, 154, 1583-1585.

Kahneman, D. & Beatty, J. Pupillary response in a pitch discrimination task. Perception & Psychophysics, 1967, 2, 101-105.

Kahneman, D., Beatty, J. & Pollack, F. Perceptual deficit during a mental task. Science, 1967, 157, 218-219.

Kahneman, D., Onuska, L. & Wolman, R. Effects of grouping on pupillary response in a short-term memory task. *Quarterly Journal of Experimental Psychology*, 1968, *20*, 309-311.

Kahneman, D. & Peavler, W. S. Incentive effects and pupillary changes in association learning. *Journal of Experimental Psychology*, 1969, *79*, 312-318.

Kahneman, D., Peavler, W. S. & Onuska, L. Effects of verbalization and incentive on the pupillary response to mental activity. *Canadian Journal of Psychology*, 1968, *22*, 186-196.

Kahneman, D., Tursky, B., Shapiro, D. & Crider, A. Pupillary, heart rate and skin resistance changes during a mental task. *Journal of Experimental Psychology*, 1969, *79*, 164-167.

Karlin, L. Cognition, preparation, and the sensory-evoked potentials. *Psychological Bulletin*, 1970, *73*, 122-136.

Karlin, L., Martz, M. J., Brauth, S. E. & Mordkoff, A. M. Auditory evoked potentials, motor potentials and reaction time. *Electroencephalography and Clinical Neurophysiology*, 1971, *31*, 129-136.

Karrer, R., Kohn, H. & Ivins, J. Effects of varying the stimulus and response contingencies on the CNV. In W. C. McCallum & J. R. Knott (Eds.), *Event-related slow potentials of the brain: their relations to behavior*. Amsterdam: Elsevier, 1973, 39-43 (EEGJ., Suppl. 33).

Kerkhof, G. A. Decision latency: the P3 component in auditory signal detection. *Neuroscience Letters*, 1978, *8*, 289-294.

Klinke, R., Fruhstorfer, H. & Finkenzeller, P. Evoked responses as a function of external and stored information. *Electroencephalography and Clinical Neurophysiology*, 1968, *25*, 119-122.

Kohn, M. & Clynes, M. Color dynamics of the pupil. *Annals of the N. Y. Academy of Sciences*, 1969, *156*, 931-950.

Koikegami, H. & Yoshida, K. Pupillary dilation induced by stimulation of amygdaloid nuclei. *Folia Psychiatrica Neurologica Japonica*, 1953, *7*, 109-125.

Kooi, K. A., Tipton, A. C. & Marshall, R. E. Polarities and field configurations of the vertex components of the human auditory evoked response. *Electroencephalography and Clinical Neurophysiology*, 1971, *31*, 166-169.

Lefcourt, H. M. *Locus of control: current trends in theory and research*. Hillsdale, New Jersey: Erlbaum, 1976.

Leifer, L. J., Otto, D. A., Hart, S. G. & Huff, E. M. Slow potential correlates of predictive behavior during a complex learning task. In W. C. McCallum & J. R. Knott (Eds.), The responsive brain. Bristol: Wright & Sons, 1976, 65-70.

Levine, S. Pupillary dilation as a function of stimulus uncertainty. Unpublished Master's Thesis, Queens College, 1969.

Libby, W. I., Jr., Lacey, B. & Lacey, J. I. Pupillary and cardiac activity during visual attention. Psychophysiology, 1973, 10, 270-294.

Lindsley, D. B. Attention, consciousness, sleep, and wakefulness. In J. Field, H. W. Magoun and V. E. Hall (Eds.), Handbook of physiology, vol. III. Washington, D. C.: American Physiological Society, 1960, 1553-1593.

Loewenfeld, I. Mechanisms of reflex dilatation of the pupil: historical review and experimental analysis. Documenta Ophthalmologica, 1958, 12, 185-448.

Loewenfeld, I. Comment on Hess' findings. Survey of Ophthalmology, 1966, 11, 291-294.

Loewy, A. D. & Saper, C. B. Edinger-Westphal nucleus: projections to the brain stem and spinal cord in the cat. Brain Research, 1978, 150, 1-27.

Lowenstein, O. Pupillary reflex shapes and topical clinical diagnosis. Neurology, 1955, 5, 631-644.

Lowenstein, O. & Loewenfeld, I. E. Role of sympathetic and parasympathetic systems in reflex dilatation of the pupil. Archives of Neurology and Psychiatry, 1950, 64, 313-340.

Lowenstein, O. & Loewenfeld, I. E. Basic mechanisms of pupillary movements. Transactions of the New York Academy of Sciences, 1961, 23, 579-586.

Lowenstein, O. & Loewenfeld, I. The pupil. In H. Davson (Ed.), The eye: vol. 3. New York: Academic Press, 1962, 231-267.

Lynn, A. Attention, arousal and the orientation reaction. New York: Pergamon Press, 1966.

MacLean, P. D. Psychosomatic disease and the "visceral brain": recent developments bearing on the Papez theory of emotion. Psychosomatic Medicine, 1949, 11, 338-353.

Maltzman, I. Orienting reflexes and significance: a reply to O'Gorman. Psychophysiology, 1979, 16, 274-282.

Marsh, G. R., Poon, L. W. & Thompson, L. W. Some relationships between CNV, P300 and task demands. In W. C. McCallum & J. R. Knott (Eds.), The responsive brain. Bristol: Wright & Sons, 1976, 122-125.

McAdam, D. W. Slow potential changes recorded from human brain during learning of a temporal interval. Psychonomic Science, 1966, 6, 435-436.

McAdam, D. W. & Rubin, E. H. Readiness potential, vertex positive wave, contingent negative variation and accuracy of perception. Electroencephalography and Clinical Neurophysiology, 1971, 30, 511-517.

McAdam, D. W. & Seales, D. M. Bereitschaftspotential enhancement with increased level of motivation. Electroencephalography and Clinical Neurophysiology, 1969, 27, 73-75.

Milstein, V., Small, J. G., Gans, G. E. & Moore, J. E. Risk taking and the CNV. Electroencephalography and Clinical Neurophysiology, 1974, 37, 434.

Morrell, L. & Salamy, J. Hemispheric asymmetry of electrocortical responses to speech stimuli. Science, 1971, 174, 164-166.

Naatanen, R. Selective attention and evoked potentials. Annales Academiae Scientiarum Fennicae, 1967, 151, 1-226.

Naatanen, R. Orienting and evoked potentials. In H. D. Kimmel, E. H. van Olst & J. F. Orlebeke (Eds.), The orienting reflex in humans. Hillsdale: Lawrence Erlbaum, 1979, 61-75.

Naatanen, R. & Michie, P. T. Different variants of endogenous negative brain potentials in performance situations: a review and classification. In D. Lehmann & E. Callaway (Eds.), Human evoked potentials: applications and problems. New York: Plenum Press, 1979, 251-267.

Nunnally, J. C., Knott, P. D., Duchnowski, A. & Parker, R. Pupillary response as a general measure of activation. Perception & Psychophysics, 1967, 2, 149-155.

O'Gorman, J. G. The orienting reflex: novelty or significance detector? Psychophysiology, 1979, 16, 253-262.

Papez, J. W. A proposed theory of emotion. Archives of Neurology and Psychiatry, 1937, 38, 725-744.

Paivio, A. & Simpson, H. M. The effect of word abstractness and pleasantness on pupil size during an imagery task. Psychonomic Science, 1966, 5, 55-56.

Papakostopoulos, D., Winter, A. & Newton, P. New techniques for the control of eye potential artefacts in multichannel recording. Electroencephalography and Clinical Neurophysiology, 1973, 34, 651-653.

Paul, D. D. & Sutton, S. Evoked potential correlates of response criterion in auditory signal detection. Science, 1972, 177, 362-364.

Peavler, W. S. Pupil size, information overload, and performance differences. Psychophysiology, 1974, 11, 559-566.

Peavler, W. S. & McLaughlin, J. P. The question of stimulus content and pupil size. Psychonomic Science, 1967, 8, 505-506.

Peters, J. F., Hamilton, C. E. & Knott, J. R. Stimulus certainty, subject set and event related potentials. Electroencephalography and Clinical Neurophysiology, 1974, 37, 432.

Peters, J. F., Knott, J. R., Miller, L. H., Van Veen, W. J. & Cohen, S. I. Response variables and magnitude of the contingent negative variation. Electroencephalography and Clinical Neurophysiology, 1970, 29, 608-611.

Picton, T. W. & Hillyard, S. A. Human auditory evoked potentials. II: Effects of attention. Electroencephalography and Clinical Neurophysiology, 1974, 36, 191-200.

Picton, T. W., Hillyard, S. A., Krausz, H. I. & Galambos, R. Human auditory evoked potentials. I: Evaluation of components. Electroencephalography and Clinical Neurophysiology, 1974, 36, 179-190.

Picton, T. W., Hillyard, S. A. & Galambos, R. Cortical evoked responses to omitted stimuli. In M. N. Livanov (Ed.), Major problems of brain electrophysiology. Moscow: Nauka, 1974, 302-311.

Picton, T., Woods, D., Stuss, D. & Campbell, K. Methodology and meaning of human evoked-potential scalp distribution studies. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related potential research (EPIC IV). Washington: U. S. Environmental Protection Agency, 1978, 515-522.

Poon, L. W., Thompson, L. W., Williams, R. B., Jr. & Marsh, G. R. Changes of antero-posterior distribution of CNV and late positive component as a function of information processing. Psychophysiology, 1974, 11, 660-673.

Prichep, L. S. Attention and the auditory evoked potential in hyperkinetic children treated with methylphenidate and in normal children. Doctoral dissertation, City University of New York, 1974.

Prichep, L. S., Sutton, S. & Hakerem, G. Evoked potentials in hyperkinetic and normal children under certainty and uncertainty: A placebo and methylphenidate study. Psychophysiology, 1976, 13, 419-427.

Purves, S. J. & Low, M. D. Visual evoked and emitted potentials; analysis of single trial responses. Electroencephalography and Clinical Neurophysiology, 47, 21P-22P.

Rebert, C. S. & Knott, J. R. The vertex non-specific evoked potential and latency of contingent negative variation. Electroencephalography and Clinical Neurophysiology, 1970, 28, 561-565.

Renault, B. & Lesevre, N. Topographical study of the emitted potential obtained after the omission of an expected stimulus. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related potential research (EPIC IV). Washington: U. S. Environmental Protection Agency, 1978, 202-205.

Renault, B. & Lesevre, N. A trial by trial study of the visual omission response in reaction time situations. In D. Lehmann & E. Callaway (Eds.), Human evoked potentials: applications and problems. New York: Plenum Press, 1979, 317-329.

Ritter, W., Simson, R. & Vaughan, H. G., Jr. Topographic analysis of task-related cerebral potentials. In J. E. Desmedt (Ed.), Progress in clinical neurophysiology, Vol. 6. Cognitive components in cerebral event-related potentials and selective attention. Basel: Karger, 1979, 132-139.

Ritter, W. & Vaughan, H. G., Jr. Averaged evoked responses in vigilance and discrimination: a reassessment. Science, 1969, 164, 326-328.

Rohrbaugh, J. W., Syndulko, K. & Lindsley, D. B. Cortical slow negative waves following non-paired stimuli. Electroencephalography and Clinical Neurophysiology, 1978, 45, 551-567.

Rose, R. M., Teller, D. Y. & Rendleman, P. Statistical properties of staircase estimates. Perception & Psychophysics, 1970, 8, 199-204.

Rosenberger, P. B. Response-adjusting stimulus intensity. In W. C. Stebbins (Ed.), Animal psychophysics: the design and conduct of sensory experiments. New York: Appleton-Century-Crofts, 1970, 161-184.

Roth, W. T. How many late positive waves are there? In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related potential research (EPIC IV). Washington: U. S. Environmental Protection Agency, 1978, 170-172.

Roth, W. T., Krainz, P. L., Ford, J. M., Tinklenberg, J. R., Rothbart, R. M. & Kopell, B. S. Parameters of temporal recovery of the human auditory evoked potential. Electroencephalography and Clinical Neurophysiology, 1976, 40, 623-632.

Rotter, J. B. Generalized expectancies for internal versus external control of reinforcement. Psychological Monographs, 1966, 80 (1, Whole No. 609).

Ruchkin, D. S. & Sutton, S. Visual evoked and emitted potentials and stimulus significance. Psychonomic Science, 1973, 2, 144-146.

Ruchkin, D. S. & Sutton, S. Emitted P300 potentials and temporal uncertainty. Electroencephalography and Clinical Neurophysiology, 1978a, 45, 268-277.

Ruchkin, D. S. & Sutton, S. Equivocation and P300 amplitude. In D. A. Otto (Ed.), *Multidisciplinary perspectives in event-related potential research (EPIC IV)*. Washington: U. S. Environmental Protection Agency, 1978b, 175-177.

Ruchkin, D. S. & Sutton, S. Latency characteristics and trial by trial variation of emitted potentials. In J. E. Desmedt (Ed.), *Progress in clinical neurophysiology, Vol. 6. Cognitive components in cerebral event-related potentials and selective attention.* Basel: Karger, 1979a, 106-118.

Ruchkin, D. S. & Sutton, S. CNV and P300 relationships for emitted and for evoked cerebral potentials. In J. E. Desmedt (Ed.), *Progress in clinical neurophysiology, Vol. 6. Cognitive components in cerebral event-related potentials and selective attention.* Basel: Karger, 1979b, 119-131.

Ruchkin, D. S., Sutton, S., Kietzman, M. L. & Silver, K. Slow wave and P300 in signal detection. *Electroencephalography and Clinical Neurophysiology*, 1980, *50*, 35-47.

Ruchkin, D. S., Sutton, S., Munson, R., Silver, K. & Macar, F. P300 and feedback provided by absence of the stimulus. *Psychophysiology*, 1981, *18*, 271-282.

Ruchkin, D. S., Sutton, S. & Stega, M. Emitted P300 and slow wave event-related potentials in guessing and detection tasks. *Electroencephalography and Clinical Neurophysiology*, 1980, *49*, 1-14.

Ruchkin, D. S., Sutton, S.& Tueting, P. Emitted and evoked P300 potentials and variation in stimulus probability. *Psychophysiology*, 1975, *12*, 591-595.

Rusinov, V. S. Electroencephalographic studies in conditional reflex formation in man. In M. A. B. Brazier (Ed.), *The central nervous system and behavior*. New York: Josiah Macy Foundation, 1959, 249-256.

Shiga, N. & Ohkubo, Y. Pupillary movments during shadowing. *Tohoku Psychologica Folia*, 1978, *37*, 16-24.

Shiga, N.& Ohkubo, Y. Pupillary responses to auditory stimuli. *Tohoku Psychologica Folia*, 1979, *38*, 57-65.

Simpson, H. M. Effects of a task relevant response on pupil size. *Psychophysiology*, 1969, *6*, 115-121.

Simpson, H. M. & Climan, M. H. Pupillary and electromyographic changes during an imagery task. *Psychophysiology*, 1971, 8, 483-490.

Simson, R., Vaughan, H. G., Jr,. & Ritter, W. The scalp topography of potentials associated with missing auditory or visual stimuli. *Electroencephalography and Clinical Neurophysiology*, 1976, *40*, 33-42.

Sokolov, Y. N. (sic) Perception and the conditioned reflex. Oxford: Pergamon Press, 1963.

Sokolov, E. N. The modeling properties of the nervous system. In M. Cole & I. Maltzman (Eds.), A handbook of contemporary soviet psychology. New York: Basic Books, 1969, 671-704.

Squires, K. C., Donchin, E., Herning, R. I. & McCarthy, G. On the influence of task relevance and stimulus probability on event-related potential components. Electroencephalography and Clinical Neurophysiology, 1977, 42, 1-14.

Squires, K., Hillyard, S. A. & Lindsay, P. Cortical potentials evoked by confirming and disconfirming feedback following an auditory discrimination. Perception & Psychophysics, 1973a, 13, 25-31.

Squires, K., Hillyard, S. A. & Lindsay, P. Vertex potentials evoked during auditory signal detection: relation to decision criteria. Perception & Psychophysics, 1973b, 14, 265-272.

Squires, K., Squires, N. & Hillyard, S. A. Vertex evoked potentials in a rating-scale detection task: Relation to signal probability. Behavioral Biology, 1975a, 13, 21-34.

Squires, K., Squires, N. & Hillyard, S. A. Decision-related cortical potentials during an auditory signal detection task with cued observation intervals. Journal of Experimental Psychology: Human Perception and Performance, 1975b, 1, 268-279.

Squires, K. C., Wickens, C., Squires, N. K. & Donchin, E. The effect of stimulus sequence on the waveform of the cortical event-related potential. Science, 1976, 193, 1142-1146.

Squires, K. C., Wickens, C., Squires, N. K. & Donchin, E. Sequential dependencies of the waveform of the event-related potential: a preliminary report. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related potential research (EPIC IV). Washington: U. S. Environmental Protection Agency, 1975, 178-221.

Squires, N. K., Donchin, E., Squires, K. C. & Grossberg, S. Bisensory stimulation: inferring decision-related processes from the P300 component. Journal of Experimental Psychology: Human Perception and Performance, 1977, 3, 299-315.

Squires, N., Squires, K. & Hillyard, S. A. Two varieties of long-latency positive waves evoked by unpredictable auditory stimuli in man. Electroencephalography and Clinical Neurophysiology, 1975, 38, 387-401.

Squires, N., Squires, K. & Hillyard, S. A. Functional equivalence of signal-present, signal-absent, and threshold-detect P3s. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related potential research (EPIC IV). Washington: U. S. Environmental Protection Agency, 1978, 218-221.

Steinhauer, S. R., Hakerem, G. & Spring, B. The pupillary response as a potential indicator of vulnerability to schizophrenia. Psychopharmacology Bulletin, 1979, 15, 44-45.

Stelmack, R. M. & Mandelzys, N. Extroversion and pupillary response to affective and taboo words. Psychophysiology, 1975, 12, 536-540.

Stuss, D. T. & Picton, T. W. Neurophysiological correlates of human concept formation. Behavioral Biology, 1978, 23, 135-162.

Sutton, S. The specification of psychological variables in an average evoked potential experiment. In E. Donchin & D. B. Lindsley (Eds.), Average evoked potentials: Methods, results and evaluations. Washington, D. C.: U. S. Government Printing Office, 1969, 237-262.

Sutton, S. The sensitivity of the evoked potential to psychological variables. Presented at the meetings of the EEG Society, Washington, D. C., 1970. Electroencephalography and Clinical Neurophysiology, 1971, 31, 302 (abstract).

Sutton, S. P300 -- thirteen years later. In H. Begleiter (Ed.), Evoked brain potentials and behavior. New York: Plenum Press, 1979, 107-126.

Sutton, S., Braren, M., Zubin, J. & John, E. R. Evoked-potential correlates of stimulus uncertainty. Science, 1965, 150, 1187-1188.

Sutton, S. & Tueting, P. The sensitivity of the evoked potential to psychological variables. In P. H. Venables & M. J. Christie (Eds.), Research in psychophysiology. New York: Wiley & Sons, 1975, 351-363.

Sutton, S., Tueting, P., Hammer, M. & Hakerem, G. Evoked potentials and feedback. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related potential research (EPIC IV). Washington: U. S. Environmental Protection Agency, 1978, 184-187.

Sutton, S., Tueting, P., Zubin, J. & John, E. R. Information delivery and the sensory evoked potential. Science, 1967, 155, 1436-1439.

Tecce, J. J. Contingent negative variation (CNV) and psychological processes in man. Psychological Bulletin, 1972, 77, 73-108.

Truex, R. C. & Carpenter, M. B. Human neuroanatomy, sixth edition. Baltimore: Williams & Wilkins, 1969.

Tryon, W. W. Pupillometry: a survey of sources of variation. Psychophysiology, 1975, 12, 90-93.

Tueting, P. Event-related potentials, cognitive events, and information processing. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related potential research (EPIC IV). Washington: U. S. Environmental Protection Agency, 1978, 159-169.

Tueting, P. & Sutton, S. The relationship between pre-stimulus and post-stimulus components of the averaged evoked potential. In S. Kornblum (Ed.), Attention and performance IV. New York: Academic Press, 1973, 185-207.

Tueting, P., Sutton, S. & Zubin, J. Quantitative evoked potential correlates of the probability of events. Psychophysiology, 1970, 7, 385-394.

Vacchiano, R. B., Strauss, P. S., Ryan, S. & Hochman, L. Pupillary response to value linked words. Perceptual and Motor Skills, 1968, 27, 207-210.

Vaughan, H. G., Jr. The analysis of scalp recorded brain potentials. In R. F. Thompson & M. M. Patterson (Eds.), Bioelectric recording techniques. Part B: Electroencephalography and human brain potentials. New York: Academic Press, 1974, 158-207

Vaughan, H. G., Jr. & Ritter, W. The sources of auditory evoked responses recorded from the human head. Electroencephalography and Clinical Neurophysiology, 1970, 28, 360-367.

Velden, M. Some necessary revisions of the neuronal model concept of the orienting response. Psychophysiology, 1978, 15, 181-185.

Walter, W. G., Cooper,, R., Aldridge, V. J., McCallum, W. C. & Winter, A. L. Contingent negative variation: an electric sign of sensorimotor association and expectancy in the human brain. Nature, 1964, 203, 380-384.

Wastell, D. G. On the independence of of P300 and the CNV: a short critique of the principal components analysis of Donchin et al. (1975). Biological Psychology, 1979, 9, 171-176.

Weinberg, H., Walter, W. G., Cooper, R. & Aldridge, V. J. Emitted cerebral events. Electroencephalography and Clinical Neurophysiology, 1974, 36, 449-456.

Weinberg, H., Walter, W. G. & Crow, H. J. Intracerebral events in humans related to real and imaginary stimuli. Electroencephalography and Clinical Neurophysiology, 1970, 29, 1-9.

White, G. L. & Maltzman, I. Pupillary activity while listening to verbal passages. Journal of Research in Personality, 1978, 12, 361-369.

Wilkinson, R. T. & Morlock, H. C., Jr. Auditory evoked response and reaction time. Electroencephalography and Clinical Neurophysiology, 1967, 23, 50-56.

Winer, B. J. Statistical principles in experimental design. New York: McGraw Hill, 1971.

Woodmansee, J. Methodological problems in pupillographic experiments. Proceedings, 74th meeting of the American Psychological Association, 1966, _1_, 133-134.

Woody, C. D. Characterization of an adaptive filter for the analysis of variable latency neuroelectric signals. _Medical and Biological Engineering_, 1967, _5_, 539-553.

Zinn, K. M. _The pupil_. Springfield: Charles C. Thomas, 1972.

APPENDIX

Analysis of Variance Summary Tables for Pupillary and Event-Related Potential Amplitudes and Eye Artifact Analysis

All F-tests based on df=(1,7)

Error term used is the interaction of each factor with the factor "Subjects."

ANOVA FOR BASELINE PUPIL DIAMETER ACROSS STIMULI

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.001	1970.4	4547.13	1,7	4547.13
2	B=BET SOURCE	BS	p<.05	10.288	.199839	1,7	.199839
3	S=SUBJECTS				16.1539	7	2.30770
4	O=OUTCOME	SO		0.392	.759375E-01	1,7	.759375E-01
5	V=BET VALUE	SV		0.477	.108957E-02	2,14	.544787E-03
6	BS				.135962	7	.194231E-01
7	BO	BSO		0.033	.202087E-03	1,7	.202087E-03
8	SO				1.35496	7	.193566
9	BV	BSV		3.754	.952950E-02	2,14	.476475E-02
10	SV				.159764E-01	14	.114117E-02
11	OV	SOV		1.505	.410640E-02	2,14	.205320E-02
12	BSO				.428629E-01	7	.612328E-02
13	BSV				.177696E-01	14	.126926E-02
14	BOV	BSOV		0.722	.241664E-02	2,14	.120832E-02
15	SOV				.190943E-01	14	.136388E-02
16	BSOV				.234167E-01	14	.167262E-02

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(diameter in millimeters)

	...Days 2 - 4...			...Days 5 - 7...			CER-	
	0	25	50	0	25	50	TAIN	S
SUBJECT	6.71	6.77	6.82	7.00	7.02	7.07	7.03	1
BET	6.56	6.61	6.66	6.54	6.56	6.56	6.37	2
	7.05	7.09	7.06	7.08	7.06	7.06	7.06	3
	6.29	6.29	6.28	6.34	6.36	6.34	6.27	4
	6.94	7.03	7.07	6.90	6.96	6.97	6.95	5
	7.68	7.70	7.67	7.86	7.85	7.82	7.88	6
	7.28	7.44	7.33	7.13	7.02	7.11	7.33	7
	6.88	6.88	6.82	6.31	6.32	6.39	6.53	8
MEAN	6.92	6.98	6.96	6.90	6.89	6.92	6.93	
s.d.	0.43	0.45	0.42	0.51	0.49	0.48	0.53	
COMPUTER	6.62	6.64	6.65	6.87	6.94	6.89		1
BET	6.58	6.63	6.62	6.61	6.58	6.58		2
	7.08	7.05	6.97	6.98	7.03	7.03		3
	6.30	6.25	6.27	6.31	6.30	6.31		4
	7.00	6.93	6.97	6.82	6.68	6.78		5
	7.59	7.61	7.63	7.82	7.78	7.80		6
	7.02	7.02	7.05	6.98	6.95	6.86		7
	6.78	6.76	6.77	6.18	6.15	6.14		8
MEAN	6.87	6.86	6.87	6.82	6.80	6.80		
s.d.	0.39	0.40	0.40	0.50	0.51	0.51		

ANALYSIS OF VARIANCE FOR PEAK DILATION COMBINED ACROSS STIMULI

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	12.473	.849384	1,7	.849384
2	B=BET SOURCE	BS	p<.01	17.624	.255234	1,7	.255234
3	S=SUBJECTS				.476657	7	.068093
4	O=OUTCOME	SO		0.456	.142604E-02	1,7	.142604E-02
5	V=BET VALUE	SV	p<.05	7.733	.290437E-01	2,14	.145218E-01
6	BS				.101373	7	.144819E-01
7	BO	BSO		0.470	.551033E-03	1,7	.551033E-03
8	SO				.218822E-01	7	.312604E-02
9	BV	BSV		0.851	.206873E-02	2,14	.103436E-02
10	SV				.262895E-01	14	.187782E-02
11	OV	SOV		0.469	.827082E-03	2,14	.413541E-03
12	BSO				.819067E-02	7	.117009E-02
13	BSV				.169979E-01	14	.121413E-02
14	BOV	BSOV		0.680	.977094E-03	2,14	.488547E-03
15	SOV				.123395E-01	14	.881398E-03
16	BSOV				.100561E-01	14	.718296E-03

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(change in diameter in millimeters)

	...Days 2 - 4...			...Days 5 - 7...			CER-	
	0	25	50	0	25	50	TAIN	S
SUBJECT BET	0.02	0.03	0.03	0.02	0.04	0.04	-0.02	1
	0.09	0.08	0.08	0.10	0.13	0.13	-0.03	2
	0.17	0.14	0.17	0.10	0.12	0.12	0.01	3
	0.14	0.14	0.15	0.13	0.14	0.12	-0.01	4
	0.33	0.34	0.36	0.30	0.32	0.36	-0.08	5
	0.05	0.06	0.10	0.02	0.03	0.04	-0.04	6
	0.07	0.11	0.14	0.06	0.11	0.12	-0.06	7
	0.22	0.24	0.27	0.25	0.30	0.36	-0.07	8
MEAN	0.14	0.14	0.16	0.12	0.15	0.16	-0.04	
s.d.	0.10	0.10	0.11	0.10	0.11	0.13	0.03	
COMPUTER BET	-0.03	0.01	0.02	0.00	-0.02	0.00		1
	-0.01	0.03	0.06	0.05	0.07	0.11		2
	0.00	0.17	0.03	0.00	0.00	-0.01		3
	0.09	0.08	0.11	0.05	0.05	0.08		4
	0.05	0.12	0.16	0.06	0.16	0.22		5
	0.01	0.01	0.03	0.00	-0.01	0.00		6
	0.00	-0.01	-0.01	-0.04	-0.03	-0.06		7
	0.06	0.07	0.12	-0.08	0.09	0.18		8
MEAN	0.02	0.06	0.07	0.01	0.04	0.07		
s.d.	0.04	0.06	0.06	0.05	0.07	0.10		

ANALYSIS OF VARIANCE FOR EMITTED RESPONSE: PEAK DILATION

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.05	9.975	.690204	1,7	.690204
2	B=BET SOURCE	BS	p<.01	16.183	.234037	1,7	.234037
3	S=SUBJECTS				.484329	7	.691898E-01
4	O=OUTCOME	SO		0.057	.150000E-03	1,7	.150000E-03
5	V=BET VALUE	SV		5.410	.167270E-01	2,14	.836354E-02
6	BS				.101229	7	.144613E-01
7	BO	BSO		0.913	.135000E-02	1,7	.135000E-02
8	SO				.183500E-01	7	.262142E-02
9	BV	BSV		0.071	.156250E-03	2,14	.781252E-04
10	SV				.216395E-01	14	.154568E-02
11	OV	SOV		0.283	.693750E-03	2,14	.346875E-03
12	BSO				.103499E-01	7	.147856E-02
13	BSV				.152770E-01	14	.109121E-02
14	BOV	BSOV		3.811	.225625E-02	2,14	.112812E-02
15	SOV				.171062E-01	14	.122187E-02
16	BSOV				.414378E-02	14	.295984E-03

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(change in diameter in millimeters)

	Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CER-TAIN	S
SUBJECT BET	0.03	0.04	0.03	0.01	0.02	0.01	-0.02	1
	0.08	0.04	0.08	0.07	0.08	0.12	-0.03	2
	0.17	0.14	0.14	0.11	0.12	0.10	-0.04	3
	0.13	0.13	0.10	0.12	0.13	0.15	-0.03	4
	0.35	0.33	0.33	0.29	0.30	0.35	-0.07	5
	0.03	0.02	0.03	0.03	0.06	0.10	-0.05	6
	0.05	0.07	0.10	0.02	0.12	0.13	-0.10	7
	0.23	0.33	0.36	0.18	0.22	0.26	-0.12	8
MEAN	0.13	0.14	0.15	0.10	0.13	0.15	-0.06	
s.d.	0.11	0.13	0.13	0.10	0.09	0.11	0.04	
COMPUTER BET	0.00	-0.03	0.01	-0.01	0.00	0.01		1
	-0.01	0.02	0.05	0.05	0.08	0.11		2
	0.02	0.02	0.02	0.00	0.00	-0.02		3
	0.04	0.04	0.05	0.07	0.05	0.09		4
	0.08	0.12	0.15	0.04	0.17	0.20		5
	-0.01	0.00	0.02	0.00	0.02	0.01		6
	0.01	0.00	-0.04	-0.05	-0.04	-0.08		7
	-0.04	0.13	0.14	0.08	0.04	0.09		8
MEAN	0.01	0.04	0.05	0.02	0.04	0.05		
s.d.	0.04	0.06	0.07	0.04	0.06	0.09		

ANALYSIS OF VARIANCE FOR SINGLE CLICK: PEAK DILATION

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	13.380	.976066	1,7	.976066
2	B=BET SOURCE	BS	p<.01	16.942	.279504	1,7	.279504
3	S=SUBJECTS				.510616	7	.729452E-01
4	O=OUTCOME	SO		1.616	.453749E-02	1,7	.453749E-02
5	V=BET VALUE	SV	p<.05	5.993	.336583E-01	2,14	.168291E-01
6	BS				.115479	7	.164970E-01
7	BO	BSO	p<.05	6.057	.374999E-02	1,7	.374999E-02
8	SO				.196458E-01	7	.280654E-02
9	BV	BSV		1.587	.335831E-02	2,14	.167915E-02
10	SV				.393083E-01	14	.280773E-02
11	OV	SOV		1.254	.269999E-02	2,14	.134999E-02
12	BSO				.433335E-02	7	.619051E-03
13	BSV				.148083E-01	14	.105774E-02
14	BOV	BSOV		0.193	.300014E-03	2,14	.150007E-03
15	SOV				.150666E-01	14	.107619E-02
16	BSOV				.108666E-01	14	.776186E-03

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(change in diameter in millimeters)

	Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CER-TAIN	S
SUBJECT BET	0.02	0.04	0.04	0.02	0.04	0.03	-0.01	1
	0.11	0.16	0.13	0.05	0.11	0.08	-0.03	2
	0.10	0.11	0.12	0.16	0.12	0.17	0.01	3
	0.15	0.16	0.15	0.14	0.15	0.14	0.00	4
	0.32	0.37	0.37	0.30	0.36	0.39	-0.06	5
	0.08	0.07	0.09	0.02	0.05	0.04	-0.05	6
	0.13	0.09	0.11	0.10	0.11	0.22	-0.03	7
	0.25	0.27	0.29	0.26	0.28	0.36	-0.06	8
MEAN	0.15	0.16	0.16	0.13	0.15	0.18	-0.03	
s.d.	0.10	0.11	0.11	0.11	0.11	0.14	0.03	
COMPUTER BET	0.03	0.02	0.03	0.00	-0.01	-0.01		1
	0.05	0.08	0.10	-0.01	0.04	0.06		2
	0.01	0.01	0.02	0.03	-0.01	0.03		3
	0.10	0.09	0.14	0.07	0.06	0.10		4
	0.08	0.19	0.25	0.03	0.08	0.16		5
	0.00	0.01	0.06	0.00	-0.03	0.00		6
	-0.05	-0.02	-0.04	0.01	-0.04	-0.01		7
	0.06	0.09	0.13	-0.08	0.09	0.25		8
MEAN	0.04	0.06	0.09	0.01	0.02	0.07		
s.d.	0.05	0.07	0.09	0.04	0.05	0.09		

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: PEAK DILATION

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	12.500	.815859	1,7	.815859
2	B=BET SOURCE	BS	p<.01	17.964	.304876	1,7	.304876
3	S=SUBJECTS				.456882	7	.652689E-01
4	O=OUTCOME	SO		0.012	.260415E-04	1,7	.260415E-04
5	V=BET VALUE	SV	p<.05	6.569	.272250E-01	2,14	.136125E-01
6	BS				.118799	7	.169712E-01
7	BO	BSO		1.852	.338439E-02	1,7	.338439E-02
8	SO				.146156E-01	7	.208794E-02
9	BV	BSV		0.802	.263333E-02	2,14	.131666E-02
10	SV				.290083E-01	14	.207202E-02
11	OV	SOV		1.415	.215832E-02	2,14	.107916E-02
12	BSO				.127906E-01	7	.182722E-02
13	BSV				.229666E-01	14	.164047E-02
14	BOV	BSOV		4.789	.389998E-02	2,14	.194999E-02
15	SOV				.106750E-01	14	.762499E-03
16	BSOV				.570002E-02	14	.407144E-03

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(change in diameter in millimeters)

CANCEL when:	Evoked = Win			Emitted = Win			CER-	
	0	25	50	0	25	50	TAIN	S
SUBJECT BET	0.03	0.03	0.02	0.01	0.03	0.05	-0.03	1
	0.13	0.14	0.14	0.16	0.09	0.09	-0.02	2
	0.10	0.13	0.13	0.18	0.16	0.20	0.06	3
	0.14	0.14	0.16	0.13	0.14	0.13	0.01	4
	0.31	0.30	0.35	0.34	0.33	0.35	-0.10	5
	0.04	0.05	0.09	0.02	0.03	0.05	-0.03	6
	0.02	0.11	0.11	0.05	0.14	0.11	-0.05	7
	0.24	0.23	0.27	0.26	0.30	0.37	-0.02	8
MEAN	0.13	0.14	0.16	0.14	0.15	0.17	-0.02	
s.d.	0.10	0.09	0.10	0.12	0.11	0.13	0.05	
COMPUTER BET	0.00	0.00	0.02	0.00	-0.01	0.00		1
	0.06	0.06	0.11	0.00	0.03	0.06		2
	0.01	-0.01	-0.05	-0.05	0.01	0.02		3
	0.08	0.08	0.12	0.04	0.05	0.08		4
	0.05	0.12	0.19	0.04	0.15	0.20		5
	0.01	0.00	0.01	0.01	0.00	0.00		6
	-0.02	-0.03	-0.08	-0.02	0.02	0.03		7
	0.04	0.09	0.13	-0.12	0.04	0.15		8
MEAN	0.03	0.04	0.06	-0.01	0.04	0.07		
s.d.	0.03	0.06	0.10	0.05	0.05	0.07		

ANALYSIS OF VARIANCE FOR EMITTED RESPONSE: PUPIL RECOVERY SLOPE

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	26.138	.698709	1,7	.698709
2	B=BET SOURCE	BS		0.004	.937498E-05	1,7	.937498E-05
3	S=SUBJECTS				.187115	7	.267308E-01
4	O=OUTCOME	SO		0.104	.651041E-03	1,7	.651041E-03
5	V=BET VALUE	SV		0.502	.943749E-03	2,14	.471874E-03
6	BS				.161156E-01	7	.230223E-02
7	BO	BSO		1.758	.362604E-02	1,7	.362604E-02
8	SO				.436406E-01	7	.623437E-02
9	BV	BSV		3.203	.266875E-02	2,14	.133437E-02
10	SV				.131562E-01	14	.939731E-03
11	OV	SOV	p<.05	9.247	.119145E-01	2,14	.595729E-02
12	BSO				.144322E-01	7	.206175E-02
13	BSV				.583125E-02	14	.416518E-03
14	BOV	BSOV		1.927	.191458E-02	2,14	.957291E-03
15	SOV				.901874E-02	14	.644196E-03
16	BSOV				.695207E-02	14	.496577E-03

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(change in diameter in millimeters)

	Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN	S
SUBJECT	0.03	-0.02	-0.04	-0.01	0.05	0.05	0.01	1
BET	0.02	0.05	0.10	-0.01	0.06	0.08	0.01	2
	0.11	0.11	0.08	0.12	0.12	0.11	0.08	3
	0.08	0.07	0.05	0.06	0.11	0.12	-0.01	4
	0.11	0.11	0.10	0.11	0.15	0.18	0.06	5
	-0.01	-0.02	-0.01	0.03	0.05	0.08	0.04	6
	0.11	0.11	0.08	0.13	0.16	0.13	0.07	7
	0.26	0.15	0.20	0.08	0.14	0.15	0.08	8
MEAN	0.09	0.07	0.07	0.06	0.11	0.11	.04	
s.d.	0.08	0.06	0.07	0.06	0.05	0.04	0.04	
COMPUTER	0.06	0.04	0.04	0.06	0.09	0.07		1
BET	0.05	0.01	0.06	0.08	0.09	0.13		2
	0.20	0.12	0.12	0.09	0.07	0.08		3
	0.01	0.03	0.03	0.06	0.04	0.08		4
	0.07	0.11	0.09	0.11	0.09	0.13		5
	0.03	0.00	0.02	0.05	0.04	0.03		6
	0.16	0.09	0.17	0.05	0.12	0.09		7
	0.26	0.18	0.19	0.12	0.12	0.08		8
MEAN	0.11	0.07	0.09	0.08	0.08	0.09		
s.d.	0.09	0.06	0.06	0.03	0.03	0.03		

ANALYSIS OF VARIANCE FOR SINGLE CLICK: PUPIL RECOVERY SLOPE

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	23.478	.855037	1,7	.855037
2	B=BET SOURCE	BS		1.063	.240000E-02	1,7	.240000E-02
3	S=SUBJECTS				.254929	7	.364184E-01
4	O=OUTCOME	SO		3.677	.253500E-01	1,7	.253500E-01
5	V=BET VALUE	SV		1.866	.277500E-02	2,14	.138750E-02
6	BS				.158000E-01	7	.225714E-02
7	BO	BSO		0.391	.337497E-03	1,7	.337497E-03
8	SO				.482500E-01	7	.689285E-02
9	BV	BSV		0.390	.325000E-03	2,14	.162500E-03
10	SV				.104083E-01	14	.743452E-03
11	OV	SOV	p<.05	6.838	.422499E-02	2,14	.211249E-02
12	BSO				.602917E-02	7	.861310E-03
13	BSV				.582500E-02	14	.416071E-03
14	BOV	BSOV		1.364	.257500E-02	2,14	.128750E-02
15	SOV				.432500E-02	14	.308929E-03
16	BSOV				.132083E-01	14	.943450E-03

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(change in diameter in millimeters)

	Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CER-TAIN	S
SUBJECT BET	0.02	-0.01	-0.03	0.03	0.06	0.06	0.05	1
	0.02	0.03	0.01	0.04	0.13	0.09	0.06	2
	0.11	0.10	0.08	0.16	0.17	0.17	0.10	3
	0.11	0.09	0.10	0.06	0.07	0.08	0.02	4
	0.17	0.16	0.14	0.09	0.11	0.20	0.06	5
	0.04	0.04	0.03	-0.01	0.00	0.02	0.00	6
	0.17	0.11	0.12	0.16	0.14	0.17	0.07	7
	0.10	0.15	0.18	0.25	0.24	0.24	0.04	8
MEAN	0.09	0.08	0.08	0.10	0.12	0.13	0.05	
s.d.	0.06	0.06	0.07	0.09	0.07	0.08	0.03	
COMPUTER BET	0.04	0.04	0.01	0.11	0.11	0.08		1
	0.05	0.06	0.02	0.07	0.06	0.09		2
	0.04	0.07	0.07	0.18	0.14	0.20		3
	0.08	0.06	0.12	0.03	0.03	0.05		4
	0.02	0.12	0.10	0.09	0.11	0.12		5
	0.01	0.02	0.03	0.01	0.01	0.03		6
	0.10	0.15	0.09	0.16	0.12	0.17		7
	0.15	0.12	0.14	0.16	0.19	0.26		8
MEAN	0.06	0.08	0.07	0.10	0.10	0.13		
s.d.	0.05	0.05	0.05	0.06	0.06	0.08		

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: PUPIL RECOVERY SLOPE

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S	p<.01	29.044	1.47510	1,7	1.47510
2 B=BET SOURCE	BS		0.192	.704166E-03	1,7	.704166E-03
3 S=SUBJECTS				.355512	7	.507875E-01
4 O=OUTCOME	SO		1.956	.198375E-01	1,7	.198375E-01
5 V=BET VALUE	SV	p<.01	17.133	.380770E-01	2,14	.190385E-01
6 BS				.256458E-01	7	.366369E-02
7 BO	BSO		0.245	.204169E-03	1,7	.204169E-03
8 SO				.709791E-01	7	.101398E-01
9 BV	BSV		1.885	.195208E-02	2,14	.976041E-03
10 SV				.155562E-01	14	.111116E-02
11 OV	SOV		0.393	.693752E-03	2,14	.346876E-03
12 BSO				.581249E-02	7	.830356E-03
13 BSV				.724791E-02	14	.517708E-03
14 BOV	BSOV		0.027	.270786E-04	2,14	.135393E-04
15 SOV				.123395E-01	14	.881398E-03
16 BSOV				.690625E-02	14	.493303E-03

Data for Subjects 1-8, Mean and St. Dev. Across Subjects
(change in diameter in millimeters)

CANCEL when:	Evoked = Win			Emitted = Win			CER-TAIN	S
	0	25	50	0	25	50		
SUBJECT	0.04	0.04	0.05	0.05	0.03	0.06	0.05	1
BET	0.02	0.07	0.11	0.11	0.12	0.13	0.06	2
	0.15	0.14	0.18	0.19	0.17	0.17	0.11	3
	0.09	0.08	0.11	0.08	0.10	0.10	0.01	4
	0.14	0.19	0.22	0.16	0.19	0.26	0.08	5
	0.04	0.05	0.08	0.03	0.01	0.03	0.03	6
	0.16	0.11	0.17	0.13	0.15	0.20	0.05	7
	0.15	0.16	0.18	0.29	0.23	0.36	0.10	8
MEAN	0.10	0.11	0.14	0.13	0.13	0.16	0.06	
s.d.	0.06	0.05	0.06	0.08	0.08	0.11	0.04	
COMPUTER	0.07	0.11	0.11	0.12	0.08	0.11		1
BET	0.04	0.09	0.10	0.11	0.07	0.11		2
	0.09	0.12	0.21	0.13	0.13	0.18		3
	0.08	0.08	0.11	0.04	0.05	0.07		4
	0.05	0.09	0.18	0.09	0.16	0.20		5
	0.02	0.04	0.06	0.03	0.01	0.03		6
	0.13	0.14	0.16	0.17	0.16	0.21		7
	0.13	0.15	0.17	0.24	0.36	0.43		8
MEAN	0.08	0.10	0.14	0.12	0.13	0.17		
s.d.	0.04	0.04	0.05	0.07	0.11	0.12		

ANALYSIS OF VARIANCE FOR MISSING STIMULUS: EMITTED P366 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.001	37.2936	32757.47	1,7	32757.47
2	L=SCALP LOC	LS	p<.01	15.9198	3001.277	3,21	1000.426
3	B=BET SOURCE	BS	p<.01	19.2521	1745.195	1,7	1745.195
4	S=SUBJECTS				6148.567	7	878.3667
5	O=OUTCOME	SO		0.9346	40.29388	1,7	40.29388
6	V=BET VALUE	SV		7.6761	256.7025	2,14	128.3513
7	LB	LBS		5.6265	132.2891	3,21	44.09638
8	LS				1319.678	21	62.84179
9	BS				634.5464	7	90.64949
10	LO	LSO		0.2193	4.595252	3,21	1.531751
11	BO	BSO		0.2369	2.945776	1,7	2.945776
12	SO				301.7838	7	43.11197
13	LV	LSV		3.1540	41.30573	6,42	6.884288
14	BV	BSV		0.2063	2.326752	2,14	1.163376
15	SV				234.0920	14	16.72086
16	OV	SOV		1.9685	36.72088	2,14	18.36044
17	LBS				164.5829	21	7.837280
18	LBO	LBSO		2.4477	15.87078	3,21	5.290261
19	LSO				146.6575	21	6.983693
20	BSO				87.05629	7	12.43661
21	LBV	LBSV		1.0356	9.411591	6,42	1.568598
22	LSV				91.67419	42	2.182719
23	BSV				78.93198	14	5.637998
24	LOV	LSOV		0.9988	10.92747	6,42	1.821244
25	BOV	BSOV		4.2072	75.06072	2,14	37.53036
26	SOV				130.5794	14	9.327101
27	LBSO				45.38789	21	2.161328
28	LBSV				63.61606	42	1.514668
29	LBOV	LBSOV		0.1476	2.399516	6,42	.3999193
30	LSOV				76.58670	42	1.823493
31	BSOV				124.8872	14	8.920512
32	LBSOV				113.8164	42	2.709915

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CER-TAIN
SUBJECT BET	Frontal	MEAN	8.94	7.86	8.63	7.31	9.24	10.36	2.64
		s.d.	4.68	4.22	5.59	4.63	4.42	6.24	2.63
	Vertex	MEAN	15.38	13.64	15.78	13.63	16.06	17.56	3.76
		s.d.	7.18	7.92	8.99	7.50	6.77	8.34	2.70
	Parietal	MEAN	13.42	12.96	14.69	12.43	14.30	16.05	4.04
		s.d.	6.80	7.34	7.81	6.10	7.63	6.95	2.09
	Occipital	MEAN	7.20	6.33	6.65	6.75	8.35	9.33	2.31
		s.d.	4.69	3.05	4.63	3.72	4.98	3.91	1.87
COMPUTER BET	Frontal	MEAN	3.76	3.30	5.53	4.70	5.30	6.75	
		s.d.	2.42	2.25	2.44	3.62	3.40	3.51	
	Vertex	MEAN	7.88	8.53	11.89	8.49	9.64	11.21	
		s.d.	3.98	5.22	5.24	4.92	4.99	6.72	
	Parietal	MEAN	7.69	8.51	10.89	8.70	8.69	10.03	
		s.d.	3.66	5.79	5.47	5.47	5.37	6.51	
	Occipital	MEAN	3.77	5.18	5.51	4.60	5.19	4.78	
		s.d.	3.13	3.54	2.80	2.57	3.64	4.36	

ANALYSIS OF VARIANCE FOR SINGLE CLICK: EVOKED P53 COMPONENT

All F-tests non-significant

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S		11.6454	752.3882	1,7	752.3882
2	L=SCALP LOC	LS		1.4525	20.17867	3,21	6.726223
3	B=BET SOURCE	BS		5.1276	18.44945	1,7	18.44945
4	S=SUBJECTS				452.2567	7	64.60809
5	O=OUTCOME	SO		0.0076	.0180128	1,7	.0180128
6	V=BET VALUE	SV		0.3045	6.256820	2,14	3.128410
7	LB	LBS		4.6100	7.635844	3,21	2.545281
8	LS				97.24538	21	4.630733
9	BS				25.18634	7	3.598049
10	LO	LSO		1.3259	5.899981	3,21	1.966660
11	BO	BSO		0.0255	.2526600	1,7	.2526600
12	SO				16.68271	7	2.383244
13	LV	LSV		0.9628	7.454520	6,42	1.242420
14	BV	BSV		0.3550	2.767371	2,14	1.383685
15	SV				143.8540	14	10.27528
16	OV	SOV		7.0482	74.10895	2,14	37.05448
17	LBS				11.59448	21	.5521180
18	LBO	LBSO		1.3821	9.046864	3,21	3.015621
19	LSO				31.14800	21	1.483238
20	BSO				69.29566	7	9.899380
21	LBV	LBSV		0.4824	2.385321	6,42	.3975535
22	LSV				54.19781	42	1.290424
23	BSV				54.56128	14	3.897234
24	LOV	LSOV		1.6081	16.74733	6,42	2.791222
25	BOV	BSOV		1.5198	17.96939	2,14	8.984694
26	SOV				73.60229	14	5.257306
27	LBSO				45.81912	21	2.181863
28	LBSV				34.61220	42	.8240999
29	LBOV	LBSOV		0.1651	1.255076	6,42	.2091794
30	LSOV				72.89878	42	1.735685
31	BSOV				82.76617	14	5.911870
32	LBSOV				53.20382	42	1.266758

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN
SUBJECT BET	Frontal	MEAN	1.47	1.73	1.28	1.52	1.07	0.92	1.88
		s.d.	2.36	1.28	1.58	1.99	1.45	0.95	1.99
	Vertex	MEAN	1.43	2.28	2.14	2.67	2.94	1.53	2.06
		s.d.	2.46	1.84	1.80	3.16	3.54	1.48	2.48
	Parietal	MEAN	0.76	1.68	2.45	2.25	1.91	0.69	2.36
		s.d.	1.99	1.96	1.75	3.01	2.44	2.08	2.26
	Occipital	MEAN	0.31	1.66	1.85	1.62	1.57	1.14	1.77
		s.d.	1.28	1.76	1.20	1.57	1.79	1.19	1.41
COMPUTER BET	Frontal	MEAN	0.66	0.26	1.04	1.13	1.81	0.68	
		s.d.	2.02	0.99	1.36	1.03	1.46	1.22	
	Vertex	MEAN	1.07	0.59	1.96	1.22	2.62	0.74	
		s.d.	2.87	1.55	1.84	1.59	2.41	1.70	
	Parietal	MEAN	0.53	0.59	2.76	1.01	1.35	0.28	
		s.d.	3.21	1.83	2.42	1.99	2.32	2.19	
	Occipital	MEAN	1.03	1.28	2.61	1.24	1.57	0.29	
		s.d.	2.00	1.05	1.83	2.09	2.27	1.58	

ANALYSIS OF VARIANCE FOR SINGLE CLICK: EVOKED N93 COMPONENT

All F-tests non-significant

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S		5.7690	324.6130	1,7	324.6130
2 L=SCALP LOC	LS		3.6002	90.80887	3,21	30.26962
3 B=BET SOURCE	BS		6.1295	67.73761	1,7	67.73761
4 S=SUBJECTS				393.8776	7	56.26823
5 O=OUTCOME	SO		0.6763	7.232526	1,7	7.232526
6 V=BET VALUE	SV		2.6748	34.05031	2,14	17.02515
7 LB	LBS		0.7804	3.426847	3,21	1.142282
8 LS				176.5645	21	8.407831
9 BS				77.35712	7	11.05102
10 LO	LSO		2.4521	9.940481	3,21	3.313494
11 BO	BSO		0.0234	.1441350	1,7	.1441350
12 SO				74.85428	7	10.69347
13 LV	LSV		1.2534	12.68755	6,42	2.114591
14 BV	BSV		1.1683	17.08653	2,14	8.543267
15 SV				89.11042	14	6.365030
16 OV	SOV		2.8504	28.58693	2,14	14.29346
17 LBS				30.73883	21	1.463754
18 LBO	LBSO		2.9133	4.808320	3,21	1.602773
19 LSO				28.37714	21	1.351292
20 BSO				43.07813	7	6.154018
21 LBV	LBSV		0.7284	7.113682	6,42	1.185614
22 LSV				70.85958	42	1.687133
23 BSV				102.3759	14	7.312567
24 LOV	LSOV		0.5913	6.011258	6,42	1.001876
25 BOV	BSOV		2.7711	16.14161	2,14	8.070805
26 SOV				70.20253	14	5.014466
27 LBSO				11.55325	21	.5501547
28 LBSV				68.36118	42	1.627647
29 LBOV	LBSOV		2.5083	25.15397	6,42	4.192328
30 LSOV				71.16887	42	1.694497
31 BSOV				40.77429	14	2.912449
32 LBSOV				70.19694	42	1.671356

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN
SUBJECT BET	Frontal	MEAN	-1.54	-1.39	-0.86	-0.65	-1.35	-0.57	-3.02
		s.d.	2.09	1.43	2.86	3.13	2.00	1.86	1.46
	Vertex	MEAN	-1.47	-1.20	-0.08	-1.59	-0.49	-0.35	-4.49
		s.d.	2.45	2.16	3.14	4.05	2.76	2.09	2.09
	Parietal	MEAN	0.06	-0.88	0.52	-0.34	-0.07	0.33	-3.81
		s.d.	1.69	1.62	2.41	3.13	2.50	2.11	2.36
	Occipital	MEAN	0.06	-0.80	0.17	0.02	0.40	0.07	-2.05
		s.d.	1.61	0.83	1.19	2.10	1.05	1.15	1.58
COMPUTER BET	Frontal	MEAN	-2.78	-2.47	-1.71	-1.71	-0.01	-1.73	
		s.d.	1.47	1.63	2.13	1.03	0.80	1.07	
	Vertex	MEAN	-3.11	-1.87	-1.30	-2.29	-0.65	-2.44	
		s.d.	1.34	2.19	2.31	1.66	1.87	1.72	
	Parietal	MEAN	-2.78	-0.55	0.62	-0.98	-0.99	-1.53	
		s.d.	1.76	1.80	2.93	2.21	2.24	1.48	
	Occipital	MEAN	-1.55	-0.35	-0.11	-0.36	-1.01	-0.51	
		s.d.	1.76	1.70	1.68	1.97	1.30	2.00	

ANALYSIS OF VARIANCE FOR SINGLE CLICK: EVOKED P197 COMPONENT

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S	p<.001	41.9754	15908.22	1,7	15908.22
2 L=SCALP LOC	LS	p<.01	19.0025	2052.904	3,21	684.3014
3 B=BET SOURCE	BS	p<.01	23.7483	596.8291	1,7	596.8291
4 S=SUBJECTS				2652.922	7	378.9888
5 O=OUTCOME	SO	p<.01	24.9690	465.4104	1,7	465.4104
6 V=BET VALUE	SV	p<.01	12.9407	167.1464	2,14	83.57319
7 LB	LBS		6.4898	92.73186	3,21	30.91062
8 LS				756.2343	21	36.01116
9 BS				175.9200	7	25.13143
10 LO	LSO		0.9182	12.18623	3,21	4.062077
11 BO	BSO		0.0153	.6298714	1,7	.6298714
12 SO				130.4765	7	18.63950
13 LV	LSV		3.9073	68.24195	6,42	11.37366
14 BV	BSV		3.5089	25.00476	2,14	12.50238
15 SV				90.41440	14	6.458172
16 OV	SOV		2.3488	99.28838	2,14	49.64419
17 LBS				100.0217	21	4.762939
18 LBO	LBSO		1.6437	8.830334	3,21	2.943445
19 LSO				92.89910	21	4.423767
20 BSO				287.4538	7	41.06483
21 LBV	LBSV		1.4820	18.38675	6,42	3.064459
22 LSV				122.2568	42	2.910877
23 BSV				49.88291	14	3.563065
24 LOV	LSOV		0.2434	3.590376	6,42	.5983960
25 BOV	BSOV		0.6052	9.831884	2,14	4.915942
26 SOV				295.8990	14	21.13565
27 LBSO				37.60540	21	1.790734
28 LBSV				86.84671	42	2.067779
29 LBOV	LBSOV		0.3583	3.683346	6,42	.6138910
30 LSOV				103.2547	42	2.458446
31 BSOV				113.7165	14	8.122610
32 LBSOV				71.96311	42	1.713407

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN
SUBJECT BET	Frontal	MEAN	5.84	9.44	10.27	4.92	6.18	7.24	4.90
		s.d.	3.59	3.86	3.68	3.35	4.66	4.07	2.69
	Vertex	MEAN	10.18	13.71	14.32	8.82	10.46	11.20	5.67
		s.d.	3.48	5.10	4.23	6.35	8.04	6.32	4.09
	Parietal	MEAN	7.63	10.01	10.47	7.36	6.75	7.59	4.70
		s.d.	2.90	4.22	3.83	6.12	4.24	4.40	4.24
	Occipital	MEAN	3.91	4.16	4.98	3.30	2.65	3.01	3.00
		s.d.	2.02	2.42	3.48	2.61	3.21	2.66	2.33
COMPUTER BET	Frontal	MEAN	3.93	5.27	6.49	3.42	3.58	3.86	
		s.d.	2.80	2.97	3.64	1.96	2.89	2.52	
	Vertex	MEAN	7.45	9.34	10.65	6.17	7.06	6.29	
		s.d.	3.46	4.75	4.79	2.52	3.92	4.37	
	Parietal	MEAN	5.65	7.71	8.14	4.53	4.85	3.72	
		s.d.	4.79	4.60	4.21	2.38	3.05	3.24	
	Occipital	MEAN	2.86	4.44	4.04	2.36	2.25	0.49	
		s.d.	3.54	2.57	4.07	2.54	2.41	1.68	

ANALYSIS OF VARIANCE FOR SINGLE CLICK: EVOKED N236 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	19.3118	7006.046	1,7	7006.046
2	L=SCALP LOC	LS	p<.01	15.9571	1346.898	3,21	448.9660
3	B=BET SOURCE	BS	p<.01	28.1237	770.8367	1,7	770.8367
4	S=SUBJECTS				2539.497	7	362.7853
5	O=OUTCOME	SO	p<.001	33.4995	946.5844	1,7	946.5844
6	V=BET VALUE	SV		7.8928	200.0441	2,14	100.0221
7	LB	LBS		5.9944	97.07554	3,21	32.35851
8	LS				590.8527	21	28.13584
9	BS				191.8613	7	27.40875
10	LO	LSO		9.1338	62.58574	3,21	20.86191
11	BO	BSO		10.9771	88.95403	1,7	88.95403
12	SO				197.7969	7	28.25670
13	LV	LSV		5.1956	38.33112	6,42	6.388520
14	BV	BSV		0.3566	7.176870	2,14	3.588435
15	SV				177.4166	14	12.67262
16	OV	SOV		0.5945	19.23449	2,14	9.617244
17	LBS				113.3597	21	5.398081
18	LBO	LBSO		4.3072	15.05454	3,21	5.018181
19	LSO				47.96465	21	2.284031
20	BSO				56.72536	7	8.103622
21	LBV	LBSV		0.3435	4.209375	6,42	.7015626
22	LSV				51.64364	42	1.229611
23	BSV				140.8691	14	10.06208
24	LOV	LSOV		1.2445	19.13101	6,42	3.188501
25	BOV	BSOV		1.3380	53.64218	2,14	26.82109
26	SOV				226.4617	14	16.17584
27	LBSO				24.46628	21	1.165061
28	LBSV				85.79129	42	2.042650
29	LBOV	LBSOV		0.6183	14.51313	6,42	2.418855
30	LSOV				107.6097	42	2.562135
31	BSOV				280.6477	14	20.04626
32	LBSOV				164.3079	42	3.912093

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win <u>0</u>	Win <u>25</u>	Win <u>50</u>	Lose <u>0</u>	Lose <u>25</u>	Lose <u>50</u>	CER-TAIN
SUBJECT BET	Frontal	MEAN	6.08	8.13	8.55	2.65	3.87	5.02	0.89
		s.d.	3.36	3.91	3.64	3.18	3.44	4.92	2.29
	Vertex	MEAN	10.30	11.88	12.61	4.04	6.17	7.16	0.33
		s.d.	5.75	5.31	4.85	4.14	4.48	7.62	3.21
	Parietal	MEAN	7.38	8.36	9.42	2.96	4.14	5.22	-0.24
		s.d.	4.26	3.88	4.24	2.77	3.00	5.97	3.41
	Occipital	MEAN	3.50	3.53	3.14	0.17	0.63	1.63	-0.21
		s.d.	3.12	2.89	2.63	2.92	2.43	3.13	1.91
COMPUTER BET	Frontal	MEAN	1.95	2.85	5.03	1.19	2.49	1.49	
		s.d.	3.23	1.82	2.51	1.28	3.57	1.78	
	Vertex	MEAN	4.74	5.82	7.79	2.15	4.41	2.86	
		s.d.	5.19	4.22	3.74	2.98	4.92	2.92	
	Parietal	MEAN	2.30	5.26	6.25	2.25	2.55	2.27	
		s.d.	4.88	4.81	4.05	2.41	4.43	3.48	
	Occipital	MEAN	0.63	2.66	2.03	0.24	-0.02	-0.69	
		s.d.	3.60	3.14	3.35	3.16	2.37	2.64	

ANALYSIS OF VARIANCE FOR SINGLE CLICK: EVOKED P309 COMPONENT

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S	p<.001	34.6376	35374.66	1,7	35374.66
2 L=SCALP LOC	LS	p<.01	16.8468	3191.141	3,21	1063.714
3 B=BET SOURCE	BS	p<.01	19.9777	2227.178	1,7	2227.178
4 S=SUBJECTS				7148.954	7	1021.279
5 O=OUTCOME	SO	p<.01	14.3967	835.8235	1,7	835.8235
6 V=BET VALUE	SV	p<.01	13.6150	395.0204	2,14	197.5102
7 LB	LBS		6.2141	172.1837	3,21	57.39456
8 LS				1325.947	21	63.14034
9 BS				780.3828	7	111.4833
10 LO	LSO		2.0576	34.58175	3,21	11.52725
11 BO	BSO		3.1976	86.99178	1,7	86.99178
12 SO				406.3952	7	58.05646
13 LV	LSV		6.3298	77.23596	6,42	12.87266
14 BV	BSV		1.4038	70.50394	2,14	35.25197
15 SV				203.0957	14	14.50684
16 OV	SOV		3.0788	118.8047	2,14	59.40234
17 LBS				193.9586	21	9.236123
18 LBO	LBSO		3.1582	25.96058	3,21	8.653526
19 LSO				117.6504	21	5.602399
20 BSO				190.4360	7	27.20515
21 LBV	LBSV		0.3040	5.102631	6,42	.8504384
22 LSV				85.41321	42	2.033648
23 BSV				351.5577	14	25.11126
24 LOV	LSOV		0.9029	14.90086	6,42	2.483476
25 BOV	BSOV		0.4236	7.989662	2,14	3.994831
26 SOV				270.1156	14	19.29397
27 LBSO				57.54058	21	2.740028
28 LBSV				117.5071	42	2.797788
29 LBOV	LBSOV		0.7080	6.263908	6,42	1.043985
30 LSOV				115.5283	42	2.750675
31 BSOV				132.0337	14	9.430976
32 LBSOV				61.92713	42	1.474456

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN
SUBJECT BET	Frontal	MEAN	11.37	11.71	14.48	8.10	7.13	9.44	3.55
		s.d.	4.19	5.01	7.12	7.00	3.59	4.36	3.49
	Vertex	MEAN	16.49	18.69	21.34	12.96	13.25	14.88	4.40
		s.d.	6.91	7.71	8.97	11.36	7.38	8.98	4.29
	Parietal	MEAN	13.91	16.17	18.73	11.73	11.42	13.60	4.05
		s.d.	5.20	6.27	7.49	9.72	6.92	8.17	4.96
	Occipital	MEAN	7.83	7.52	9.25	6.17	5.08	6.91	2.88
		s.d.	3.95	3.61	4.57	5.56	4.56	5.04	2.82
COMPUTER BET	Frontal	MEAN	4.37	6.64	8.32	3.62	5.23	5.61	
		s.d.	2.90	2.64	4.19	1.59	3.38	2.58	
	Vertex	MEAN	8.10	11.85	13.02	7.38	9.23	9.42	
		s.d.	4.84	6.31	6.72	4.05	5.98	4.95	
	Parietal	MEAN	6.76	10.84	12.33	7.34	8.66	7.57	
		s.d.	5.63	5.50	7.06	5.29	6.90	5.02	
	Occipital	MEAN	4.13	5.74	6.17	3.68	3.92	2.62	
		s.d.	4.92	3.21	4.30	5.17	4.39	4.11	

ANALYSIS OF VARIANCE FOR SINGLE CLICK: EVOKED N375 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	19.4735	13181.72	1,7	13181.72
2	L=SCALP LOC	LS		7.5410	1810.262	3,21	603.4207
3	B=BET SOURCE	BS	p<.001	37.1761	1468.831	1,7	1468.831
4	S=SUBJECTS				4738.344	7	676.9063
5	O=OUTCOME	SO		0.5371	56.85684	1,7	56.85684
6	V=BET VALUE	SV		7.3321	225.3757	2,14	112.6879
7	LB	LBS		6.0634	89.36758	3,21	29.78919
8	LS				1680.384	21	80.01830
9	BS				276.5703	7	39.51004
10	LO	LSO		0.4573	13.74784	3,21	4.582613
11	BO	BSO		1.4746	49.60779	1,7	49.60779
12	SO				740.9785	7	105.8541
13	LV	LSV		2.1794	23.29053	6,42	3.881756
14	BV	BSV		1.0907	15.48339	2,14	7.741694
15	SV				215.1662	14	15.36902
16	OV	SOV		0.6496	16.30981	2,14	8.154903
17	LBS				103.1713	21	4.912919
18	LBO	LBSO		0.9763	4.781214	3,21	1.593738
19	LSO				210.4428	21	10.02109
20	BSO				235.4943	7	33.64204
21	LBV	LBSV		0.4258	4.517023	6,42	.7528372
22	LSV				74.80755	42	1.781132
23	BSV				99.37288	14	7.098063
24	LOV	LSOV		0.3302	7.262613	6,42	1.210436
25	BOV	BSOV		1.5025	45.27844	2,14	22.63922
26	SOV				175.7604	14	12.55431
27	LBSO				34.28070	21	1.632414
28	LBSV				74.26185	42	1.768139
29	LBOV	LBSOV		0.7363	9.304298	6,42	1.550716
30	LSOV				153.9772	42	3.666123
31	BSOV				210.9503	14	15.06788
32	LBSOV				88.45542	42	2.106082

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN
SUBJECT BET	Frontal	MEAN	6.53	5.13	5.74	4.21	4.74	5.86	-0.46
		s.d.	3.90	5.75	6.30	5.05	3.80	4.72	3.31
	Vertex	MEAN	11.13	10.07	11.65	7.96	9.31	11.32	-1.57
		s.d.	5.38	6.67	8.17	7.53	8.47	9.24	3.99
	Parietal	MEAN	11.58	10.90	12.35	7.83	9.37	11.89	-2.65
		s.d.	4.68	5.90	5.20	4.51	7.01	8.80	5.56
	Occipital	MEAN	5.73	5.36	6.53	3.37	3.41	5.59	-2.05
		s.d.	3.15	3.63	3.22	4.01	5.15	5.38	2.02
COMPUTER BET	Frontal	MEAN	1.03	1.56	3.20	1.80	1.96	2.54	
		s.d.	3.25	3.30	3.95	2.66	2.77	3.34	
	Vertex	MEAN	3.99	5.45	6.40	4.96	5.49	6.87	
		s.d.	3.98	5.24	5.32	4.06	5.68	4.78	
	Parietal	MEAN	3.94	6.24	7.01	4.79	5.01	7.18	
		s.d.	4.18	4.63	6.63	4.58	5.93	4.55	
	Occipital	MEAN	1.67	3.43	3.24	0.98	1.83	3.13	
		s.d.	3.45	3.56	5.05	4.17	3.14	4.32	

ANALYSIS OF VARIANCE FOR SINGLE CLICK: EVOKED P438 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.001	37.7955	32151.09	1,7	32151.09
2	L=SCALP LOC	LS	p<.01	13.8550	3689.043	3,21	1229.681
3	B=BET SOURCE	BS	p<.01	25.3764	1598.912	1,7	1598.912
4	S=SUBJECTS				5954.619	7	850.6599
5	O=OUTCOME	SO		0.5722	45.25822	1,7	45.25822
6	V=BET VALUE	SV		8.3483	249.6904	2,14	124.8452
7	LB	LBS		4.8981	73.74519	3,21	24.58173
8	LS				1863.821	21	88.75337
9	BS				441.0547	7	63.00781
10	LO	LSO		1.0348	35.05651	3,21	11.68550
11	BO	BSO		0.5366	12.26543	1,7	12.26543
12	SO				553.6331	7	79.09044
13	LV	LSV		2.7798	37.04996	6,42	6.174993
14	BV	BSV		0.9828	27.83462	2,14	13.91731
15	SV				209.3647	14	14.95462
16	OV	SOV		1.0369	30.15381	2,14	15.07691
17	LBS				105.3913	21	5.018633
18	LBO	LBSO		0.1960	1.708296	3,21	.5694319
19	LSO				237.1339	21	11.29209
20	BSO				159.9932	7	22.85618
21	LBV	LBSV		0.3643	5.909975	6,42	.9849958
22	LSV				93.29856	42	2.221394
23	BSV				198.2516	14	14.16083
24	LOV	LSOV		0.1782	3.867519	6,42	.6445866
25	BOV	BSOV		5.2162	70.28720	2,14	35.14360
26	SOV				203.5640	14	14.54029
27	LBSO				60.99703	21	2.904620
28	LBSV				113.5666	42	2.703968
29	LBOV	LBSOV		1.0512	11.89716	6,42	1.982860
30	LSOV				151.8993	42	3.616649
31	BSOV				94.32414	14	6.737438
32	LBSOV				79.22120	42	1.886219

Means and Standard Deviations (microvolts) Across 8 Subjects

			Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN
SUBJECT BET	Frontal	MEAN	7.22	6.35	8.07	5.69	7.07	7.91	2.43
		s.d.	3.22	4.74	5.39	4.36	4.14	4.28	3.28
	Vertex	MEAN	13.86	12.87	15.89	11.32	14.36	14.43	1.85
		s.d.	6.15	6.68	7.61	8.14	8.46	9.17	3.61
	Parietal	MEAN	16.19	14.49	17.61	12.61	15.34	16.40	2.88
		s.d.	7.11	6.59	7.58	6.86	7.20	8.74	3.76
	Occipital	MEAN	9.81	7.88	10.32	6.59	7.33	8.99	3.25
		s.d.	5.63	3.92	5.28	5.37	5.25	5.69	2.75
COMPUTER BET	Frontal	MEAN	2.30	3.71	4.01	2.85	4.08	4.50	
		s.d.	2.54	2.96	2.85	3.04	3.30	3.17	
	Vertex	MEAN	7.46	9.58	10.17	8.02	9.23	10.04	
		s.d.	3.93	5.21	4.52	5.06	5.91	5.07	
	Parietal	MEAN	8.37	10.79	12.27	8.94	10.36	11.01	
		s.d.	5.26	5.32	5.68	5.74	6.42	5.25	
	Occipital	MEAN	5.37	6.36	6.90	4.89	4.98	4.44	
		s.d.	4.03	4.30	4.95	5.36	4.95	5.35	

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: P40 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.001	47.2365	1358.639	1,7	1358.639
2	L=SCALP LOC	LS		7.6291	75.92605	3,21	25.30868
3	B=BET SOURCE	BS		8.5160	14.50039	1,7	14.50039
4	S=SUBJECTS				201.3372	7	28.76246
5	O=OUTCOME	SO		0.3943	2.362538	1,7	2.362538
6	V=BET VALUE	SV		2.2016	28.38642	2,14	14.19321
7	LB	LBS		0.3252	1.176231	3,21	.3920768
8	LS				69.66515	21	3.317388
9	BS				11.91909	7	1.702727
10	LO	LSO		1.3342	4.427089	3,21	1.475696
11	BO	BSO		1.2535	8.724189	1,7	8.724189
12	SO				41.94330	7	5.991900
13	LV	LSV		0.7384	3.775255	6,42	.6292092
14	BV	BSV		0.0346	.5012498	2,14	.2506249
15	SV				90.25539	14	6.446813
16	OV	SOV		1.5573	16.59249	2,14	8.296247
17	LBS				25.31593	21	1.205520
18	LBO	LBSO		0.9773	3.702868	3,21	1.234289
19	LSO				23.22734	21	1.106064
20	BSO				48.71812	7	6.959732
21	LBV	LBSV		0.8278	6.714898	6,42	1.119150
22	LSV				35.78999	42	.8521427
23	BSV				101.4586	14	7.247044
24	LOV	LSOV		0.2090	1.576491	6,42	.2627484
25	BOV	BSOV		0.1765	1.780773	2,14	.8903863
26	SOV				74.58325	14	5.327375
27	LBSO				26.52138	21	1.262923
28	LBSV				56.78528	42	1.352030
29	LBOV	LBSOV		0.4799	4.421097	6,42	.7368495
30	LSOV				52.79949	42	1.257131
31	BSOV				70.64453	14	5.046038
32	LBSOV				64.49276	42	1.535542

Means and Standard Deviations (microvolts) Across 8 Subjects

	CANCEL when:		Evoked = Win			Emitted = Win			CER-
			0	25	50	0	25	50	TAIN
SUBJECT BET	Frontal	MEAN	1.93	1.37	2.92	1.16	1.72	2.08	1.46
		s.d.	1.09	1.14	1.63	1.56	1.46	1.49	1.69
	Vertex	MEAN	3.31	2.13	3.64	2.04	2.82	3.26	0.14
		s.d.	1.85	1.70	2.31	1.77	2.44	1.17	2.36
	Parietal	MEAN	2.42	1.40	2.35	1.35	1.78	2.25	0.53
		s.d.	1.06	1.96	1.87	1.76	2.81	1.52	2.34
	Occipital	MEAN	1.90	0.80	1.58	2.12	1.78	1.68	-0.26
		s.d.	1.59	1.37	1.21	1.40	1.97	1.30	1.21
COMPUTER BET	Frontal	MEAN	1.31	0.88	1.55	1.31	1.65	1.37	
		s.d.	1.05	1.50	1.09	1.25	0.68	1.82	
	Vertex	MEAN	2.19	1.83	2.67	2.47	2.44	2.84	
		s.d.	1.54	1.67	2.11	2.42	1.38	1.54	
	Parietal	MEAN	1.20	0.64	1.69	1.95	1.72	2.39	
		s.d.	1.46	1.90	2.01	2.49	1.30	1.82	
	Occipital	MEAN	1.48	0.73	1.35	1.08	1.59	2.19	
		s.d.	2.10	2.32	1.27	1.60	1.86	2.24	

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: EVOKED N86 COMPONENT

All F-tests non-significant

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S		9.9317	677.8220	1,7	677.8220
2 L=SCALP LOC	LS		2.6321	86.96442	3,21	28.98814
3 B=BET SOURCE	BS		7.3237	115.2159	1,7	115.2159
4 S=SUBJECTS				477.7398	7	68.24854
5 O=OUTCOME	SO		0.2935	1.131004	1,7	1.131004
6 V=BET VALUE	SV		2.0043	41.99801	2,14	20.99901
7 LB	LBS		0.3574	2.674080	3,21	.8913600
8 LS				231.2779	21	11.01323
9 BS				110.1228	7	15.73183
10 LO	LSO		4.2929	15.39074	3,21	5.130247
11 BO	BSO		0.4986	2.413009	1,7	2.413009
12 SO				26.97244	7	3.853206
13 LV	LSV		0.5937	4.318014	6,42	.7196689
14 BV	BSV		0.0782	1.660572	2,14	.8302858
15 SV				146.6758	14	10.47684
16 OV	SOV		0.0472	.4713216	2,14	.2356608
17 LBS				52.37090	21	2.493852
18 LBO	LBSO		1.1286	2.822572	3,21	.9408573
19 LSO				25.09585	21	1.195041
20 BSO				33.87414	7	4.839163
21 LBV	LBSV		0.2469	1.867989	6,42	.3113315
22 LSV				50.91437	42	1.212247
23 BSV				148.6211	14	10.61579
24 LOV	LSOV		2.6916	19.89197	6,42	3.315328
25 BOV	BSOV		2.9713	13.31624	2,14	6.658121
26 SOV				69.97185	14	4.997989
27 LBSO				17.50738	21	.8336849
28 LBSV				52.96185	42	1.260997
29 LBOV	LBSOV		1.3247	10.43185	6,42	1.738641
30 LSOV				51.73353	42	1.231751
31 BSOV				31.37084	14	2.240775
32 LBSOV				55.12528	42	1.312507

Means and Standard Deviations (microvolts) Across 8 Subjects

	CANCEL when:		Evoked = Win			Emitted = Win			CER-TAIN
			0	25	50	0	25	50	
SUBJECT BET	Frontal	MEAN	-1.40	-2.17	-1.11	-1.33	-1.01	-0.68	-4.53
		s.d.	1.84	1.33	1.92	2.11	1.67	1.50	2.82
	Vertex	MEAN	-1.75	-1.52	-1.53	-1.18	-1.40	0.08	-5.75
		s.d.	2.32	2.53	2.48	2.92	3.06	2.80	3.50
	Parietal	MEAN	-0.85	-0.49	-0.18	-1.03	-1.71	0.81	-4.61
		s.d.	1.42	1.92	2.76	2.42	3.75	3.13	2.72
	Occipital	MEAN	-0.43	0.25	0.20	0.17	-0.90	0.41	-2.90
		s.d.	1.30	1.63	1.95	1.34	1.41	1.82	3.14
COMPUTER BET	Frontal	MEAN	-2.61	-3.13	-1.46	-1.72	-1.43	-2.27	
		s.d.	1.89	1.00	1.79	1.47	1.01	2.25	
	Vertex	MEAN	-3.06	-2.64	-1.62	-2.47	-2.38	-2.34	
		s.d.	2.61	2.14	1.53	2.83	1.98	2.57	
	Parietal	MEAN	-1.53	-1.81	-1.03	-2.68	-2.21	-1.65	
		s.d.	2.32	2.23	1.42	2.35	2.99	2.39	
	Occipital	MEAN	-1.17	-1.22	-0.95	-1.77	-1.46	-0.45	
		s.d.	2.27	2.87	1.12	1.99	2.00	1.49	

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: EVOKED P211 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.001	56.8780	27553.25	1,7	27553.25
2	L=SCALP LOC	LS	p<.01	28.8120	3751.506	3,21	1250.502
3	B=BET SOURCE	BS	p<.01	25.5274	775.9455	1,7	775.9455
4	S=SUBJECTS				3390.993	7	484.4276
5	O=OUTCOME	SO		5.8765	158.3635	1,7	158.3635
6	V=BET VALUE	SV		7.7017	173.3595	2,14	86.67977
7	LB	LBS	p<.01	17.3504	197.2537	3,21	65.75123
8	LS				911.4431	21	43.40205
9	BS				212.7758	7	30.39655
10	LO	LSO		0.2991	3.818747	3,21	1.272916
11	BO	BSO		0.1273	.8494320	1,7	.8494320
12	SO				188.6418	7	26.94883
13	LV	LSV		2.0142	17.11753	6,42	2.852922
14	BV	BSV		1.5180	24.10839	2,14	12.05419
15	SV				157.5640	14	11.25457
16	OV	SOV		0.4085	6.973515	2,14	3.486757
17	LBS				79.58183	21	3.789611
18	LBO	LBSO		0.6030	3.101618	3,21	1.033873
19	LSO				89.38587	21	4.256470
20	BSO				46.72116	7	6.674451
21	LBV	LBSV		0.5632	6.685713	6,42	1.114285
22	LSV				59.48928	42	1.416412
23	BSV				111.1705	14	7.940754
24	LOV	LSOV		1.2214	16.04303	6,42	2.673838
25	BOV	BSOV		2.0713	20.61065	2,14	10.30533
26	SOV				119.4993	14	8.535664
27	LBSO				36.00803	21	1.714668
28	LBSV				83.09414	42	1.978432
29	LBOV	LBSOV		1.1229	16.16279	6,42	2.693798
30	LSOV				91.94247	42	2.189106
31	BSOV				69.65315	14	4.975225
32	LBSOV				100.7530	42	2.398882

Means and Standard Deviations (microvolts) Across 8 Subjects

	CANCEL when:		Evoked = Win			Emitted = Win			CER-TAIN
			0	25	50	0	25	50	
SUBJECT BET	Frontal	MEAN	8.77	9.44	10.05	8.02	7.30	8.48	3.49
		s.d.	4.38	3.81	2.54	4.20	3.00	4.91	1.06
	Vertex	MEAN	16.16	16.06	15.79	13.33	13.87	17.02	5.21
		s.d.	6.71	4.78	5.43	5.03	4.98	5.82	2.22
	Parietal	MEAN	11.66	11.17	11.39	9.43	9.36	11.33	3.43
		s.d.	4.77	3.93	4.91	4.27	3.71	5.14	2.92
	Occipital	MEAN	5.87	4.22	5.26	4.54	3.94	4.96	2.47
		s.d.	2.72	1.11	2.27	1.63	2.54	2.87	
COMPUTER BET	Frontal	MEAN	5.44	5.79	7.40	4.62	4.51	6.13	
		s.d.	2.29	3.25	4.29	3.84	3.28	4.22	
	Vertex	MEAN	10.34	10.69	13.02	8.99	9.28	11.17	
		s.d.	3.90	4.15	5.88	3.57	3.85	6.97	
	Parietal	MEAN	7.38	8.32	10.42	6.44	6.40	8.55	
		s.d.	3.85	3.67	5.34	4.44	4.17	5.38	
	Occipital	MEAN	4.05	4.36	5.66	2.86	3.28	4.10	
		s.d.	2.23	3.54	2.61	2.80	3.27	3.28	

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: EVOKED N270 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	12.4298	8605.578	1,7	8605.578
2	L=SCALP LOC	LS		10.2335	1650.817	3,21	550.2723
3	B=BET SOURCE	BS	p<.001	30.8628	1095.256	1,7	1095.256
4	S=SUBJECTS				4846.323	7	692.3319
5	O=OUTCOME	SO		3.5042	85.78710	1,7	85.78710
6	V=BET VALUE	SV		6.3001	277.1516	2,14	138.5758
7	LB	LBS	p<.01	15.2423	240.8578	3,21	80.28593
8	LS				1129.206	21	53.77169
9	BS				248.4152	7	35.48788
10	LO	LSO		1.6831	24.94258	3,21	8.314193
11	BO	BSO		0.3514	7.746520	1,7	7.746520
12	SO				171.3672	7	24.48103
13	LV	LSV		2.0131	23.76852	6,42	3.961421
14	BV	BSV		0.2986	7.365921	2,14	3.682961
15	SV				307.9403	14	21.99574
16	OV	SOV		0.5324	5.077442	2,14	2.538721
17	LBS.				110.6137	21	5.267319
18	LBO	LBSO		0.3877	2.597139	3,21	.8657131
19	LSO				103.7376	21	4.939885
20	BSO				154.3038	7	22.04340
21	LBV	LBSV		0.8590	10.58351	6,42	1.763919
22	LSV				82.64938	42	1.967842
23	BSV				172.6617	14	12.33298
24	LOV	LSOV		0.8354	11.40970	6,42	1.901616
25	BOV	BSOV		0.1437	2.502500	2,14	1.251250
26	SOV				66.76179	14	4.768699
27	LBSO				46.89265	21	2.232984
28	LBSV				86.24675	42	2.053494
29	LBOV	LBSOV		0.7449	10.52931	6,42	1.754886
30	LSOV				95.59883	42	2.276163
31	BSOV				121.8657	14	8.704695
32	LBSOV				98.94378	42	2.355804

Means and Standard Deviations (microvolts) Across 8 Subjects

	CANCEL when:		Evoked = Win			Emitted = Win			CER-TAIN
			0	25	50	0	25	50	
SUBJECT BET	Frontal	MEAN	6.23	5.38	7.67	4.18	4.92	7.42	-0.10
		s.d.	4.92	5.23	5.86	4.59	3.12	6.09	1.51
	Vertex	MEAN	10.17	10.55	11.88	7.78	8.07	11.03	0.43
		s.d.	6.67	8.27	7.80	6.18	6.59	6.67	2.59
	Parietal	MEAN	7.36	8.45	9.61	5.50	6.17	8.48	-0.34
		s.d.	4.54	5.43	6.22	4.77	5.05	6.17	3.33
	Occipital	MEAN	1.74	2.42	2.99	2.26	1.48	2.41	-0.27
		s.d.	3.17	2.22	2.67	3.15	2.86	2.27	2.08
COMPUTER BET	Frontal	MEAN	1.68	1.76	3.19	1.08	1.46	2.83	
		s.d.	3.71	3.20	4.34	2.86	3.13	5.07	
	Vertex	MEAN	3.62	5.33	6.31	2.96	4.12	5.71	
		s.d.	4.58	5.58	6.49	4.81	4.22	6.66	
	Parietal	MEAN	3.52	5.00	5.85	3.11	2.94	4.61	
		s.d.	3.88	4.84	5.79	4.11	3.99	6.11	
	Occipital	MEAN	0.68	1.37	2.20	0.10	1.56	2.09	
		s.d.	1.48	2.87	2.03	1.70	2.74	3.66	

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: EVOKED P336 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	23.0743	37463.58	1,7	37463.58
2	L=SCALP LOC	LS		11.6248	2908.895	3,21	969.6317
3	B=BET SOURCE	BS	p<.01	13.4601	2595.996	1,7	2595.996
4	S=SUBJECTS				11365.23	7	1623.604
5	O=OUTCOME	SO		0.0001	.0044688	1,7	.0044688
6	V=BET VALUE	SV		11.5580	410.2381	2,14	205.1191
7	LB	LBS	p<.01	15.7632	291.7934	3,21	97.26445
8	LS				1751.624	21	83.41065
9	BS				1350.062	7	192.8660
10	LO	LSO		0.5146	9.068285	3,21	3.022762
11	BO	BSO		0.0600	1.441790	1,7	1.441790
12	SO				328.6498	7	46.94998
13	LV	LSV		2.1265	40.73227	6,42	6.788711
14	BV	BSV		0.3190	5.926022	2,14	2.963011
15	SV				248.4573	14	17.74695
16	OV	SOV		0.1348	4.265087	2,14	2.132544
17	LBS				129.5772	21	6.170345
18	LBO	LBSO		0.7684	6.873764	3,21	2.291255
19	LSO				123.3582	21	5.874202
20	BSO				168.3247	7	24.04638
21	LBV	LBSV		0.5011	5.181431	6,42	.8635718
22	LSV				134.0818	42	3.192424
23	BSV				130.0480	14	9.289142
24	LOV	LSOV		1.0897	13.30706	6,42	2.217843
25	BOV	BSOV		0.2093	2.962576	2,14	1.481288
26	SOV				221.5550	14	15.82536
27	LBSO				62.62271	21	2.982034
28	LBSV				72.38144	42	1.723368
29	LBOV	LBSOV		0.5801	9.069706	6,42	1.511618
30	LSOV				85.48447	42	2.035344
31	BSOV				99.06456	14	7.076040
32	LBSOV				109.4433	42	2.605793

Means and Standard Deviations (microvolts) Across 8 Subjects

		CANCEL when:	Evoked = Win			Emitted = Win			CER-TAIN
			0	25	50	0	25	50	
SUBJECT BET	Frontal	MEAN	11.21	11.35	12.87	10.90	11.25	12.43	3.01
		s.d.	6.36	5.16	7.04	7.06	5.05	6.38	2.49
	Vertex	MEAN	15.00	17.10	18.35	15.28	15.48	18.31	3.74
		s.d.	9.55	9.57	10.49	10.34	11.10	10.78	2.72
	Parietal	MEAN	12.85	14.18	16.11	13.63	14.17	16.48	2.29
		s.d.	10.03	10.27	10.02	9.38	9.98	10.25	2.19
	Occipital	MEAN	6.31	6.21	7.43	7.52	7.10	7.96	0.64
		s.d.	5.11	5.98	5.79	3.91	5.98	6.10	2.10
COMPUTER BET	Frontal	MEAN	5.17	4.96	7.38	5.01	5.40	7.62	
		s.d.	3.14	3.36	3.83	3.93	2.37	5.48	
	Vertex	MEAN	8.25	9.39	11.92	8.48	8.62	11.29	
		s.d.	5.58	6.07	6.63	5.64	4.39	7.99	
	Parietal	MEAN	7.43	8.69	10.42	7.65	7.44	10.75	
		s.d.	6.76	6.19	7.16	5.95	5.29	8.56	
	Occipital	MEAN	4.64	4.42	5.35	3.48	4.54	6.37	
		s.d.	4.14	4.22	4.20	3.37	3.50	4.84	

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: EVOKED N404 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S		10.5202	11785.69	1,7	11785.69
2	L=SCALP LOC	LS		7.3414	1482.300	3,21	494.1001
3	B=BET SOURCE	BS	p<.01	18.2972	1759.123	1,7	1759.123
4	S=SUBJECTS				7842.024	7	1120.289
5	O=OUTCOME	SO		1.8799	197.7000	1,7	197.7000
6	V=BET VALUE	SV		6.9238	457.4175	2,14	228.7088
7	LB	LBS		7.5674	109.1925	3,21	36.39749
8	LS				1413.375	21	67.30359
9	BS				672.9930	7	96.14185
10	LO	LSO		2.1343	32.59146	3,21	10.86382
11	BO	BSO		0.0897	2.311060	1,7	2.311060
12	SO				736.1491	7	105.1642
13	LV	LSV		2.2621	37.29359	6,42	6.215598
14	BV	BSV		0.2811	8.716896	2,14	4.358448
15	SV				462.4546	14	33.03247
16	OV	SOV		0.4105	6.998196	2,14	3.499098
17	LBS				101.0049	21	4.809755
18	LBO	LBSO		0.6385	3.549826	3,21	1.183275
19	LSO				106.8937	21	5.090177
20	BSO				180.4078	7	25.77254
21	LBV	LBSV		1.0863	8.241325	6,42	1.373554
22	LSV				115.4014	42	2.747653
23	BSV				217.0887	14	15.50634
24	LOV	LSOV		2.2361	31.09316	6,42	5.182193
25	BOV	BSOV		0.0119	.2946987	2,14	.1473494
26	SOV				119.3271	14	8.523363
27	LBSO				38.91652	21	1.853168
28	LBSV				53.10759	42	1.264466
29	LBOV	LBSOV		0.8593	20.33953	6,42	3.389922
30	LSOV				97.33558	42	2.317514
31	BSOV				173.6910	14	12.40650
32	LBSOV				165.6884	42	3.944963

Means and Standard Deviations (microvolts) Across 8 Subjects

	CANCEL when:		Evoked = Win			Emitted = Win			CER-
			0	25	50	0	25	50	TAIN
SUBJECT BET	Frontal	MEAN	4.72	7.27	8.88	4.22	4.71	5.33	-0.61
		s.d.	4.21	6.17	6.62	4.93	5.95	5.14	1.36
	Vertex	MEAN	7.71	10.99	11.77	7.03	8.73	10.51	-1.11
		s.d.	5.75	8.09	8.42	5.30	11.36	8.38	2.72
	Parietal	MEAN	9.69	11.33	13.08	7.71	9.99	12.34	-2.09
		s.d.	7.65	9.22	9.78	4.52	10.48	9.07	3.03
	Occipital	MEAN	4.31	:34.16	5.95	3.82	4.82	5.28	-3.31
		s.d.	4.94	4.94	5.29	2.32	6.32	5.32	2.67
COMPUTER BET	Frontal	MEAN	1.83	2.30	4.34	0.29	1.19	1.23	
		s.d.	2.83	3.56	5.74	2.20	2.63	5.36	
	Vertex	MEAN	3.84	5.72	7.84	2.90	3.71	4.68	
		s.d.	3.71	6.22	7.75	5.73	3.84	6.73	
	Parietal	MEAN	4.22	5.81	7.32	3.22	3.59	5.81	
		s.d.	4.88	6.80	8.71	5.11	3.76	6.37	
	Occipital	MEAN	1.87	2.24	3.02	0.21	1.47	2.97	
		s.d.	2.70	4.21	4.07	3.03	2.74	3.98	

ANALYSIS OF VARIANCE FOR DOUBLE CLICK: EVOKED P466 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.01	25.5352	29103.43	1,7	29103.43
2	L=SCALP LOC	LS		10.1246	2610.360	3,21	870.1199
3	B=BET SOURCE	BS	p<.001	30.4307	1516.025	1,7	1516.025
4	S=SUBJECTS				7978.157	7	1139.737
5	O=OUTCOME	SO		2.7397	208.6095	1,7	208.6095
6	V=BET VALUE	SV		6.7657	272.8938	2,14	136.4469
7	LB	LBS		7.5112	139.4508	3,21	46.48360
8	LS				1804.770	21	85.94144
9	BS				348.7329	7	49.81899
10	LO	LSO		1.7380	35.36369	3,21	11.78790
11	BO	BSO		0.2865	2.913666	1,7	2.913666
12	SO				532.9976	7	76.14251
13	LV	LSV		4.4229	58.98554	6,42	9.830924
14	BV	BSV		1.0651	22.35334	2,14	11.17667
15	SV				282.3456	14	20.16754
16	OV	SOV		0.2639	3.838306	2,14	1.919153
17	LBS				129.9601	21	6.188575
18	LBO	LBSO		0.6380	5.908203	3,21	1.969401
19	LSO				142.4309	21	6.782423
20	BSO				71.18924	7	10.16989
21	LBV	LBSV		0.8978	11.77282	6,42	1.962137
22	LSV				93.35527	42	2.222745
23	BSV				146.9068	14	10.49334
24	LOV	LSOV		0.2803	4.011059	6,42	.6685098
25	BOV	BSOV		0.0534	1.581417	2,14	.7907085
26	SOV				101.8032	14	7.271655
27	LBSO				64.82813	21	3.087054
28	LBSV				91.78907	42	2.185454
29	LBOV	LBSOV		0.3425	4.751469	6,42	.7919114
30	LSOV				100.1860	42	2.385380
31	BSOV				207.3573	14	14.81124
32	LBSOV				97.10181	42	2.311948

Means and Standard Deviations (microvolts) Across 8 Subjects

		CANCEL when:	Evoked = Win			Emitted = Win			CER-TAIN
			0	25	50	0	25	50	
SUBJECT BET	Frontal	MEAN	7.18	8.58	8.69	5.33	6.68	5.74	1.04
		s.d.	4.04	4.97	4.50	4.52	4.70	5.18	2.54
	Vertex	MEAN	12.30	14.69	14.56	9.78	13.13	12.79	1.59
		s.d.	5.21	6.44	6.87	5.60	9.04	7.64	3.44
	Parietal	MEAN	13.38	16.35	16.89	11.56	14.40	15.27	1.35
		s.d.	7.76	8.85	8.30	6.29	9.87	7.90	2.54
	Occipital	MEAN	8.06	8.73	8.79	7.64	8.00	8.11	1.19
		s.d.	4.76	6.22	5.60	4.17	6.57	4.92	2.49
COMPUTER BET	Frontal	MEAN	3.79	4.76	5.89	1.96	2.79	3.46	
		s.d.	2.20	3.15	5.17	2.25	2.59	5.10	
	Vertex	MEAN	7.76	8.91	10.88	6.01	7.56	8.30	
		s.d.	5.22	6.20	7.39	4.42	5.50	7.79	
	Parietal	MEAN	7.82	9.21	11.12	7.84	8.85	9.67	
		s.d.	6.72	7.20	9.63	5.73	6.06	7.83	
	Occipital	MEAN	5.59	5.96	6.73	4.95	5.28	6.16	
		s.d.	3.88	2.72	5.55	4.24	3.76	4.85	

ANALYSIS OF VARIANCE FOR WARNING CLICK: P68 COMPONENT

All F-tests non-significant

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S		0.4679	7.177735	1,7	7.177735
2	L=SCALP LOC	LS		1.6201	45.28755	3,21	15.09585
3	B=BET SOURCE	BS		0.0737	.3094012	1,7	.3094012
4	S=SUBJECTS				107.3764	7	15.33949
5	D=3 DAY PER.	SD		0.0330	.1086759	1,7	.1086759
6	V=BET VALUE	SV		2.5033	12.60089	2,14	6.300446
7	LB	LBS		2.2991	5.200794	3,21	1.733598
8	LS				195.6730	21	9.317762
9	BS				29.39053	7	4.198647
10	LD	LSD		2.3201	5.198672	3,21	1.732891
11	BD	BSD		0.1925	.2370094	1,7	.2370094
12	SD				23.02748	7	3.289640
13	LV	LSV		1.0327	5.109822	6,42	.8516369
14	BV	BSV		2.0118	4.086070	2,14	2.043035
15	SV				35.23639	14	2.516885
16	DV	SDV		0.1815	.5150512	2,14	.2575256
17	LBS				15.83452	21	.7540249
18	LBD	LBSD		2.3648	5.308992	3,21	1.769664
19	LSD				15.68506	21	.7469075
20	BSD				8.616729	7	1.230961
21	LBV	LBSV		1.4980	7.805554	6,42	1.300926
22	LSV				34.63632	42	.8246742
23	BSV				14.21716	14	1.015511
24	LDV	LSDV		0.4742	1.404444	6,42	.2340740
25	BDV	BSDV		0.2950	.9476283	2,14	.4738141
26	SDV				19.86144	14	1.418675
27	LBSD				15.71492	21	.7483295
28	LBSV				36.47387	42	.8684255
29	LBDV	LBSDV		0.3142	1.023839	6,42	.1706398
30	LSDV				20.73078	42	.4935899
31	BSDV				22.48761	14	1.606258
32	LBSDV				22.80727	42	.5430302

Means and Standard Deviations (microvolts) Across 8 Subjects

			...Days 2 - 4...			...Days 5 - 7...		
			0	25	50	0	25	50
SUBJECT BET	Frontal	MEAN	-0.10	0.09	0.01	-0.21	-0.08	0.14
		s.d.	1.09	0.84	1.09	1.24	1.36	0.79
	Vertex	MEAN	0.74	0.92	0.55	0.53	0.59	0.56
		s.d.	1.09	1.93	1.13	1.65	1.84	1.36
	Parietal	MEAN	-0.40	0.68	-0.20	-0.14	0.84	0.00
		s.d.	1.59	2.43	0.78	1.53	1.62	1.14
	Occipital	MEAN	-0.38	0.47	-0.30	-0.62	0.73	-0.45
		s.d.	1.55	1.45	1.16	1.77	2.32	1.02
COMPUTER BET	Frontal	MEAN	0.07	0.16	-0.02	0.42	0.57	0.15
		s.d.	1.43	1.11	1.26	1.15	1.52	0.66
	Vertex	MEAN	0.54	0.39	0.60	0.62	1.31	0.92
		s.d.	0.91	1.03	1.18	1.53	1.43	1.71
	Parietal	MEAN	-0.58	-0.12	-0.02	-0.17	0.10	0.13
		s.d.	0.88	1.24	1.21	1.13	1.04	1.36
	Occipital	MEAN	-0.50	-0.21	0.50	-1.06	-0.60	-0.60
		s.d.	1.11	1.03	1.50	1.24	1.29	1.01

ANALYSIS OF VARIANCE FOR WARNING CLICK: N115 COMPONENT

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S	p<.001	48.1231	3916.751	1,7	3916.751
2 L=SCALP LOC	LS		4.7207	391.2142	3,21	130.4047
3 B=BET SOURCE	BS	p<.01	14.4507	44.11203	1,7	44.11203
4 S=SUBJECTS				569.7313	7	81.39018
5 D=3 DAY PER.	SD		0.3761	4.745923	1,7	4.745923
6 V=BET VALUE	SV		0.4114	2.030288	2,14	1.015144
7 LB	LBS		3.6298	13.49665	3,21	4.498885
8 LS				580.1077	21	27.62418
9 BS				21.36810	7	3.052585
10 LD	LSD		5.9297	39.95735	3,21	13.31912
11 BD	BSD		0.0007	.0007865	1,7	.0007865
12 SD				88.31974	7	12.61711
13 LV	LSV		1.7896	6.776070	6,42	1.129345
14 BV	BSV		0.0412	.2210560	2,14	.1105280
15 SV				34.54521	14	2.467515
16 DV	SDV		0.4673	1.564946	2,14	.7824729
17 LBS				26.02815	21	1.239436
18 LBD	LBSD		2.5367	5.462032	3,21	1.820677
19 LSD				47.16948	21	2.246166
20 BSD				7.625373	7	1.089339
21 LBV	LBSV		0.8075	2.872496	6,42	.4787493
22 LSV				26.50486	42	.6310681
23 BSV				37.56156	14	2.682969
24 LDV	LSDV		1.0109	4.123439	6,42	.6872399
25 BDV	BSDV		0.7031	1.625327	2,14	.8126637
26 SDV				23.44377	14	1.674555
27 LBSD				15.07270	21	.7177476
28 LBSV				24.90084	42	.5928772
29 LBDV	LBSDV		0.9137	2.415772	6,42	.4026287
30 LSDV				28.55342	42	.6798433
31 BSDV				16.18202	14	1.155859
32 LBSDV				18.50702	42	.4406434

Means and Standard Deviations (microvolts) Across 8 Subjects

			...Days 2 - 4...			...Days 5 - 7...		
			0	25	50	0	25	50
SUBJECT BET	Frontal	MEAN	-4.36	-4.69	-4.75	-4.13	-4.21	-3.90
		s.d.	2.13	2.01	2.63	2.02	2.16	1.89
	Vertex	MEAN	-5.01	-5.52	-5.41	-4.36	-4.65	-4.67
		s.d.	2.15	2.24	2.36	2.57	2.42	2.55
	Parietal	MEAN	-3.37	-2.64	-2.45	-2.67	-3.36	-2.78
		s.d.	1.55	2.79	2.24	1.35	1.73	1.94
	Occipital	MEAN	-2.16	-1.81	-1.57	-2.37	-2.05	-1.94
		s.d.	3.13	1.83	2.05	1.76	1.55	2.37
COMPUTER BET	Frontal	MEAN	-3.85	-3.61	-3.99	-2.90	-2.90	-3.09
		s.d.	1.38	1.29	1.05	1.79	1.54	2.02
	Vertex	MEAN	-4.51	-5.00	-4.23	-3.23	-3.37	-3.60
		s.d.	2.54	2.34	2.16	2.75	2.09	2.21
	Parietal	MEAN	-1.91	-2.54	-1.73	-1.83	-2.45	-2.13
		s.d.	2.50	2.37	1.93	1.96	2.34	2.15
	Occipital	MEAN	-1.74	-1.50	-1.00	-2.48	-2.35	-2.58
		s.d.	1.96	2.14	1.92	2.37	2.90	2.49

ANALYSIS OF VARIANCE FOR WARNING CLICK: P201 COMPONENT

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S	p<.001	36.8132	5600.426	1,7	5600.426
2	L=SCALP LOC	LS	p<.01	14.3361	2856.400	3,21	952.1332
3	B=BET SOURCE	BS		0.0538	.8588228	1,7	.8588228
4	S=SUBJECTS				1064.917	7	152.1310
5	D=3 DAY PER.	SD		2.1822	31.75150	1,7	31.75150
6	V=BET VALUE	SV		0.7852	3.544383	2,14	1.772192
7	LB	LBS		1.3664	5.607333	3,21	1.869111
8	LS				1394.715	21	66.41498
9	BS				111.6841	7	15.95488
10	LD	LSD		3.2737	37.89993	3,21	12.63331
11	BD	BSD		0.6874	3.006868	1,7	3.006868
12	SD				101.8523	7	14.55033
13	LV	LSV		0.5027	2.636433	6,42	.4394056
14	BV	BSV		0.0091	.0333183	2,14	.0166591
15	SV				31.59858	14	2.257041
16	DV	SDV		3.3295	28.36991	2,14	14.18495
17	LBS				28.72520	21	1.367867
18	LBD	LBSD		2.1011	2.931639	3,21	.9772131
19	LSD				81.03946	21	3.859022
20	BSD				30.61841	7	4.374058
21	LBV	LBSV		0.3787	2.844489	6,42	.4740815
22	LSV				36.71444	42	.8741533
23	BSV				25.60122	14	1.828658
24	LDV	LSDV		0.3230	1.816523	6,42	.3027539
25	BDV	BSDV		0.7976	4.529826	2,14	2.264913
26	SDV				59.64567	14	4.260405
27	LBSD				9.767243	21	.4651068
28	LBSV				52.57615	42	1.251813
29	LBDV	LBSDV		0.2516	1.155111	6,42	.1925185
30	LSDV				39.36510	42	.9372644
31	BSDV				39.75713	14	2.839795
32	LBSDV				32.13174	42	.7650414

Means and Standard Deviations (microvolts) Across 8 Subjects

			...Days 2 - 4...			...Days 5 - 7...		
			0	25	50	0	25	50
SUBJECT BET	Frontal	MEAN	2.58	3.47	2.76	3.69	3.11	3.56
		s.d.	3.32	3.05	3.08	3.06	2.54	3.06
	Vertex	MEAN	7.05	8.14	7.43	7.81	6.57	7.03
		s.d.	4.08	4.58	4.73	3.33	2.83	3.34
	Parietal	MEAN	4.83	5.68	4.99	4.88	3.47	3.71
		s.d.	2.96	4.73	4.15	2.33	1.80	2.23
	Occipital	MEAN	-0.08	0.61	0.21	0.20	-0.50	-0.65
		s.d.	3.75	3.27	2.22	3.40	2.52	2.80
COMPUTER BET	Frontal	MEAN	3.27	3.57	3.39	4.12	3.54	3.69
		s.d.	3.39	3.29	3.63	2.59	3.21	2.84
	Vertex	MEAN	8.11	7.94	7.71	7.77	6.73	7.05
		s.d.	3.34	3.48	3.50	2.15	3.07	2.86
	Parietal	MEAN	4.99	5.50	5.07	4.20	3.94	3.93
		s.d.	2.53	2.51	2.52	1.98	1.92	2.21
	Occipital	MEAN	0.14	0.81	0.43	-0.89	-0.90	-1.29
		s.d.	2.25	1.41	2.30	2.58	2.61	3.21

ANALYSIS OF VARIANCE FOR WARNING CLICK: N284 COMPONENT

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S	p<.01	27.0297	1090.532	1,7	1090.532
2 L=SCALP LOC	LS		1.2574	165.1951	3,21	55.06505
3 B=BET SOURCE	BS		1.3332	12.47042	1,7	12.47042
4 S=SUBJECTS				282.4201	7	40.34573
5 D=3 DAY PER.	SD		2.0276	10.42143	1,7	10.42143
6 V=BET VALUE	SV		0.1888	1.858215	2,14	.9291075
7 LB	LBS		6.3120	18.24020	3,21	6.080066
8 LS				919.6404	21	43.79240
9 BS				65.47384	7	9.353406
10 LD	LSD		0.3815	5.797626	3,21	1.932542
11 BD	BSD		0.3222	.6987091	1,7	.6987091
12 SD				35.97839	7	5.139770
13 LV	LSV		1.2102	6.624672	6,42	1.104112
14 BV	BSV		1.8275	15.83773	2,14	7.918865
15 SV				68.88147	14	4.920105
16 DV	SDV		0.6872	5.071974	2,14	2.535987
17 LBS				20.22848	21	.9632610
18 LBD	LBSD		4.1707	7.691696	3,21	2.563899
19 LSD				106.3915	21	5.066262
20 BSD				15.17923	7	2.168462
21 LBV	LBSV		0.9244	3.894050	6,42	.6490084
22 LSV				38.31914	42	.9123605
23 BSV				60.66391	14	4.333136
24 LDV	LSDV		0.2962	1.583693	6,42	.2639488
25 BDV	BSDV		0.0666	.1758111	2,14	.0879056
26 SDV				51.66437	14	3.690312
27 LBSD				12.90948	21	.6147372
28 LBSV				29.48764	42	.7020866
29 LBDV	LBSDV		0.8456	4.026676	6,42	.6711126
30 LSDV				37.42868	42	.8911590
31 BSDV				18.47666	14	1.319762
32 LBSDV				33.33515	42	.7936940

Means and Standard Deviations (microvolts) Across 8 Subjects

			...Days 2 - 4...			...Days 5 - 7...		
			0	25	50	0	25	50
SUBJECT BET	Frontal	MEAN	-2.21	-2.18	-1.93	-1.49	-1.58	-1.63
		s.d.	1.27	1.30	2.12	1.93	1.87	1.86
	Vertex	MEAN	-1.78	-2.35	-1.64	-1.34	-2.05	-1.70
		s.d.	2.01	2.46	2.86	2.70	2.79	2.81
	Parietal	MEAN	-1.52	-1.12	-0.60	-1.32	-1.70	-0.61
		s.d.	1.72	1.24	2.47	2.37	1.78	1.82
	Occipital	MEAN	-3.56	-2.94	-2.01	-2.45	-2.90	-2.16
		s.d.	3.27	1.92	2.46	3.45	3.28	3.74
COMPUTER BET	Frontal	MEAN	-1.54	-1.01	-1.66	-0.43	-0.64	-1.02
		s.d.	1.41	0.93	1.50	0.55	1.66	1.57
	Vertex	MEAN	-1.48	-1.30	-1.44	-0.72	-0.53	-0.84
		s.d.	2.68	1.41	2.59	2.27	1.25	2.11
	Parietal	MEAN	-2.00	-0.86	-1.12	-0.64	-0.46	-0.95
		s.d.	2.83	1.44	2.70	1.96	1.40	1.36
	Occipital	MEAN	-2.70	-2.56	-2.90	-3.22	-2.64	-3.49
		s.d.	2.24	1.89	2.66	3.96	2.77	3.94

ANALYSIS OF VARIANCE FOR WARNING CLICK: P328 COMPONENT

All F-tests non-significant

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S		0.0018	.1679192	1,7	.1679192
2	L=SCALP LOC	LS		1.5242	266.6749	3,21	88.89164
3	B=BET SOURCE	BS		0.8083	11.65175	1,7	11.65175
4	S=SUBJECTS				636.9218	7	90.98883
5	D=3 DAY PER.	SD		0.1391	1.513782	1,7	1.513782
6	V=BET VALUE	SV		1.7548	6.814953	2,14	3.407477
7	LB	LBS		4.0345	22.21393	3,21	7.404644
8	LS				1224.692	21	58.31865
9	BS				100.9059	7	14.41513
10	LD	LSD		2.9472	43.45282	3,21	14.48427
11	BD	BSD		7.3084	9.910565	1,7	9.910565
12	SD				76.16376	7	10.88054
13	LV	LSV		1.6640	9.235462	6,42	1.539244
14	BV	BSV		0.4459	6.728809	2,14	3.364405
15	SV				27.18550	14	1.941821
16	DV	SDV		0.6521	3.693357	2,14	1.846679
17	LBS				38.54155	21	1.835312
18	LBD	LBSD		0.2225	1.237651	3,21	.4125503
19	LSD				103.2072	21	4.914626
20	BSD				9.492396	7	1.356057
21	LBV	LBSV		0.4498	2.859490	6,42	.4765817
22	LSV				38.85213	42	.9250507
23	BSV				105.6390	14	7.545645
24	LDV	LSDV		0.3459	2.033720	6,42	.3389534
25	BDV	BSDV		0.3765	1.787463	2,14	.8937314
26	SDV				39.64595	14	2.831853
27	LBSD				38.93155	21	1.853883
28	LBSV				44.50220	42	1.059576
29	LBDV	LBSDV		0.8093	3.088938	6,42	.5148230
30	LSDV				41.15075	42	.9797798
31	BSDV				33.23170	14	2.373693
32	LBSDV				26.71850	42	.6361548

Means and Standard Deviations (microvolts) Across 8 Subjects

			...Days 2 - 4...			...Days 5 - 7...		
			0	25	50	0	25	50
SUBJECT BET	Frontal	MEAN	-1.18	-1.36	-0.89	0.29	0.09	0.31
		s.d.	1.19	1.52	2.13	2.02	1.64	2.20
	Vertex	MEAN	0.23	-1.05	-0.60	0.77	0.19	0.50
		s.d.	2.19	3.36	3.65	2.18	2.24	2.57
	Parietal	MEAN	0.94	0.80	1.20	0.71	0.40	0.85
		s.d.	1.97	3.66	3.93	2.77	1.81	2.60
	Occipital	MEAN	-1.48	-0.91	-0.73	-1.31	-0.77	-1.68
		s.d.	4.54	3.88	3.53	4.31	4.56	4.06
COMPUTER BET	Frontal	MEAN	0.23	0.19	-0.20	0.87	0.74	0.14
		s.d.	2.26	1.57	1.58	0.90	1.96	1.54
	Vertex	MEAN	0.80	0.44	0.66	1.66	0.84	0.34
		s.d.	2.15	1.62	2.28	2.06	0.96	1.89
	Parietal	MEAN	1.36	1.38	1.21	1.30	0.41	0.43
		s.d.	3.24	2.66	2.71	2.87	1.42	2.14
	Occipital	MEAN	-0.99	-0.79	-1.28	-1.55	-2.01	-2.51
		s.d.	2.48	2.04	3.47	4.09	3.80	4.20

ANALYSIS OF VARIANCE FOR CONTINGENT NEGATIVE VARIATION

SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1 MEAN	S	p<.01	20.1393	5444.416	1,7	5444.416
2 L=SCALP LOC	LS		1.1201	248.1806	3,21	82.72686
3 B=BET SOURCE	BS		8.7870	202.2607	1,7	202.2607
4 S=SUBJECTS				1892.363	7	270.3375
5 D=3 DAY PER.	SD	p<.01	13.4336	314.7047	1,7	314.7047
6 V=BET VALUE	SV		1.3542	7.391512	2,14	3.695756
7 LB	LBS		2.4026	36.36001	3,21	12.12000
8 LS				1550.943	21	73.85443
9 BS				161.1281	7	23.01830
10 LD	LSD		1.9226	19.20895	3,21	6.402983
11 BD	BSD		0.0330	.1842384	1,7	.1842384
12 SD				163.9866	7	23.42665
13 LV	LSV		1.4555	8.092508	6,42	1.348751
14 BV	BSV		1.6902	18.52065	2,14	9.260327
15 SV				38.20878	14	2.729199
16 DV	SDV		0.6666	2.100839	2,14	1.050420
17 LBS				105.9342	21	5.044485
18 LBD	LBSD		2.2397	10.44314	3,21	3.481046
19 LSD				69.93623	21	3.330296
20 BSD				39.12745	7	5.589636
21 LBV	LBSV		1.1556	5.239439	6,42	.8732398
22 LSV				38.92060	42	.9266810
23 BSV				76.70260	14	5.478757
24 LDV	LSDV		0.7138	2.583148	6,42	.4305246
25 BDV	BSDV		0.2990	1.206591	2,14	.6032956
26 SDV				22.06153	14	1.575824
27 LBSD				32.63911	21	1.554243
28 LBSV				31.73832	42	.7556743
29 LBDV	LBSDV		0.9768	3.501150	6,42	.5835249
30 LSDV				25.33126	42	.6031253
31 BSDV				28.25260	14	2.018043
32 LBSDV				25.09032	42	.5973886

Means and Standard Deviations (microvolts) Across 8 Subjects

			...Days 2 - 4...			...Days 5 - 7...			CERTAIN
			0	25	50	0	25		
SUBJECT BET	Frontal	MEAN	-5.55	-6.44	-6.33	-4.18	-4.68	-3.88	-1.03
		s.d.	2.49	2.42	3.02	4.43	2.88	3.32	2.74
	Vertex	MEAN	-6.48	-6.66	-6.51	-4.33	-4.49	-4.49	-0.74
		s.d.	3.93	3.62	4.82	5.59	4.22	5.53	2.44
	Parietal	MEAN	-5.75	-5.28	-5.19	-3.89	-3.64	-3.55	-0.44
		s.d.	4.00	3.88	3.95	4.60	3.45	4.08	3.16
	Occipital	MEAN	-4.28	-3.42	-3.15	-2.28	-1.34	-2.02	-0.57
		s.d.	4.05	2.66	3.21	4.06	3.73	3.64	3.61
COMPUTER BET	Frontal	MEAN	-4.51	-4.84	-5.18	-2.44	-2.50	-3.28	
		s.d.	3.29	2.39	2.84	2.92	3.57	2.92	
	Vertex	MEAN	-4.06	-4.91	-5.17	-1.73	-1.90	-2.68	
		s.d.	3.64	2.65	3.89	3.55	3.37	3.67	
	Parietal	MEAN	-2.84	-3.73	-4.24	-1.64	-1.40	-2.30	
		s.d.	3.75	2.65	3.77	3.37	3.15	2.96	
	Occipital	MEAN	-2.30	-2.50	-2.81	-1.82	-1.68	-2.52	
		s.d.	2.64	1.97	3.04	3.99	3.44	2.98	

ANALYSIS OF VARIANCE FOR EYE MOVEMENT ARTIFACTS
All F-tests non-significant

	SOURCE	ERROR TERM	SIG. df=1,7	F	SUM OF SQUARES	DEG. OF FREEDOM	MEAN SQUARE
1	MEAN	S		8.1381	2144.470	1,7	2144.470
2	C=STIMULI	CS		3.2166	54.11511	2,14	27.05755
3	B=BET SOURCE	BS		0.1751	1.394449	1,7	1.394449
4	S=SUBJECTS				1844.570	7	263.5099
5	O=OUTCOME	SO		0.0182	.0460050	1,7	.0460050
6	V=BET VALUE	SV		2.2066	8.538254	2,14	4.269127
7	CB	CBS		2.2579	6.174034	2,14	3.087017
8	CS				117.7650	14	8.411783
9	BS				55.74428	7	7.963468
10	CO	CSO		2.4755	20.85450	2,14	10.42725
11	BO	BSO		0.0376	.0566743	1,7	.0566743
12	SO				17.72984	7	2.532835
13	CV	CSV		0.1715	.9375865	4	.2343966
14	BV	BSV		0.1322	.3124410	2,14	.1562205
15	SV				27.08617	14	1.934726
16	OV	SOV		3.2942	6.214278	2,14	3.107139
17	CBS				19.14126	14	1.367233
18	CBO	CBSO		0.3382	.9771192	2,14	.4885596
19	CSO				58.97035	14	4.212168
20	BSO				10.56135	7	1.508765
21	CBV	CBSV		0.4709	2.496104	4	.6240260
22	CSV				38.27538	28	1.366978
23	BSV				16.54437	14	1.181741
24	COV	CSOV		0.1726	1.208987	4	.3022468
25	BOV	BSOV		0.7920	1.881759	2,14	.9408793
26	SOV				13.20520	14	.9432286
27	CBSO				20.22660	14	1.444757
28	CBSV				37.10302	28	1.325108
29	CBOV	CBSOV		0.9987	6.632871	4	1.658218
30	CSOV				49.02817	28	1.751006
31	BSOV				16.63095	14	1.187925
32	CBSOV				46.49181	28	1.660422

Means and Standard Deviations (microvolts) Across 8 Subjects

	Stimulus		Win 0	Win 25	Win 50	Lose 0	Lose 25	Lose 50	CERTAIN
SUBJECT BET	Emitted	MEAN	3.18	2.83	3.42	3.99	3.83	4.23	0.91
		s.d.	2.12	2.63	3.23	2.34	2.58	2.43	3.12
	Single	MEAN	2.51	3.30	2.98	2.28	1.68	2.73	1.07
		s.d.	2.45	1.72	2.55	2.88	2.96	2.53	2.81
	Double	MEAN	2.25	2.33	2.37	1.82	2.40	2.23	-0.16
		s.d.	2.90	3.40	2.90	2.83	4.85	2.88	3.39
COMPUTER BET	Emitted	MEAN	2.13	3.19	2.98	3.25	2.70	3.92	
		s.d.	2.70	3.51	3.34	3.73	2.86	2.92	
	Single	MEAN	2.74	2.99	2.99	2.38	2.33	2.56	
		s.d.	2.41	2.04	2.60	3.72	3.42	2.90	
	Double	MEAN	2.17	2.61	2.38	2.06	1.68	2.81	
		s.d.	3.83	4.54	3.93	3.10	2.64	3.85	