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ANNUAL REPORT
A PSYCHOPHYSIOLOGICAL MAPPING OF COGNITIVE PROCESSES
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April 30, 1985

*We would like to acknowledge Kevin Socha, M.S., and Marla Cage, B.S., for their work in developing the software used in this project.
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<td>This is the second annual report to be completed under Air Force Office of Scientific Research contract F49620-83-C-0059. The report details the background, preliminary findings, and conclusions of a study conducted in the Washington University Behavior Research Laboratory over the past year. The experiment was concerned with the effects of varied cognitive and perceptual (i.e., monitoring) demands on patterns of physiological responding. Cognitive demands were varied by manipulating the number of letters (1, 3, or 5) comprising a briefly-presented set which the subject was instructed to encode, rehearse, and,</td>
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5 sec later, compare to a single test letter. Perceptual demands were varied by presenting the subject with a cue stimulus (the numeral "1", "3", or "5") 5 sec prior to the set, informing him of the number of letters contained therein. Several physiological measures were recorded, including HR, EOG and "probe" evoked potentials sampled from the intervals preceding and following the letter set, and "task" evoked potentials and blinks elicited by the cue, letter set, and test stimuli. Performance data, i.e., RT and error rates, were also recorded.

Unique patterns of physiological activity characterized the intervals preceding and following the presentation of the letter set. Within the interval preceding the letter set, where, we presume, that perceptual demands were varied, probe ERP P2-N2 amplitude increased with increasing set size. Within the subsequent interval, where, we presume, that a different, higher level process was being tapped, a later component of the probe ERP, viz., P4-N4, proved sensitive to set size. There was evidence for an interval effect on HR as well.

Changes in task ERPs and RT generally replicated the findings of other investigators. The letter set elicited a P2-N2 component which increased with increasing set size. This was attributed to greater storage requirements associated with larger letter sets. The test stimulus elicited a N2-P3 component which decreased with increasing set size. This was attributed to greater uncertainty associated with more difficult judgments and its presumed effect on P3 amplitude. RT increased as a simple, linear function of set size for both positive and negative responses.
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1.0 Overview

The following report represents the effort, to date, of the Washington University Behavior Research Laboratories (WUBRL) on the contract, *A Psychophysiological Mapping of Cognitive Processes*.

1.1 Hardware

The apparatus for conducting the experiments for this contract has been constructed. It is depicted schematically in Figure 1 and described below in conjunction with the experimental protocol.

Insert Fig. 1 about here

1.2 Software

Stimulus generation and display programs have been completed and will be described below as well. Physiological responses are digitized on-line and stored on disk, as indicated in Figure 1. For purposes of back-up and monitoring, respectively, raw data are also recorded on analog tape and polygraph strip chart.

Development of programs for computer analysis of digitized physiological data is still in process. Accordingly, not all physiological measures can be reduced from disk as yet. For the experimental work reported here, twenty subjects were run for each of whom we have five channels of information: Event-related Potentials (Fz and Pz), EOG, Heart Rate, and
Stimulus/Response occurrence. For purposes of the present report, a sample of these data (the first of four experimental sessions for each of eight subjects) has been reduced, collated and analyzed statistically. The limiting factor for the remaining subjects was the (electrical) noise in the stimulus channel on analog tape which precluded reducing data from all subjects from tape. This will not present a problem when reducing the digitized data since the timing of events there is referenced to the known clock time at which stimuli were presented to the subjects. Thus, when reduction programs are completed, which is imminent, the remainder of the data will be processed. All physiological and response data for the 8 subjects have been analyzed with one exception: for the blink data there was only a partial overlap (about half) with the subjects represented in the other measures. Thus, these data are not strictly comparable with the others. For present purposes we will ignore this point.

2.0 Introduction

Components of the transient change in the electroencephalogram immediately following the presentation of task relevant stimuli, i.e., the Event Related Potential (ERP or EP), have been reported to show systematic changes in amplitude and latency in many situations in which the internal state of the subject is manipulated. Almost as many constructs have been used to explain these observations as experiments have been carried out. As a consequence, many of these
hypothesized constructs overlap to a large extent and others are so vague as to be of little scientific value.

There is accumulating evidence that in many situations, the later cortical potentials, among those that operationally define these constructs, are less closely coupled to stimulus parameters than are earlier components. A clear definition of the psychological dimensions which determine the late components has proven to be elusive. It has been suggested, for example, that a positive wave about 300 msec after stimulus onset (the P300) is associated with the resolution of uncertainty. If a subject makes a judgment about such a stimulus, then a P300 will be evoked at an amplitude which is inversely related to the difficulty of that judgment (Andreassi & Juszcak, 1984; Gomer, Spicuzza, & O'Donnell, 1976; Squires, Squires, & Hillyard, 1975), and at a latency which is directly related to rated difficulty (Adam & Collins, 1978; Ford, Roth, Mohs, Hopkins, & Kopell, 1979; Hillyard & Kutas, 1983; Kutas, McCarthy, & Donchin, 1977). Task relevance has also been found to affect the amplitude of this wave (Donchin et al., 1978; Hillyard, Hink, Schvent, & Picton, 1973). However, these notions of "resolution of uncertainty" and "task relevance" may be less closely related than is usually thought (cf. Ford, Roth, & Kopell, 1976). Furthermore, the classic P300 wave is now known to be comprised of a complex of orthogonal but temporally overlapping components (Chapman et al., 1979; Ford et al., 1972; Friedman, Vaughan, & Erlenmeyer-Kimling, 1981;
Squires, Squires, & Hillyard, 1975), which may each be related to a unique process. Unfortunately, the isolation of these components presents both statistical (Hunt & Tianwattanatata, 1984; Wood & McCarthy, 1984) and theoretical problems that preclude a simple interpretation on the basis of variations in this one measure.

Complexities have arisen with the interpretation of components of the event-related heart rate response as well. HR deceleration in anticipation of a task-relevant stimulus has been noted by several authors, including Bernstein, Taylor, Weinstein, & Riedel (1984), Coles, & Duncan-Johnson (1975), Jennings & Hall (1980) and Walter & Porges (1976), but the data on the relationship between the magnitude of the deceleration and either the difficulty of the imminent demand or the accuracy of the response are inconclusive. The accelerative change following the presentation of a task-relevant stimulus is also well-documented and similarly enigmatic.

In general, therefore, it seems reasonable to suppose that an array of physiological measures, taken together, could more adequately characterize a psychological process than any one measure, such as P300 or HR, taken alone. We have chosen to accept this supposition as our working hypothesis: that psychological processes can be identified by unique physiological patterns. The very fact that psychological constructs can be logically and functionally discriminated from others implies that this is the case. This hypothesis also
suggests that the interpretation of a change in one measure may be simplified by consideration of concomitant variation in other measures.

To these ends, we have utilized a 3 stimulus paradigm incorporating aspects of a fixed foreperiod design into a paradigm originally developed by Sternberg (1966). It involves the presentation of a cue stimulus (S1) which, itself, requires minimal processing, although it informs the subject of the perceptual demands to be imposed by the subsequent task. There must be, we assume, a unique pattern of preparatory (i.e., monitoring) activity triggered by this stimulus whose strength varies along some continuous multivariate dimension, as others, examining individual measures in other paradigms, have suggested (e.g., Coles, 1974; Naatanen, 1982; Tecce, 1972).

The second stimulus (S2) is a set of 1, 3, or 5 consonant letters which the subject is instructed to rehearse for later comparison with a test item (S3). Within the interval between the presentations of the memory set and the test item, qualitatively different processes might be evoked, the strengths of which may vary with the cognitive demands imposed by the antecedent task, i.e., the number of items which must be retained and subsequently compared to the test item. We would expect this to be manifested in a pattern of physiological activity distinctly different from that occurring in the previous interval (Bauer, Keen, & Mouton, 1983; Walter & Porges, 1976).
Capitalizing on the multivariate psychophysiological approach to the study of human information processing, we have chosen some measures not commonly studied by other investigators in this context, in the hope that these might enhance our understanding of the underlying processes. One of these is the EEG response evoked by simple task-irrelevant visual stimuli presented at random times within the trial. These noninformative "probe" stimuli (see Papanicolaou & Johnstone, 1984, for a review), if they utilize processing resources accessed by the task-relevant stimuli, would be expected to elicit responses which vary with the degree of communality and the capacity of the shared resource pool. Accordingly, in the present context, we expect to demonstrate modulation of the early components of the probe ERP as perceptual (i.e., monitoring) demands are heightened (in the warning or cue interval), and modulation of the late components when cognitive demands are pressing (as in the memory interval).

Another measure, not previously used in this context, is the timing of eyeblinks. We have, in other contexts, suggested that blink timing, like P300 latency, indexes a momentary relaxation of information processing demands following the presentation of task relevant stimuli (Stern, Walrath & Goldstein, 1984). This was demonstrated by our finding (Bauer, Strock, Goldstein, Stern & Walrath, 1985) that blinks which occurred between trials of stimulus presentation did not occur...
at random. When stimuli were delivered at a rate of approximately 1 every 2.3 seconds, blinks occurred at an average latency of approximately 600 msec, but only when active attention to the stimuli was required. Under "no-task" conditions, the average blink latency increased to approximately 1100 msec.

Applying these results to the present design, we would expect that if the anticipation, encoding, and scanning of larger memory sets is a relatively more difficult task, and that if blinking is delayed until information processing demands momentarily subside, then blink latency should be generally greater for trials with larger memory sets than smaller ones. This effect, we believe, should be particularly marked in the interval following the presentation of the memory set, when information processing demands are at their peak.

3.0 Method

3.1 Subjects

The data for eight, right-handed, college age men who took part in the experiment are reported here. All had normal or corrected-to-normal vision and reported unremarkable medical histories. Each was paid $15 for his participation.

3.2 Apparatus

Task-relevant stimuli were presented by activation of an LED alphanumeric display unit (IEE, Inc., 1 X 20 Dot Matrix Display Module) located behind a 1.3 cm clear slit running horizontally across the length of a 0.6 m x 1.9 m black plastic
sheet (the full apparatus consisted of seven display modules, as indicated in Figure 1; only the center one was used for the present experiment). The sheet was flexed along its length into a 120 degree circular arc and fixed to the surface of a table. The subject was seated 1.5 m in front of this table such that his eyes were level with the stimulus display. A joystick, with which the subject could indicate his response, was attached to the table and was accessible to the subject's right hand.

Centered 42 cm above the display was a 34cm X 34 cm translucent panel which could be backlit (100 msec, 4 fL flashes). Illumination of this panel served as the probe stimulus.

This apparatus was located in a 2.3 m x 2.75 m, sound attenuated, electrically shielded room. Illumination was provided by two overhead incandescent lights located to either side and slightly behind the subject chair. Ambient light intensity was 6.14 fc.

The electroencephalogram (EEG) was recorded from chlorided Grass silver cup electrodes attached by means of gauze pads impregnated in collodion to two midline scalp sites: frontal (Fz) and parietal (Pz). Each of these electrodes was referenced to linked chlorided silver Grass clips attached to the earlobes. Beckman miniature biopotential electrodes were taped to the center of the forehead (ground) as well as above and below the left eye, the source of the electrooculographic
signal (EOG). Finally, for the electrocardiogram (EKG), Beckman miniature electrodes were positioned on the lateral aspects of the rib cage. Impedances at all capital sites were kept below 5 Kilohms.

The EEG and EKG signals were amplified by Tektronix Model AM 502 differential amplifiers (EEG: gain = 10K, nominal bandpass = 0.1 to 1000 Hz; EKG: gain = 2K, bandpass = 0.1 to 1000 Hz), and the EOG by a specially constructed amplifier (gain = 1.5K, bandpass = DC to 1 kHz). Each of these physiological signals, along with stimulus and response event markers, were stored on analog tape (Ampex) and, in digital form (sampling rate = 200 Hz), on computer disk, for off-line analysis.

3.3 Procedure

The cue stimuli (Si) were the numerals "1", "3", or "5" (avg. luminance = 45 fc) projected for 700 msec centered on the display unit. The memory set and test stimuli were presented for the same duration and at the same location. They were selected at random, without replacement, from a set of 18 consonant, upper-case letters (excluding the letters, "Y", "W", and "V") with the restrictions that in each sequence of 150 trials, the three set sizes occurred equally often, the test stimulus occurred with equal frequency at each position in the memory set, and the test stimulus was a member of the memory set on half the trials.
Subjects were tested on 2 days (modal interval = 2 days) at approximately the same clock time. Each experimental day was divided into three periods. The first was a 5 minute block consisting of thirty practice trials. This was followed by two 31 minute blocks consisting of 113 and 112 trials separated by a 3-5 min rest period.

Each trial consisted of the presentation of a cue stimulus, a memory set, and a test stimulus, in sequence, at regular intervals (SOA = 5700 msec). On a given trial the irrelevant probe stimuli might be presented at one of nine temporal locations in the interval following the cue stimulus and one of nine in the interval following presentation of the memory set. In each interval they were grouped into three sets of three, one set at the outset of the interval (1000, 1300, or 1600 msec after stimulus offset), one in the middle of the interval (2200, 2500, or 2800 msec after stimulus offset), and one at the end of the interval (3400, 3700, or 4000 msec after offset). On 10% of the trials (i.e., 45/450), the probe stimuli were omitted from the cue and memory intervals entirely and inserted, instead, in the intertrial interval, i.e., following the test stimulus. The durations and onset times of all stimuli were under the control of an LSI 11/23 minicomputer.

On each trial, the value of the cue stimulus indicated that 5700 msec later, a 1, 3, or 5 item memory set would occur, centered horizontally about the same location. Five seconds
after the memory set turned off, a test item was presented, to which subjects were instructed to make a discriminative response with the right hand. For one half of the subjects, this meant that they were to move a joystick to the right if the test item was an element of the memory set (a "positive" response), and to the left if it was not (a "negative" response). For the remaining subjects, left was positive and right was negative.

3.4 Data Reduction

3.4.1 Event-Related Potentials (ERPs). The EEG and EOG signals were digitized from analog tape at a rate of 250 Hz on a PDP 11/40 computer with appropriate software. Epochs of EEG contaminated by eye movements or A/D converter overflow were rejected. When not characterized by such artifacts, epochs of EEG 100 msec preceding and 500 msec following task stimulus onset and 100 msec preceding and 700 msec following probe stimulus onset were retained. For each subject, these data were combined into time point averages, temporally locked to the stimuli. The averages were computed separately for Fz and Pz leads. For the task stimulus ERPs, i.e., those elicited by the cue, memory set, and test stimuli, the averages were further subdivided by stimulus type and set size. For the probe stimulus ERPs, the averages were subdivided by set size and interval (i.e., cue and memory interval probes). For present purposes, responses to probe stimuli were pooled for each interval producing one probe ERP for the cue interval and
one for the memory interval. (In the full data analysis, probe ERPs will be sorted by location (3) within each of these intervals.) Probe ERPs from the test interval were discarded. Each average was based on 20-25 trials.

Before components of the task stimulus ERPs were measured, the ERPs were digitally filtered. This was accomplished by 20 iterations of an algorithm that forms a weighted average (following a binomial function) of a sampling point and the points before and after it. The probe ERPs were passed through an analog filter (Krohn-Hite Model 3342, bandpass = DC to 8 Hz, roll-off = 24 db/octave).

Six components were identified in each of the averaged ERPs. Probe stimulus ERPs were characterized by a complex of 6 alternate positive and negative going waves occurring within latency ranges of 160-260, 260-340, 340-440, 440-500, 500-560, and 560-700 msec post-stimulus onset. The maximum or minimum voltage of the averaged ERP occurring within each of these windows was determined to be the amplitude, with respect to a 100 msec prestimulus baseline, of P2, N2, P3, N3, P4, and N4, respectively. (Note: The early components of the probe stimulus ERPs, i.e., P1 and N1, were obscured by a switch artifact associated with the offset of the probe stimulus and could not be measured. Measurement of these components will be possible when programs for the reduction of ERP data stored on computer disk are complete). Six components were identified in the task stimulus ERPs as well. Here, peaks occurring within
latency ranges of 68-112, 112-152, 152-200, 200-260, 260-400, and 400-500 ms were identified as P1, N1, P2, N2, P3, and N3, respectively. Prior to analysis, all peak-to-baseline amplitudes were converted to peak-to-peak amplitudes. This conversion simplifies the interpretation of a change in component amplitude when the baseline voltage of the ERP is changing over time.
3.4.2 EKG and Performance. EKG and performance data were digitized on-line and later subjected to averaging. The digitized EKG signal was converted to heart rate, expressed as the number of whole and fractional beats per minute (bpm) occurring in each of eighteen 950 msec bins throughout the trial. It was averaged over trials of the same type (i.e., set size). Reaction times, calculated from response signal onset, were segregated by response type (positive and negative) and set size prior to averaging.

3.4.3 Blink Latency. Stimulus and EOG signals, stored on analog tape, were digitized off-line at a rate of 200 Hz on a PDP-11/40 computer. A reduction program applied to the digitized data identified as blinks, those voltage deflections that met specified polarity, amplitude, duration, and velocity criteria and calculated their onset times with respect to the onset of the preceding task stimulus. Only the first blink following onset of a task-relevant stimulus was accepted and only if the blink was not preceded by a probe stimulus. Blink latencies were sorted by interval (cue, memory, and test) and set size and averaged.

4.0 Results

4.1 Heart Rate (HR)

Each of the three task intervals in a trial was divided into six bins commencing, respectively, with the onset of the task (cue, memory or test) stimulus. Heart rate in each of the six bins of each interval was then expressed as a difference
score based on the prestimulus HR of that interval. Thus, HR in the final bin of each interval served also as the baseline rate for the ensuing interval. These data are displayed in Figure 2. A 3 (task interval) X 6 (time bins) X 3 (set size) ANOVA was performed on these data with all variables within. Whereas HR exhibited a decelerative trend in the cue period, it was mainly accelerative in the memory and test intervals. This was reflected in a significant interval effect, $F(1,9)=9.08$, $p=.0103$. The time bin (pooled intervals) effect was also significant, $F(2,14)=29.18$, $p<.0001$. In general, we may describe the time course of the HR effect as triphasic. The initial effect is either nil or decelerative. This is followed by an accelerative phase of varying proportion and ends in a deceleration as the next stimulus is due. The significant time bin by interval interaction, $F(2,18)=12.53$, $p=.0002$, suggests that these components are not represented in equal proportion in the three intervals. This was ascertained by establishing confidence limits (set size pooled) around a zero change null, i.e., the baseline; any mean change from prestimulus baseline of more than $+1.15$ bpm is significant by t-test (using the Greenhouse-Geisser adjusted df and alpha = .05). Accordingly, the deceleration in the first bin of the cue interval was not significant though the final two cue points were. The
accelerative portions in the memory and test phases were both significant as was the initial decelerative change in the test interval.

4.2 Event-Related Potentials (ERPs)

Event-related potentials elicited by the task stimuli, i.e., the cue, memory set, and test stimuli, and those evoked by the probe stimuli will be discussed separately. Note that at this point in the data analysis, the task stimulus ERP data have not yet been segregated into groups based on match or mismatch trials nor the probe stimulus ERP data, by location in the interval.

The analytic procedure for both task and probe EPs was a multivariate ANOVA (MANOVA) in which independent variables were Stimulus Type (cue, memory, and test stimuli for the task EPs; only the first two for probe EPs), set size (1, 3, 5), and Lead (Fz vs Pz). Dependent variables were the five EP components. Univariate analyses are reported for a variable only when the test for that variable was significant in the overall MANOVA.

4.2.1 Task Stimuli. Group averaged tracings for task EPs are presented in Figure 3. There was a significant overall effect of Stimulus Type on the collective components, 

\[ F(10, 20) = 3.93, \ p = .0044. \]

Univariate analyses revealed significant Stimulus Type effects on two components: N2-P2
(F(1,11)=5.13, p=.0312), and P3-N3 (F(1,8)=5.48, p=.0402).

These effects are displayed in Figure 4A. For both components amplitudes were greater for the memory and test stimuli than for the cue stimuli.

Though set size exhibited no independent overall effect in the MANOVA it did interact with stimulus type, F(20,80)=2.49, p=.0021. Univariate Fs for both N2-P3 (F(2,18)=3.44, p=.0417) and P2-N2 (F(2,15)=5.81, p=.0112) were significant contributors to this effect. With respect to N2-P3 (Figure 4B), the data suggest that the response to the test stimulus is the only one of the three task stimuli that reflects set size; simple effects tests bore this out (CUE: F(1,12)= 0.53, p=.58; MEMORY: F(1,9)=1.72, p=.22; TEST: F(1,9)=7.12, p=.0184). In contrast to the N2-P3 component, which was sensitive to set size in the test stimulus ERP only, the P2-N2 component, as can be seen in panel C of Figure 4, showed the set size effect only to the memory set (simple effects (F(1,9)= 9.45, p=.0085)).

Set size was positively related to P2-N2 amplitude in response to memory stimuli. Although there was an apparent inverse relationship of amplitude and set size in the test stimulus ERP this did not approach significance, F(1,10)=2.05, p=.18.

Electrode locus, i.e., Fz vs Pz, was also a significant variable in the MANOVA, F(5,3)=27.08, p=.0106. Univariate
analyses revealed that the N2-P3 component differed as a function of recording derivation, the amplitude at Pz significantly exceeding that at Fz, $F(1,7)=23.99$, $p=.0018$. The P3-N3 component was also distinguished by recording origin but here Fz amplitude was significantly greater than Pz amplitude.

4.2.2 Probe Stimuli. As will be recalled, probe EPs were obtained only for the intervals following the cue and memory stimuli. Group averaged EPs are displayed in Figure 5. None of the main effects: stimulus type (cue, memory), electrode locus, or set size, was significant in the overall MANOVA. The stimulus type X set size interaction, however, was significant in the MANOVA, $F(10,24)=2.27$, $p=.0490$. Univariate analyses of the data displayed in Figure 6 indicated that amplitude was a direct function of set size for P2-N2 in the cue interval, $F(1,10)=7.06$, $p=.0184$, but not in the memory interval. Though P4-N4 amplitude was also positively related to set size (Figure 7), this held for the memory interval only, unlike the P2-N2 component.
4.3 Blink Latency

Blink latency data were subjected to a 3 (Interval) X 3 (Set size) ANOVA, with both factors within. As can be seen in Figure 8, blinks occurred earlier in the cue interval than in either of the other intervals. This was manifested as a significant Interval effect, $F(1,10)=6.78, p=.0215$. Neither the Set size effect nor the Set size by Interval interaction was significant. Since there was no \textit{a priori} reason to expect a set size effect for blink latency in the cue interval, a second, exploratory, analysis was performed excluding the cue interval data. In the revised analysis, the set size effect was significant. This will have to be explored further in the full analysis.

4.4 Reaction Time (RT)

Performance by subjects was virtually flawless: of the 113 trials presented in a session, error rated averaged 1.2% (range = 0 - 3.5%). Such trials have been excluded from the present analysis.

Reaction time data were subjected to a reciprocal transformation for normalization and then submitted to ANOVA.
Variables were set size and stimulus class, i.e., for the latter, trials were sorted by whether the test stimulus matched the memory items or not. For ease of interpretation, the non-transformed data are presented in graphical form (Figure 9).

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Insert Fig. 9 about here
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Both the set size and the stimulus class effect were significant ($F(1, 9) = 13.94, p = .0023; F(1, 7) = 19.36, p = .0032$, respectively). For matching items the mean increase in RT per item was 42.4 msec; for mismatched items, this value was 26.6. The lower slope for the mismatch items was due apparently to the aberrant latency for set size 5. Nevertheless, this did not produce an interaction of set size with stimulus class, $F(1, 10) = 0.88, p = .4225$, nor were the slopes of the RT by set size functions for match and mismatch trials significantly different, $t(7) = 0.91, p > .05$. With respect to the stimulus class effect, judgments of mismatch took 103.9 msec more, on the average, than judgments of match. The differences between match and mismatch means at each set size were significant, as suggested in the graph, for set sizes 1 and 3 ($t(7) = 3.32, p < .05$ and $t(7) = 2.61, p < .05$, respectively) but not for the set size of 5, $t(7) = 1.70, p > .05$. 
5.0 Discussion

With some notable exceptions, the heart rate data reported here follow patterns not unlike that reported in the literature. In contrast to the memory and test periods, heart rate following the cue stimulus was solely decelerative. There are suggestions, in the latter period, of the triphasic effect described in other studies (Bernstein, 1984; Jennings & Hall, 1980; Walter & Porges, 1976) by virtue of the acceleration in bins 2 and 3. The effect was minimal, however, and progressive deceleration was the trend, achieving significance as the memory set became imminent. If the accelerative phase is interpreted, along with Coles and Duncan-Johnson (1975), in terms of stimulus significance, then the absence of such a component in the cue period is consistent with the minor inherent significance of that stimulus. The deceleration at the termination of this period, a relatively substantial effect, suggests a fair degree of attention in anticipation of the memory set. Note, however, that one would expect that the heavier the anticipated load the greater this deceleration should be, according to this interpretation. This was not the case here; there was no set size effect or any interaction of other variables with set size. Although the full complement of data may yet alter this conclusion, the high probability values do not suggest hope along these lines.

The accelerations noted in both the memory and test intervals appear somewhat later (4 to 5 bins after stimulus
onset) than the incipient (and nonsignificant) acceleration observed after cue stimulus onset (2 to 3 bins poststimulus onset) which suggests a more prolonged processing period for these stimuli. On the other hand, the difference may simply be due to the larger peak heart rate in the later periods which takes longer to develop. In either event, after each instance of deceleration, the heart rate regresses toward prestimulus baseline. This reflects, we believe, not a natural cycle of acceleration-deceleration, but an anticipation of the next stimulus. Prolonging the interval, accordingly, would be expected to produce a less precipitous decline from peak rate. In addition, by removing the overriding early decelerative tendency produced by the present brief interstimulus interval, a by-product might well be a more pronounced accelerative component. In reviewing the ISI literature, Bohlin and Kjellberg (1979) arrived at the same conclusion, viz., that shorter ISIs tend to condense and truncate the triphasic cardiac response and, in so doing, reduce the latency and magnitude of the accelerative phase as well as the second deceleration. Applying this to the present data we may speculate a bit further that the absence of a set size effect here may also be a result of this artifact. That is, condensing the response may have masked a true set size effect by preventing the development of the full accelerative response. The problem posed by this issue is not simply that of determining the quantitative expression of the independent
variables but, more importantly, of ascertaining the consequences of truncation or lack of it for the underlying behavior: Does alteration of the HR response by manipulation of the ISI have either causal, incidental, or no relevance for the cognitive activity it purports to reflect?

The initial deceleration in the test period was a convincing effect observed in every subject and, indeed, at all set sizes. The first 950 msec time bin, in which this occurred maximally, was coexistent with several cognitive processes, viz., test stimulus encoding, scanning of the memory set, decision, response selection, and response execution. Data presented by Bernstein (1984) indicate that neither the stimulus itself, the detection response alone, nor a combination of the two produces a decelerative effect. Two possibilities for the decelerative effect emerge. The first concerns cognitive processes in the test period. These would include scanning of the memory set, as well as match-mismatch decision and response selection; note that the mean reaction time here was about 960 msec so that these processes were essentially completed prior to the end of the first test bin. A second alternative points to a residual from the preparatory phase (at the end of the memory period) as the origin of the deceleration. Internal events such as those occurring upon test stimulus onset have generally been associated with acceleration, which would tend to support the second alternative. Nevertheless, the sharp reversal from
deceleration to acceleration in the transition from the cue to the memory interval suggests that residual effects are not necessarily so persevering. Further speculation on the point will await full data reduction and analysis.

We contended, at the outset of this report, that multiple physiological indices, considered in combination, can enrich our understanding of cognitive processes, and of their physiological manifestations, to a greater degree than any single measure considered in isolation. A preliminary analysis of our data appears to support this thesis. That is, when subjects were instructed to anticipate a varying perceptual load (viz., the memory set) the pattern of physiological responding was markedly different from that which occurred when subjects were either encoding and rehearsing the memory set (in effect, a varying cognitive demand), or matching a memorial representation of the set to a test item.

That subjects anticipated the memory set can be inferred from the finding that their heart rate response was deceleratory and predominantly monophasic. It should be noted, however, that if we restrict our analysis to this one measure, we have only a unidimensional source of data, insensitive to the degree of anticipated perceptual load. When changes in another measure, viz., the amplitude of probe ERPs, occurring are examined within the same interval, however, we can more readily explain the absence of a set size effect on HR. That is, within the same interval where HR was found to be
Insensitive to set size, probe ERP P2-N2 amplitude was found to increase with increasing set size, a finding which is consistent with the position that the amplitude of the early negative ERP components reflects an attentional bias toward selected input channels (modalities) (Donald, 1983; Naatanen, 1982) which increases with increasing perceptual demands (Hillyard & Picton, 1979). That this increase in probe ERP P2-N2 amplitude is reflecting a selective process, as opposed to a nonselective one (such as arousal or activation), is reinforced by a consideration of the direction of HR change. We might therefore conclude that the modulation of probe ERP P2-N2 amplitude within the cue interval is reflecting a selective channeling of attention toward a particular sensory analyzer, while HR deceleration is merely an index of the availability of this capacity (cf. Bauer, 1982; Silverstein, Graham, & Bohlin, 1981).

It is interesting to contrast this pattern of physiological activity with a qualitatively different pattern occurring in the subsequent memory interval in which cognitive, rather than perceptual, processes were demanded. This manipulation was manifested in the modulation of a late component of the probe ERP and an acceleratory trend in the HR response, alluded to earlier. The demonstrated increase in probe ERP P4-N4 amplitude with increased memory set size, however, is somewhat problematic. A large body of evidence now indicates that the amplitude of another late component, viz.,
the P3, measured with respect to baseline, indexes the amount of processing resources allocated to the recognition and classification of task relevant stimuli. These studies have found that when the stimuli which elicit the P3 are deemed of secondary importance (Kramer et al., 1981), fewer resources are allocated to their processing and P3 amplitude is reduced. Since the probe stimuli used in the present study were of no instructed importance, we can only infer that they did not gain access to the same resource pool used by the task relevant stimuli. Therefore, our seemingly enigmatic finding of an increase in the amplitude of a late component of the probe ERP with increasing cognitive demands might be attributed to a nonselective activation of these subsidiary resource pools. This speculation is reinforced by our finding that the topographical distribution of P4-N4 is markedly different from that demonstrated for P3: the P4-N4 elicited by task relevant stimuli was largest frontally, while the P3 has been shown to be largest parietally (Hillyard & Picton, 1979).

Turning to the ERPs elicited by the task-relevant memory and test stimuli, we again have evidence for an association between a qualitatively different information processing demand and a qualitatively distinct pattern of physiological activity. The amplitude of the P2-N2 component of the memory set ERP increased with increasing set size. This finding parallels the now classic results of Chapman (reviewed in Chapman, 1981), who found that the amplitude of a similar component, viz., the
P250, elicited by a memory item, was correlated with behavioral measures of recall. As such, it was interpreted as a sign of storage in short term memory. In the present investigation, a similar interpretation could apply for the demonstrated increase in the amplitude of the P2-N2 component of the memory set ERP with increasing set size. However, the fact that the set size manipulation was confounded with the luminance of the ERP-eliciting stimulus implies caution in such an interpretation. It may be observed in this respect, however, that N1-P2 amplitude did not vary with set size, an effect which would be expected on the basis of a luminance difference.

The amplitude of a late component of the test stimulus ERP was found to vary with the number of items, memorally represented, to which the test item was compared. This decrease in test stimulus ERP N2-P3 amplitude with increasing set size has been demonstrated by many investigators (e.g., Andreassi & Juszcak, 1984; Gomer et al., 1976) in the past. It is consistent with interpretations of P3 amplitude reduction as being related to the subject's lack of confidence in his/her decision (Sutton, Braren, & Zubin, 1965).

The hypothesis, with respect to the blink latency data, would hold that set size should be positively related to blink latency, or that a Set Size X Interval interaction would occur in which set size would at least be a significant variable in the memory and test intervals. Neither of these was the case. Set size did not exhibit a significant effect nor did the
p-value (.69) suggest that the cause was a lack of statistical power. On the other hand, the interaction probability (.0795) points to the potential benefit of the forthcoming increase in the volume of data.

The significant interval effect is consistent with previous work from this laboratory (Stern et al., 1984). The longer blink latency to the memory and test stimuli indicate a greater processing load than to the cue stimulus, a result which reflects the nature of the cognitive task presented by these stimuli. Further suggested by the longer blink latency to the test than to the memory stimuli, although not as strongly, is that the test stimulus creates a greater processing demand than does encoding the memory set. These inferences must remain tentative for the present. They will be examined more fully when the data set is complete. Similarly, a bin by bin analysis of blink frequency should also be useful in tracking shifts in processing demands within intervals as an adjunct to the ERP and HR changes over these periods.

The reaction time data for our subjects parallel the reduction in test stimulus N2-P3 amplitude and confirm the oft-demonstrated finding that RT increases linearly with memory set size for both correct "positive" and correct "negative" responses. As noted by Sternberg (1966, 1975), this pattern of results indicates a sequential and exhaustive memory search. Further, the differing intercepts of the RT-set size functions for positive and negative responses are suggestive of greater
uncertainty (or hesitancy) in the emission of a negative response. Once the test stimulus ERPs are sorted into negative and positive responses, we would expect this greater degree of uncertainty to be evident in generally lower N2-P3 amplitudes on negative than on positive (response) trials.
6.0 References


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Figure Captions

Figure 1. Schematic diagram of display apparatus, information flow, and output devices.

Figure 2. Mean heart rate (bpm) change in each (950 msec) time bin of the three intervals. Baseline for each interval was the prestimulus rate for that interval.

Figure 3. Group-averaged ERPs as a function of stimulus type (cue, memory, or test), set size, and electrode location. Polarity = positive up.

Figure 4. Task ERP amplitudes as a function of stimulus type (cue, memory or test). Panel A: P3-N3 and P2-N2 components pooled across set size. Panel B: N2-P2 amplitudes as a function of set size. Panel C: P2-N2 amplitudes as a function of set size.

Figure 5. Group-averaged probe ERPs as a function of interval (cue or memory), set size, and electrode locus. Polarity = positive up.

Figure 6. Mean Probe ERP amplitude for P2-N2 as a function of interval (cue or memory) and set size.

Figure 7. Mean Probe ERP amplitude for P4-N4 as a function of interval (cue or memory) and set size.

Figure 8. Mean Blink Latency as a function of stimulus type (cue, memory, or test) and set size.

Figure 9. Mean Reaction Time as a function of match/mismatch and set size.
8.0 Figures
Figure 1
Figure 3
Figure 4
Figure 5
Figure 6
Figure 7