THE EFFECTS OF DEPRESSION ON SELECTIVE ATTENTION AND LATE COGNITIVE PROCESSES

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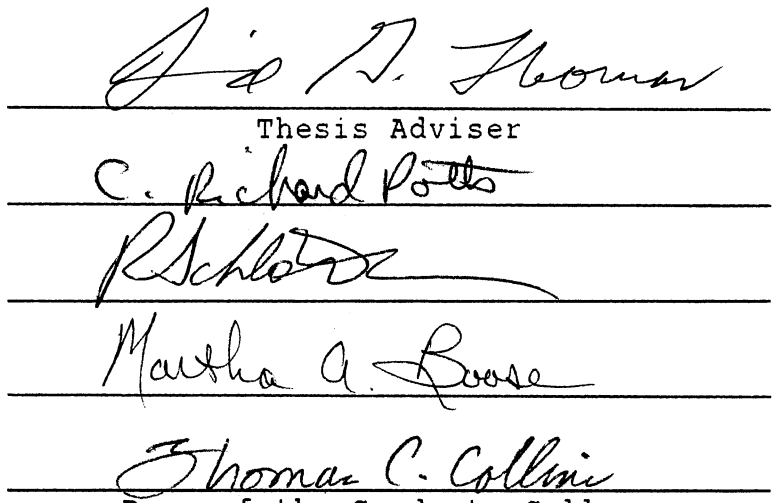

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CHAPTER I

INTRODUCTION

The investigation of depression and its associated symptoms has been a topic of major importance in the field of clinical psychology. For many years, clinicians treating depressed patients have noted a variety of additional symptoms beyond the affective aspects of the disorder. Symptoms including psychomotor retardation, difficulties in attention and concentration, low levels of motivation and arousal, and poor memory have been the subject of clinical "lore."

In recent years, there has been a change in perspective in viewing the complexity of depression. While this syndrome was originally conceptualized as only affecting emotional functioning, recent empirical findings point to deficits in cognitive processing, as well. The following section includes research concerning the effects of depression on cognitive functioning.

The most basic research investigating cognitive functioning has focused on psychomotor retardation and depression. While psychomotor retardation does not implicate one specific cognitive stage of process, it is

nonetheless an indication of some type of processing impairment. Generally, most studies which explore psychomotor retardation, define this concept operationally by measuring reaction time to various tasks. For instance, Payne and Hewlett (1960) administered six psychomotor tasks to depressives, schizophrenics, hysterics, neurotics, and controls. They reported significant differences in reaction time in tasks of psychomotor speed and recognition, with the depressives being the slowest to respond. The authors went on to speculate that the depressives performed most slowly because they were distracted by depressive thoughts.

Giedke, Their, and Bolz (1981) found similar results with respect to depression and its effect on reaction time. These authors presented depressives and controls with an auditory discrimination task. Subjects were instructed to press a microswitch as soon as they perceived a target tone and were told to ignore a standard tone of a different frequency. The results indicated that depressed subjects reacted significantly slower to the target tones. Similar findings have been consistently documented in the literature on depression (Miller, 1974; Miller, 1975; Hall & Stride, 1964; Huston & Senf, 1952; Plooij-Van Gorsel, 1984; El Massioui & Lesevre, 1988; and Thier, Axmann, & Giedke, 1986).

The abundance of data relating increased reaction time to perceptual/motor tasks in depressed subjects lends strong

support to the idea that psychomotor retardation is a rather robust symptom in many types of depression. Nelson and Charney (1981) reviewed 20 factor analytic studies, nine cluster analytic studies, four discriminate function studies, and a variety of other research focusing on factors associated with depressive illnesses. They reported that while severity of depressed mood, lack of reactivity, self reproach, loss of interest, and depressive delusions were all factors in depressive disorders, the symptom most strongly associated with depression was psychomotor retardation.

Research on Cognitive Processing Impairments

With the evidence of slower reaction times established, the next logical question is, which level(s) of information processing is most affected by depression? Weingartner and Silberman (1982) suggested that "depressed patients experience and demonstrate difficulties in concentration, attention, memory and other aspects of information processing" (pp. 82). In a literature review of deficits in depression, Miller (1975) concluded that impairments in the areas of cognitive, motor, and perceptual processing, as well as communication were indicated. The following studies are just a few examples of the impairments indicated in specific stages of information processing.

In a study investigating the effects of depression on the encoding process, Weingartner, Cohen, Murphy, and Gerdt (1988) utilized patients diagnosed with depression, affective disorders other than depression, and controls and administered memory tasks designed to assess the organizational strategies used to process information. The results indicated that depressives used a weak or insufficient encoding strategy making it more difficult to recall learned material. In addition, the data suggested that when information was organized and structured for the depressed subjects, they demonstrated marked improvement in their abilities to encode and recall information. Weingartner et al. concluded that depression was associated with impairments in encoding strategies which resulted from disruptions in the levels of activation and a general lack of energy for cognitive processing. Similar findings concerning the encoding process have been replicated (Berndt & Berndt, 1980).

Henry, Weingartner, and Murphy (1973) investigated impairments in the encoding of information specific to short term and long term memory. The data indicated that while depressives showed no deficits in short term memory tasks, they demonstrated significantly poorer performances on tasks assessing the encoding of information into long term memory. From their results, Henry et al. concluded that depression

interferes with the encoding and transfer of information into long term memory.

Hasher and Zacks (1979) reported that deficits in attentional capacity were associated with depression. Depressed subjects showed little or no impairments in attention to tasks requiring automatic processing. However, a reduced attentional capacity was indicated for tasks requiring effortful processing. These results suggested that depression reduces the capacity for attention, thereby allocating fewer resources for effortful processing.

Breslow, Kocsis, and Belkin (1980) also investigated the effects of depression on attention and later cognitive processes, such a memory. Hospitalized depressives and matched controls were administered the Wechsler Memory Scale. The data indicated that depressed subjects performed significantly worse on all areas indicating memory function impairment. Of particular importance was the finding that depressives recorded their poorest performance on the scales assessing mental control. Breslow et al. noted that these mental control subtests are strongly associated with attention and alerting mechanisms. Thus, the data suggested impaired memory functioning stemming from attentional deficits resulting from depression.

In a later study, Breslow, Kocsis, and Belkin (1981) presented hospitalized depressed patients and matched controls with a story which featured positive, negative, and

neutral affective themes. The results showed significant differences between groups in their abilities to recall information from the entire story. Interestingly, no differences were indicated in the recall of negative or neutral themes, Thus, the majority of the differences found between the depressives and controls were due to decrement in the recall of positive themes by the depressed patients. Breslow et al. speculated that depressives selectively attend to certain types of information (negative) and consequently are not as able to make use of their overall concentrational abilities.

Thus, while the findings of these studies have indicated overall deficits in memory functioning, each study suggested that impairments occurring earlier in information processing were the casual factors for the observed memory difficulties. The results of Henry et al. (1973) and Weingartner et al. (1988) suggest that impairments in recall ability of depressed subjects were due to poor encoding processes. Breslow et al. (1980, 1981) cited deficits in attentional capabilities as the possible cause of decrements of memory recall.

In considering the results of these studies, the methodological limitations of behavioral research become apparent. Each study cited a deficit in an earlier-occurring cognitive process as the potential source of memory impairment. However, none of the investigations

provide a direct method to evaluate these earlier processes. The present study proposes to overcome this limitation through the use of evoked potentials (EPs).

In an effort to address this specific issue, Tueting, Kaskey, Buchsbaum, Connelly, Perris, and Roemer (1984) suggested that in many instances, behavioral research provides little assistance in understanding the internal processes of cognitive functioning. EPs can "serve as an intermediate level of analysis" (pp. 254). EP research uses sequential analysis (processing events as a function of time) to establish electrophysiological correlates of cognitive processes. Tueting et al. also reported that the use of EPs provides a reliable means of investigating these correlates in psycho- pathology such as depression. The following section reviews the EP literature which has supported the notion that depression has a significant effect on cognitive processing.

Evoked Potential Research on Depression

One of the originators in the use of EPs for the study of psychopathology has been Charles Shagass. Shagass has employed EPs to study a variety of psychopathology. In an early study, Shagass (1955) investigated quantitative differences in the photically activated electroencephalogram (EEG) which resulted from different emotional states. Psychiatric patients diagnosed with anxiety hysteria,

neurotic depression, depression, paranoid schizophrenia and simple schizophrenia were compared to nonpatient controls on two conditions in which the rates of light flashes were altered. Results indicated that depressives demonstrated a significantly lower flash gradient than the other clinical groups with the exception of the paranoid schizophrenics. From these results, it was concluded that the photically-activated EEG varied significantly as a result of differences in emotional states with depressives exhibiting the smallest amplitude levels of stimulation.

In a later study Shagass, Roemer, Straumanis, and Amadeo (1978) presented patients with a number of different psychiatric difficulties as well as a control group with stimuli in three different sensory modalities (somatosensory, visual, and auditory). Shagass et al. noted significant differences in the late EP activity of neurotic and psychotic depressives. The results were interpreted as a potential indication of cognitive impairment, specifically at the processing level of attention.

In a follow up study, Shagass, Roemer, Straumanis, and Amadeo (1980) again presented three types of sensory stimuli to various psychiatric patients and non patient controls. The results indicated that for the somatosensory EPs, psychotic depressives showed higher peak amplitudes in the posterior brain regions than controls. Nonpsychotic

depressives, on the other hand, demonstrated greater amplitudes anteriorly than did controls.

Thus, Shagass and his colleagues have utilized EPs to demonstrate the potential usefulness of the EEG (electroencephalogram) in exploring differences among various types of psychopathology. Other authors have followed this type of methodology and employed EPs for the study of depression in particular.

Buchsbaum, Goodwin, Murphy, and Borge (1971) utilized EPs to investigate the intensity of sensory input on the functioning of the central nervous system in depression. Bipolar (manic-depressives), unipolar (depressives), and control subjects were presented with a series of light flashes of four increasing intensities. The results indicated that the bipolar groups demonstrated greater peak amplitudes in the components of the visual evoked potentials than the control and unipolar groups. Interestingly, the visual evoked potentials of the unipolar depressed group featured significant reduction in comparison to the control group. The authors suggested a parallel between the resultant intensity of processing of incoming information and the clinical behaviors which have been observed in these groups of patients.

In a follow up, Buchsbaum, Landau, Murphy, and Goodwin (1973) again recorded visual evoked potentials of bipolar, unipolar, and control subjects to four varying light

intensities. The results indicated that male and female bipolar patients had significant augmentation in EP amplitude with increasing intensity of stimuli. However, only males fitting the unipolar classification demonstrated a reduction in EP amplitude with increasing stimulus intensity. Additional data indicated that smaller averaged evoked potential (AEP) amplitudes were associated with higher levels of depression.

Thus, Buchsbaum and his colleagues provided data which indicated that patients suffering from affective disorders (such as unipolar disorder and bipolar disorder) displayed respective diminished or heightened effects on EP components which were consistent with the symptoms characteristic of each dysfunction. Of particular interest to the present study was the finding that depression resulted in the reduction of the overall EP components (Buchsbaum et al., 1971, 1973).

While research has been conducted on the effects of depression on the overall EP wave, other studies have addressed individual EP components associated with specific cognitive processes. In particular, peak P300 (or P3) has been the focus of a number of investigations exploring the physiological correlates of information processing in a variety of psychopathologies including depression.

P300 and Depression

P300 is a slow, positive wave which generally occurs at around 300 msec (see Figure 1). It has been associated with higher cognitive functions, and consequently has been classified as an "endogenous" component. Generally, experimenters investigating the P300 component make use of the "oddball" paradigm. In the "oddball" paradigm, subjects are required to detect infrequent auditory stimuli which result in slow, positive waves in the 250-450 msec range of the average evoked potential (AEP).

Insert Figure 1 about here

While the exact nature of the occurrence of the P300 has not yet been determined, it is generally accepted that P300 is indicative of late cognitive processes. In recent years, P300 has been associated with a variety of cognitive processes including: stimulus evaluation and categorization time (Kutas, McCarthy, & Donchin, 1977; Hillyard & Kutas, 1983; McCarthy & Donchin, 1981), updating of internal models of environment (Donchin, Ritter, & McCallum, 1978), encoding of material into long term memory storage (Karis, Fabiani, & Donchin, 1984), and level of mental workload (Donchin, 1985).

Several studies have utilized P300 to investigate the information processing associated with depression.

Blackwood, Whalley, Christie, Blackburn, St. Clair, and McInnes (1987) recorded the EPs of depressives, schizophrenics, and control subjects to a two-tone discrimination task. The data indicated that the P300 amplitude was significantly smaller in the depressives and schizophrenics as compared to the controls. The authors went on to note that while the reduction of the P300 amplitude may be indicative of impairments of auditory information processing, depressives showed a marked improvement as evidence by a rise in P300 amplitude following treatment (psychotropic, psychotherapy, and/or ECT).

Pfefferbaum, Wenegrat, Ford, Roth, and Kopell (1984) compared the P300 components of subjects diagnosed with dementia, depression, and schizophrenia, with a control group. The authors noted that a number of the patients were or had previously been using psychotropic medication. EPs were collected to auditory and visual discrimination tasks. While Pfefferbaum et al. reported a number of significant results, the most pertinent to the present study was that non-medicated depressives displayed significantly diminished P300 amplitudes as compared to controls. Similar results concerning reduced P300 amplitudes of depressed subjects have been reported (Levit, Sutton, & Zubin, 1973; Roth, Pfefferbaum, Kelly, Berger, & Kopell, 1981).

In regard to P300 latency, Johnson, Pfefferbaum. Hart, and Kopell (1984) recorded the EPs of chronic alcoholics, depressives, and control subjects to an easy discrimination task, a difficult classification task, and a recognition task. Results indicated that both depressives and chronic alcoholics manifested significantly later P300 latencies and reaction times than did the controls.

A number of studies have shown anomalies in P300 amplitude or latency (Blackwood et al., 1987; Johnson et al., 1984; and Pfefferbaum et al., 1984). Such results in association with behavioral studies (Weingartner et al., 1988) suggest underlying impairments in the late processing stages of depressives. While a great deal of research has implicated P300 as indicative of these deficits, a number of studies have reported contrary results.

Giedke, Their, and Bolz (1981) investigated peak P300 as a measure of stimulus evaluation time in cognitive processing. As described previously, depressed and control subjects were presented an "oddball" paradigm task. While depressives showed significantly longer reaction times in responding to targets, no differences in P300 amplitude or latency were present. Thus, the results suggested that while differences in information processing were evident, the differences were not due to the cognitive processes associated with P300. Similar lack of significant evidence in the P300 components of depressed subjects have been

reported (El Massioui & Lesevre, 1988; Thier, Axmann, & Giedke, 1986; and Plooij-Van Gorsel, 1984).

Thus, the indication of late processing deficits associated with P300 is still uncertain at this time. The conflicting evidence in the literature leaves the exploration of this issue as one of the major focusses of the present study.

Just as P300 has been shown to be indicative of late cognitive processing, other EP components have been associated with different stages of information processing. Of particular importance to the present study is the discussion of the components of the EP related to the selective attention process.

Evoked Potential Research and Selective Attention

In reviewing the EP research on selective attention, the focus begins with the study by Hillyard, Hink, Schwent, and Picton (1973). These authors hypothesized that peak N100, a negative component peaking at 80 to 130 msec (see Figure 1), was affected by the process of selective attention. Hillyard et al. theorized that the selective attention process would act to enhance stimuli perceived from a chosen source and at the same time, suppress irrelevant information. Two experiments were conducted to

assess the relationship between peak N100 and selective attention processes.

In Experiment 1, subjects were presented sequences of two standard tones of different frequencies to each ear. About one-tenth of the tones presented to each ear featured target tones of a slightly higher frequency than the standards. The subjects were presented the tone sequences four times in succession under two different attentional conditions. In one condition, subjects were instructed to attend to the left ear, and count the number of deviant tones presented to that ear. In the other condition, subjects were instructed to attend to the right ear and count the number of deviant tones presented to that ear. Experiment 2 was similar to the first experiment with a few minor changes in methodology. Results for both studies indicated that N100 was substantially larger for the attended versus nonattended tones.

Hillyard et al. reported three features which distinguished their study from previous research directed at investigating selective attention. First, the relevant (attended to) and irrelevant (ignored) stimuli differed in both pitch and spatial localization which assisted subjects in distinguishing them. Second, stimuli were delivered at such a quick rate that it was impossible to attend and discriminate in one ear and fully appreciate stimuli presented to the other. Finally, the discrimination of

deviant stimuli was difficult. The authors interpreted the results for N100 as indicating that "the underlying attentional process is a tonically maintained set favoring one ear over the other rather than an active discrimination and recognition of each individual stimulus"(pp. 179). Thus, the amplitude of N100 was reflective of basic attentional processes which blocked out the relevant stimuli and admitted sensory input for further processing.

In addition, analysis of the slightly higher pitched tones elicited P300 components (peaking between 250 to 450 msec). Hillyard et al. combined the results regarding N100 and P300 to suggest that both components represent "fundamentally different selective attention processes" (pp. 179). Corresponding to an attention model described by Broadbent (1970), N100 represents the stimulus set mode of attention, in which all sensory input is admitted to an attended channel for further analysis. The authors suggested that P300 was analogous to the response set mode of attention which compares sensory information against prototypes or templates in storage.

Similar results concerning N100 amplitude and selective attentional processes were reported in a later study (Parasuraman, 1978). In this study, subjects attended to three different frequencies of tones presented at three different spatial locations (right ear, left ear, and centrally). Two different presentation rates were used for

the tones. In the slow presentation rate condition, interstimulus intervals (ISIs) varied between 700 and 1400 msec. The fast presentation rate condition had a range of 350 to 800 msec. Subjects participated in 14 different conditions which required them to monitor one, two, or all three auditory channels at both stimulus rate presentations for the detection of an occasionally louder tone. These target tones occurred randomly within each condition with a probability of 0.1 in each channel.

Results indicated no significant differences in N100 in the slow presentation rate of the tones. However, in the fast presentation rate, N100 amplitude was significantly larger when a channel was attended to that when it was ignored. These data replicated the results of Hillyard et al. (1973), and supported the hypothesis that the amplitude of N100 was related to the process of selectively attending to a given channel. Additionally, Parasuraman reported that in the fast presentation condition, the amplitude of peak N100 decreased in both the attended and unattended channels with an increase in the number of channels monitored. Thus, as subjects were required to allocate more attentional resources to monitor an increasing number of input channels, the amplitude of N100 was consequently diminished. Parasuraman concluded that "the N100 component of the vertex evoked potential reflects the distribution of attentional capacity among competing auditory inputs" (pp. 464). Other

findings have produced similar results suggesting that N100 amplitude serves as a indication of allocation of attentional resources (Hink, Van Voorhis, Hillyard, & Smith, 1977). Thus, the data from these studies lend support for the association of N100 amplitude and the process of selective attention.

Naatanen, Gaillard, and Mantysalo (1978) proposed a different interpretation to the increase in the amplitude of peak N100 to tasks requiring more attentional resources. These authors speculated on the presence of a slow negative wave, which they labelled processing negativity (PN). This PN wave could occur as early as 50 msec and remain as late as 350 msec into the EP. Naatanen et al. suggested that the utilization of short ISIs in the paradigms of Hillyard et al. (1973) and Parasuraman (1978) resulted in a consequent overlap of PN with N100 thereby increasing the apparent amplitude of N100.

To investigate the possibility of this PN overlap, Naatanen et al. conducted two experiments. In Experiment 1, subjects listened to tones of the same frequency which varied in intensity (loudness). In Experiment 2, the intensity of the tones remained constant, but in this instance, the frequency was varied. Unlike Hillyard et al. (1973) which used randomized short ISIs, the tone pips in both experiments were presented at a constant ISI rate of 800 msec. In each experiment, subjects participated in two

conditions: one which required them to attend to stimuli in the right ear and ignore the left; the other which required attention to stimuli in the left ear, ignoring the right. In both conditions, subjects were told to monitor and count the number of signal tones presented only to the attended ear.

The data showed no N100 enhancement to the attended stimuli in comparison to those which were ignored. However, there was a later negative shift which was superimposed on the EPs of the attended stimuli. The authors reported that the effect of this wave, hypothesized to be PN, was small but systematic beginning at the usual downward slope of N100 (latency around 100 msec) and lasting at least 500 msec. Naatanen et al. went on to speculate that the N100 effect reported by Hillyard et al. (1973) and Parasuraman (1978) was not a "true" enhancement of the N100 component but rather the superimposition of PN on N100.

Thus, uncertainties arose concerning whether the changes in the amplitude of N100 were due to a "true" N100 effect, as suggested by Hillyard et al. (1973) and Parasuraman (1978) or were the result of the simultaneous occurrence of another slow negative wave, which was actually an indication of selective attention.

Hansen and Hillyard (1980) set out to investigate these questions. Subjects were presented binaural standard (51 msec in duration) and target (102 msec in duration) tone pips. Standards and targets occurred with probabilities of

0.8 and 0.2, respectively. Attention conditions consisted of tones of two different frequencies, presented in a randomized order. The low frequency was in all cases 300 Hz. The higher frequency varied across the three frequency separations. In one frequency separation, 350 Hz tones acted as the higher frequency (50 Hz frequency separation from the lower tone). The second frequency separation employed 400 Hz for the higher pitched tones (100 Hz frequency separation). Finally, the third frequency separation used 700 Hz as the higher pitched frequency (400 Hz frequency separation).

Thus, subjects participated in six different conditions, two in succession for each respective frequency separation. Subjects were instructed to attend to the high (or low, varying across successive conditions) tones and to press a microswitch when they perceived a target of the frequency to which they were attending. A major alteration of the paradigm of Hansen and Hillyard was that while previous research had designated that attention be focussed on a particular ear (right or left), Hansen and Hillyard instructed subjects to attend to a particular tone frequency.

EPs were recorded from four electrode sites: FPz, Fz, Cz, and Pz. For each frequency separation (comprised of two conditions), a difference wave was calculated for the standard tones. This wave, labelled Nd by the authors, was

calculated by subtracting (point by point) the AEP to the respective stimuli when subjects were not attending from the AEP of when they were attending. In calculating Nd, the authors hoped to assess the presence of the PN wave hypothesized by Naatanen et al. (1978).

Results indicated that the effects of selective attention were seen in the Nd wave as a broad negativity which began before the peak of the N100 components at the 400 Hz frequency separation and continued throughout the analysis epoch. The latency of the onset of Nd progressively increased (with a decrease in amplitude) as the separations between the frequencies of the channels were reduced. Thus, Hansen and Hillyard reported "a temporal dissociation between the evoked N100 wave and the attention sensitive Nd, brought about by manipulating the interchannel discriminability "(pp. 280). In regards to peak amplitude, the data showed a significant Nd amplitude increase as frequency separation increased.

Hansen and Hillyard suggested that the overlap between Nd and N100 is most likely responsible for the N100 enhancement in the previous literature (Hillyard et al., 1973, Parasuraman, 1978). Further, the authors suggested that Nd has an "endogenous character" as evidenced by its difference in wave shape from N100 (Nd is characteristically a broad component versus the sharpness of the N100 peak), temporal dissociation from N100 brought about by altering

the interchannel frequency separation, and its more anterior distribution on the scalp as compared to the more posterior-originating N100. In conclusion, Hansen and Hillyard indicated that the utilization of Nd was reliable as a correlate of selective attention.

Other studies have supported the indication that the Nd wave is associated with tasks that require attention (Okita, 1988; Hansen & Hillyard, 1984). The use of Nd as a reliable correlate of selective attention has also been employed to investigate the result of administering differential doses of adrenocorticotropic hormone (ACTH) (Born, Brauninger, Fehm-Wolfsdorf, Voigt, Pauschinger, & Fehm, 1987). ACTH has been demonstrated to have an impairing influence on attention. The results indicated a near linear dose-dependent effect on the Nd amplitude of the AEPs. Similarly, Satterfield, Schell, Nicholas, and Backs (1988) investigated the EPs of controls and children with attention deficit disorder with hyperactivity (ADDH) to a two-choice discrimination task. The findings showed significantly smaller Nd waves in the children diagnosed with ADDH, suggesting impairments in attentional processing. Thus, research utilizing Nd has reliably established its association with attentional processes.

Selective Attention and Depression

The use of Nd as an indicator of selective attentional processes was recently employed by Burkhart (1989) to assess impairments resulting from depressive states. Burkhart used much of the same methodology as Hansen and Hillyard (1980). The rationale for the use of this selective attention paradigm included its proficiency at "measuring attention relatively unconfounded by other task functions such as memory or motor responses" (pp. 35). Much of the behavioral research cited required encoding or recall of previously learned material. The utilization of stimulus perception eliminates this memory component and permits a more direct assessment of attention. The selective attention paradigm also allows for the comparison of EPs in both the attended and ignored channels by way of the Nd wave, which Hansen and Hillyard (1980) suggested was a reliable indicator of selective attention.

While previous research in psychopathology has defined the term "depression" in a variety of ways and subtypes (e.g. psychotic, neurotic, reactive, endogenous, etc.), Burkhart elected to classify depression operationally as a manifestation of particular symptoms; i.e. behaviors and attitudes associated with depressive states rather than treating it as a psychological disorder. The assignment of subjects to the respective control or depressed groups was

determined by means of empirical scores on two reliable inventories devised to assess levels of depression.

Sixteen depressed and 16 control subjects were selected on the basis of their scores on the Zung Self-Rating Depression Scale (SDS) (Zung, 1965) and the Beck Depression Inventory (BDI)(Beck et al., 1961). Electrodes were affixed to the scalp at the Cz and Fz sites. In utilizing the selective attention paradigm of Hansen and Hillyard (1980) Burkhart elected to present subjects with the conditions of the 50 Hz and 400 Hz frequency separations. Thus, subjects participated in four different conditions. In all of these conditions, 300 Hz tone pips served as the low frequency tones. In two of the four conditions, a frequency of 350 Hz was utilized for the higher frequency tones. In the remaining two conditions, 700 Hz tones served as the high frequency tones. Subjects were presented short standard tones and long target tones at both frequencies during a condition. Subjects were instructed to attend to a particular tone frequency within a given condition, and press a thumb switch when they perceived a target tone of the attended frequency. AEPs and consequent Nd components were calculated for all standard tones.

Significant Nd amplitude differences were reported for frequency separation, with a 400 Hz separation producing a greater amplitude than the 50 Hz separation. This finding was consistent with the results of Hansen and Hillyard

(1980). The results of both studies indicated that an increase in Nd amplitude was associated with greater allocation of selective attentional resources. No significant main effects for mean or peak amplitude were found for group or scalp location. In addition, no significant interactions for Nd amplitude were indicated. In assessing peak latency of Nd, a significant main effect for electrode site was reported for Nd Latency. The results indicated that site Cz had an earlier Nd than the more anterior Fz. Analysis of peak latency and 25% latency produced no main effects for the variables of subject group or frequency separation. No significant interactions were reported as well.

In an effort to assess the effects of depression on attentional capacity, four Dunn's comparisons (Kirk, 1968) between the variable of groups (control versus depressed) and frequency separation (400 Hz versus 50 Hz) at four different dependent measures (mean amplitude, peak amplitude, peak latency, 25% latency) were proposed. None of these comparisons yielded a significant difference between the groups.

One interesting result reported by Burkhart was that while virtually no significant differences were reported for Nd as a function of group (depressed versus controls), significant differences were indicated for the variable of group for peak N100. Burkhart found that N100 amplitude was

significantly greater in the controls versus the depressed subjects for the frontal electrode site. These results were interpreted as an increased level of arousal in the control subjects which accompanied the selective attention process. Thus, while several of the previously cited studies suggested possible deficits in the attentional processing of depressives, the results in Burkhart's study concerning Nd as a measure of selective attention did not support these findings.

Purpose of the Present Study

The purpose of the present study is to investigate the effects of depression on the selective attention process. In addition, several alterations to the methodology of Burkhart (1989) were added in order to assess potential hemispheric differences and possible impairments in late cognitive processes associated with depression.

The first alteration implemented in the present study concerned the criterion scores employed by Burkhart (1989) for the determination of depression. In establishing the depressed group, Burkhart set scores of 0.45 or greater on the Zung SDS and 10 or greater on the BDI as criteria for inclusion in the depressed sample of the subjects. With specific reference to the BDI, Burkhart reported a mean score of 16.6 (s.d.=6.02) with a range of 10 to 33 for the depressed group. In addition, the control group had a mean

score of 3.25 on the BDI (s.d.=2.5) with a range of 0 to 8. However, Kendall, Hollon, Beck, Hammen, and Ingram (1987) reported that the classification of depression "should probably be reserved for individuals with BDI scores over 20" (pp. 298). Thus, it appears that the standard score of 10 used by Burkhart appeared to be too low for a reliable determination of depression. It is possible that this low criterion for the BDI produced a sample of depression too mild to be affected with the types of cognitive deficits described in the previous research. This lack in severity of depression may have been a major factor contributing to the nonsignificant results.

To alleviate these potential problems, the present study proposed to once again utilize the Zung SDS and the BDI in the selection of subjects. However, the criteria for determination of depression was raised on both instruments. In the initial screening of students for potential participation, a score of 0.50 or above on the Zung SDS was required to classify students as depressed. In addition, a score of 16 or above, which is one s.d. above the mean of the depressed group in Burkhart's study, was used as the criterion for inclusion in the depressed group. This criterion of 16 was more in agreement with the standards suggested by Kendall et al. (1987).

A second alteration concerned the placement of electrodes on the scalp. Burkhart reported a significant

main effect for the electrode site on the measures of Nd peak latency. From these results, it was concluded that site Cz had an earlier Nd peak latency than site Fz. While these findings have relevance in the theorizing of information processing, Davidson and Tomarken (1989) utilized Fourier transformation of the EEG to discover asymmetries in the hemispheric processing of emotional information. In particular, their research has indicated that certain left hemispheric regions are stimulated during the expression and experiencing of positive emotions. Conversely, corresponding right hemispheric regions are activated during the expression and experiencing of certain negative emotions. Ahern and Schwartz (1985) drew similar conclusions from their results which indicated that the anterior regions of the right hemisphere were more activated during the processing of negative emotional material in comparison to positive emotional material.

The implications of this research have particular relevance when considering the processing of information in affective disorders such as depression. Henriques and Davidson (1990) also utilized Fourier analysis to conduct a study investigating hemispheric asymmetries in depressives. Their results were consistent with the findings of Ahern and Schwartz (1985). Depressives showed less left hemisphere activation in comparison to the right in the anterior electrodes than did nondepressed subjects.

Thus, with data suggesting the presence of potential hemispheric asymmetries, the present study proposed to again record the EPs to tone pips from the central and frontal regions of the scalp (Burkhart, 1989). However, in an effort to assess possible asymmetries, electrodes were placed on both sides of the midline in the central and frontal areas. Therefore, sites F3 and F4 monitored the frontal region and C3 and C4 the more posterior region. The choice of these particular sites allowed for evaluation across hemispheric regions (F3 versus F4 and C3 versus C4).

In applying the paradigm used by Hansen and Hillyard (1980) to the study of depression, Burkhart (1989) narrowed the focus of her investigation to the evaluation of selective attention by way of the Nd wave. However, studies like that of Weingartner et al. (1988) and Berndt and Berndt (1980) indicated that possible deficits have been manifested in later-occurring cognitive functions such as the encoding process. Similarly, EP studies which reported significant differences in the P300 waves of depressed subjects (Pfefferbaum et al., 1984; Blackwood et al., 1987; and Johnson et al., 1984) also lend support to the hypothesis that depression impairs late cognitive processes. In an effort to evaluate this hypothesis, the present study proposed a third alteration. EPs were recorded to all target tones to investigate P300 differences associated with depression.

Finally, in addition to the investigation of P300, the present study also measured reaction time (RT) and false alarms in addition to the behavioral measure of hit rate utilized in Burkhart (1989). With regard to RT, a number of studies have reported increased RTs associated with depression (Payne & Hewlett, 1960; Giedke et al., 1981; Miller, 1974). Furthermore, the recording of RT has also been established as a potential determinator of the severity of depression present in depressed subjects (Martin & Rees, 1966).

Hypotheses of the Present Study

Using the selective attention paradigm employed in the study of Burkhart (1989) with the alterations of a higher criterion score on the BDI, differential placement of the electrodes allowing for the assessment of hemispheric asymmetries, analysis of the later stages of cognitive processing by way of the evaluation of P300, and the measurement of RT and false alarms, a number of hypotheses were generated. With regards to Nd, it was hypothesized that all subjects would demonstrate less difficulty allocating their attentional abilities to detect target tones in the 400 Hz separation than the 50 Hz separation. Consequently, these differences in selective attention would be manifested by greater Nd amplitudes and earlier onset latencies for the 400 Hz separation.

In investigating attentional effects due to depression, the second hypothesis posited that depressed subjects would show attentional differences compared to control subjects. These differences would be manifested in smaller absolute amplitudes and later onset latencies for the Nd component. These results would indicate that depressed subjects were not able to allocate as many selective attentional resources to the attended channels as controls. Data suggesting no differences in Nd between the groups would indicate that changes in attention due to depression may be situation specific, or that cognitive impairments associated with depression may exist but that these deficits are a result of difficulties in processing not related to attention.

The third hypothesis of the present study addressed the issue of levels of task arousal, as indicated by the amplitude and latency of N100. It was hypothesized that control subjects would exhibit significantly greater absolute amplitudes and earlier onset latencies than depressives. Findings consistent with this hypothesis would support the results found for the Fz site in Burkhart (1989), and suggest that controls exhibited higher levels of arousal than depressives.

With regard to the assessment of late cognitive processes, the amplitude and latency of the P300 component were employed to investigate possible processing impairments. The fourth hypothesis addressed in the present

investigation posited that the P300 waves of the depressives would be characterized by smaller amplitudes and later onset latencies than the control subjects. Results consistent with this hypothesis would lend support to possible impairments in later cognitive processes such as stimulus evaluation and categorization time (Kutas, McCarthy, & Donchin, 1977), updating of internal representations of the environment (Donchin, Ritter, & McCallum, 1978), and encoding into long term memory (Karis, Fabriani, & Donchin, 1984).

The fifth hypothesis addressed differences in hemispheric processing for depressives and controls (as indicated by the amplitudes and latencies of Nd, N100, and P300). More specifically, it was posited that depressives would evidence smaller amplitudes and later onset latencies of the various EP components in the left hemisphere electrodes. Results consistent with this prediction would lend support to the findings of Henriques and Davidson (1990), that depressives have shown less left hemisphere activation in comparison to the right in the anterior regions than controls. Contrary results would indicate that depressed subjects process information in similar ways as controls with regards to hemispheric activation levels.

Finally, with regard to the behavioral measures of the selective attention paradigm, it was hypothesized that depressives would demonstrate fewer hits, more false alarms, and longer reaction times to the perception of target tones

in the attended channel. These results would provide data consistent with previous research that depression is associated with psychomotor retardation (Payne & Hewlett, 1960; Miller, 1974; Hall & Stride, 1964; Huston & Senf, 1952; and Plooij-Van Gorsel, 1984).

CHAPTER II

METHOD

Subjects

Subjects were 32 undergraduate students enrolled in psychology courses at Oklahoma State University. Subjects were selected with consideration to the following criteria: 1) no known neurological impairments; 2) not presently participating in any form of psychotropic therapy for depression or using street drugs; 3) must be right hand dominant; and 4) no hearing deficits or imbalances.

Subjects were divided into two groups with 16 subjects in each group (8 females and 8 males). The first group consisted of subjects who reported moderate to severe depressive symptoms. The other group was comprised of subjects expressing minimal symptoms of depression. The determination of the presence of the depressive symptoms was assessed by means of the Zung Self-Rating Depression Scale (SDS) (Zung, 1965) and the Beck Depression Inventory (BDI) (Beck et al., 1961).

In the initial screening of subjects, the Zung SDS was administered to a large pool of undergraduates from introductory and upper division psychology courses. Students

scoring between 0.50 and 1.00 on the Zung SDS were classified as potential subjects for the depressed group. Similarly, students scoring between 0.25 and 0.40 were considered as potential control group subjects. Consequently, scores which fell into one of these respective groups were rank ordered from the highest to the lowest score. Students whose scores fell in the highest (indicating depression) and lowest (indicating lack of depression) ranges were contacted for further participation in the study. The experimenter then contacted the students on the determination of rank until the desired number of students was obtained. Subjects in the depressed sample recorded a mean score of 0.61 on the Zung SDS (s.d. of 0.07) with a range of 0.50 to 0.71. Subjects in the control group scored a mean of 0.33 (s.d. of 0.04) with a range of 0.25 to 0.40.

Students selected on the basis of the Zung scale were then administered the BDI. A criterion score of 16 or above on the BDI was established for qualification as potential depressed subjects. Subjects whose BDI scores fell within the 0 to 9 range qualified for potential inclusion in the nondepressed-control group. Subjects in the depressed group recorded a mean score of 22.75 (s.d. of 6.52) with a range of 16 to 35. Control subjects had a mean BDI score of 1.88 (s.d. of 2.03) and a range of 0 to 8. It is important to note that only students who demonstrated consistent

assessment scores across the Zung SDS and the BDI were included in the selective attention paradigm.

Upon completion of the BDI, prospective subjects were required to undergo a brief audiological examination. The hearing sensitivity of each subject was tested by using an audiometer. Professional audiologists reviewed each of the audiograms for the 250, 500, 750, 1000, 2000, and 4000 Hz frequencies. If the shape of the audiogram was suspect, if the hearing levels were elevated to the point that equipment compensation was not possible, or if there was a sensitivity difference greater than plus or minus 5 dB between ears, potential subjects were dismissed.

Materials

Depression Measures In replicating the procedure of Burkhart (1989), the Zung SDS and BDI were utilized for the empirical assessment of the depressive and control subjects. The BDI is composed of 21 Likert-type questions. The BDI was created to assess behavioral manifestations of depression along a continuum of severity (Beck et al., 1961). Scores on the BDI can range from 0, indicating lack of depressive symptoms, to 63, indicating severe depressive symptoms. In keeping with the methodology of Burkhart (1989), subjects attaining a score of 0 to 9 on the BDI were classified as nondepressed. However, in an effort to increase the potential severity level of depression of the

subjects in the depressed group, a score of 16 or above was used for inclusion. This was a changed from the methodology of Burkhart (1989) which required a score of 10 or above on the BDI.

The Zung SDS was developed to provide a quantitative, self-administered, brief form of assessment for depression (Zung, 1965). It is comprised of 20 items, each of which requires the subject to rate the contents on a four point scale in terms of frequency. Scores on the Zung SDS can range from 0.25 to 1.00. As was the case for the BDI, a more stringent criterion score was used in the present study in comparison to Burkhart (1989). Students who obtained a score between 0.50 and 1.00 were classified as potential depressed subjects. Scores of 0.25 to 0.40 served as the criterion range for potential control subjects. Blumenthal (1975) reported that the BDI and Zung SDS share a correlation of 0.76.

Stimuli The stimuli of all four selective attention conditions consisted of the simultaneous presentation of tone pips and white noise presented binaurally. This study used stimuli identical in frequency and duration to that of Burkhart (1989) however, the stimuli intensity level was controlled and presented at 60 dB SL. This sensation level was established in an effort to control for individual hearing differences within the subjects. A sound level meter was used to calibrate the intensity of the tones

produced by the sound amplifier. Due to the utilization of a sensation level, the sound amplifier was adjusted for each subject to present stimuli corresponding to the appropriate hearing level.

Four selective attention conditions were constructed for the two different frequency separations. In each of these conditions, tones that were 51 msec in duration were designated as standards, and tones that were 102 msec were designated as targets. In all four conditions across both frequency separations, a 300 Hz tone served as the low frequency tone. In one of the frequency separations, the higher tones consisted of 350 Hz tone pips (frequency separation difference of 50 Hz). The other frequency separation utilized 700 Hz tones as the higher frequency (frequency separation difference of 400 Hz). For each frequency separation, the subjects were presented one condition which required monitoring of targets in the low frequency and a second condition requiring that they monitor the higher pitched targets . Thus, in frequency separation 1 (e.g. the 400 Hz separation), condition 1a would have subjects monitoring for high targets (700 Hz tones) and condition 1b would require subjects to monitor for low targets (300 Hz tones). Similarly, frequency separation 2 (the 50 Hz separation in this example) would have the subjects monitoring for high targets (350 Hz tones) in condition 2a, and low targets (300 Hz tones) in condition

2b. Consequently, subjects participated in two conditions (attend-low and attend-high) for each frequency separation (50 Hz and 400 Hz) for a total of four conditions.

Within each of the conditions, both high and low frequency tones were presented in a random order with equal probability. In keeping with the paradigm of Burkhart (1989), tones were presented at ISI rates of 600, 675, 750, 825, and 900 msec randomly ordered with a rectangular distribution.

EEG Data The EEG data was recorded by means of four gold plated disc electrodes affixed to sites F3, F4, C3 and C4, according to the International 10-20 System. These electrodes were referenced separately to linked earlobes (A1 and A2), with an electrode placed on the forehead serving as a ground. The electrode impedance was equal to or below 5 kohms and was verified at initial placement and immediately following each condition. Eye movement artifact was monitored by means of two electrodes; one placed over the outer canthus of the left eye and the other super-orbitally. A grass Model 79 polygraph with band passes of 0.1 to 100 Hz with 60 Hz notch filters was used to amplify the data of the EEG. A MetraByte DASH 16 A/D conversion board digitized the EEG. Stimulus presentation and data acquisition were synchronized by an IBM PC-XT. EEG data were sampled every 6 msec. For each of the standard tones, EEG data were collected 30 msec prior to stimulus onset and continued

until 357 msec after stimulus onset. For data collected to the target tones, the EEG again collected 30 msec prior to stimulus onset but continued until 800 msec after stimulus onset.

Procedure

Undergraduate psychology students were screened with the Zung SDS (see Appendix A for the consent form which accompanied this questionnaire). Students selected on the basis of their scores on the Zung scale were contacted for further assessment with the BDI. Students that were contacted for further participation were administered the BDI within two weeks of the administration of the Zung scale. Students demonstrating consistency across both instruments (either consistently depressed or nondepressed) participated in a hearing sensitivity test. Students with adequate hearing abilities were recruited to participate in the four selective attention conditions. Students presenting inconsistent scores across the Zung SDS and the BDI or hearing impairments were dismissed without administration of the selective attention paradigm. Twenty-three students were dismissed from participation; 21 students for inconsistent scores on the Zung SDS and BDI and 2 students for hearing deficits.

Students who met the above criteria for depression levels and hearing abilities were next brought into the lab

to participate in the selective attention paradigm. A requirement within the present study was established that students who qualified as subjects had to be administered the attentional paradigm within seven days of the administration of the BDI. Each subject was seated in a comfortable, reclining chair in a sound-attenuated and electronically shielded room. At this point, informed consent for each student was obtained (see Appendix B), directions concerning the experimental procedure were given (see Appendix C), and the electrodes were placed at the appropriate sites on the scalp.

In an attempt to familiarize the subjects with the durations and frequencies of the tones, students were presented a practice discrimination trial. In this task, 10 standard and 10 target stimuli of 300 Hz were presented in an alternating fashion. Similar procedures were employed for standards and targets of 700 Hz and 350 Hz. An additional practice sequence was given to each subject, consisting of 100 tones which simulated the first condition. Subjects were instructed to press a microswitch when they perceived a target tone in the attended frequency. The various orders of practice trials were counterbalanced across all subjects, as were the orders of the conditions presented.

The experiment proper consisted of four attentional conditions. Each condition was comprised of 400 tones,

i.e., 200 tones presented at two different frequencies. Within each of the frequencies, standard (51 msec) tones were presented 80% of the time (40% high standards and 40% low standards), with the targets (102 msec) occurred the other 20% (10% high targets and 10% low targets) of the time. Within each of the four attentional conditions, subjects were instructed to focus their attention on one frequency of tones (either the high or low tones), ignore tones of the other frequency, and respond by pressing the thumb switches when they perceived target tones in the attended frequency. Thus, an example of a possible sequence of conditions might be to have a subject attend to the 700 Hz frequency and respond by pressing a thumb switch to the perception of 700 Hz targets for the condition 1a. Condition 1b consequently had the subject attending to the 300 Hz frequency, monitoring for 300 Hz targets, and ignoring tones in the 700 Hz frequency. Following this same example, conditions 2a and 2b again used the 300 Hz tones, but this time in combination with 350 Hz tones. Therefore, a subject was instructed to attend to tones of 350 Hz in condition 2a and 300 Hz tones in condition 2b.

In keeping with the procedure of Burkhart (1989), subjects were randomly assigned to different orders of condition presentation as determined by a Latin Square for the frequency separations. Brief, five minute breaks were

provided between each condition, at which time the impedances of the electrodes were measured.

Data Reduction

Single trial EPs containing excessive eye movement artifact were excluded from the average evoked potentials (AEPs). For each subject, a total of 32 AEPs were calculated. Sixteen of these AEPs were calculated to the standard tones and the remaining 16 to the targets. In reference to both the standard and target tones, a total of two AEPs were computed for each of the two frequency separations. Thus, four AEPs were calculated for each of the four electrodes for both the standards and targets.

Using the 16 AEPs calculated to the standard tones, an Nd wave for each of the frequency separations was computed, for each subject, for each electrode site (F3, F4, C3, C4). The Nd wave is the difference between the AEP generated to the standards when ignored subtracted from the AEP to the attended standards (Burkhart, 1989; Hansen & Hillyard, 1980, 1984; Parasuraman, 1980). For conditions 1 and 2 of the example above, the EPs to the attended tones in both conditions (300 Hz standards in condition 1 and 700 Hz standards in condition 2) were combined and averaged together to formulate an AEP for the attended tones of the 400 Hz frequency separation. Similarly, the EPs to the nonattended tones for conditions 1 and 2 were combined and

averaged to produce an AEP for the nonattended tones of the 400 Hz frequency separation. Thus, the AEP of the nonattended tones was subtracted from the AEP of the attended tones to render an Nd wave for the 400 Hz frequency separation. Similar averaging and subtraction procedures were conducted on conditions 3 and 4 of the example to generate an Nd wave for the 50 Hz frequency separation. Thus, eight Nd waves were calculated for each subject, one for both of the frequency separations at all four electrode sites.

For each of the Nd waves, the following four dependent variables were measured: Peak amplitude, mean amplitude, latency at peak amplitude, and 25% latency. Peak amplitude is defined as the most negative peak in the 50-357 msec poststimulus range (Burkhart, 1989; Hansen & Hillyard, 1980; Parasuraman, 1980). The second dependent measure, mean amplitude, was calculated by averaging across all data points of the Nd wave within the interval of 0-357 msec poststimulus (Burkhart, 1989). In assessing the latency of Nd, the latency corresponding to the peak amplitude (labelled the peak latency) was utilized as the first dependent measure of latency (Burkhart, 1989; Hansen & Hillyard, 1980; Parasuraman, 1980). The 25% latency measure was also used. Twenty-five percent latency was defined as the latency poststimulus onset at which the leading edge of

the Nd wave had reached 25% of its peak amplitude (Hansen & Hillyard, 1980).

A total of 16 AEPs were also generated from the EPs elicited from the target stimuli. In calculating the AEPs to the targets, the EPs to the attended targets of a given frequency separation were combined and averaged to produce an AEP to the attended targets of the respective frequency separation. Similarly, the EPs elicited from the nonattended targets of a given frequency separation were also combined and averaged to render an AEP. These same mathematical procedures were conducted on the EPs generated from the attended and nonattended targets of the other frequency separation. Thus, for each subject, two AEPs (one in which the subject attended to relevant targets and a second in which they ignored irrelevant targets) were calculated for each frequency separation at four different scalp locations for a total of 16 AEPs.

Peak P300 is of particular interest in analyzing the AEPs of the target tones. The following two dependent measures were recorded to assess the amplitude and latency of P300: Peak amplitude and peak latency. Peak P300 was defined as the most positive peak in the 250-450 msec poststimulus range (Giedke, Thier, & Bolz, 1981). The amplitude and latency were measured at the maximum positive deflection of this peak. Amplitude was measured baseline to peak with baseline being the mean amplitude of the EEG for

the 30 msec period prior to stimulus onset. Latency was measured from the time of stimulus onset.

In analyzing the AEPs to the standards across all conditions, a measurement of peak N100 was also calculated. As was the case with P300, the dependent measures of peak amplitude and peak latency were undertaken for the purpose of assessing task arousal in the present study. Peak amplitude was defined as the most negative peak in the 80-130 msec range. Peak latency was defined as the latency corresponding to the peak amplitude.

Finally, the recording of the behavioral measures of hit rate, false alarms, and reaction time (RT) was also conducted. Hit rate was defined as the number of correct responses via the thumb switches to target tones in the attended frequency separation. False alarms were calculated for the number of incorrect responses to stimuli which were not targets in the attended channel. RT was defined as the duration of time from the onset of the tones required for subjects to correctly respond to targets in the attended channel.

CHAPTER III

RESULTS

In an effort to facilitate a better understanding of the data, the results section has been organized according to the various behavioral and EP component measures which were utilized. The hypotheses which were originally proposed in the latter part of the literature review are addressed within the subsections of the respective dependent measures to which they pertain.

The Nd Component

As was the case for the analysis carried out in Burkhart (1989), the present study made use of four dependent measures of the Nd component (peak amplitude, mean amplitude, peak latency, and 25% latency). A four-factor ANOVA (factors of group, gender, frequency separation, and electrode site placement) was calculated for each of the four dependent variables.

In addressing the first hypothesis of the present study that all subjects regardless of group would demonstrate greater selective attentional capabilities in the 400 Hz separations than in the 50 Hz conditions, the results

indicated a significant main effect for frequency separation across all four measures of Nd (peak amplitude: $F(1,28)=42.06$, $p< .0001$; mean amplitude: $F(1,28)=27.12$, $p< .0001$; peak latency: $F(1,28)=5.22$, $p< .03$; 25% Latency: $F(1,28)=7.77$, $p< .009$).The direction of these results indicated that the subjects had significantly greater absolute amplitudes and earlier onset latencies for the 400 Hz separation, thereby supporting the first hypothesis (See Figures 2-5). These results were also consistent with those of Hansen and Hillyard (1980) and Burkhart (1989).

Insert Figures 2, 3, 4, and 5 about here

In addition to the effects for frequency separation, the ANOVAs computed for the Nd measures also revealed significant main effects for electrode site placement in three of the four dependent measures. These differences were indicated in mean amplitude ($F(3,84)=5.67$, $p< .001$) with the frontal sites (F3 and F4) evidencing greater amplitudes; and peak latency ($F(3,84)=14.21$, $P< .0001$), and 25% latency ($F(3,84)=9.10$, $p< .0001$) with the frontal sites (F3 and F4) having later latency scores than the central sites (C3 and C4). See Figures 3, 4, and 5 for these results as well.

In an effort to evaluate the second proposed hypothesis that differences exist in the selective attention abilities

of depressives and controls (as determined in the various measures of Nd amplitude and latency), eight a priori Dunn's comparisons were calculated for peak amplitude, mean amplitude, peak latency, and 25% latency of Nd. These eight contrasts were comprised of comparisons between depressives and controls at each combination of frequency separation (400 Hz and 50 Hz) and electrode site placement (F3, F4, C3, and C4), thereby yielding eight possible combinations. The results of these comparisons showed that in the 400 Hz separation at site C3, controls evidenced significantly greater absolute peak amplitude in Nd than did the depressed subjects (Dunn's $t=2.86$, $p< .05$) and in the 400 Hz separation at site F3, controls recorded earlier peak latencies than depressives (Dunn's $t=2.64$, $p< .05$). There were no other significant comparisons indicated in the other dependent measures of Nd. See Tables 1-4.

Insert Tables 1, 2, 3, 4 about here

Further analysis of the ANOVAs conducted on the measures of Nd revealed significant group X gender X electrode site placement interactions for the dependent variables of peak amplitude ($F(3,84)=5.72$, $p< .02$) and peak latency ($F(3,84)=3.15$, $p< .03$). A significant interaction was also found in the Nd measure of mean amplitude for group X gender X frequency separation ($F(1,28)= 5.72$, $p< .02$). In

an attempt to determine potential group differences (depressives vs. controls) in the measures of peak amplitude and peak latency related to these significant interactions, eight post hoc comparisons (Tukey's HSD, Tukey, 1953) for each combination of the two levels of gender with the four electrode site placements were calculated. Similarly, four post hoc comparisons (Tukey's HSD, Tukey, 1953) were also computed for each combination of gender and frequency separation for the dependent measure of mean amplitude. There were no significant differences between the depressed and control subjects for any of these comparisons. See Tables 5, 6, and 7.

Insert Tables 5, 6, and 7 about here

N100 Data

While Burkhart (1989) reported no significant differences between depressives and controls for the various measures of Nd, significant group differences were indicated for the absolute amplitude of component N100 at the Fz electrode site (with controls having greater absolute amplitudes). In an effort to address the third hypothesis that depressed subjects would demonstrate decreased levels of arousal (as measured by the peak amplitude and peak latency of N100), eight <u>a priori</u> Dunn's comparisons were

calculated for each combination of frequency separation (400 Hz and 50 Hz) and electrode site (F3, F4, C3, and C4) for the peak amplitude and latency of N100. The results reflected no significant differences in N100 amplitude or latency between the groups for any of the comparisons. These results are illustrated in Tables 8 and 9.

Insert Tables 8 and 9 about here

Five-factor ANOVAs (factors of group, gender, frequency separation, electrode site placement, and attending vs. ignoring) were also calculated for the peak amplitude and peak latency measures of N100. Analysis of peak amplitude revealed no significant main effects or interactions. See Figure 6.

Insert Figure 6 about here

Analysis of peak latency revealed a significant main effect for electrode site placement ($F(3,84)=5.81$, $p< .001$) with the more central sites (C3 and C4) evidencing earlier peak latencies than the frontal (F3 and F4) placements. In addition, a significant electrode site X attending vs. ignoring interaction ($F(3,84)=2.73$, $p< .05$) was also found. There were no other significant main effects or interactions indicated. See Figure 7.

Insert Figure 7 about here

P300 Data

In an effort to assess late cognitive processing, the P300 components, which were extracted from the EPs recorded to the target tones, were scored and analyzed. The fourth hypothesis posited in the present study stated that depressives would demonstrate impairments in the later cognitive processes as defined as decreased amplitude and later onset latencies in the P300 component. Once again, eight a priori Dunn's comparisons between depressed and control subjects were calculated, combining each level of frequency separation (400 Hz and 50 Hz) with each of the electrode site placements (F3, F4, C3, and C4). These contrasts also reflected no significant differences between the subject groups for either dependent measures of P300. See Tables 9 and 10 for the results.

Insert Tables 10 and 11 about here

Five-factor ANOVAs (factors of group, gender, frequency separation, electrode site placement, and attending vs.

ignoring) were also calculated for the measures of peak amplitude and peak latency of the P300 component. Analysis of the measure of peak amplitude revealed significant main effects for frequency separation ($F(1,28)=9.15$, $p< .005$) and attending vs. ignoring target tones ($F(1,28)=8.93$, $p< .006$). These results indicated that subjects demonstrated significantly greater P300 amplitudes in the 400 Hz separation than the 50 Hz separation, and when they were attending to the tones rather than ignoring them. A significant main effect was also noted for electrode site placement ($F(3,84)=13.09$, $p< .0001$). These results indicated that the central placement sites (C3 and C4) evidenced greater peak amplitude than the frontal sites (F3 and F4). No other significant main effects or interactions were indicated. See Figure 8.

Insert Figure 8 about here

With regard to P300 peak latency, significant main effects were once again indicated for frequency separation ($F(1,28)=4.98$, $p< .03$) and attending vs. ignoring ($F(1,28)=8.15$, $p< .008$). These results revealed that subjects had earlier onset latencies in the 400 Hz vs. 50 Hz condition and when they ignored targets rather than attending to them. In addition, the results also indicated that females had significantly earlier onset latencies of

P300 than males ($F(1,28)=6.17$, $p< .02$). No other significant main effects or interactions were noted for the measure of peak latency. See Figure 9.

Insert Figure 9 about here

Data for Hemispheric Differences

The fifth hypothesis proposed in the present study suggested that hemispheric differences would be present in depressives in the various amplitude and latency measures of Nd, N100, and P300. In an effort to assess for hemispheric asymmetries, four a priori Dunn's comparisons were conducted contrasting the electrodes in the respective cranial regions (i.e. F3 vs. F4 and C3 vs. C4) for each combination of subject group (depressed and controls) and frequency separation (400 Hz and 50 Hz) at the dependent measures of Nd (peak amplitude, mean amplitude, peak latency, and 25% latency), P300 (peak amplitude and peak latency), and N100 (peak amplitude and peak latency). There were no significant differences between hemisphere sites for any of the dependent variables of Nd, P300, or N100. Tables 12-19 present these results.

Insert Tables 12-19 about here

Behavioral Data

Within the selective attention paradigm, the assessment of the behavioral responses of the subjects included the measurement of hits (correct responses via the thumb switches to target stimuli in the attended channel), false alarms (incorrect responses via the thumb switches to stimuli which were not targets in the attended channel), and reaction time (the duration of time required for subjects to correctly respond to targets in the attended channel). The final hypothesis proposed in the present study suggested that depressives would demonstrate task impairments evidenced by significantly fewer hits, more false alarms, and slower reaction times than the controls.

Three-factor ANOVAs (variables of group, gender, and frequency separation) were calculated for each of the three dependent measures (hit rate, false alarms, and reaction time). With regards to the final proposed hypothesis, no significant main effects or interactions were found which revealed differences between depressives and controls for any of the dependent measures. See Figures 10, 11, and 12.

Insert Figures 10, 11, and 12 about here

However, a number of other significant results were found. In exploring the data corresponding to hit rate, males had significantly more hits than females ($F(1,28)=4.99$, $p< .03$)

and subjects had a significantly easier time demonstrating hits in the 400 Hz separation than in the 50 Hz conditions ($F(1,28)=12.79$, $p< .001$). In addition, a significant gender X frequency separation interaction was also indicated ($F(1,28)=4.98$, $p< .03$). Data regarding false alarms also indicated that subjects had fewer false alarms in the 400 Hz frequency separation than in the 50 Hz separation ($F(1,28)=6.83$, $p< .01$). Finally, the analysis of the results pertaining to reaction time revealed that males were significantly faster than females ($F(1,28)=7.45$, $p< .01$). Thus, the results of the behavioral measures of the selective attention paradigm showed that males were significantly faster and had more hits than females. Furthermore, subjects demonstrated more hits and fewer false alarms in the 400 Hz separation.

CHAPTER IV

DISCUSSION

The present study made use of a selective attention paradigm which was first utilized by Hansen and Hillyard (1980) and was later employed by Burkhart (1989) to investigate possible differences in attention between depressed and control subjects. As predicted in the first hypothesis of the present study, subjects exhibited significantly greater absolute amplitude (as measured by peak amplitude and mean amplitude) and earlier onset latency (as measured by peak latency and 25% latency) for the Nd component in the 400 Hz frequency separation than in the 50 Hz separation across all electrode sites (F3, F4, C3, and C4). These results, which are consistent with the Nd data of Hansen and Hillyard (1980) and Burkhart (1989), indicate the subjects were better able (as assessed via the dependent measures of Nd) to selectively attend when the physical difference between the attended and unattended stimulus channels was large (400 Hz separation). Subjects experienced significantly greater difficulty in attending to the target channel when the difference was small (50 Hz separation). The robustness of this frequency separation main effect

evident within the four dependent measures of the present study, as well as in the results of Hansen and Hillyard (1980) and Burkhart (1989), confirms the present EP paradigm as a reliable indicator (via the Nd component) of selective attention processes.

The primary intention of the present study was to examine potential differences in the attentional abilities and later cognitive processes of depressed and control individuals. The second principal hypothesis of the present investigation posited that depressives would have poorer selective attentional abilities than controls. These differences would be reflected in the amplitudes and latencies of the Nd component. Of the multitude (eight comparisons for each of four dependent measures) of a priori Dunn's comparisons which were conducted to evaluate this hypothesis, only two significant comparisons between the groups were indicated; one in the 400 Hz separation at electrode site C3 for peak amplitude, and the other in the 400 Hz separation at site F3 for peak latency. All other planned comparisons were nonsignificant. While the presence of two significant comparisons must not be disregarded, the overwhelming lack of significant findings regarding group differences would suggest the presence of Type I (Alpha) errors as a possible explanation for these noted significant comparisons. Furthermore, analysis of group main effects within the various four-factor ANOVAs also showed no

significant differences. Post hoc tests (Tukey HSD) conducted on the significant interaction of group X gender X site for the Nd measures of peak amplitude and peak latency, as well as the significant interaction of group X gender X frequency separation for the mean amplitude of Nd also revealed no significant group differences. It was noted that in most of the cases of the a priori and post hoc tests, the group means were in the hypothesized direction with the depressives having smaller Nd amplitudes and later latencies. Consequently, there appeared to be some trends in the data possibly suggesting attentional differences. However, rigorous statistical analysis did not indicate true significant differences. Thus, the data was conservatively interpreted as reflecting no differences in the amplitude and latency measures of Nd and subsequently suggest no differences in the selective attentional processes of depressed and control subjects. These results are once again consistent with the data of Burkhart (1989).

The third hypothesis which was examined in the present investigation proposed that depressives would also feature lower levels of task arousal as defined by decreased amplitude and later onset latency measures of the N100 component. Similar findings would be consistent with data reported by Burkhart (1989) for the Fz scalp locations and by El Massioui and Lesevre (1988). However, the present study found no significant group differences for N100

amplitude or latency, reflecting no differences in the level of task arousal between depressed and control subjects for the present study.

The lack of statistically significant N100 differences for the groups points to an apparent discrepancy in the findings of the present study with the results reported by Burkhart (1989). If the methodology which was utilized in the present investigation was a replication of Burkhart's study, a logical question follows: What, if any, differences existed between the methodologies which would account for the differences in the results? One possible explanation for the discrepant findings may be in the choice of electrode site placement in each study. The sites employed in Burkhart (1989) were the midline locations of Fz and Cz. However, in an effort to be able to assess hemispheric differences, the present study made use of F3, F4, C3, and C4. It is possible that in moving the electrode sites laterally (away from the midline), the significant N100 differences detected for Fz in Burkhart, were no longer present for the sites chosen for the present study.

A second plausible explanation for the discrepant findings addresses the level of arousal within the experimental process. In the present study, subjects were screened with the BDI up to seven days prior to participation in the selective attention paradigm. At no time were subjects administered the BDI immediately prior to

the experimental paradigm. However, subjects selected for participation by Burkhart (1989) were assessed with the BDI immediately before the selective attention paradigm. The ramifications of administering the BDI prior to an attentional task may be that, while this practice produced little or no effect on nondepressed controls, it required the depressed subjects to consider the presence and extent of their depressed symptoms, thereby lowering their levels of arousal for the task at hand. While it is impossible to speculate with any absolute certainty concerning the effects that these differences in methodology could produce, the possibility exists that these differences had the potential to alter the arousal levels corresponding to N100 measurement.

The fourth hypothesis addressed in the present study involved an expansion of the paradigm used in Burkhart (1989). In addition to selective attention, the present study included the recording and analysis of the P300 component to the target tones in an effort to assess the presence of group differences in later cognitive processes. Previous EP research (Kutas, McCarthy, & Donchin, 1977; Hillyard & Kutas, 1983; McCarthy & Donchin, 1981; Donchin, Ritter, & McCallum, 1978) has suggested a strong association between the amplitude and latency of P300 and processes such as stimulus encoding, stimulus evaluation, and the updating of environmental schemata. The present study hypothesized

that depressives would demonstrate significantly lower amplitudes and longer onset latencies in P300. Such results would be consistent with the findings reported in Whalley et al. (1987), Pfefferbaum et al. (1984), Levit, Sutton, and Zubin (1973), and Roth et al. (1981) for amplitude measures and with those in Johnson et al. (1984) for measures of latency.

The results of the present investigation indicated no significant differences between depressives and controls for P300 amplitude or latency. These results failed to confirm the findings of the studies cited above and suggest that no significant differences exist in the late cognitive processes which are associated with the P300 component. The lack of significant P300 amplitude and latency differences is consistent with data reported in Plooij-van Gorsel (1984) which indicated that "...depressives do not have disturbed cognitive functioning" (p. 614). Similar nonsignificant P300 findings have also been reported in the literature (Giedke, Thier, & Bolz, 1981; El Massioui & Lesevre', 1988; Thier, Axmann, & Giedke, 1986). Thus, according to the P300 data collected with the selective attention paradigm, there are no differences in the encoding and stimulus evaluation processes between depressed and control subjects.

In looking at the various significant main effects noted for the measures of P300 peak amplitude and latency, an unusual finding was discovered for the variable of

attending vs. ignoring for peak latency. The results indicated that subjects had overall earlier peak latencies in the conditions when they ignored the target tones. While this effect was unexpected, it is probable that such results were influenced by the large main effect seen for the peak amplitude of P300. Consequently, while P300 latency differences were reflected in the data, these differences were most likely due to EEG artifact.

The present study also attempted to explore hemispheric differences (as measured by the amplitudes and latencies of Nd, N100, and P300) reflecting asymmetrical processing in the brains of depressed subjects. As mentioned in the introduction, Henriques and Davidson (1990) utilized a Fourier transformation of the EEG to detect that depressives had more left-sided anterior and less right-sided posterior activation. Furthermore, additional EEG data cited in Davidson and Tomarken (1989) and Ahern and Schwartz (1985) provided further indications of asymmetric processing in depressives. However, the electrode site placement utilized in Burkhart (1989) which included the centrally-placed Fz and Cz sites, would not allow for the assessment of hemispheric differences. Consequently, the present study recorded data from the laterally located sites of F3, F4, C3, and C4. The fifth hypothesis, therefore, was that hemispheric differences would exist for the depressed subjects but not for the controls. Within the 64 different

planned comparisons (eight comparisons for each of the four dependent measures of Nd, two dependent measures of N100, and two dependent measures of P300) computed to assess differences in hemispheric processing, there were no significant differences found between hemispheres for any of the dependent measures of Nd, N100, or P300.

In comparing the nonsignificant results of the present study to the significant hemispheric findings reported in Henriques and Davidson (1990), Davidson and Tomarken (1989), and Ahern and Schwartz (1985), the vast procedural differences in EEG measurement must be noted. The studies cited above made use of a specialized form of EEG analysis (Fourier transformation) in arriving at the results indicating asymmetrical processing for depressives. In addition, the significant hemispheric differences noted in the above studies also involved the recording of brain activity while the subjects were at rest. These hemispheric differences were not evident in the EP paradigm of the present study. Thus, while the significant findings of Davidson and Tomarken (1989) and the other studies served as the basis for the original hypothesized hemispheric differences, the results of the present investigation revealed no asymmetries in the processing of depressed subjects.

The final hypothesis posited in this study addressed the behavioral components to the selective attention

paradigm. As was the case for the inclusion of the assessment of P300 and hemispheric differences, the present study attempted to expand on the sole behavioral measure of hits (correctly identified targets) used in Burkhart (1989) by including the assessment of false alarms and reaction time (RT) to hits. A number of studies (Miller, 1974; Huston & Senf, 1952; Plooij-van Gorsel, 1984; El Massioui & Lesevre', 1988, to cite a few) have utilized reaction time as a behavioral estimate of psychomotor slowing and reported results showing depressed subjects responding significantly more slowly. Based on these findings, the present study hypothesized significantly slower reaction times for depressed subjects. Furthermore, it was also posited that depressives would have fewer hits and more false alarms than the control counterparts.

As was found for the Nd, N100, and P300 components, there were no significant differences between depressed and control subjects for reaction time, hits, or false alarms. Significant main effects were indicated for gender for the measures of hits and RT (with males having faster times and more hits) and for frequency separation for the measures of hits and false alarms (subjects had more hits and fewer false alarms in the 400 Hz separation). A significant interaction between gender and frequency separation was also noted for the measure of hits. Thus, the present study confirmed the results of Burkhart (1989) which also failed

to show a group difference for the measure of hits. However, the data from the present study reflect disparity with the data presented in Miller (1974), Huston and Senf, (1952), Plooij-van Gorsel (1984), El Massioui and Lesevre, (1988) (and a number of others) all of which indicated an increase in RT for depressives. While a number of explanations are posited for the disparity between the present results and the previous research, these explanations are presented at the end of the discussion section as the possibilities for the overall lack of differences between the depressed and control subjects.

Thus, in reviewing the results of the present investigation, with the exception of two significant comparisons (in the 400 Hz separation at site C3 of the peak amplitude and the 400 Hz separation at site F3 of the peak latency of Nd), there were no differences between depressed and control subjects for measures of selective attention, task arousal, and late cognitive processing (as reflected in the various measures of Nd, N100, and P300, respectively). Furthermore, differences were also not indicated for measures of hit rate, false alarms, and reaction time. Finally, there was no evidence of hemispheric asymmetry for depressed subjects in any of the components (Nd, N100, and P300) assessed in this study.

In an attempt to account for the nonsignificant findings between depressives and controls reported in this

investigation, four explanations have been generated. The first of these is that there are no actual differences in the selective attentional abilities of the depressed and control subjects, and that the deficits noted in the previous research were not directly attributable to selective attention.

The second possibility was posited in the discussion section of Burkhart (1989) and addresses the low criterion cutoff scores on the BDI for inclusion into the depressed subject sample. In selecting depressed subjects for the Burkhart study, a score of 10 or greater on the BDI qualified subjects for participation. Thus, a mean BDI score of 16.6 (range of 10 to 33) was reported for depressives. However, as cited previously, Kendall et al. (1987) indicated that, in order to accurately classify subjects as "depressed," scores of 20 to 30 are needed to "reflect moderate depression," with scores greater than 30 indicating severe depression. Thus, Burkhart concluded that perhaps the sample of depressed subjects utilized in her study did not feature a level of depressive symptoms severe enough to detect differences in selective attentional abilities.

The present study attempted to rectify the low criterion scores by raising the cutoff score for depressives on the BDI from 10 (in the previous study) to 16. This resulted in a mean BDI increase of approximately one standard deviation to 22.75 (scores ranging from 16 to 35)

for the depressed subjects in the present study. This BDI mean falls within the moderate range of depression (as indicated by Kendall et al., 1987) and represents an increase in the severity of depression as compared to the sample used in Burkhart (1989). Yet, no differences were indicated in selective attention abilities of depressives and controls.

However, it should be noted that subjects within the present study were administered the BDI up to seven days prior to their participation in the selective attention paradigm. And while it is unlikely that a large percentage of the depressed sample would experience significant elevations in their mood to the extent that it would affect attentional levels, this factor may have contributed in part to the lack of group differences.

It is possible that while the depressed subjects utilized in the present study reported a more severe degree of depressive symptoms than those of Burkhart (1989), they were not as severely depressed as those which were utilized in studies reporting significant cognitive impairments (Weingartner et al., 1981; Henry, Weingartner, & Murphy, 1973; Breslow, Kocsis, & Belkin, 1981, 1980). These studies made use of depressives from a clinical population. In most of these cases, subjects were not only diagnosed as depressed, but had also been hospitalized at some point due to the severity of their depression. Thus, it is possible

that the studies which reported significant impairments in the attentional abilities utilized a sample which featured a more severe degree of depression.

However, a more plausible explanation (regarding depression levels) for this disparity between the present results and those reported in the previous research may lie in factors separate from the severity of depression featured in the experimental groups. Other variables such as age, psychiatric treatment (psychotropic drugs, electroconvulsive therapy, etc.), and the presence of other psychological dysfunctions such as psychosis will definitely affect the cognitive processing of the depressed subjects. For instance, the present study made use of college undergraduates between the ages of 18 and 35. However, the majority of the studies that reported cognitive deficits for depressives (Weingartner et al., 1981; Henry, Weingartner, & Murphy, 1973; Breslow, Kocsis, & Belkin, 1981, 1980) sampled inpatient populations with subjects generally ranging in ages from 40 to 70 years old. Thus, it is possible that factors associated with increased aging such as dementia may have confounded the significant results reported by these studies. Recent findings have pointed out the difficulties in differentiating cognitive impairments due to affective disorders (such as depression) from deficits due to dementia and other degenerative processes (Harper, Kotik-Harper, & Kirby, 1990).

Furthermore, the samples utilized in the previous studies have also featured subjects with a high prevalence of other psychological difficulties such as psychosis as well as individuals who at the time of participation required psychotropic medication for the treatment of their psychological symptoms. These factors would also be likely to contribute to impairments in cognitive functioning. Therefore, while the present study and the previous investigation by Burkhart reflected no differences in selective attention for the depressed and control groups, it is possible that factors other than depression (i.e. age, dementia, psychosis, psychotropic medication) may have contributed to the notable deficits reported in those studies.

A third possibility for the lack of differences involves the nature and specificity of the task utilized in the selective attention paradigm. In studies such as those by Weingartner et al. (1988), Henry, Weingartner, and Murphy (1973), Breslow, Kocsis, and Belkin (1980, 1981), and Berndt and Berndt (1980), inferences were made concerning the attentional deficits reported for depressives based on performances on behavioral tasks such as encoding strategy tests, measures of short term and long term memory, and recall of affectively-laden themes. In all cases, conclusions concerning attentional abilities were extrapolated from a variety of other information processes

(such as memory, retrieval, encoding, rehearsal, etc.). In utilizing the selective attention paradigm of Hansen and Hillyard (1980), the present study attempted to attain a less confounded measure of the process of attention. This paradigm makes use of emotionally-neutral stimuli (tone pips) and evaluates selective attention on the basis of a physiological measure (EPs). Furthermore, the employment of this paradigm in several studies (Hansen & Hillyard, 1980; Burkhart, 1989; and the present study) has revealed it as a robust measure which specifically assesses selective attention. Other paradigms such as that utilized in Krames and MacDonald (1985) were able to detect deficits in depressives for divided attentional abilities.

In addition, while several of the behavioral studies (Dunbar & Lishman, 1984; Breslow, Kocsis, & Belkin, 1981) have reported attentional deficits for depressives, these investigations made use of emotionally-laden stimuli to assess this cognitive process. For instance, Breslow et al. (1981) indicated that depressed subjects demonstrated no attentional differences from controls for stories which contained neutral or emotionally- negative themes. However, depressives recalled significantly fewer emotionally-positive themes. Similarly, the results in Dunbar and Lishman (1984) revealed that depressives tended to recall negative information more easily. The present study however, made use of neutral stimuli (tone pips) in

assessing the selective attentional capabilities of depressives and controls. This lack of emotional connotation associated with task stimuli offers another possible explanation for the lack of differences between the groups. Thus, while it appears that a number of studies have reported impairments in attention for depressed subjects, these studies have frequently been confounded by other cognitive processes (such as short and long term memory tasks) and have not utilized methodologies and stimuli which specifically addressed selective attentional capacities.

A final explanation for the lack of differences between depressives and controls is applicable not only to the measures of selective attention, but also to the nonsignificant group findings for the late cognitive processes (as assessed by P300), and the behavioral measures of hit rate, false alarms, and RT. This explanation involves the level of task difficulty required of the subjects in the selective attention paradigm.

In the selective attention paradigm, subjects are required to not only discriminate tones of various frequency pitches but also of different time durations (51 ms for standards and 102 ms for targets). A large majority of the subjects who participated in the present study reported that they had great difficulty distinguishing the standard tones from the targets. It therefore appears that, while a number of studies have reported attentional deficits, late

cognitive processes, and RT slowing for depressives, the nature of the tasks utilized in these studies were not as cognitively taxing as that of the selective attention paradigm.

Furthermore, there are data which suggests that depressives actually perform worse (as compared to controls) on easy tasks than on tasks which are cognitively more taxing. In assessing the divided attentional abilities of depressives, Krames and MacDonald (1985) reported that while depressed subjects performed significantly worse on a task which had a moderate level of difficulty, there were no differences between the depressives and controls on a similar task requiring greater attentional abilities. Such results suggest that while depressed subjects may not utilize as many attentional resources for easy tasks, they are able to demonstrate adequate capabilities for difficult tasks. Therefore, the lack of group differences reported in the present investigation may be reflective of a task which required greater attentional resources and a higher degree of effort on the part of the depressed subjects and consequently resulted in no detectable differences between the groups.

Thus, in assessing how the results of the present study contribute to an attention deficit hypothesis for depression, the data suggest that, with regard to tasks which specifically require selective attention, there are no

differences indicated for individuals with a mild to moderate degree of depression. These findings are therefore in support of those reported in Burkhart (1989).

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APPENDICES

APPENDIX A

TABLE 1

GROUP MEANS, AND STANDARD DEVIATIONS () OF ND PEAK AMPLITUDE FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	-3.21	-3.86	-2.49	-2.21
	(0.86)	(1.51)	(1.00)	(1.28)
F4	-3.48	-3.73	-2.44	-2.60
	(1.03)	(1.40)	(1.08)	(1.50)
C3	-2.85	-4.11	-2.14	-1.94
	(1.27)	(1.40)	(1.04)	(0.96)
C4	-3.04	-3.94	-2.39	-2.29
	(1.14)	(1.67)	(1.03)	(1.18)

TABLE 2

GROUP MEANS, AND STANDARD DEVIATIONS () OF ND MEAN AMPLITUDE FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	-1.16	-1.11	-0.68	-0.28
	(0.73)	(1.05)	(0.51)	(0.82)
F4	-1.37	-1.03	-0.74	-0.60
	(0.74)	(1.10)	(0.57)	(0.82)
C3	-0.86	-1.12	-0.43	-0.07
	(0.73)	(0.93)	(0.73)	(0.60)
C4	-1.02	-1.15	-0.62	-0.34
	(0.77)	(1.02)	(0.66)	(0.76)

TABLE 3

GROUP MEANS, AND STANDARD DEVIATIONS () OF ND PEAK LATENCY FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	326	244	339	317
	(78.71)	(60.73)	(94.22)	(86.47)
F4	336	264	353	343
	(87.11)	(78.01)	(98.03)	(109.04)
C3	257	230	285	258
	(86.62)	(69.37)	(97.03)	(76.44)
C4	258	241	292	308
	(83.45)	(74.58)	(124.74)	(99.50)

TABLE 4

GROUP MEANS, AND STANDARD DEVIATIONS () OF ND 25% LATENCY FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	206	138	226	230
	(122.38)	(50.20)	(90.94)	(107.28)
F4	233	176	253	250
	(117.63)	(46.78)	(115.70)	(112.60)
C3	174	115	208	192
	(87.21)	(33.12)	(105.13)	(87.69)
C4	151	147	198	228
	(32.82)	(33.45)	(111.50)	(75.81)

TABLE 5

GROUP MEANS, AND STANDARD DEVIATIONS () UTILIZED IN POST-HOC COMPARISONS OF ND PEAK LATENCY FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR MALES AND FEMALES

	Males		Females	
Site	Depressed	Control	Depressed	Control
F3	342.81	304.69	322.19	256.56
	(83.80)	(92.85)	(88.92)	(64.88)
F4	325.63	353.13	363.44	253.75
	(80.74)	(103.12)	(100.36)	(73.02)
C3	273.13	250.94	268.44	237.50
	(90.20)	(82.10)	(95.82)	(65.04)
C4	273.44	305.31	275.94	243.13
	(113.85)	(106.12)	(100.93)	(67.43)

TABLE 6

GROUP MEANS, AND STANDARD DEVIATIONS () UTILIZED IN POST-HOC COMPARISONS OF ND PEAK AMPLITUDE FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR MALES AND FEMALES

	Males		Females	
Site	Depressed	Control	Depressed	Control
F3	-2.94	-3.19	-2.77	-2.88
	(1.01)	(1.69)	(0.99)	(1.57)
F4	-2.88	-3.41	-3.04	-2.92
	(1.09)	(1.73)	(1.27)	(1.33)
C3	-2.61	-2.92	-2.37	-3.15
	(1.35)	(1.48)	(1.06)	(1.78)
C4	-2.96	-3.02	-2.47	-3.21
	(1.12)	(1.62)	(1.09)	(1.72)

TABLE 7

GROUP MEANS, AND STANDARD DEVIATIONS () UTILIZED IN POST-HOC COMPARISONS OF ND MEAN AMPLITUDE FOR EACH OF THE FREQUENCY SEPARATIONS (400 HZ, 50 HZ) FOR MALES AND FEMALES

	Males		Females	
Freq. Sep.	Depressed	Control	Depressed	Control
400 Hz	-1.14	-1.61	-1.04	-0.59
	(0.88)	(0.81)	(0.59)	(0.93)
50 Hz	0.96	-0.28	0.54	0.36
	(0.65)	(0.83)	(0.59)	(0.70)

TABLE 8

GROUP MEANS, AND STANDARD DEVIATIONS () OF N100 PEAK AMPLITUDE FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	-3.69	-4.47	-5.51	-4.81
	(6.15)	(4.71)	(3.38)	(3.55)
F4	-3.25	-4.49	-6.21	-4.28
	(6.40)	(4.71)	(1.85)	(4.39)
C3	-4.53	-4.65	-5.17	-5.47
	(4.99)	(4.68)	(3.42)	(1.60)
C4	-4.53	-4.95	-5.13	-5.27
	(4.89)	(3.37)	(3.20)	(1.48)

TABLE 9

GROUP MEANS, AND STANDARD DEVIATIONS () OF N100 PEAK LATENCY FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	100.81	100.13	100.09	100.22
	(13.80)	(15.98)	(9.93)	(15.06)
F4	100.94	99.66	103.25	102.23
	(15.93)	(15.66)	(13.42)	(16.26)
C3	97.97	95.97	98.28	97.47
	(12.74)	(11.39)	(10.22)	(10.87)
C4	95.43	95.88	96.75	97.25
	(13.76)	(14.54)	(11.04)	(11.11)

TABLE 10

GROUP MEANS, AND STANDARD DEVIATIONS () OF P300 PEAK AMPLITUDE FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	0.49	1.14	-0.23	0.20
	(2.24)	(3.38)	(1.81)	(3.49)
F4	-0.08	1.15	-0.94	0.20
	(2.43)	(3.25)	(1.75)	(3.17)
C3	1.79	2.38	1.03	1.44
	(2.03)	(2.66)	(1.72)	(2.18)
C4	1.07	2.30	0.58	1.31
	(2.00)	(2.79)	(1.95)	(2.39)

TABLE 11

GROUP MEANS, AND STANDARD DEVIATIONS () OF P300 PEAK LATENCY FOR EACH OF THE SCALP LOCATIONS (F3, F4, C3, C4) FOR THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	400 Hz		50 Hz	
Site	Depressed	Control	Depressed	Control
F3	309.00	289.19	316.44	306.53
	(30.82)	(33.37)	(37.48)	(32.65)
F4	312.22	290.03	312.44	304.63
	(29.31)	(36.63)	(37.34)	(30.77)
C3	306.47	293.19	320.03	303.53
	(32.73)	(38.50)	(39.76)	(29.91)
C4	300.59	289.78	311.25	304.56
	(27.23)	(40.06)	(32.85)	(33.52)

TABLE 12

SITE MEANS, AND STANDARD DEVIATIONS () OF ND PEAK AMPLITUDE FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	-3.21	-3.48	-2.85	-3.04
	(0.86)	(1.03)	(1.27)	(1.14)
Control	-3.86	-3.73	-4.11	-3.94
	(1.51)	(1.40)	(1.40)	(1.67)
50 Hz				
Depressed	-2.49	-2.44	-2.14	-2.39
	(1.00)	(1.08)	(1.04)	(1.03)
Control	-2.21	-2.60	-1.94	-2.29
	(1.28)	(1.49)	(0.96)	(1.18)

TABLE 13

SITE MEANS, AND STANDARD DEVIATIONS () OF ND MEAN AMPLITUDE FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	-1.16	-1.37	-0.86	-1.02
	(0.73)	(0.74)	(0.73)	(0.77)
Control	-1.11	-1.03	-1.12	-1.15
	(1.05)	(1.10)	(0.93)	(1.02)
50 Hz				
Depressed	-0.68	-0.74	-0.43	-0.62
	(0.51)	(0.57)	(0.73)	(0.66)
Control	-0.28	-0.60	-0.07	-0.34
	(0.82)	(0.82)	(0.60)	(0.76)

TABLE 14

SITE MEANS, AND STANDARD DEVIATIONS () OF ND PEAK LATENCY FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	326	336	257	258
	(78.71)	(87.11)	(86.62)	(83.45)
Control	244	264	230	241
	(60.73)	(78.01)	(69.37)	(74.58)
50 Hz				
Depressed	339	353	285	292
	(94.22)	(98.03)	(97.03)	(124.74)
Control	317	343	258	308
	(86.47)	(109.04)	(76.44)	(99.50)

TABLE 15

SITE MEANS, AND STANDARD DEVIATIONS () OF ND 25% LATENCY FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	206	233	174	151
	(122.38)	(117.63)	(87.21)	(32.82)
Control	138	176	115	147
	(50.20)	(46.78)	(33.12)	(33.45)
50 Hz				
Depressed	226	253	208	198
	(90.94)	(115.70)	(105.13)	(111.50)
Control	230	250	192	228
	(107.28)	(112.60)	(87.69)	(75.81)

TABLE 16

SITE MEANS, AND STANDARD DEVIATIONS () OF N100 PEAK AMPLITUDE FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	-3.69	-3.25	-4.53	-4.53
	(6.15)	(6.40)	(4.99)	(4.89)
Control	-4.47	-4.49	-4.65	-4.95
	(4.71)	(4.71)	(4.68)	(3.37)
50 Hz				
Depressed	-5.51	-6.21	-5.17	-5.13
	(3.38)	(1.85)	(3.42)	(3.21)
Control	-4.81	-4.28	-5.47	-5.27
	(3.55)	(4.39)	(1.60)	(1.48)

TABLE 17

SITE MEANS, AND STANDARD DEVIATIONS () OF N100 PEAK LATENCY FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	100.81	100.94	97.97	95.43
	(13.80)	(15.92)	(12.74)	(13.76)
Control	100.13	99.66	95.97	95.88
	(15.98)	(15.66)	(11.39)	(14.55)
50 Hz				
Depressed	100.09	103.25	98.28	96.75
	(9.93)	(13.42)	(10.22)	(11.04)
Control	100.22	102.23	97.47	97.25
	(15.06)	(16.26)	(10.87)	(11.11)

TABLE 18

SITE MEANS, AND STANDARD DEVIATIONS () OF P300 PEAK AMPLITUDE FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	0.49	-0.08	1.79	1.07
	(2.24)	(2.43)	(2.03)	(2.00)
Control	1.14	1.15	2.38	2.30
	(3.38)	(3.25)	(2.66)	(2.79)
50 Hz				
Depressed	-0.23	-0.94	1.03	0.58
	(1.81)	(1.75)	(1.72)	(1.95)
Control	0.20	0.20	1.44	1.31
	(3.49)	(3.17)	(2.18)	(2.39)

TABLE 19

SITE MEANS, AND STANDARD DEVIATIONS () OF P300 PEAK LATENCY FOR DEPRESSED AND CONTROL SUBJECTS IN THE 400 HZ AND 50 HZ FREQUENCY SEPARATIONS

	F3	F4	C3	C4
400 Hz				
Depressed	309.00	312.22	306.47	300.59
	(30.82)	(29.31)	(32.73)	(27.23)
Control	289.19	290.03	293.19	289.78
	(33.37)	(36.63)	(38.50)	(40.06)
50 Hz				
Depressed	316.44	312.44	320.03	311.25
	(37.48)	(37.34)	(39.76)	(32.85)
Control	306.53	304.63	303.53	304.56
	(32.65)	(30.77)	(29.91)	(33.52)

APPENDIX B

FIGURE CAPTION

Figure 1. An example of an evoked potential (EP) indicating the N100 and P300 components.

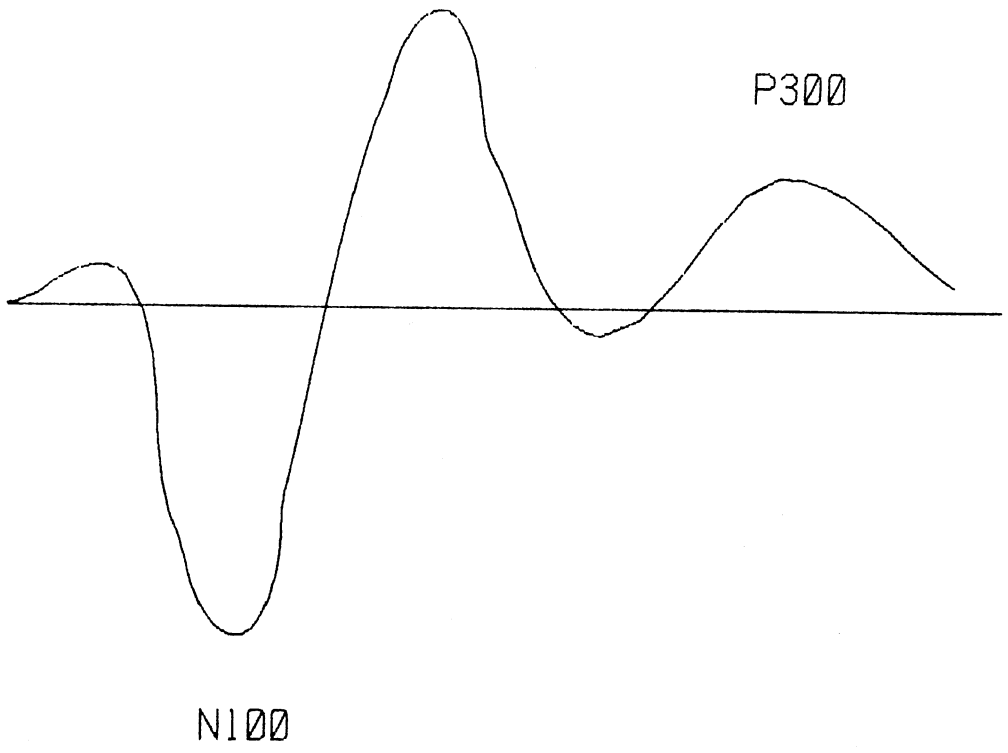
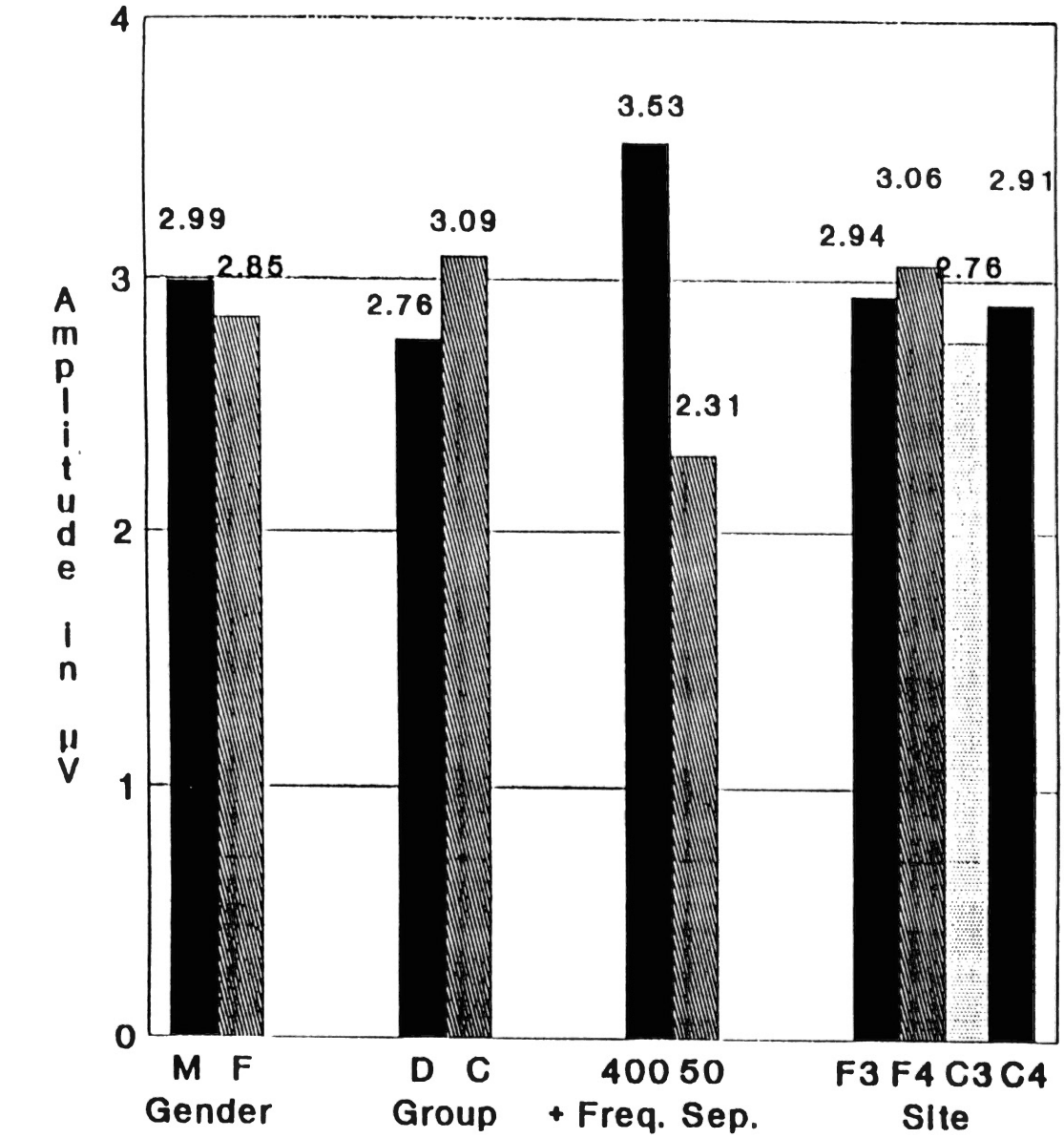

P300
N100

FIGURE CAPTION

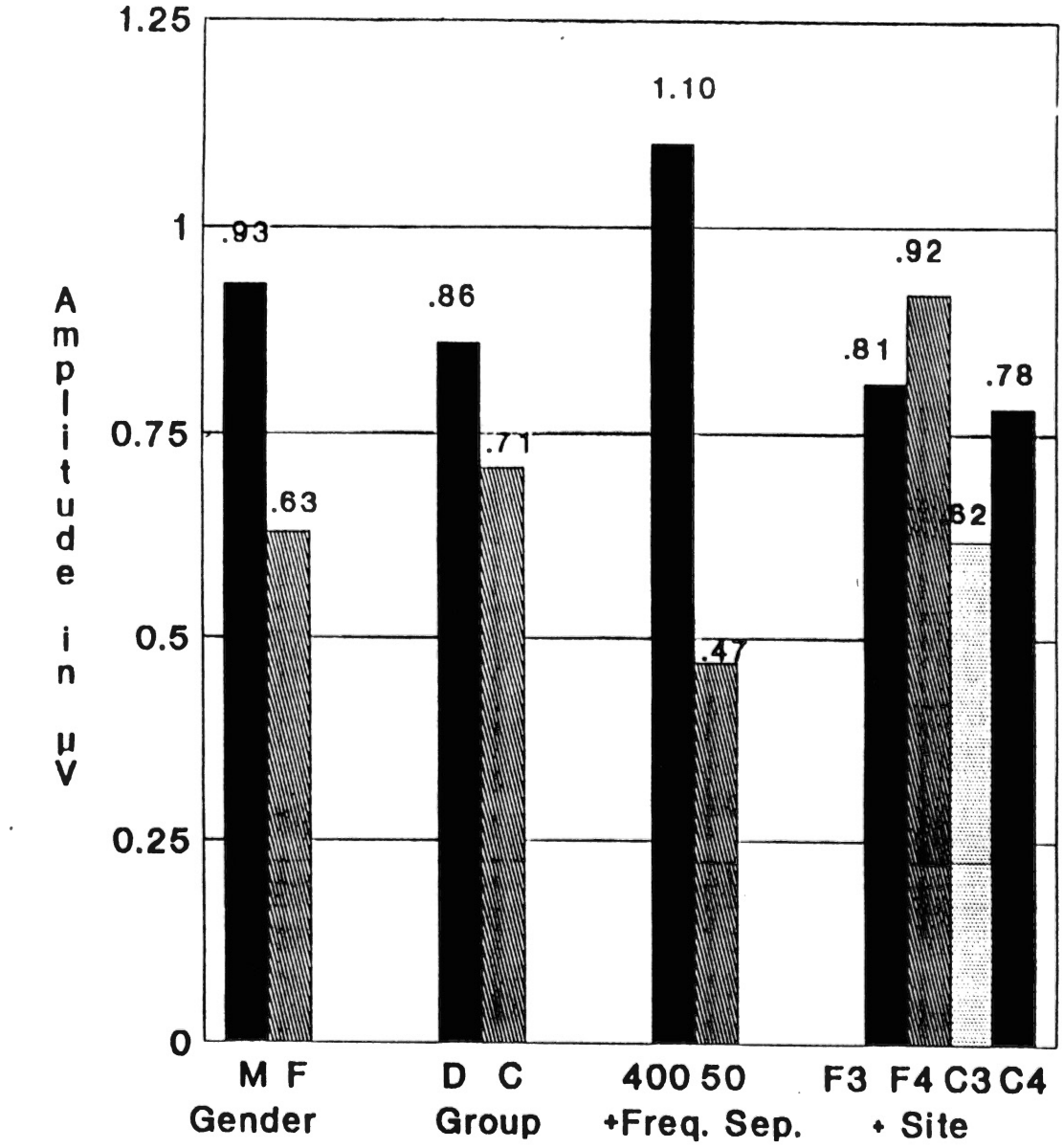
Figure 2. Main effects of Nd peak amplitude.



Note: • = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

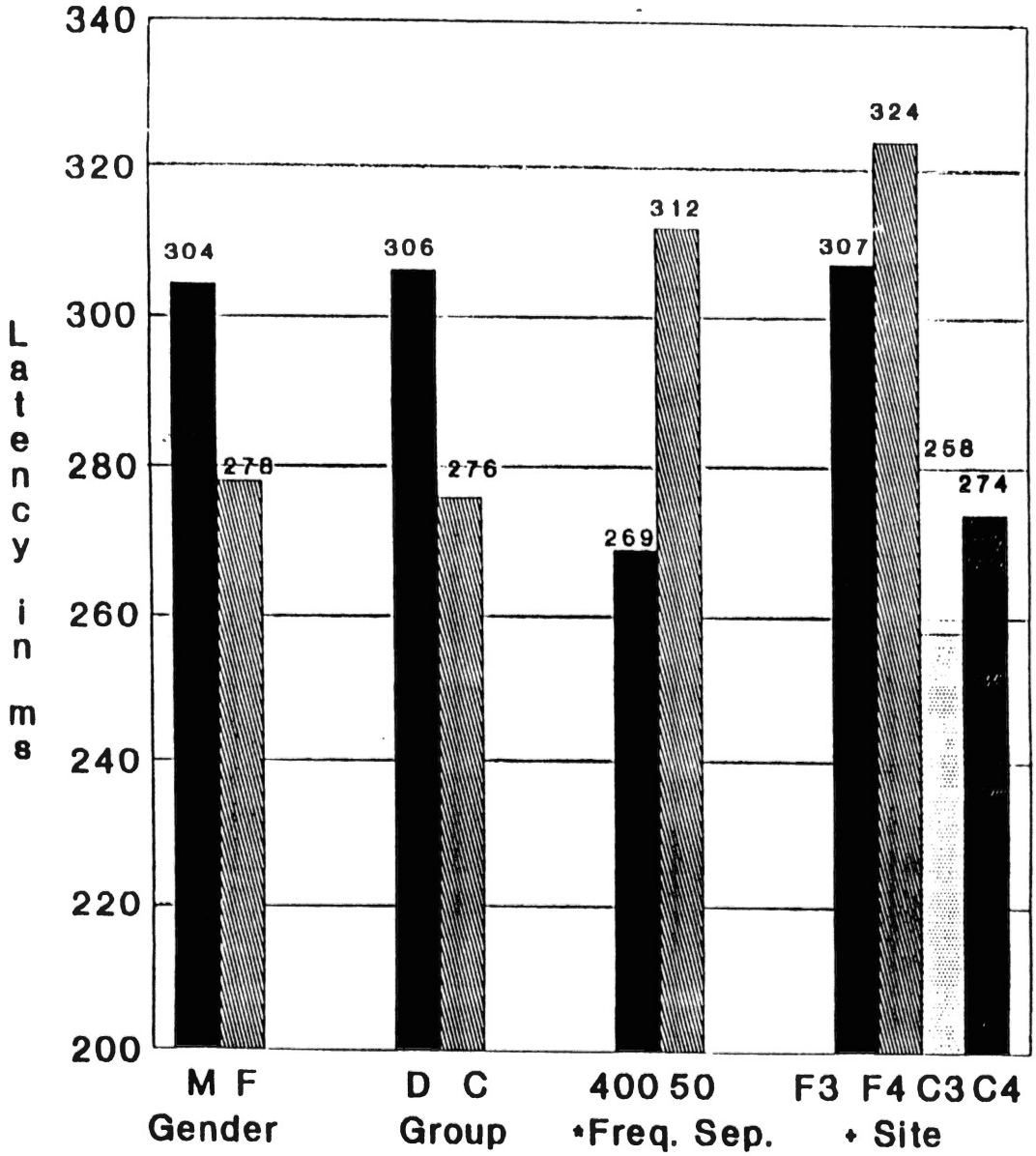
Figure 3. Main effects of Nd mean amplitude.



Note: • = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

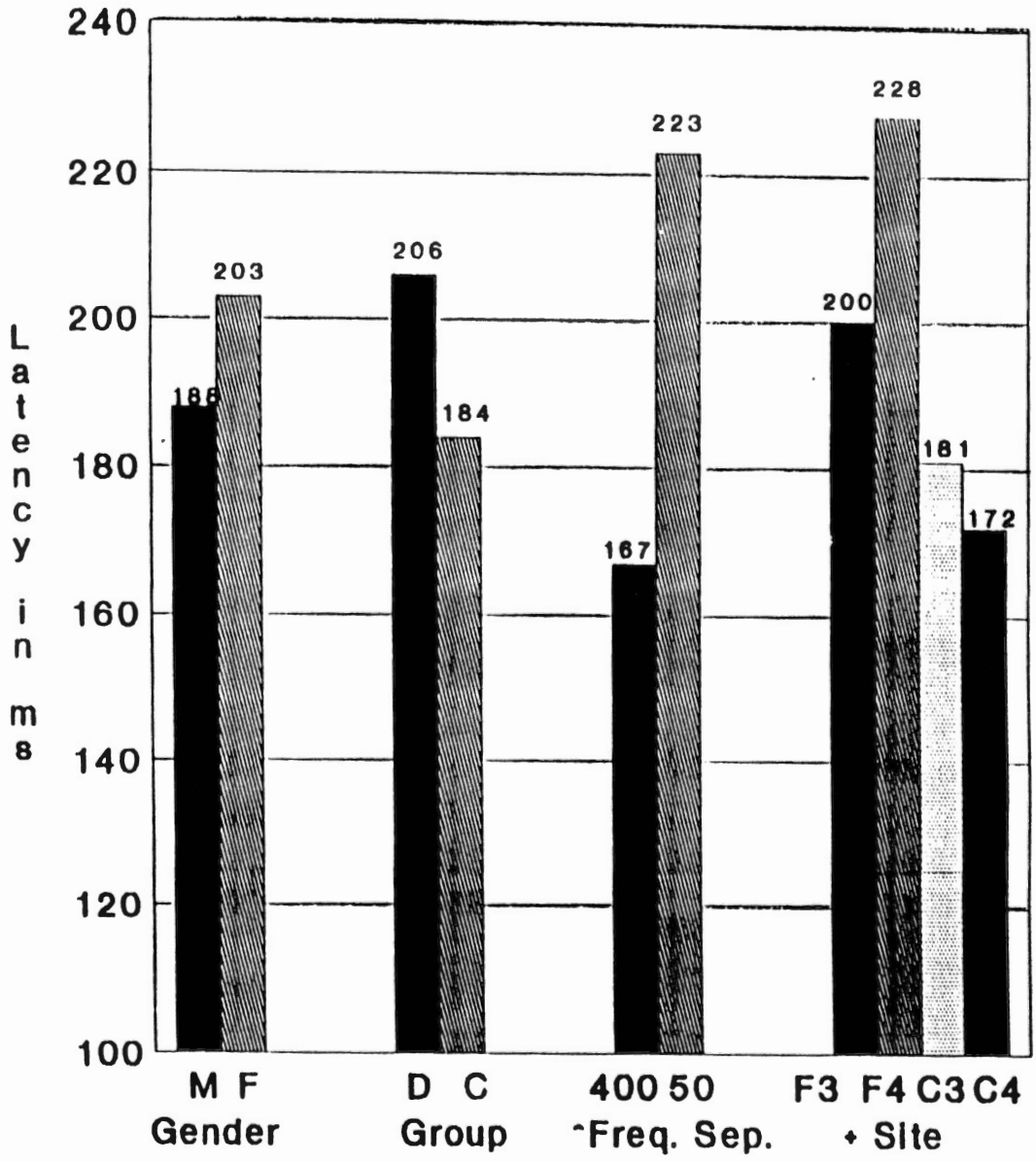
Figure 4. Main effects of Nd peak latency.



Note: • = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

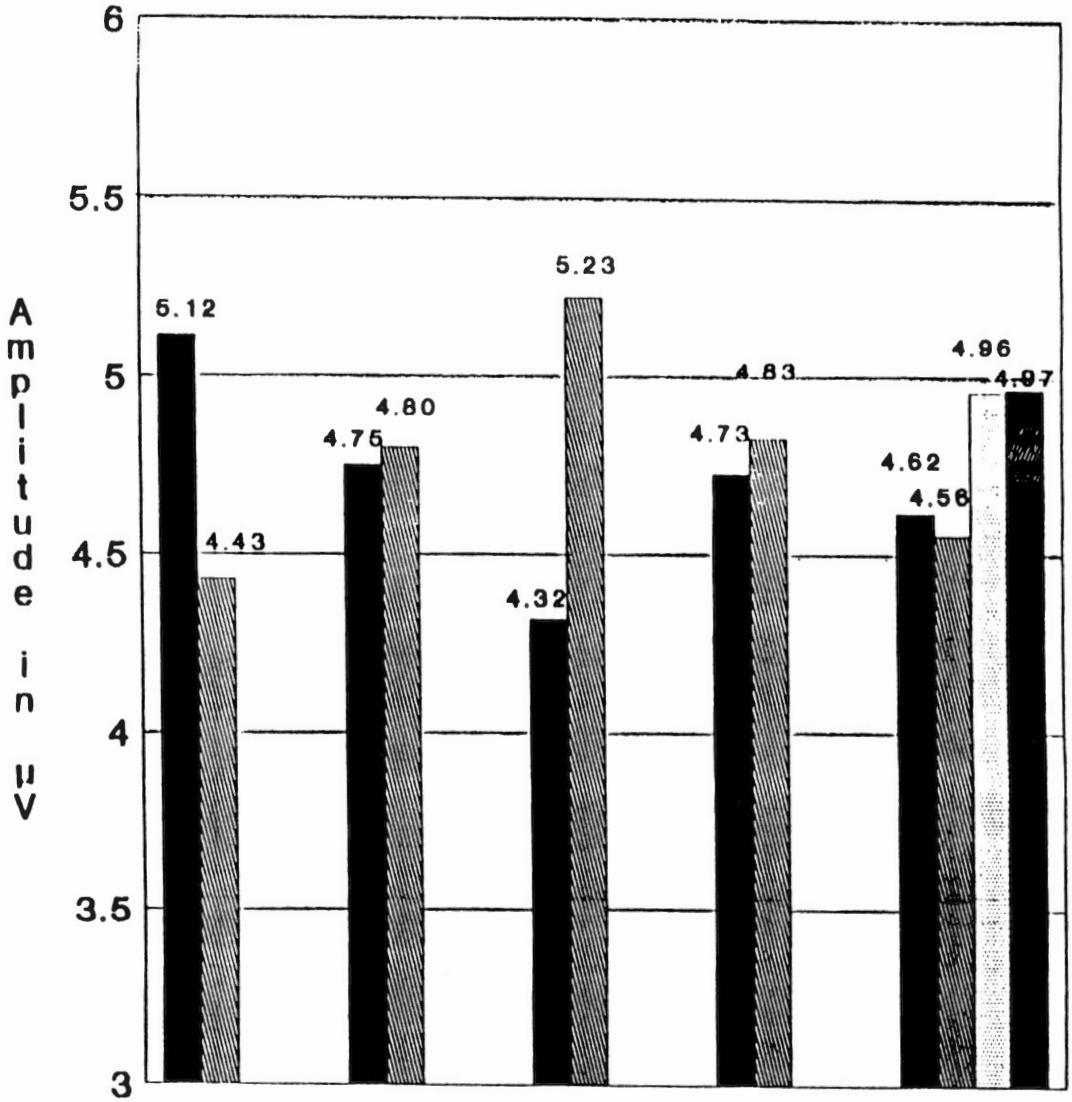
Figure 5. Main effects of Nd 25% latency.



Note: * = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

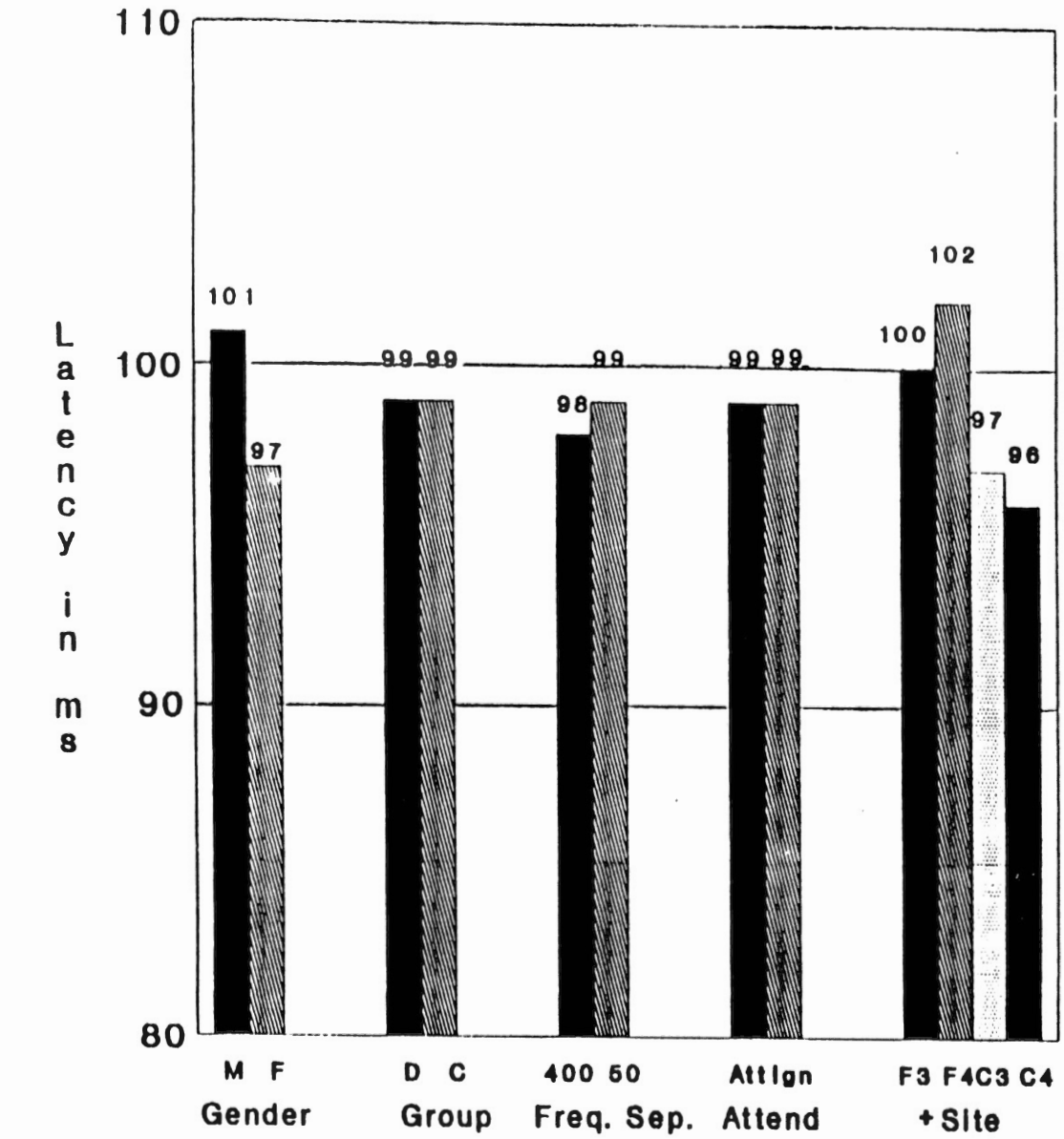
Figure 6. Main effects of N100 peak amplitude.



Note: • = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

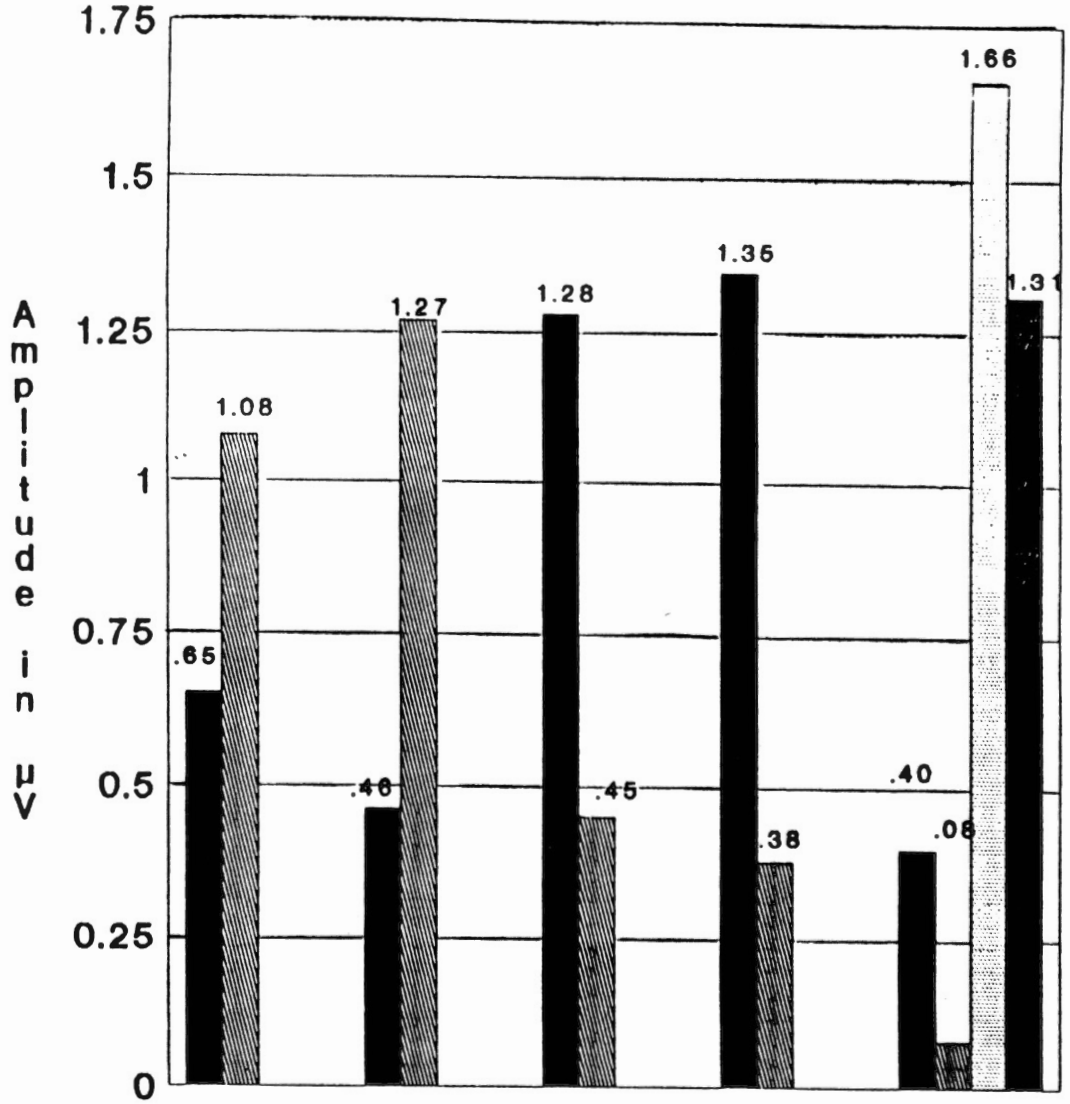
Figure 7. Main effects of N100 peak latency.



Note: • = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

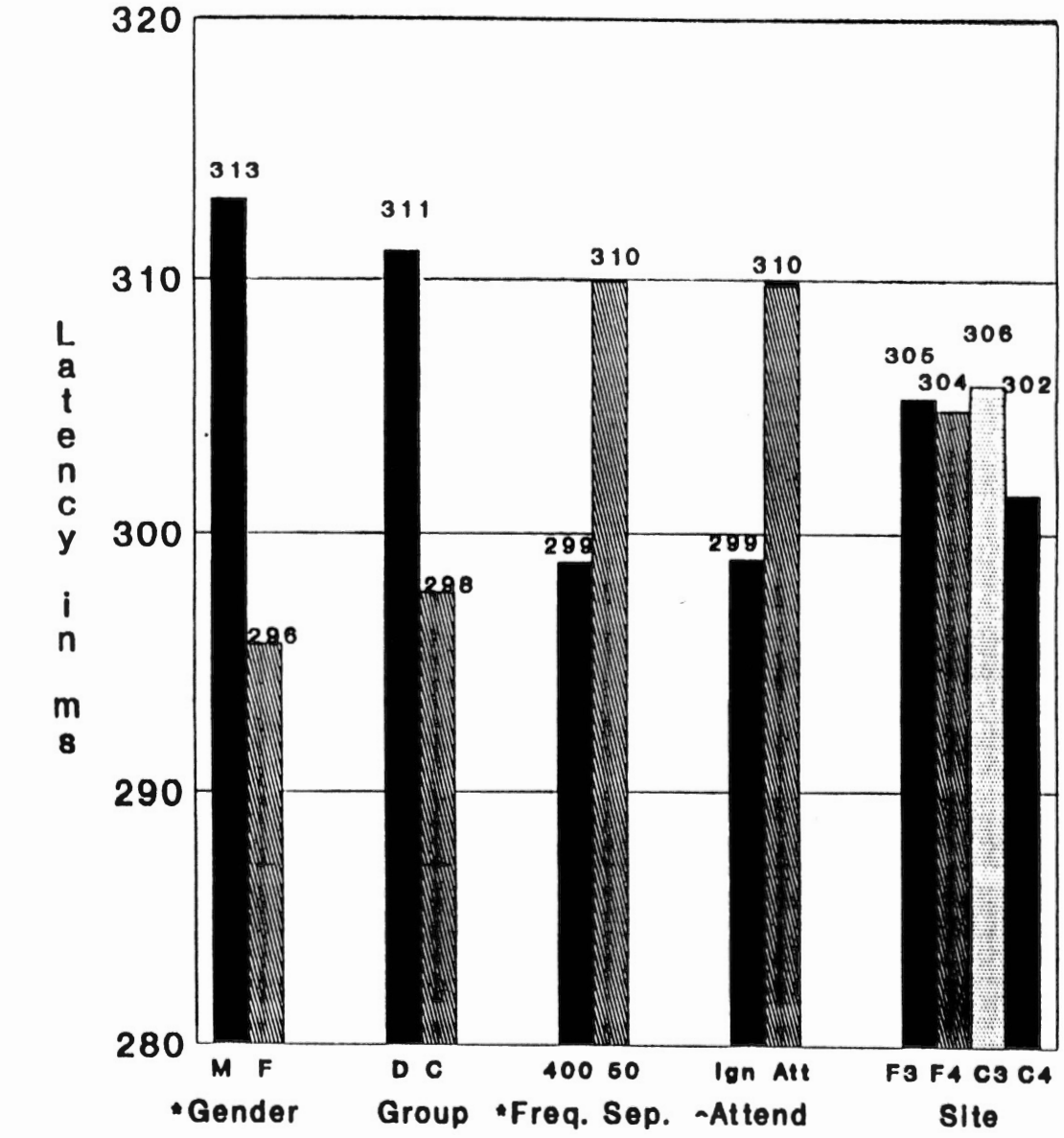
Figure 8. Main effects of P300 peak amplitude.



Note: * = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

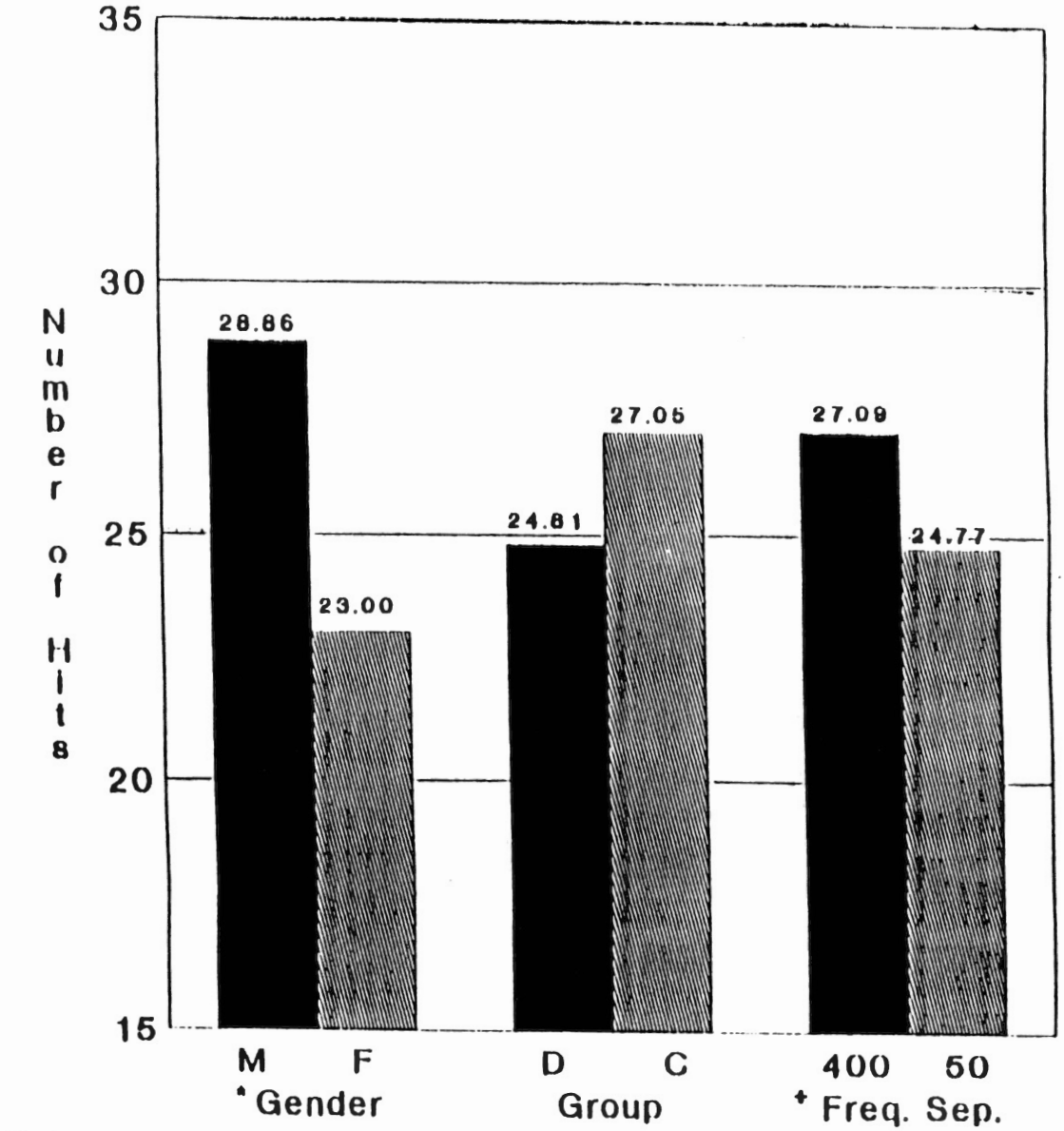
Figure 9. Main effects of P300 peak latency.



Note * = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

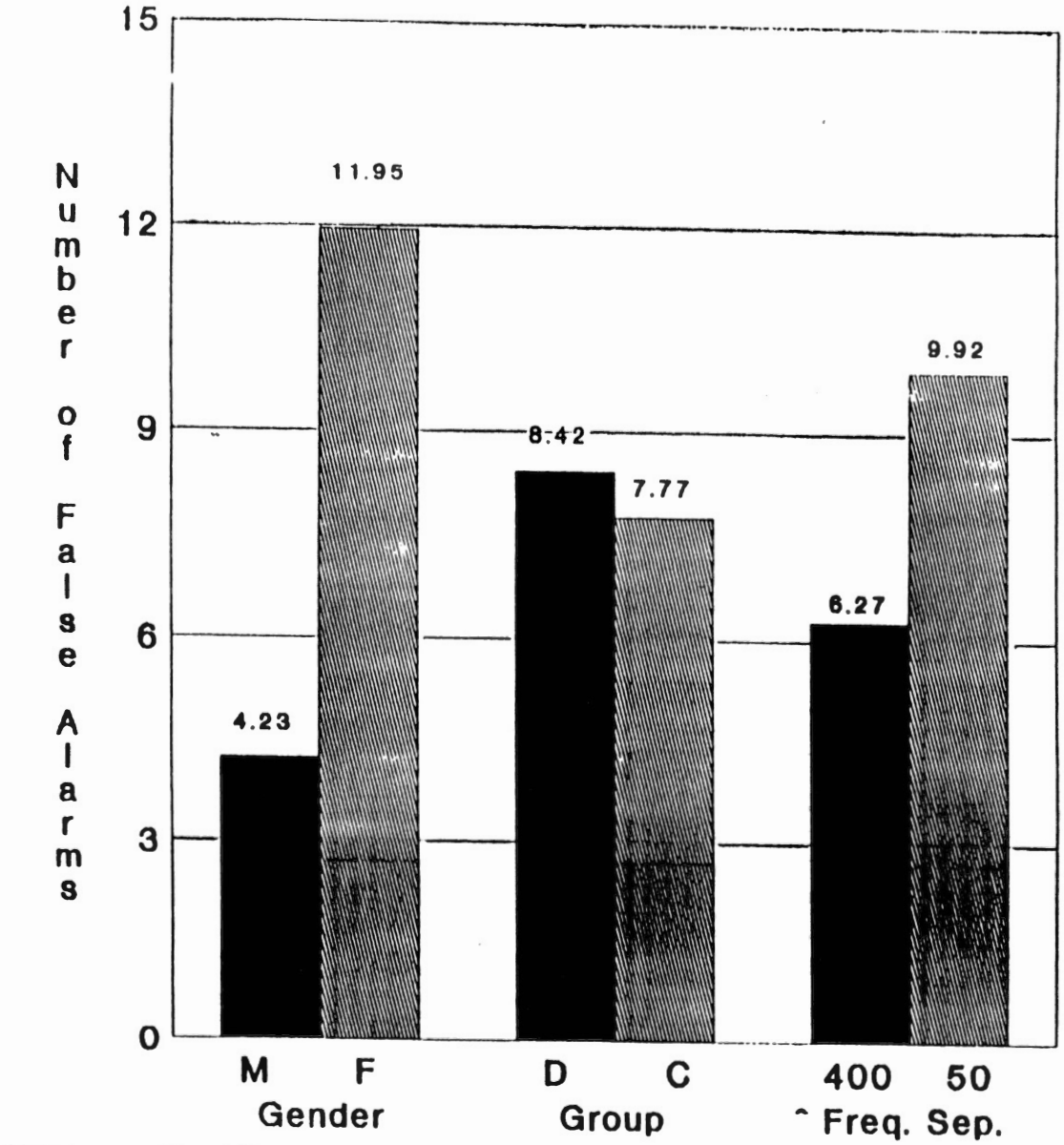
Figure 10. Main effects of the hit rate.



Note: * = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

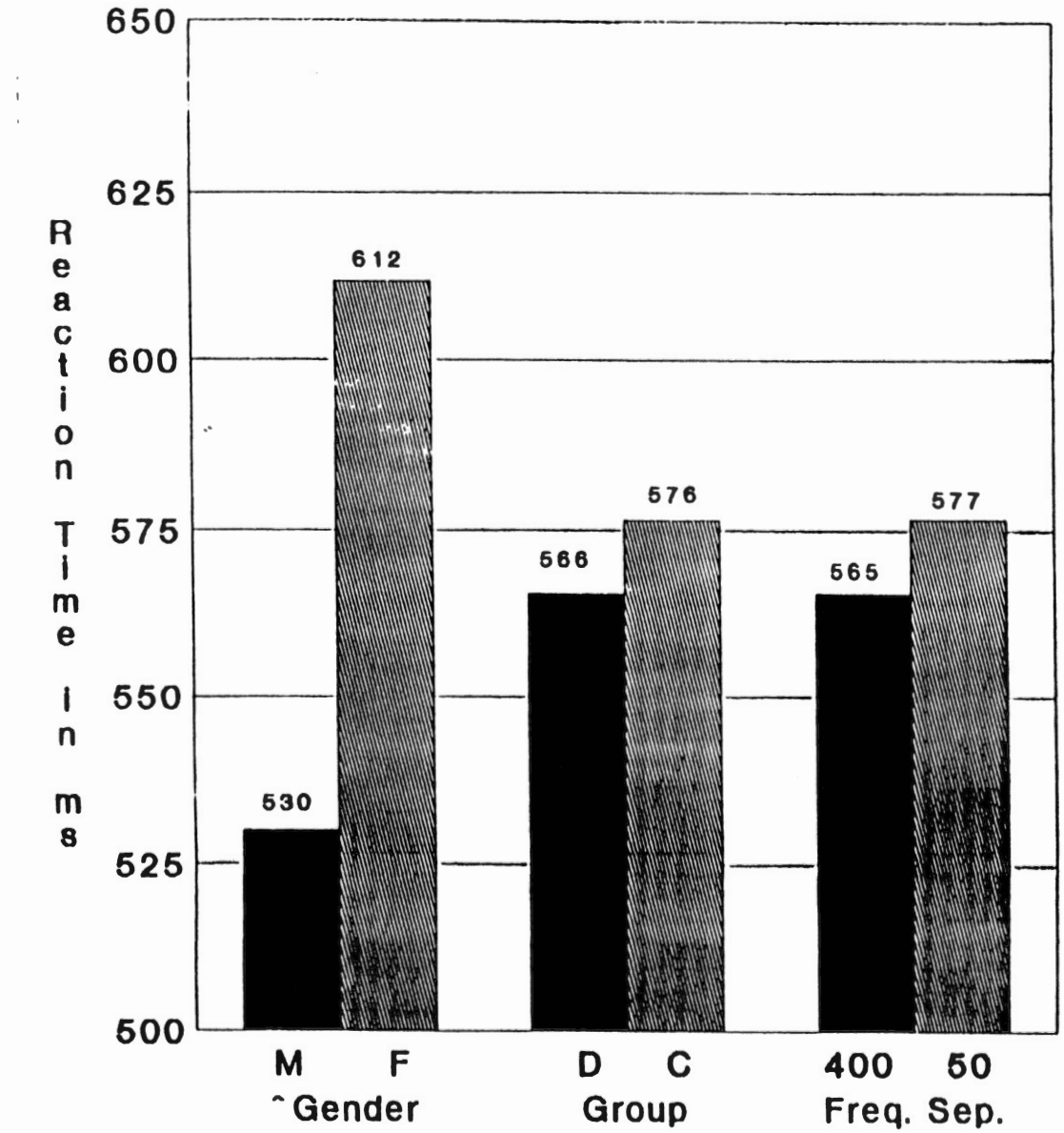
Figure 11. Main effects of the false alarm rate.



Note: • = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

FIGURE CAPTION

Figure 12. Main effects of reaction time (RT).



Note: • = Significance at 0.05
^ = Significance at 0.01
+ = Significance at 0.001

APPENDIX C

CONSENT FORM 1

I, _____ (name of participant), voluntarily consent to participate in the investigation of the auditory evoked potential correlates of emotional states, the purposes of which have been explained to me by Tom Misukanis, M.S. I thereby authorize Tom Misukanis, M.S. or associates or assistants of his choosing, to perform the following treatments or procedures:

I understand that the research requires measurement of my levels of emotion and that two separate instruments may be administered. I have been informed that my participation in the study is contingent on the results obtained in the initial screening questionnaire which will take approximately 10 minutes to complete. If I am asked to participate further, I understand that I will be administered another questionnaire assessing my levels of emotions, which will take approximately 15 minutes to complete. I have been informed that further participation in the study will then be contingent upon the results of this second screening device.

I understand that any data collected as a part of my participation in this experiment will be treated as

confidential and will receive a code number so that it will remain anonymous. In no case will any use be made of these data other than as research results. If data from my participation are ever displayed, my identity will remain anonymous.

I understand that although my participation will not be personally beneficial to me, I realize that the information gained may contribute to an understanding of the physiological basis of emotional states.

I understand that my participation is voluntary, that there is no penalty for refusal to participate, and that I am free to withdraw my consent and participation in this project at any time without penalty, after notifying the project director.

I may contact Dr. David Thomas, Psychology Department, 215 North Murray, Oklahoma State University, at telephone number (405) 744-7492 should I wish further information about the research. I may also contact Terry Macuila, University Research Services, 001 Life Sciences East, Oklahoma State University, Stillwater, OK 74078; Telephone: (405) 744-5700. Should any problems arise during the course of the study, I may take them to Dr. Vicki Green, Psychology Department

Head, 215 North Murray, Oklahoma State University, at telephone number (405) 744-6027.

I have read and fully understand this consent form. I sign it freely and voluntarily. I affirm that I am 18 years of age or older. A copy has been given to me.

Date:_____ Time:_____

Signed:_____

(signature of subject)

Signed:_____

(signature of project director)

APPENDIX D

CONSENT FORM 2

I, _____ (name of participant), voluntarily consent to participate in the investigation of the auditory evoked potential correlates of emotional states, the purposes of which have been explained to me by Tom Misukanis, M.S. I thereby authorize Tom Misukanis, M.S. or associates or assistants of his choosing, to perform the following treatments or procedures:

I understand that the research requires the measurement of my levels of my hearing by means of an audiometer. I have been informed that the risk of discomfort or adverse effects is at an absolute minimum and I have been given the opportunity to ask questions. I understand that this procedure will take approximately 20 minutes and that my further participation is also contingent on the results of the hearing assessment.

I understand that the research requires the measurement of the electroencephalogram using disk recording leads attached to the scalp using electrolyte cream. I have been informed that the risk of discomfort or adverse effects is at an absolute minimum and I have been given the opportunity to

ask questions. I understand that this procedure will take approximately 90 minutes in duration.

I understand that any data collected as a part of my participation in this experiment will be treated as confidential and will receive a code number so that it will remain anonymous. In no case will any use be made of these data other than as research results. If data from my participation are ever displayed, my identity will remain anonymous.

I understand that although my participation will not be personally beneficial to me, I realize that the information gained may contribute to an understanding of the physiological basis of emotional states.

I understand that my participation is voluntary, that there is no penalty for refusal to participate, and that I am free to withdraw my consent and participation in this project at any time without penalty, after notifying the project director.

I may contact Dr. David Thomas, Psychology Department, 215 North Murray, Oklahoma State University, at telephone number (405) 744-7492 should I wish further information about the research. I may also contact Terry Macuila, University

Research Services, 001 Life Sciences East, Oklahoma State University, Stillwater, OK 74078; Telephone: (405) 744-5700. Should any problems arise during the course of the study, I may take them to Dr. Vicki Green, Psychology Department Head, 215 North Murray, Oklahoma State University, at telephone number (405) 744-6027.

I have read and fully understand this consent form. I sign it freely and voluntarily. I affirm that I am 18 years of age or older. A copy has been given to me.

Date:_____ Time:_____

Signed:_____

(signature of subject)

Signed:_____

(signature of project director)

APPENDIX E

INSTRUCTIONS

"Thank you for your participation in this study. Today, we will be using an electroencephalograph, more commonly called an EEG, to look at the waveforms that your brain produces to certain tasks. The EEG measures the electrical activity produced by your brain. The machine does not introduce any electricity into your brain, thus you are in no danger to be shocked. Rather, it measures your electrical output. The EEG is not able to read your thoughts and will not harm you in any way. At this time, I will be measuring your head so that I can place electrodes on your scalp. I will be placing four electrodes on the front part of your head, one on your forehead, two around your left eye, and one on each ear lobe. A white cream is used to make a good connection between your skin and the electrode. I will first clean each of these areas to insure the best possible connection. Do you have any questions before we begin?

You will be listening to four sets of tones through these headphones. We will take short breaks between each set so that you will not become tired or bored. Each set of tones will contain high pitch tones and low pitch tones. In each condition, you will be asked to pay close attention to only one pitch level of the tones, either the high tones or

the low tones. In addition to the high and low tones, the tones will be of two different time lengths, short and slightly longer tones. Thus, in each condition, you will hear short and long low tones and short and long high tones. You are requested to listen carefully to the particular pitch of tones that I tell you and press your thumb switches as quickly as you can when you hear a longer tone of the particular pitch that I tell you. For instance, in this first condition, I want you to listen to the high (or low) tones, ignore the low (or high) tones, and press your thumb switches when you hear a slightly longer, high (or low) pitch tone.

Before we begin, I am going to give you some practice to familiarize you to the tones which you will hear. Let's start by presenting the tone pitch that you will focus on in the first condition. I will present five high (or low) pitch, short tones, followed by five high (or low) pitch, long tones. Then I will alternate between short and long, high (or low) pitch tones so you will be able to tell the difference in the time lengths (consequently subject is presented the tones).

Next, you will hear the low (or high) tones which you are instructed to ignore in this first condition. You will first hear five short, low (or high) tones, followed by five longer, low (or high) tones, followed by five pairs which

will alternate between short and long, low (or high) tones (consequently, subject is presented the tones).

At this point, is the task clear to you? Do you have any questions? I will now present you with a practice condition. Whereas the conditions in the experiment will take about five minutes, this practice condition will take about a minute and a half. Remember, you are to listen to the high (or low) tones, ignore the low (or high) tones, and press your thumb switches as quickly as you can when you perceive a slightly longer, high (or low) pitch tone. In addition, I will ask that throughout the experimental conditions, you keep your head and eyes as still as possible. Do you have any questions?" Begin the first condition.

Following each condition, instructions were given to prepare the subject for the next condition. These directions were: "Now, in this condition I would like you to listen to the low (or high) tones, ignore the high (or low) tones, and press your thumb switches to the slightly longer, low (or high) pitch tones. Before we begin, would you like to hear the sample tones again?"

The experiment then proceeded through the four conditions. Following the final condition, each subject was debriefed as the electrodes were cleaned off of their scalp. Debriefing instructions were as follows: "Thank you again for your participation in this study. What I am attempting

to investigate is the relationship between depression and attention. It is generally accepted that people who are feeling depressed have less attentional abilities. I am attempting to utilize a physiological measure, the EEG, to see if there is evidence for this belief. You were assessed several weeks ago in your psychology class for your level of depression. These measures indicated that you are (or, are not) presently experiencing depressive symptoms. I want to see if your EEG measures will be different from others who are not (or, are) experiencing depressive symptoms. Please keep in mind that all information utilized in this study is confidential." Subjects who reported depressive symptoms on the Zung SDS and BDI were also given a referral to local counseling services if they desired.

VITA

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THESIS: THE EFFECTS OF DEPRESSION ON SELECTIVE ATTENTION AND LATE COGNITIVE PROCESSES

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