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THE EFFECT OF ALPHA UPON THE P300

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THE EFFECT OF ALPHA UPON THE P300

by

PAUL JASIUKAITIS

A dissertation submitted to the Graduate Faculty in
Psychology in partial fulfillment of the requirements
for the degree of Doctor of Philosophy, The City
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1987

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

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date

Gad Hakerem Ph.D.
Chairman of Examining Committee

March 2 1987
date

Robert D. B. [Signature]
Executive Officer

Prof. Gad Hakerem

Prof. James Tweedy

Prof. John Antrobus

Supervisory Committee

The City University of New York

Abstract

THE EFFECT OF ALPHA UPON THE P300

by

Paul Jasiukaitis

Adviser: Professor Gad Hakeren

The overlap between alpha-blocking and P300-eliciting stimulus sets is considerable. Both phenomena require novel, target or salient stimulation and are not found upon stimuli which fail to engage the subject's attention. We had noticed that the blocking of EEG alpha spindles by auditory stimulation was often accompanied by a sharp positive shift in the EEG baseline. We hypothesized that this positive shift is a facet of alpha-blocking and that it might be sufficiently time-locked after stimulus presentation to contribute to, or even be responsible for, the formation of the P300.

The experiments described below are what we call alpha sorts. Trials on which highly discrepant 'oddball' stimuli were presented, were sorted into two bins on the basis of pre-stimulus alpha band RMS magnitude. The trial bins were then separately averaged to produce a 'high alpha' evoked potential and a 'low alpha' evoked potential for each subject. The rare stimulus trials were embedded within a near-threshold auditory intensity discrimination task. The purpose of this was to constrain

the subject's EEG to frequencies above delta/theta and to prevent fluctuations in wakefulness from contributing to effects upon the evoked potential.

Study 1 found that larger amplitude P300s were produced by selectively averaging for high pre-stimulus alpha. No effect of alpha was found upon the N100. These findings established specificity of the alpha effect for the P300 component. Study 2 employed the extra factors of direction of stimulus intensity change (increases and decreases) and separate trial sorting based on alpha RMS both pre-stimulus and post-stimulus. The purpose of the post-stimulus sort was to determine whether it was pre-stimulus alpha alone which enhanced the P300 or pre-stimulus alpha in conjunction with low alpha post-stimulus, i.e. alpha-blocking. It was found that P300 enhancement by pre-stimulus alpha did not interact with stimulus intensity and appeared to be independent of alpha post-stimulus. The data are discussed in terms of cascaded inhibition from the mesencephalic reticular formation to nucleus reticularis of the thalamus to a thalamo-cortical system responsible for both alpha and the P300.

"And this is all my creating and striving, that I create and carry together into One what is fragment and riddle and dreadful accident. And how could I bear to be a man if man were not also a creator and guesser of riddles and redeemer of accidents?"

- Friedrich Nietzsche,
Thus Spoke Zarathustra

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INTRODUCTION

Event related potentials (ERPs) have become a major focus of human psychophysiological research. It is believed that they reflect the massed electrical activity of neuronal populations beneath the skull and therefore offer a window upon the intact and normally functioning human brain. ERPs are obtained from ensembles of scalp-recorded voltage fluctuations time-locked to an eliciting stimulus or event. Because of this derivation, it might be expected that ERPs should bear some systematic relationship to the ongoing record of human electrical cerebral activity known as the electroencephalograph or EEG. The alpha rhythm, a waxing and waning 10/sec sinusoid, was the first prominent feature of the EEG described by Berger in 1930. The study of stimuli or events which induced its abrupt cessation (alpha blocking) constituted an important topic in psychophysiology up until the 1950s. Given the overlap between the stimulus sets used to elicit alpha blocking and those now prevalent in cognitive ERP research (i.e., salient, surprising or informative stimuli), it would be worthwhile to consider a relationship between classical EEG phenomena and the more recently discovered ERPs. Indeed, such an observation has already been provided in the early EEG literature. At the 1936 Cold Spring Harbor Symposium on Quantitative Biology, Herbert Jasper presented a paper on the human EEG in which he notes;

The most obvious effect of the light stimulus is the complete blocking of the occipital alpha rhythm from this region, but we note that this blocking is usually associated with a marked slow positive swing during the first part of the blocked period. (p. 320)

Further on in the same paper;

...the magnitude and duration of the slow positive wave is a function of the intensity of the light stimulation. There are some prolonged blocked periods following a brief light stimulation which are apparently due to uncontrolled additional stimuli within the subject which complicate the effect of the light itself (very marked with the "attempt to see" a dim light). (p. 323)

What Jasper (1936) had described was an endogenously generated late positive event related potential. Interesting as the Jasper (1936) paper is, there is no mention of it in the contemporary ERP literature. This may be due in part to the advent of signal averaging techniques which have induced a view of the unaveraged EEG as noise from which the data must be extracted. Although there are contemporary explanations of ERP phenomena based on pre-stimulus states (Karlin, 1970; Naatanen, 1975), they identify the pre-stimulus states with signal averaged waveforms such as the CNV or the readiness potential. They do not consider that

the evidence for some pre-stimulus states might disappear as a consequence of the averaging process itself. The value of investigation into EEG/ERP relationships is that certain questions about ERPs, such as their physiological function, might well remain unanswerable if ERPs are only regarded in isolation from their electrophysiological environment. This environment is the background EEG. To paraphrase thermodynamics, 'isolated systems run down'. Conceptualization about ERPs has taken a drearily recursive and self-defining trend since the excitement of their original discovery (Sutton, 1979). If a link can be established between ERPs and the background EEG, new models of both could develop from the junction of their respective data bases.

Alpha Blocking

Shortly after the discovery of the alpha rhythm and its blocking by external stimulation, controversy arose over whether alpha blocking was an endogenous event. Adrian and Matthews (1934) found that alpha blocking failed after the first few eye openings in a dark room or in a field of featureless uniform illumination. These investigators proposed that pattern vision induced desynchronization among occipital lobe neurons and that alpha blocking was more the result of visual input than

general attention. If pattern vision was absent the rhythm would persist in spite of other attention demanding activities, such as listening to a sound, gripping a pair of pliers, etc. However, Loomis, Harvey and Hobart (1936) found that the onset of near threshold illumination could effectively abolish alpha if the subject was attempting to detect the onset. They believed that alpha blocking was caused by the "effort to see" rather than visual input per se. Bagchi (1937) suggested that it was the "attempt to see" or the expectation of seeing, rather than pattern vision, which was responsible for alpha blocking. Eye opening itself could become associated with the experience of patterned vision through conditioning, thus producing alpha blocking as a conditioned response. Bagchi (1937) likened the failure of alpha blocking upon repeated eye openings in an unchanged visual field to the extinction of a conditioned response.

Berger (1930) first noticed that stimulus repetition usually reduced or abolished the alpha blocking response. Jasper, Cruikshank and Howard (1935) and Bagchi (1937) found that auditory or somatosensory stimulation which initially suppressed alpha would fail to do so after several presentations. Visual stimulation, however, would remain an effective suppressor over repeated presentations. Jasper et al. (1935) proposed that although alpha blocking may be intrinsically sensitive to visual input, it can

also be triggered by activity in a generalized, modality non-specific arousal system.

Not only does a distinction exist between generalized (non-visual) and specific (visual) alpha blocking responses with regard to rate of extinction, but also a further distinction can be made along the dimension of scalp topography. Gastaut (1954) and Sokolov (1963) claimed that the initial suppression of alpha rhythm upon visual stimulation can be found over both occipital and central scalp areas. With repetition of the visual stimulus, alpha will cease to be affected over the central area yet continue to block over the occipital area. The initial reaction of alpha to visual input over both occipital and central areas would indicate a generalized reaction while the persisting response in the occipital area would be a modality specific reaction. Likewise with somatosensory stimulation, a non-extinguishing localized response was found over the central scalp. Gastaut (1954) believed that activity in the 10 Hz range over the somatosensory cortex was independent from alpha and bears the same relationship to the somatosensory afferent system as alpha does to the visual system. Gastaut termed this activity the "mu" rhythm. Later, Gastaut (1958) hypothesized that the generalized alpha blocking response is mediated by the diffusely projecting mesencephalic reticular system while the more persistent localized types of blocking are mediated

by the thalamic reticular system which has segregated projections to the primary cortical sensory receiving areas.

Another controversy about alpha blocking centers around the question of what it represents on the neuronal level. Adrian and Matthews (1934) were the first to suggest that alpha blocking indicated desynchronization. Neuronal activities continue unabated, however the majority of neurons are out of step with each other thus reducing the amplitude of the gross summated signal that is the scalp recorded EEG. Jasper (1936) brought up the possibility that alpha blocking might actually represent the suppression of graded potential activity at the level of the individual neuron. He recorded differentially from electrodes 40 microns apart on the surface of feline cortex. In the record derived from these electrodes, stimulation would typically produce arrest of rhythmic activity. Given that the differential record was obtained from electrodes separated by a magnitude equivalent to single neuronal elements, Jasper argued that neuronal synchrony should produce equipotentiality between the electrodes and hence a reduced amplitude in the differential record. Desynchronization between single neuronal elements should produce, if anything, an increase in the potential difference of electrodes spaced 40 microns apart. Yet the same picture was obtained on the microanatomical level as in the gross

scalp recorded EEG. As mentioned previously, Jasper (1936) observed that a slow post-stimulus positivity in the human EEG was associated with a complete dropping out of the alpha rhythm. He also noticed that when this positive wave was followed by a slow negative wave, alpha remained blocked until the peak of the negative wave and then returned only after the baseline EEG had reached its pre-stimulus level. Jasper (1936) concluded that rhythmic activity is permitted only between certain cortical tissue polarization limits. Drawing on an analogy with the mammalian myelinated axon, wherein sustained negativity builds up during high frequency stimulation and is followed by a long positivity during a refractory period of reduced excitability, Jasper proposed that there are two mechanisms which can produce alpha blocking. One he called cathodal block which will appear as a cortical surface negativity and is due to increased excitation and neuronal desynchronization. The other is anodal depression which represents physiological inhibition of individual neurons and will appear as cortical surface positivity.

Alpha and the CNV

The CNV is a sustained negative shift in the EEG baseline that appears when a subject prepares to respond to an imperative stimulus (Walter, Cooper, Aldridge, McCallum and Winter, 1964). This preparation may be for a motor response to the imperative stimulus or even just

to receive information from the stimulus event. The CNV is optimized if the imperative stimulus is signaled by a warning stimulus that occurs around 1 sec earlier. In this case the CNV is thought to be a summed composite of an orienting potential ("O Wave") that occurs after the warning stimulus (S1) and an expectancy potential ("E Wave") that develops prior to the imperative stimulus (S2) (Loveless, 1976). If the contingency between S1 and S2 is removed while S1 still possesses signal value, the CNV will be reduced to a long latency slow negative O Wave in response to S1 (Rohrbaugh, Syndulko, Sanquist and Lindsley, 1980). The E Wave immediately prior to S2 reveals a time course similar to that of the readiness potential preceding voluntary motor action (Kornhuber and Deecke, 1965). Whether this terminal portion of the CNV can represent cognitive or sensory preparation aside from pure motoric readiness is currently under debate (Rohrbaugh and Gaillard, 1983; Ruchkin, Sutton, Mahaffey and Glaser, 1986).

Intracerebral recordings in humans have found that sustained cortical negativities are also represented subcortically in the mesencephalon (McCallum, Papakostopoulis and Griffith, 1976). This could indicate that the CNV is concomitant with activation mediated by the mesencephalic reticular formation. Given that stimulation of the mesencephalon in the cat produces arrest of cortical rhythmicity (Moruzzi and Magoun, 1949), the human

intracerebral work would suggest that there should be a negative correlation between CNV amplitude and the alpha rhythm.

Pfurtscheller and Aranibar (1976) obtained spectral power estimates of EEG activity during the interval between S1 and S2 in a CNV paradigm. Three levels of task complexity were used. In the first, the subject was not required to respond to S2 and passively awaited its occurrence. In the second, the subject responded to S2 whether it was an auditory or a visual stimulus. Finally, the subject would respond to S2 if it was a visual stimulus but not if it was auditory. CNV amplitude increased with experimental complexity, however the magnitude of decrease in the power of the alpha band (7-13 Hz) during the S1-S2 interval did not. Alpha power decreased after S1 to the same extent in all three task conditions. Across subjects, alpha power displayed marked variability in the interval immediately preceding S2. In one individual alpha power actually increased above their pre-S1 baseline amount during the period prior to S2.

Grunewald, Grunewald-Zuberbier and Netz (1980) had subjects respond to S2 with either slow ("ramplike") or fast ("ballistic") movements. Spectral power in the alpha band (8-12 Hz) during the S1-S2 interval was significantly lower for the fast movement condition at frontal, central, parietal and occipital electrodes. This difference between movement conditions was most pronounced in the

middle of the S1-S2 interval, tending to disappear by the time of S2. CNVs were identical between movement conditions except at Cz, where the fast movement condition was associated with a larger CNV. Unlike the alpha power difference, the CNV difference at Cz tended to increase towards the time of S2 presentation.

Denoth, Zappoli, Navona and Ragazzoni (1984) ran subjects in S1-S2 paradigms with eyes open and closed. Over blocks, S1 and S2 were in either the visual or auditory modalities. In all conditions they found that the maximum alpha power decrease occurred around 500 msec post-S1. This initial alpha decrement was followed in the remaining part of the S1-S2 interval by marked intra- and intersubject variability. In most cases alpha power returned to the pre-S1 level but occasionally an increase ("hypersynchronization") above that level occurred prior to S2. CNVs were identical over all conditions in the first half of the S1-S2 interval. In the second half, visual modality CNVs tended to be larger than those found in the auditory tasks.

In light of the above work, it seems that alpha activity is maximally suppressed in the CNV paradigm at a point roughly 500 msec after the occurrence of S1. This corresponds with the latency of the first CNV sub-component or O Wave. The lack of a consistent mode of alpha response immediately prior to S2 suggests that

alpha abundance is independent of the E Wave. It would appear that it is the O Wave subcomponent of the CNV which is associated with EEG desynchronization and alpha suppression. If the O Wave and alpha suppression may both be considered indicators of generalized arousal, their association at 500 msec post-S1 in the CNV paradigm could reflect the alerting action of that stimulus. The lack of consistent alpha suppression prior to S2 indicates that the generalized arousal or alerting mechanism does not necessarily remain engaged as the specific response set is being prepared.

Before concluding this section a group of studies should be considered which, although they do not address the question directly, shed some light on the relationship between alpha, CNV and P300. Thompson and Obrist (1964) and Thompson and Thompson (1965) noted a general increase in low voltage fast activity (beta) and a corresponding decrease in alpha in the human EEG during the early stages of a serial order learning task. During overlearning, there was a tendency for both alpha and beta to return to control levels. These authors concluded that learning is accompanied by decreased arousal levels as reflected by the return in alpha. Peters, Billinger and Knott (1977) recorded ERPs during a consonant-vowel-consonant paired associate task. They looked at changes in CNV and P300 amplitude as a function of im-

proved associate recall over trial blocks. At the beginning, CNV between the cue CVC and the recall prompt was large and correct recall was 3 to 25%. As subjects approached 86% recall, the CNV disappeared and a large P300 to the cue CVC had developed. These investigators suggested that the CNV in the early part of the task indicated arousal while the appearance of a P300 in the later part reflected the subject's increasing confidence that they "knew" the associate CVC as soon as the cue appeared. If the above study is taken in conjunction with the Thompson and Obrist work, it might be said that CNV and sparse alpha will be found upon tonic arousal. Returning alpha abundance and the P300 will appear associated when processing becomes more phasic and delimited to discrete periods around stimulus events.

Questions About the Averaging Process

It has been proposed that signal extraction is not the actual action that is taking place during the averaging process. Sayers, Beagley and Henshall (1974) theorized that an effective stimulus acts mainly to phase-constrain spontaneous EEG activity. Thus the evoked response is formed from various ongoing harmonic components of the spontaneous EEG which are re-aligned in a consistent manner for a limited period of time after every stimulus presentation. Sayers et al. (1974) obtained power spectra from epochs before and after an

auditory evoked response. Power spectra of the post-stimulus epoch did not differ significantly from power spectra of the immediate pre-stimulus period. Hence a stimulus that evokes a cortical ERP does not do so by addition of a new spectral component to the EEG. Also there were no harmonic amplitude differences between the spectra of evoked responses to high intensity auditory stimulation and spectra of the EEG following ineffective low intensity stimulation. There were marked differences in the distribution of phase spectral values between post-stimulus epochs according to stimulus intensity. For their final demonstration, Sayers et al. (1974) imposed the phase-spectral properties of high stimulus intensity evoked responses on arbitrarily selected segments from a subject's spontaneous EEG. The 'manufactured' waveform of the average phase-modified spontaneous EEG closely resembled that of the subject's averaged evoked response to auditory stimulation.

Parvin, Torres and Johnson (1980) compared the additive response model of ERP generation to what they called a spontaneous EEG response model. The additive response model assumes that a constant response is added to the EEG after each stimulus presentation. The spontaneous EEG response model works through a re-ordering of ongoing EEG activity induced by stimulation in much the same way as proposed by Sayers et al. (1974). In ad-

dition, Parvin et al. (1980) imply that their spontaneous EEG response model may include amplitude changes in ongoing activity along with phase re-organization, the basic distinction between additive and spontaneous models still being that what is modulated or re-ordered in the latter case are pre-existing frequencies in the EEG. A consequence of this widened definition is that signal extraction becomes a special case of re-ordered spontaneous activity. If changes in amplitude or phase of the ongoing EEG are restricted to a particular family of frequencies, other frequencies will remain in random phase to the stimulus and will be averaged out. Parvin et al. (1980) performed digital filtering of single trial evoked response epochs into three bandwidths, 1-7 Hz (delta/theta), 7-14 Hz (alpha) and 14-25 Hz (beta). The filtered epochs were separately averaged to obtain evoked potentials representing simultaneous responses within the three major EEG bands. The 1-7 Hz band always had a positive wave occurring after the stimulus. This wave varied in its latency, with the peak usually occurring 150-300 msec post-stimulus. The 7-14 Hz band was more variable than the 1-7 Hz band. The time at which its maximum activity occurred could be from shortly after the stimulus to late in the epoch. There appeared to be little important activity in the 14-25 Hz band. The investigators also obtained plots of average power

for the 1-7 Hz and 7-14 Hz bands over the post-stimulus epoch duration. Power in the 1-7 Hz band increased up until 300 msec and then subsided. In the 7-14 Hz band power decreased after stimulus presentation, mirroring the increase in low frequency power. Parvin et al. (1980) argued, as did Sayers et al. (1974), that for the additive model to be appropriate there should always be an increase in epoch power after the stimulus. Comparing the differences in average epoch power before and after stimulation, the more recent authors suggested that the low frequency (delta/theta) band obeys an additive model while the middle frequency (alpha) band responds with phase re-organization.

Even more recently, Basar, Basar-Eroglu, Rosen and Schutt (1984) averaged stimulus omission responses filtered into separate bandwidths similar to what Parvin et al. did for regular evoked responses. Basar et al. (1984) found that the stimulus omission response in some subjects was characterized by a phase re-ordering and amplitude enhancement of harmonics in the delta/theta range. They proposed that low frequency EEG activity is entrained by the application of repetitive stimuli and that this activity manifests as an endogenous response upon stimulus omission. To emphasize its pre-stimulus aspect, they labeled the entrained low frequency harmonic "-P300". Basar et al. (1984) mentioned that the subjects who had

shown delta/theta enhancement upon stimulus omission were the same subjects who could be characterized by an abundance of alpha. Alpha activity for these subjects was diminished at the point of stimulus omission. In contrast, the low alpha subjects displayed alpha enhancement upon stimulus omission with little or no change in low frequency power.

The functions of power over time presented by Parvin et al. (1980) and the reciprocal relationship between post-stimulus delta/theta and alpha power mentioned by Basar et al. (1984) are exactly what Jasper (1936) had described some 40 years earlier. Parvin et al. (1980) found a post-stimulus increase in delta/theta power coincident with a decrease in alpha power. Basar et al. (1984) found an increase in delta/theta power after a salient event (stimulus omission) if the subject's pre-event EEG could be characterized by abundant alpha power. Again, in this situation, the post-event delta/theta appearance is associated with alpha disappearance. Jasper (1936) observed in the ongoing EEG that alpha blocking was associated with a slow positive wave. In terms of spectral power, the slow positive wave would manifest as an increase in post-stimulus delta/theta power.

In our own laboratory we had averaged small numbers of trials in order to study the evoked potential

correlates of the orienting response. While looking at single trial evoked responses in the EEG, we noticed the phenomenon exemplified by the waveforms presented in Figure 1. This figure shows a single trial response of the background EEG to a 100 msec duration 1000 Hz tone pip. The tone pip was of a rare (10%) 90 dB intensity in a sequence of (90%) 80 dB tones. As can be seen, the stimulus was preceded for about 500 msec by a 20-40 uV alpha spindle. The alpha activity is blocked after the last positive phase of the alpha spindle around 300 msec post-stimulus. The last two cycles of the alpha rhythm also seem to ride on a slow positive shift. We hypothesized that this positive shift is a facet of alpha blocking, as originally suggested by Jasper (1936), and that it might be sufficiently time-locked after stimulus presentation to contribute to, or even be responsible for, the formation of the P300.

Rationale

The overlap between alpha-blocking and P300-eliciting stimulus sets is considerable. Both phenomena require novel, target, orienting or salient stimulation and are not found upon stimuli that fail to engage the subject's attention. Given our observations of single trial ERP data and the suggestion of a unique relationship between pre-stimulus alpha and a post-stimulus positive slow wave

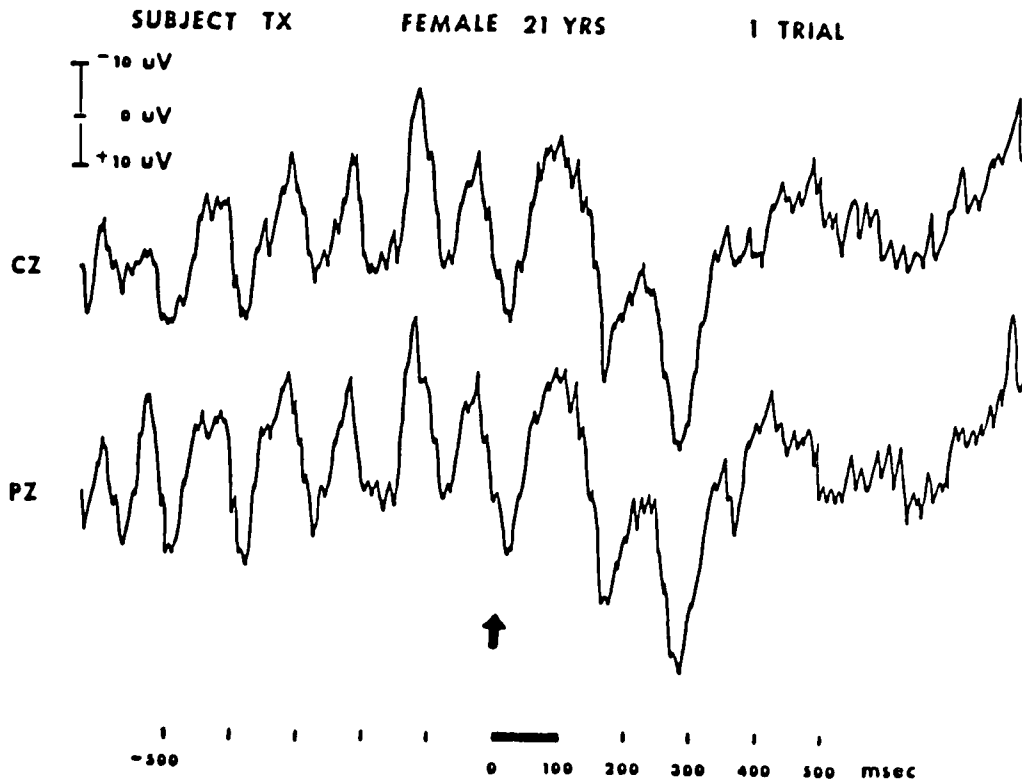


Figure 1. Single trial auditory evoked response to a deviant tone stimulus. Prominent alpha activity in both electrodes precedes the stimulus. Desynchronization follows after the last positive phase at 300 msec post-stimulus. Negative is up in this and all other waveform tracings.

from the above literature, we decided to conduct a preliminary investigation into the effect of abundant pre-stimulus alpha activity upon the P300 elicited by a rarely occurring stimulus. We must emphasize that this is not a "central intermittency" hypothesis (Harter, 1969, for review) purporting that the alternating negative and positive phases of the alpha rhythm represent a cortical shutter of excitatory and inhibitory states. The presence of alpha represents one state while its absence indicates another. Within the 10 per sec oscillation of the alpha rhythm the former state is resident, unqualified by the negativity or positivity of the individual waves. Nor is our hypothesis a phase re-ordering of background activity as described by Sayers, Beagley and Henshall (1974) or Basar, Basar-Eroglu, Rosen and Schutt (1984). The post-stimulus slow positivity is not in the same frequency range as alpha. As indicated by our single trial EEG data (Figure 1.), the slow positivity is associated with abrupt cessation of the alpha rhythm and cannot be considered a continuation of phase re-ordered alpha activity.

The experiment described below is what we call an alpha sort. Trials which are likely to evoke a P300 are sorted into two bins on the basis of the amount of pre-stimulus alpha band RMS. The trial bins are then separately averaged to produce a "high pre-stimulus alpha"

evoked potential and a "low pre-stimulus alpha" evoked potential for each subject. To evoke the P300, highly discrepant intensity changes were presented during a near-threshold intensity difference discrimination. It was hoped that these intensity changes would evoke a surprise reaction in the subject and thus a P300 (Duncan-Johnson and Donchin, 1977), while the near-threshold intensity discrimination would keep the subject sufficiently activated to constrain the background EEG to the alpha and higher frequency bands during the experiment. Our hypothesis assumes that it is a fairly abrupt termination of an alpha spindle that leads to a positive shift in the EEG (Figure 1.). We therefore chose to limit the period of pre-stimulus alpha RMS assessment to 500 msec in order to be sure that any high RMS value could not reflect an alpha spindle waning of its own accord.

STUDY 1

Method

Subjects. The subjects were seven volunteer graduate students from Queens College in Flushing, New York. They were four women and three men with a median age of 29 years. They had participated in EEG experiments before. Six of the seven were known to have well developed alpha rhythms in the eyes-open state.

Procedure. Each subject sat in a chair while wearing headphones through which binaurally simultaneous tone pips of 100 msec duration were presented. The tone pips were generated by a Hewlet Packard 200 CDR audio-oscillator and were of a constant 1000 Hz frequency. Rise time for tone onset and fall time for tone offset were both .25 msec. These stimuli were gated through switches under the control of a Heathkit H8 micro-processor and presented in pairs separated by 1.5 sec. Subjects were instructed to respond by differential key press whether the second stimulus (S2) of each pair was of the same intensity as the first (S1) or slightly louder. They were allowed to use their preferred hand and were told that response speed was not important while accuracy was. An LED four feet in front of the subject served as a visual fixation point when illuminated. The LED went on 1 sec prior to S1 and was turned off upon the subject's response key press. Feedback was

provided in that the fixation light would be briefly re-illuminated .5 sec after the subject's response if S2 was actually louder than S1. An S1-S2 tone pip pair, along with the duration for which the LED was illuminated, comprised a single trial. Trial presentation was manually initiated on the microprocessor with an inter-trial-interval between 5 to 15 sec.

On every trial S1 was set to 70 dB SPL. Prior to data collection each subject's intensity difference detection threshold was determined by a descending staircase procedure. On a random half of the staircase trials, S2 was set to 70 dB. On the remaining half, S2 was initially set to 80 dB and then decreased by 1 dB on each trial of that half until the first subject failure to report S2 as different from S1. Thereafter, S2 was adjusted by .1 dB steps across the "different" trials until the subject achieved a criterion of three trials correctly reported out of four or roughly 75% correct performance. This final value was used as the difference threshold in the ensuing data collection block.

The total number of trials for each subject during EEG recording was 160. On a random 40% of the trials the intensity of S2 was set equal to that of S1 at 70 dB. On another random 40%, S2 was set to a higher intensity which equalled 75% correct detection of difference or no difference from 70 dB. On the remaining 20% (32) trials

S2 was set to 10 dB above the threshold increase. Subjects were informed that an occasional "much louder" S2 would be presented but were given no indication on which trials the highly discrepant stimulus would occur. Subjects were instructed to respond to the rare S2s as "different" in the same manner as they did for the threshold intensity changes. The only constraints on the above randomization were that none of the 32 rare intensity increase trials could follow another or be one of the first five trials in a block.

EEG Recording. EEG activity was recorded from central (Cz) and parietal (Pz) midline sites according to the ten-twenty system of electrode placement. These sites were referenced to linked earlobes. The electro-oculograph (EOG) was measured from supra- and suborbital placements for the right eye. The EOG placements were referenced to each other. The subject was grounded through an electrode affixed to the left mastoid. Beckman Ag-AgCl electrodes were used for all placements and had a measured resistance of 5K Ohms. All channels were amplified on Princeton Applied Research amplifiers with EEG channels set to a gain of 10K and the EOG to a gain of 5K. All channels had a lower bandpass (-3 dB attenuation) of 30 Hz and an upper bandpass (-3 dB attenuation) of .03 Hz. The amplified EEG from the two scalp channels along with the EOG were stored on three separate channels

of a Sangamo eight channel FM tape recorder. A fourth channel was used to store an external synchronizing pulse which was generated 500 msec prior to the onset of S1 on each trial.

EEG and ERP Analysis. The EEG from the Pz electrode was played back off-line through a series of three Krohn-Hite model 3700R analog filters. The filters were adjusted to pass activity in the alpha bandwidth (8-12 Hz) with 30% attenuation at 7.8 and 12.5 Hz on final output. Normalized RMS of the filtered EEG from Pz was obtained by A-to-D conversion with the Heathkit H8 microprocessor on each trial for a period of 500 msec immediately prior to S2. The normalized RMS was calculated from the standard deviation of the filtered input so as to eliminate contribution by any DC offset throughout the digitized period.

Within each subject's session, 8 of the 32 rare trials (S2 set to threshold plus 10 dB increased intensity above S1) with no associated EOG activity were selected as having the highest filtered alpha band RMS values for 500 msec prior to S2 (high alpha). Another eight rare trials free of EOG activity were selected as having the lowest filtered alpha band RMS prior to S2 (low alpha). The unfiltered EEG at Cz and Pz for the above selected trials was then digitized on the H8 for a period from 200 msec before to 1000 msec after S2 at a rate of 77 samples per sec (13 msec resolution). These data were then averaged

to produce, for each subject at both electrode sites, separate 8-trial averaged evoked responses associated with high and low levels of alpha density at the Pz electrode prior to stimulus presentation.

Results

The grand mean waveforms of the seven individual subjects' ERPs to rare stimulus intensity increase are displayed in Figure 2. Separate grand mean ERPs associated with high and low pre-stimulus alpha conditions are superimposed. Also displayed are the grand means of each subject's mean pre-stimulus alpha band RMS for the eight trials in the high and low alpha conditions.

The waveforms of Figure 2 reveal greater positivity in the 300 msec region of the high pre-stimulus alpha ERPs. At Cz the enhanced positivity appears mostly in the P300 peak while at Pz it appears distributed throughout the epoch from 100 msec to roughly 800 msec post-stimulus. There does seem to be a smaller N100 peak in the high alpha ERP in comparison to the low alpha ERP at Cz. The N100 is poorly developed for both high and low alpha conditions at Pz. The difference between the grand mean alpha band RMS levels for the high and low sorted trials is on the order of 34 uV.

P300 amplitude scores were obtained for each subject's data by baseline-to-peak measurement of the most positive point within the waveform from 250 to 500 msec post-stimulus.

NOVEL STIMULUS TRIALS SORTED BY ALPHA RMS.
RMS MEASURED 500 MSEC PRE-STIMULUS.

LOW ALPHA TRIALS (8/SUBJECT) —————
 \bar{X} RMS FOR 7 SUBJECTS = 21 μ V

HIGH ALPHA TRIALS (8/SUBJECT)
 \bar{X} RMS FOR 7 SUBJECTS = 55 μ V

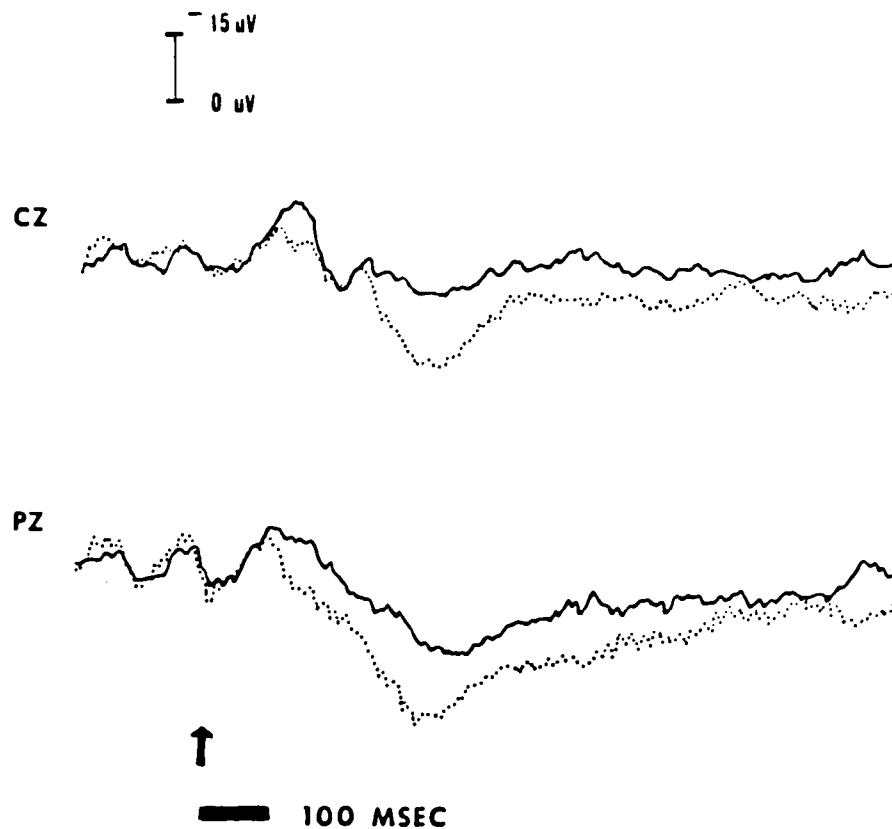


Figure 2. Grand mean waveforms of averaged evoked potentials to rare increased intensity auditory stimuli. The evoked potentials were averaged from separate high and low pre-stimulus alpha trial 'bins'.

N100 amplitudes were obtained by similarly measuring the most negative point from 75 to 150 msec post-stimulus. The measurements for both peaks were submitted to separate ANOVAs with two levels of a Pre-Stimulus Alpha factor (high vs. low) and two levels of an Electrode factor (Cz vs. Pz). The ANOVA on the P300 measure (Appendix A) produced a significant main effect of Pre-Stimulus Alpha ($F = 46.71$, $p < .0005$) with no significant effect of Electrode or interaction between Pre-Stimulus Alpha and Electrode. The ANOVA for the N100 measure (Appendix B) produced a significant main effect for Electrode ($F = 13.32$, $p < .02$) while there was no significant effect of Pre-Stimulus Alpha or interaction between Alpha and Electrode factors.

Though the above analyses demonstrate an effect of pre-stimulus alpha on the P300, they do not determine that this effect is specific to pre-stimulus activity in the alpha band. It might be that if trials were sorted by high and low EEG RMS regardless of frequency band, the same effect would obtain. We therefore re-analysed the data by playing back the recorded EEG of each subject through the Krohn-Hites with filters re-adjusted to pass activity in the delta/theta band (30% attenuation at 1.2 and 7.8 Hz on final output). We were thus able to perform a second sort of the rare stimulus trials on the basis of high vs. low delta/theta RMS from the 500 msec pre-stimulus period. After this second sort, two of the seven subjects pre-

sented with greater high vs. low RMS differences for pre-stimulus delta/theta band activity than they did for activity in the alpha band. For these two subjects, the rare stimulus trials were re-averaged into separate high and low pre-stimulus delta/theta ERPs. These subjects' delta/theta sorted ERPs, along with their original alpha sorted ERPs, are displayed in Figures 3 and 4. Inspection of the waveforms shows that P300 amplitude is the same for high pre-stimulus delta/theta trials in comparison with low pre-stimulus delta/theta trials, while P300 is greater for high pre-stimulus alpha trials than for low alpha trials even though the alpha criterion results in the smaller pre-stimulus RMS difference. From this preliminary evidence, it can be suggested that the P300 enhancement found upon high levels of pre-stimulus alpha is specific to activity in the alpha band and is not the result of differences in general EEG RMS.

Study 1 provides evidence for a positive relationship between the amount of pre-stimulus alpha activity and the amplitude of the P300 component. The amplitude of the N100 was not affected by pre-stimulus alpha. These findings establish two important points. First, that the enhancement of the P300 by pre-stimulus alpha is not due to the fortuitous summation of alpha waves with evoked response peaks. The N100 should stand an equal or even

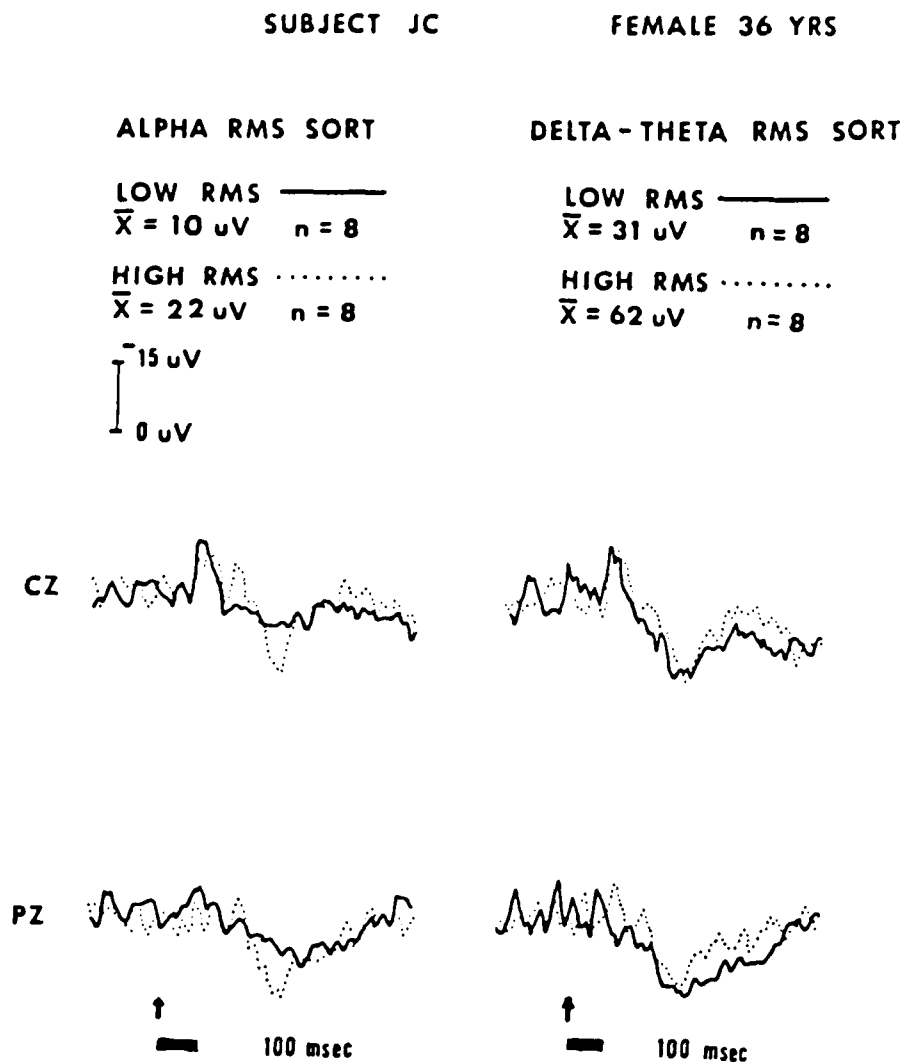


Figure 3. ERPs to rare increased intensity tones for subject JC. Left column ERPs were averaged from separate high and low pre-stimulus alpha trial 'bins'. Right column ERPs were averaged from separate high and low pre-stimulus delta/theta trial 'bins'. Mean RMS difference for this subject's high and low alpha trials was 12 μV . Mean RMS difference for her high and low delta/theta trials was 31 μV .

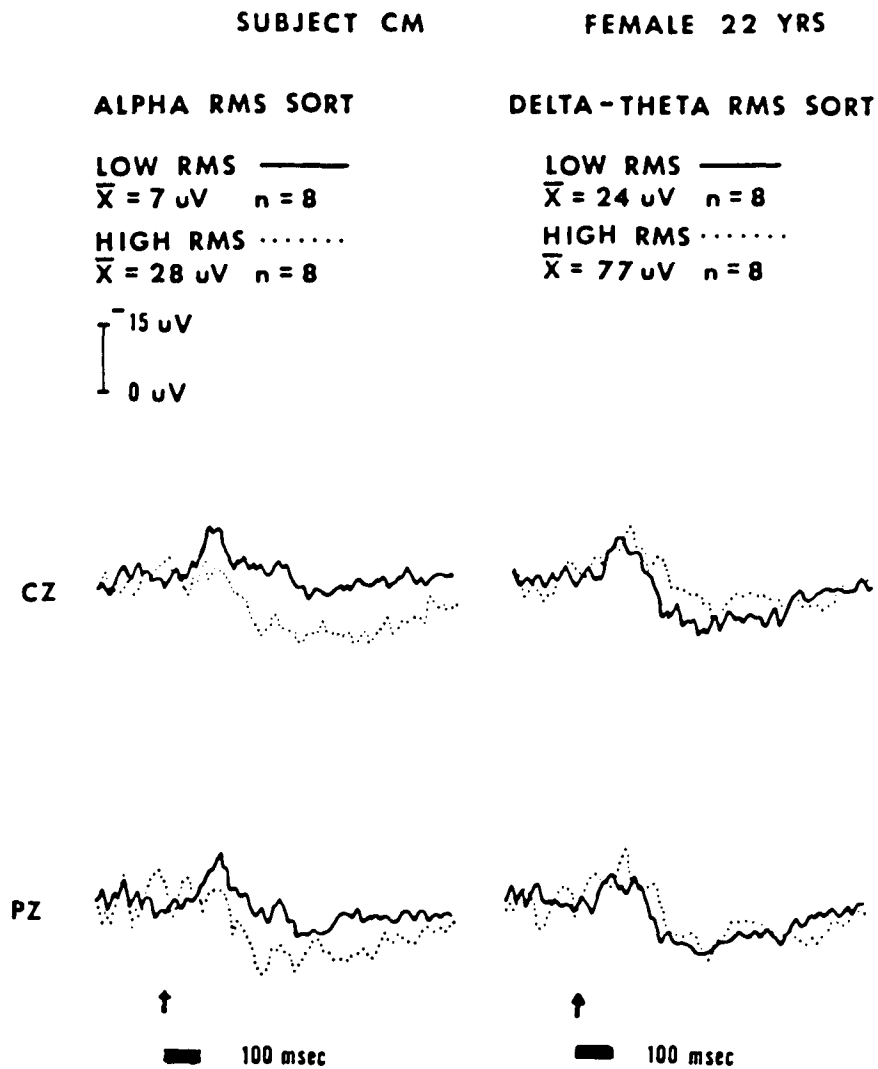


Figure 4. ERPs to rare increased intensity tones for subject CM. Left column ERPs were averaged from separate high and low pre-stimulus alpha trial 'bins'. Right column ERPs were averaged from separate high and low pre-stimulus delta/theta trial 'bins'. Mean RMS difference for this subject's high and low alpha trials was 21 μV . Mean RMS difference for her high and low delta/theta trials was 53 μV .

greater chance, by being earlier and in closer temporal proximity to any "ringing" pre-stimulus alpha, of amplitude enhancement by such a mechanism. Second, that alpha selectively enhances what is regarded as an endogenous component of the evoked response. This validates the view of Jasper (1936) and Bagchi (1937) that alpha activity is responsive to internal events such as mental activation or effort.

The failure to obtain P300 amplitude differences with pre-stimulus delta/theta manipulation argues against the idea, as proposed by Basar, Basar-Eroglu, Rosen and Schutt (1984), that long latency ERP components are derived from phase re-ordered low frequency pre-stimulus EEG activity. Further evidence that P300 is independent, if not inversely related to, the lower frequency EEG bands has been presented in a recent study by Pritchard, Brandt, Shappell, O'Dell and Barratt (1985). They obtained correlations between the amplitude of the P300 and pre-stimulus spectral power in the delta, theta and alpha bands. For rare target stimuli, the power/P300 correlation was negative in the delta band, zero in the theta and positive in the alpha. These authors postulated that P300 amplitude is coupled to "EEG arousal" as indicated by increasing frequency in the EEG bands. However, we are suggesting that there is something unique to the alpha band itself. If it were a question of arousal as reflected

by increasing EEG frequency, beta or desynchronized EEG would be associated with larger P300s than alpha. Yet in Study 1 above, subjects were performing a difficult discrimination task at consistent levels of accuracy. Arousal should have been constant throughout the recording period with alpha representing the lower end of the EEG arousal spectrum at that time. Therefore it is unlikely that our alpha sorting procedure had segregated moments of high "EEG arousal" into the high pre-stimulus alpha trials bin.

Since the rare stimulus ERP in Study 1 was elicited by increases in stimulus intensity, the question of whether the alpha-sensitive P300 was truly endogenous might be raised. The purpose of our second study was to replicate Study 1 as well as to include a rare decreased intensity stimulus condition to test if the alpha/P300 relationship applies when the ERP is elicited by stimulus discrepancy per se. If it should prove to do so, it would strengthen the position that pre-stimulus alpha affects the endogenous P300. In addition, a trials sort by post-stimulus alpha RMS was performed to test for an alpha density by alpha measurement period interaction. Such an interaction would become significant if it was alpha blocking, i.e. high alpha pre-stimulus followed by low alpha post-stimulus, that was associated with large P300s. Extra electrodes, Fz and Oz, were included to provide

enough data for an analysis of principle components. Finally, our selection of subjects was more random, from the college undergraduate population at large, without knowledge of whether the individual's background alpha rhythm was well developed or not. This was to test if the pre-stimulus alpha effect would generalize to the type of sample often recruited for ERP experiments.

STUDY 2

Method

Subjects. The subjects were 11 volunteer graduate and undergraduate students from Queens College in Flushing, New York with no previous experience in EEG or auditory discrimination experiments. They were seven women and four men with a median age of 27 years.

Procedure. The stimulus presentation apparatus was exactly the same as that described in Study 1. Prior to data collection each subject's intensity increase detection threshold was determined by the descending staircase procedure described for Study 1. In addition, the staircase was repeated with S1 set to 80 dB SPL and S2 initially set to 70 dB on 50% of the trials. S2 was increased by 1 dB and .1 dB steps on the "different" trials until the subject's intensity decrease detection threshold, as defined by 75% correct performance, could be determined.

The total number of trials (S1-S2 pairs) for each subject during EEG recording was 320. These were presented in four blocks of 80 trials each. For two of the blocks, S1 was set to 70 dB. On a random 40% of the trials S2 was also set to 70 dB. On another random 40% S2 was set to a higher intensity which equalled 75% correct detection of difference or no difference from 70 dB.

These two blocks comprised condition A. For the remaining two blocks, S1 was set to 80 dB. On 40% of these trials S2 was also set to 80 dB while on another 40% it was set to a lower intensity which equalled 75% correct detection of difference or no difference from 80 dB. These two blocks were condition B. The four blocks were presented in a counterbalanced ABBA or BAAB fashion across subjects.

On the remaining 20% (32) of the trials in condition A, S2 was set to 10 dB above the 75% correctly detected threshold increase. On the remaining 20% (32) of the trials in condition B, S2 was set to 10 dB below the 75% correctly detected threshold decrease. Subjects were instructed to respond to S2 by the differential keypress described for Study 1. The rare S2s were to be responded to as "different" in the same manner as the threshold intensity changes. The only constraints on the above randomization were that none of the 32 rare intensity change trials within a condition could follow another or be one of the first five trials in a block.

EEG Recording. With the exception of the inclusion of two extra scalp electrode channels, Fz and Oz, EEG recording was identical to that described in Study 1.

EEG and ERP Analysis. The measurement of filtered EEG RMS from the Pz electrode was the same as that described in Study 1. Within each condition, 8 of the

32 rare stimulus trials (S2 set to threshold plus 10 dB difference from S1) with no associated EOG activity were selected as having the highest filtered alpha band RMS values 500 msec prior to S2 (high alpha-pre S2). Another eight trials free of EOG activity were selected as having the lowest filtered alpha band RMS prior to S2 (low alpha-pre S2). In a similar manner, a second selection was performed on the same 32 trials to obtain those with the eight highest and eight lowest filtered alpha band RMS values from 500 to 1000 msec after S2 (high alpha-post S2 and low alpha-post S2).

The unfiltered EEG at each electrode for the above selected trials was then digitized on the H8 for a period from 200 msec prior to S2 to 1000 msec after S2 at a rate of 77 samples per sec (13 msec resolution). These data were then averaged separately for the rare increased and decreased intensity conditions to produce, for each subject at each electrode site, averaged evoked responses associated with both high and low levels of alpha density before and after stimulus presentation.

Experimental Analysis. The experimental design consisted of a 2 x 2 x 2 x 4 factorial experiment with repeated measures over all factors. The four independent variables were: (1) two levels of Alpha Density (high and low normalized RMS); (2) two levels of Stimulus Condition (10 dB above and 10 dB below discriminable threshold in-

tensity change); (3) two levels of Alpha Measurement Epoch (500 msec prior to S2 and 500 to 1000 msec after S2); and (4) four levels of Electrode Location (Fz, Cz, Pz and Oz).

Principal Component Analysis (PCA) of the averaged waveforms along with varimax rotation was performed. The PCA was done in two ways; (1) separately by alpha measurement epoch, the data base for these two analyses was a set of 176 waveforms (11 subjects x 4 electrode locations x 2 levels of alpha density x 2 stimulus conditions) and 94 timepoints; and (2) pooled across the entire data set, the data base for this analysis was 352 waveforms (11 subjects x 4 electrode locations x 2 levels of alpha density x 2 stimulus conditions x 2 alpha measurement epochs) and 94 timepoints. Covariance about origin was used to perform the PCAs since this matrix allows the estimated factor scores to preserve correct polarity. The factor scores were then subjected to a repeated measures analysis of variance. To correct for the possibly inflated degrees of freedom due to non-orthogonality of the electrode factor, the degrees of freedom associated with each F-test which included this factor were reduced according to the method of Jennings and Wood (1976).

Along with the PCA factor scores, baseline-to-peak measurements of the P300 were also obtained. The portion of the epoch from which these measurements were taken

was determined by the results of the PCA, i.e. that portion which corresponded to the maximal loadings of the extracted late positive factor and was most free of overlap from other extracted factors. These baseline-to-peak measurements were also submitted to a repeated measures analysis of variance.

Results

ERP Waveforms. The grand mean ERPs elicited by rare stimulus intensity changes and associated with high and low levels of pre-stimulus alpha band RMS are depicted in Figure 5. These ERPs exhibit waveforms that differ with direction of stimulus intensity change and level of pre-stimulus alpha band RMS.

Inspection of the waveforms reveals that at the frontal electrode (Fz) the auditory evoked response to a rare increase in stimulus intensity consists of the N100, P200, N200 and P300 peaks. At the more posterior electrodes (Pz and Oz) the P200-N200 deflection disappears and the waveforms consist solely of the N100 and P300 components with a slow resolution of P300 towards baseline during the last 600 msec of the epoch. For the rare decreases in stimulus intensity, the auditory evoked response consists of the N100, P200 and N200 peaks followed by a slow positive wave. In contrast with the intensity increase ERPs, the intensity decrease

GRAND MEAN ERPs AVERAGED BY PRE-STIMULUS
 ALPHA RMS HIGH RMS LOW RMS —

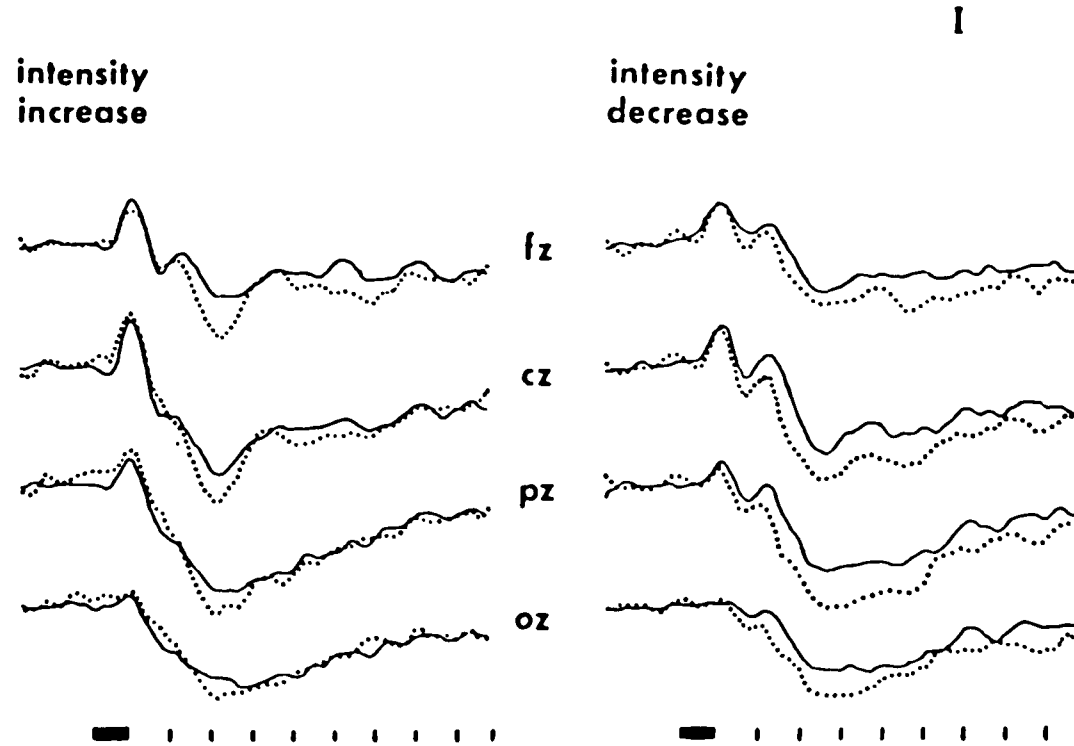


Figure 5. Grand mean ERPs from the pre-stimulus alpha sorted data. Solid bars indicate duration of the 100 msec tone burst. Vertical hatching are at 100 msec intervals. Vertical mark at the upper right equals 4 microvolts. Negative is up.

evoked response waveforms display the P200-N200 deflection is discernable at all electrodes including Oz. The intensity decrease N200 seems to occur in the 150 to 300 msec range which is primarily characterized by the P300 peak in the intensity increase condition. Thus it is possible that the presence of a P300 peak in the intensity increase condition serves to diminish the amplitude of the N200 at central and posterior electrodes.

The effect of pre-stimulus alpha band RMS upon the intensity increase ERP is seen most clearly at Fz. Here the high pre-stimulus alpha waveform exhibits a P300 that is about 6 uV more positive than the corresponding P300 of the low pre-stimulus alpha ERP. There is also more positivity associated with the high alpha ERP in the 500 to 800 msec range, although this is on the order of only 2 uV and does not appear as an enhancement of a component common to both high and low alpha waveforms. From Cz to Oz there is discernable only a small increment in positivity (2 to 3 uV) for the P300 in the high alpha condition. This enhancement seems to diminish towards the posterior area.

In the intensity decrease condition, the effect of high pre-stimulus alpha appears as an overall displacement of the waveform towards positivity starting as early as 150 msec. The enhanced positivity is maximal

at Cz and Pz while also being visible at Fz and Oz. This produces greater amplitude (4 to 6 uV) for the slow positive wave around 300 to 600 msec in the high alpha condition. Correspondingly, the amplitude of the N200 peak with respect to baseline is reduced in the high alpha condition.

The grand mean ERPs associated with high and low levels of post-stimulus alpha band RMS are depicted in Figure 6. These ERPs display the same difference with direction of rare stimulus intensity change that is seen in the pre-stimulus alpha RMS sorted ERPs. Again, there is a clearer presence of the N200 component in the intensity decrease condition and a larger amplitude P300 peak frontally in the intensity increase condition. In contrast with the pre-stimulus alpha sorted data, there appears to be no effect of alpha RMS level upon the waveforms of the post-stimulus alpha sorted ERPs.

Principal Components. The initial PCAs were to determine whether a common factor structure might be identified in the grand mean waveforms of the pre- and post-stimulus alpha sorted data and which factors might prove sensitive to the alpha density treatment. The rotated factor loadings based on the covariance matrix from the two separate analyses are depicted in Figures 7 and 8. It is evident that only the first two factors derived from the two data sets can be matched on the

GRAND MEAN ERPs AVERAGED BY POST-STIMULUS
 ALPHA RMS HIGH RMS LOW RMS -

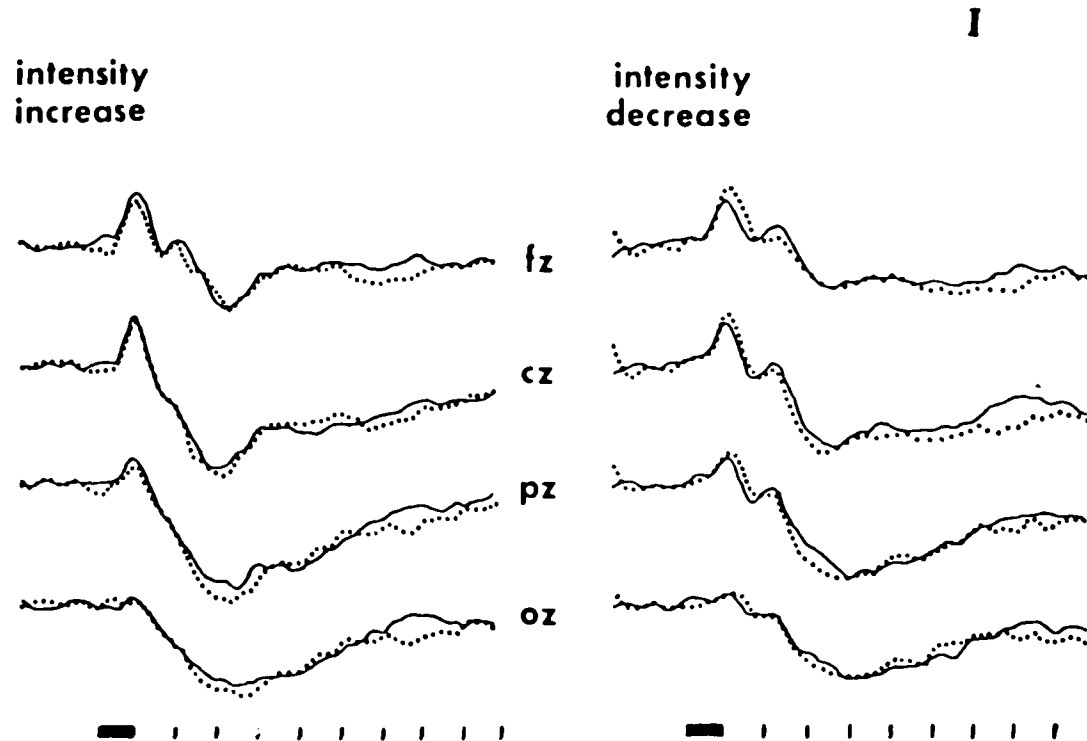


Figure 6. Grand mean ERPs from the post-stimulus alpha sorted data. Solid bars indicate duration of the 100 msec tone burst. Vertical hatching are at 100 msec intervals. Vertical mark at the upper right equals 4 microvolts. Negative is up.

Factor Loadings for Pre-Stimulus Sorted Data

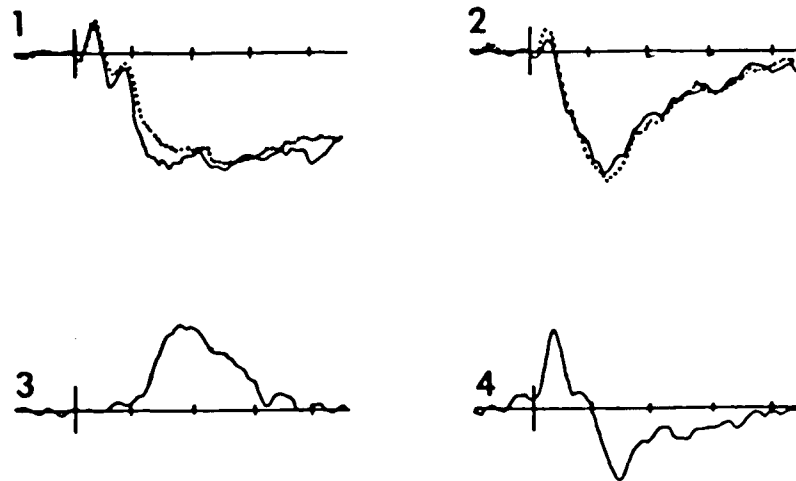


Figure 7. First four extracted loadings from the pre-stimulus alpha sorted ERP set. Large vertical hatching indicates stimulus onset, smaller hatchings are at 200 msec intervals. Dotted lines are the first two factor loadings from the post-stimulus data superimposed (see Figure 8).

Factor Loadings for Post-Stimulus Sorted Data

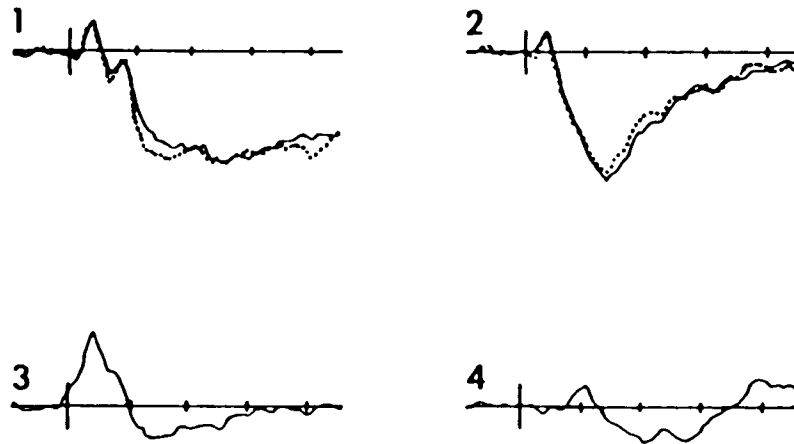


Figure 8. First four extracted loadings from the post-stimulus alpha sorted ERP set. Large vertical hatching indicates stimulus onset, smaller hatchings are at 200 msec intervals. Dotted lines are the first two factor loadings from the pre-stimulus data superimposed (see Figure 7).

basis of shape and temporal configuration of the factor loadings. However, as can be seen in Table 1, these two factors account for the greater percentage of variance after rotation in both data sets.

Factor 1 (refer to Figures 7 and 8 for discussion of this and other factor loadings), which has high positive loadings from 300 msec onwards, would appear to be associated with the late tonic positivity in the original waveforms. It is interesting to note that brief phasic peaks corresponding to the N100-P200-N200 complex are also included in this factor loading. By itself, this factor would seem to account for all of the variance in the ERPs elicited from Fz and Cz in the decreased intensity conditions. One discrepancy is that factor 1 gives no indication of a return to baseline by the end of 1000 msec. Such a trend does seem evident in the grand mean waveforms. It is possible that this factor contains the resolution to baseline of a pre-S2 CNV. Since CNV resolution would remain "tonic" change until the next CNV, its contribution to any factor would appear as a tonic positive displacement from baseline.

Factor 2 exhibits a steep positive slope from 100 to 300 msec. After 300 msec it returns to baseline during the remaining 700 msec as a negatively accelerated curve. This factor would seem to represent

TABLE 1

Percentage of Variance After Rotation for the Separate
Principal Component Analyses (PCAs)

<u>Pre-Stimulus RMS Sort</u>		<u>Post-Stimulus RMS Sort</u>	
<u>Factor</u>	<u>%</u>	<u>Factor</u>	<u>%</u>
1 (Slow Wave)	54	1 (Slow Wave)	55
2 (P300)	23	2 (P300)	32
3 (N400)	10	3 (N400)	5
4 (N100)	7	4 (not certain)	4
5 (not certain)	4	5 (not certain)	3
6 (not certain)	2	6 (not certain)	2

the classic P300. In the original data it appears most clearly in the waveforms elicited at Pz for the increased intensity conditions.

Factor 3 from the pre-stimulus alpha sorted ERP set presents a single wave with a rounded maximum at 400 msec. The factor scores for this component were of negative polarity at Fz and Cz in the increased intensity condition. Referring back to the grand mean waveforms (Figure 5), this factor would correspond to the negative hump after the P300 peak at Fz and Cz in the increased intensity, high alpha condition. In this case, what might otherwise be regarded as the negative-going return slope of the P300 in the grand mean waveform was apparently extracted by the PCA as a separate component. Since this return slope is obviously a function of the amplitude of the P300 peak, the "N400" component was not extracted from the post-stimulus sorted data set which was unaffected by alpha.

Factor 4 from the pre-stimulus alpha sorted data and factor 3 from the post-stimulus alpha sorted data may be matched with each other on the basis of their both having a large, sharp negative peak at 100 msec. They differ in that factor 4 from the pre-stimulus sort is also associated with a sharp positive wave at 300 msec, giving it a biphasic appearance. Furthermore, post-stimulus factor 3's N100 is much broader, starting earlier and more

gradual than the pre-stimulus factor 4's N100 and returning to baseline more slowly. It is obvious that both these factors represent the N100 of the original data, although it is not immediately apparent from the grand mean waveforms (Figures 5 and 6) why the two factors should differ as they do.

Factors 5 and 6 from the pre-stimulus alpha sorted data and factors 4,5 and 6 from the post-stimulus alpha sorted data display smaller loadings and are not easily discernable in the grand mean ERPs. These factors will not be considered in the following analyses.

ANOVAs for the Factor Scores from Pre- and Post-Stimulus Alpha Sorted Data. The factor score, or weighting coefficient, of a component for any given electrode and stimulus condition combination represents the contribution of that component to the ERP waveform in that particular data case (Glaser and Ruchkin, 1976). As such, factor scores can be submitted to an analysis of variance as dependent variables in a multifactor experimental design. The criterion for statistical significance in the following tests was set at a level of .05. Since the presence of an interaction indicates that conclusions based on main effects will not fully describe the data (Keppel, 1973), for the analyses below the main effects of variables participating in significant interactions are not discussed.

Appendix C presents the results of the ANOVA on the factor scores for component 1 ("Slow Wave") from the pre-stimulus alpha data set. Neither the main effects of stimulus intensity, alpha, electrode or any of their interactions reached significance. The ANOVA on the factor scores for the Slow Wave component in the post-stimulus alpha sorted data (Appendix D) reveals that the main effect of stimulus intensity did reach significance, $F(1,10) = 6.58$, $p < .03$. Plots of the Slow Wave factor scores from both data sets are presented in Figure 9. It is clear that the Slow Wave component is larger for decreased stimulus intensity at all electrode sites in the post-stimulus alpha sorted data. In the pre-stimulus sort there does seem to be a tendency for the decreased intensity, high alpha Slow Wave factor scores to be more positive than the rest. Although the stimulus intensity x alpha interaction did not reach significance, its F value (2.25) was the largest obtained in the ANOVA for the pre-stimulus alpha sorted Slow Wave factor scores.

The results of the ANOVA on the factor scores for component 2 ("P300") in the pre-stimulus sort (Appendix E) reveal a significant interaction of stimulus intensity and electrode location, $F(3,30) = 4.61$, $p < .01$, and a significant main effect of alpha, $F(1,10) = 5.46$, $p < .05$. The ANOVA on this same component's factor scores for the post-stimulus sort (Appendix F) shows again a

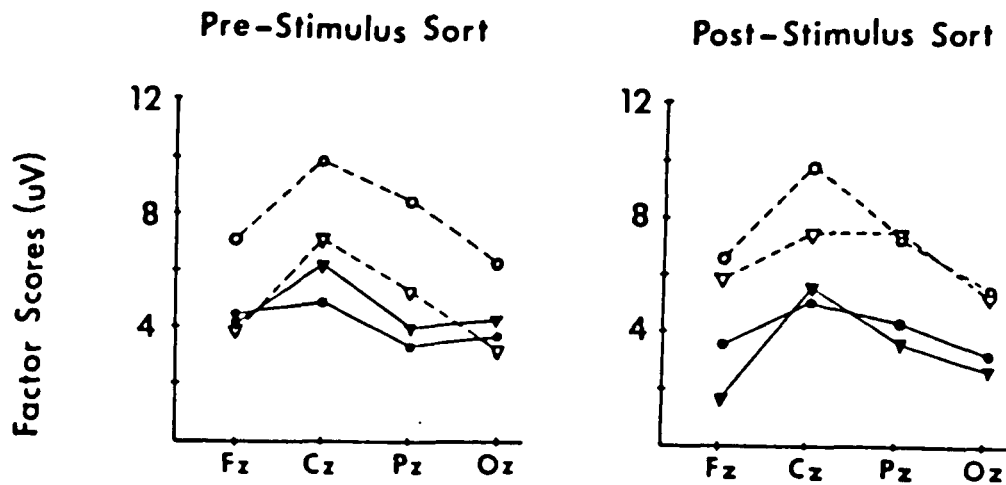


Figure 9. Slow Wave factor scores for pre- and post-stimulus alpha sorted ERP sets.

- High Alpha and Increased Intensity
- ▼—▼ Low Alpha and Increased Intensity
- High Alpha and Decreased Intensity
- ▽---▽ Low Alpha and Decreased Intensity

significant interaction of stimulus intensity and electrode site, $F(3,30) = 3.2$, $p < .05$. The main effect of alpha does not reach significance nor does alpha density interact with any other factor. Referring to the pre- and post-stimulus factor score plots in Figure 10, it can be seen that the P300 component is larger with increased stimulus intensity for both data sets. The source of the stimulus intensity and electrode interaction is best seen in the post-stimulus plot. The P300 component to increased intensity stimulation displays maximal amplitudes at Cz and Pz while for decreased intensity stimulation the maxima are more posterior at Pz and Oz. The striking difference between the pre- and post-stimulus P300 factor score plots lies in the definite separation of high and low alpha conditions at both intensity levels in the pre-stimulus RMS sorted data. This separation by alpha density is absent in the post-stimulus plot. The lack of any interaction along with the presence of a significant main effect for a given variable are indicative of that factor's additivity with all other factors in the design. Again referring to Figure 10, the P300 component's amplitude increase due to high pre-stimulus alpha density is the same for both types of stimulus change at electrodes Cz and Pz. The effect of alpha density at Pz seem limited to the increased

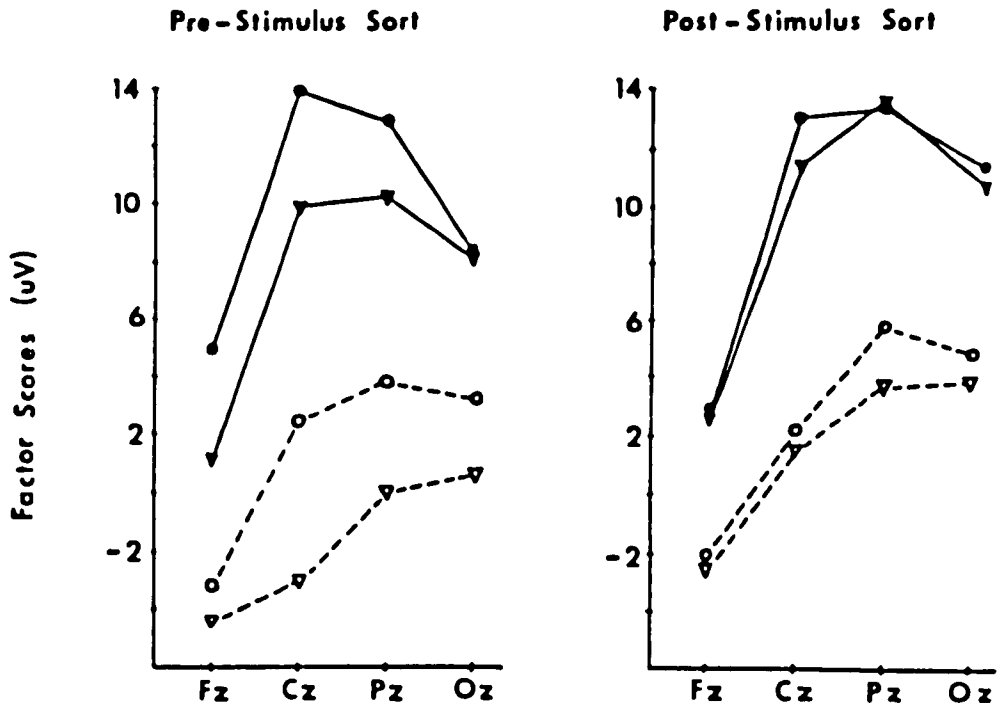


Figure 10. P300 factor scores for pre- and post-stimulus alpha sorted ERP sets.

- High Alpha and Increased Intensity
- ▼—▼ Low Alpha and Increased Intensity
- High Alpha and Decreased Intensity
- ▽---▽ Low Alpha and Decreased Intensity

intensity condition while at Oz it seems limited to the decreased intensity condition. However, the interaction of alpha and intensity and electrode for the pre-stimulus data did not reach significance, $F(3,30) = 1.47$, $p < .25$.

As mentioned above, an N400 component was extracted only from the pre-stimulus alpha sorted data. The ANOVA for its factor scores (Appendix G) presents with significant interactions of stimulus intensity and electrode, $F(3,30) = 4.18$, $p < .02$, and alpha density and electrode, $F(3,30) = 3.35$, $p < .04$. Figure 11 shows that the source of the intensity and electrode interaction is that this component's factor scores are positive overall for the decreased intensity condition while for increased intensity they are positive parieto-occipitally but are negative fronto-centrally. Within both intensity conditions, although markedly for increased intensity, factor scores for the high alpha condition are relatively more negative than those for the low alpha condition at Fz and Cz. This would be the source of the alpha density and electrode interaction.

The ANOVAs for the N100 factor scores of both data sets (component 4 in the pre-stimulus sort and component 3 in the post-stimulus sort) revealed significant main effects of electrode location. The specific F-ratios for these measures are reproduced in Appendices H and I.

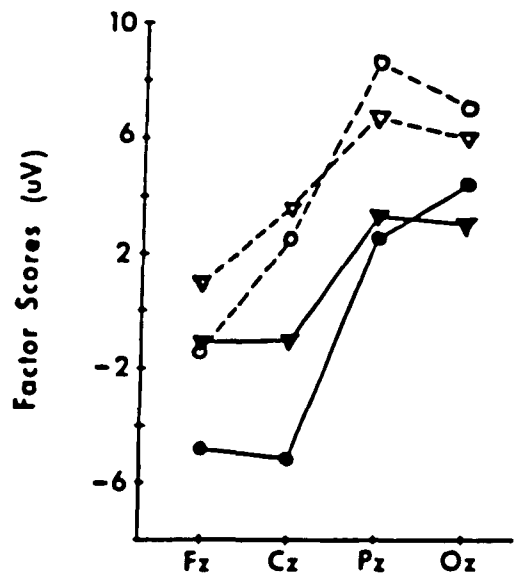


Figure 11. N400 factor scores for the pre-stimulus alpha sorted ERP data.

- High Alpha and Increased Intensity
- ▼—▼ Low Alpha and Increased Intensity
- High Alpha and Decreased Intensity
- ▽---▽ Low Alpha and Decreased Intensity

The N100 component in both ERP sets displayed a linear increase in amplitude from posterior to anterior electrode sites. No other main effect or interaction reached significance.

ANOVA on Factor Scores from Combined Data. The results of the initial PCAs demonstrated the similarity of the first two extracted components from both sets of ERPs sorted on the basis of different alpha RMS measurement epochs. It was evident that the second component of the pre-stimulus RMS sorted ERP set was sensitive to alpha density. However, the plots of this component's factor scores could not be used to assess differences due to alpha RMS measurement epoch because these measures were scaled relative to the grand mean for each ERP subset. In order to evaluate the effects of alpha measurement epoch on this component, PCA was performed on ERPs pooled across the entire data set.

The second extracted factor from the pooled data exhibited the same shape and temporal configuration as did the second factor (P300 component) that was extracted separately from both of the alpha measurement epoch data sets. It was decided on the basis of the relatively greater independence of stimulus intensity and alpha density effects at Cz and Pz (left panel, Figure 10), to utilize only these two electrodes in the following $2 \times 2 \times 2 \times 2$ (electrode \times alpha measurement epoch \times alpha density \times stimulus intensity) ANOVA.

The ANOVA on the factor scores of the extracted P300 component from the entire data set (Appendix J) presents with significant main effects of stimulus intensity, $F(1,10) = 24.87$, $p < .001$, and electrode, $F(1,10) = 9$, $p < .02$, and a significant interaction of alpha density and alpha measurement epoch, $F(1,10) = 6.18$, $p < .04$. As can be seen from the plot of these P300 factor scores (Figure 12), both the high and low alpha condition factor score amplitudes of the post-stimulus measurement condition at Cz and Pz lie midway between the amplitudes of the pre-stimulus measurement condition. This interaction confirms that alpha density post-stimulus has no effect upon the amplitude of the P300 component while alpha density pre-stimulus does.

ANOVA on Baseline-to-Peak Measurements. The ANOVA on the factor scores from the combined data set demonstrated that the alpha density factor interacted with RMS measurement epoch but not with direction of intensity change. To reinforce these findings, it was decided to perform the same analysis using a baseline-to-peak measurement of the P300 component. On the basis of the temporal configuration of the second extracted component from the above PCAs, the P300 in each subject's electrode and experimental condition combination was defined as the most positive peak between 250 and 500 msec. Baseline-to-peak amplitudes of these components were submitted to a $2 \times 2 \times 2 \times 4$ (measurement epoch x

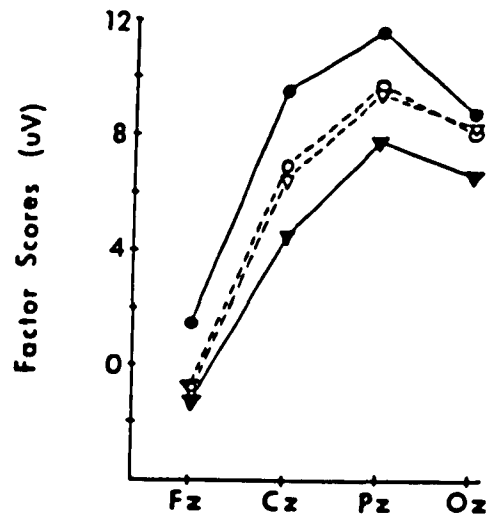


Figure 12. P300 factor scores for the combined pre- and post-stimulus alpha sorted ERP data set, collapsed across intensity.

- Pre-stimulus sorted High Alpha
- ▼—▼ Pre-stimulus sorted Low Alpha
- Post-stimulus sorted High Alpha
- ▽---▽ Post-stimulus sorted Low Alpha

alpha density x intensity change x electrode) ANOVA.

The results of the ANOVA on the baseline-to-peak P300 (Appendix K) included a significant main effect of electrode, $F(3,30) = 18.91$, $p < .01$, and a significant interaction of alpha density and alpha measurement epoch, $F(1,10) = 7.31$, $p < .03$. Interestingly, the factor of stimulus intensity only approached significance, $F(1,10) = 3.77$, $p < .10$. Figure 13 shows that the baseline-to-peak P300 is maximal at Cz and Pz and is enhanced by pre-stimulus alpha. The post-stimulus sorted P300 amplitudes no longer lie between the high and low alpha levels of the pre-stimulus data, as they did in the factor score plot (Figure 12), but are now of the same magnitude as the pre-stimulus low alpha P300. Specifically, the post-stimulus data no longer constitute a perfect "dilution" of the alpha density effect. They are now equivalent to the low pre-stimulus alpha treatment.

The above analyses establish that pre-stimulus alpha activity can affect the P300. When considering the grand mean waveforms, especially those for the intensity decrease condition (Figure 5, right panel), it also seems that the enhanced positivity associated with high pre-stimulus alpha may begin to appear earlier than 300 msec. To test for the effect of alpha on positive components earlier than the P300, we measured the most positive peak between 100 to 250 msec post-stimulus (P150) in all

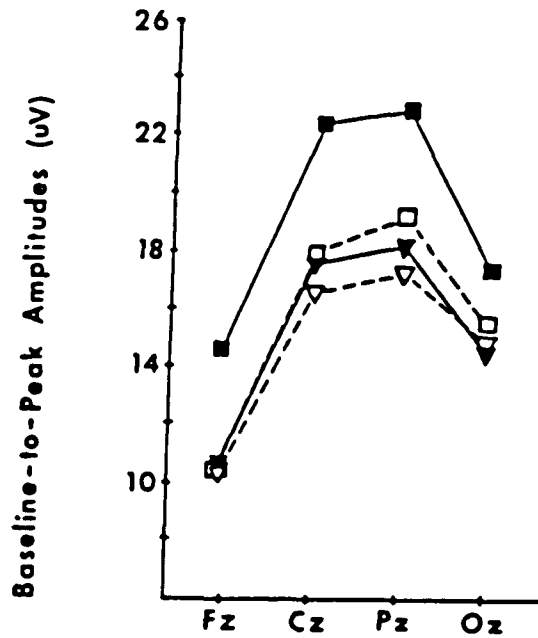


Figure 13. Baseline-to-peak P300 amplitudes for the combined pre- and post-stimulus alpha sorted ERP data set.

- Pre-stimulus sorted High Alpha
- ▼—▼ Pre-stimulus sorted Low Alpha
- Post-stimulus sorted High Alpha
- ▽--▽ Post-stimulus sorted Low Alpha

of the waveforms from Study 2. These baseline-to-peak amplitudes were then submitted to a 2 x 2 x 2 x 4 (RMS measurement epoch x alpha density x intensity change x electrode) ANOVA. The results of the ANOVA are presented in Appendix L. The main effect of intensity change, $F(1,10) = 5.61$, $p < .05$, and of electrode location, $F(3,30) = 9.35$, $p < .01$, were significant. Plots of P150 amplitudes are displayed in Figure 14. The P150 is larger with increased stimulus intensity and has a centro-parietal maximum. There does seem to be separation between high and low alpha levels for the pre-stimulus RMS sorted data that is not found in the post-stimulus sort. The P150 is larger with low pre-stimulus alpha in the increased intensity condition but is larger with high pre-stimulus alpha in the decreased intensity condition. The only indication of this in the ANOVA on P150 amplitudes is that the interaction of stimulus intensity and alpha density approached significance, $F(1,10) = 3.62$, $p < .10$.

Study 2 provides evidence that the positive relationship between pre-stimulus alpha abundance and P300 amplitude is independent of stimulus intensity. This indicates that alpha affects the endogenous or psychological processes responsible for the P300, rather than the exogenous or stimulus driven processes which can also contribute to this component (Roth, Doyle, Pfefferbaum and Kopell, 1980). Both the factor scores and baseline-

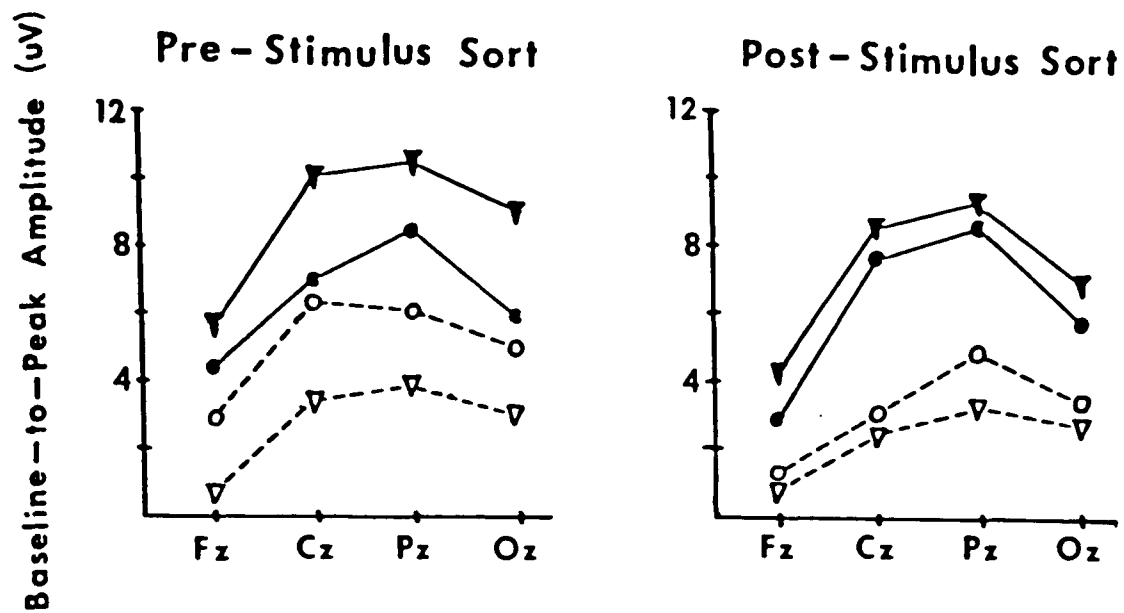


Figure 14. Baseline-to-peak P150 amplitudes from both pre- and post-stimulus alpha sorted data.

- High Alpha and Increased Intensity
- ▼——▼ Low Alpha and Increased Intensity
- High Alpha and Decreased Intensity
- ▽-----▽ Low Alpha and Decreased Intensity

to-peak amplitudes for the P300 component are increased by the presence of pre-stimulus alpha. In this regard there is convergence of the two types of measures. However, the baseline-to-peak measure (Figure 13) does not show the "dilution" produced by the post-stimulus alpha sort (Figure 12). This is the production of a middle-ranged amplitude P300 by the random selection of pre-stimulus alpha densities when sorting by alpha density that occurs post-stimulus. Thus it appears that the extracted factor scores are a more exact measure of P300 with regard to the alpha effect. It is possible that the baseline-to-peak measure was overlapped with some other component. It cannot be ascertained whether the asymmetry between pre- and post-stimulus sorts in Figure 13 is due to reduction of the post-stimulus amplitudes or elevation of the low alpha pre-stimulus amplitudes.

Even more striking is the disagreement between the formal analyses of the factor score and baseline-to-peak measurements of P300 and the appearance of the grand mean waveforms. The grand mean waveforms suggest that there is a difference in the topography of the pre-stimulus alpha effect as a function of stimulus intensity (Figure 5). Pre-stimulus alpha appears to enhance the P300 of the increased intensity ERP primarily at the frontal electrode. The effect of alpha on the P300 in

the decreased intensity condition seems to occur more at the centro-parietal electrodes. However, the ANOVAs for the P300 factor score and baseline-to-peak measures did not reveal a significant interaction between alpha density, intensity change and electrode location. The amplitude of the P150 seemed to show an interaction of alpha density and stimulus change (Figure 14, left panel). This would support the view that ERP responses to the two types of stimulus change are distinct and so are the effects of alpha upon them, but again the interaction term did not reach formal significance. At this point, the analyses do not support any difference in the pre-stimulus alpha effect as a function of stimulus intensity or that there is any other alpha-sensitive component besides the centro-parietal P300.

Study 2 failed to find a relationship between P300 and the amount of alpha activity that occurs immediately after a stimulus. This might suggest that the enhancement of the P300 by pre-stimulus alpha has little bearing upon the effectiveness of alpha blocking or suppression. However, as indicated in Table 2, the high alpha levels measured post-stimulus did not approach those found pre-stimulus. This attests to the fact that alpha was effectively blocked by most of the rare stimulus presentations. Failure of alpha suppression on any rare stimulus trial may never have actually occurred. Alternatively, it

TABLE 2

Group Mean P300 Amplitudes (uV Baseline-to-Peak)
and Group Mean Alpha RMS (uV measured at Pz)

	<u>Electrode</u>	<u>Intensity Increase</u>				<u>Intensity Decrease</u>			
		<u>Cz</u>		<u>Pz</u>		<u>Cz</u>		<u>Pz</u>	
		<u>Alpha</u>	<u>RMS</u>	<u>High</u>	<u>Low</u>	<u>High</u>	<u>Low</u>	<u>High</u>	<u>Low</u>
STUDY 1	P300:	26	15	28	16	-	-	-	-
	Alpha:			55	21	-	-	-	-
STUDY 2									
<u>Pre-Stimulus Alpha</u>									
	P300:	23	19	22	20	21	15	23	16
	Alpha:			40	13			35	13
<u>Post-Stimulus Alpha</u>									
	P300:	20	18	21	18	16	15	18	16
	Alpha:			18	10			19	10

could be that the duration of P300 amplitude-related alpha suppression may not outlast the P300 itself, i.e. it might be over by 500 msec and thus would not have been measured in our design. To sum up the above, the null hypothesis can never be proven. We cannot state that alpha blocking is unrelated to the P300, but simply that our experiment failed to detect such a relationship.

GENERAL DISCUSSION

A positive relationship between P300 and the alpha rhythm might at first seem contra-intuitive. The P300 is regarded as an indicator of an engaged processing state while alpha has been considered the hallmark of mental quiescence (Brown, 1970). One hypothesis that could reconcile this apparent dichotomy is that the enhancement of the P300 by pre-stimulus alpha activity represents a rebound phenomenon. The psychological states usually associated with alpha and P300 are certainly mutually exclusive, but the onset of one could be more pronounced if preceded by predominance of the other. This hypothesis is consistent with a recent theory of adaptive systems as proposed by Grossberg (1984). In his monograph, Grossberg attempts to account for a wide range of psychological phenomena by means of antagonistic rebound within gated dipoles. A gated dipole is composed of two mutually antagonistic systems. When one system is ascendant the other is inhibited. If non-specific input is delivered simultaneously to both systems, or if the ascendant system is turned off, the previously inhibited system becomes active to a point beyond which it would have been if it had simply received input in the absence of inhibition from its antagonistic partner. This is antagonistic rebound. A distributed gated dipole

can be conceived as operating throughout the brain in the regulation of cortical tone and activation. One member of this gated dipole could be the mesencephalic reticular formation, the output of which results in EEG desynchronization and behavioral activation (Moruzzi and Magoun, 1949). The other member of the dipole could then be the thalamo-cortical alpha generating system as described by Andersen and Andersson (1968). Ascendancy of this system would result in cortical rhythmicity and behavioral quiescence. Competition between the two systems would occur subcortically while output from one or the other produced widespread cortical electrophysiological effects. If it is assumed that output from the mesencephalic reticular system results in a cortical P300, then it would be predicted that the presence of pre-stimulus alpha will be associated with an enhanced P300 by way of antagonistic rebound in the reticular system as part of a mesencephalic reticular/thalamo-cortical dipole.

The alpha rhythm remains one of the unexplained phenomena of the human EEG. The most definitive attempt to account for it to date is the theory proposed by Andersen and Andersson (1968). They suggested a facultative pacemaker for the origin of the alpha rhythm. Rhythmic activity is assumed to be an inherent property of groups of cells in all thalamic nuclei. Connections between

these nuclei determine the degree of synchrony of rhythmic activity in the thalamus. Synchronous thalamic activity is in turn projected on to the cortex. Of crucial importance for the appearance of alpha activity in both structures is the intrathalamic spread of rhythmic activity from one thalamic nucleus to others. Andersen and Andersson postulated that a type of interneuron or nucleus existed which could act as a distributor of intrathalamic synchrony. A possible candidate for such a role is the nucleus reticularis of the thalamus. The nucleus reticularis is a thin sheet of cells surrounding the thalamus. It receives inputs from and sends efferents to specific and non-specific thalamic nuclei. Scheibel and Scheibel (1966), in pointing out this arrangement, suggested that the nucleus reticularis would be well suited to assist in synchronizing thalamic discharges. Physiological studies have shown that the nucleus reticularis exerts an inhibitory effect on thalamic neurons (Schlag and Wazak; 1970, 1971). In this way it could act as a thalamic pacemaker by synchronizing the excitatory period of thalamic cell groups through the widespread imposition of inhibitory periods.

Yingling and Skinner (1977) found that the firing of neurons in the nucleus reticularis was suppressed by electrical stimulation of the mesencephalic reticular formation (MRF). These investigators proposed that the

general facilitation of thalamic transmission by MRF stimulation (Desmedt, 1960) is the result of disinhibition of thalamic nuclei following suppression of the inhibitory influence of the nucleus reticularis. If the P300 is simultaneous with MRF activity and/or the facilitation of thalamic transmission, a larger P300 after alpha activity could be considered a form of thalamic rebound. Alpha rhythm would indicate the preponderance of the inhibitory activity of the nucleus reticularis in the thalamus. MRF inhibition of the nucleus reticularis, along with direct MRF excitation of the thalamus (Purpura, 1966), during alpha would produce a pronounced excitatory rebound in thalamic nuclei that would appear cortically as a larger P300. However, this is not an antagonistic dipole in Grossberg's (1984) sense. The inhibitory influence is cascaded from MRF to nucleus reticularis to thalamus. There is no mutually inhibitory pair, no rebounding of alpha following a period of MRF activation. According to Andersen and Andersson's (1968) model, the alpha rhythm would arise gradually as more and more thalamic neurons are phased into synchrony by some distributed inhibitory activity. Indeed, this is how alpha spindles appear to arise in scalp recordings. The sudden release of thalamic neurons from reticularis inhibition would produce a more phasic electrical sign such as the P300 component of the ERP.

Appendix A

Study 1: ANOVA on Baseline-to-Peak P300 Measure

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Alpha	858	1	858	46.7	.0005
Error	110	6	18		
Electrode	34	1	34	1.5	.2676
Error	137	6	22		
Alpha x Electrode	3	1	3	0.7	.4309
Error	24	6	4		

	<u>Cell Means (uV)</u>	<u>Standard Deviation</u>
High Alpha-Cz	26	11
High Alpha-Pz	28	9
Low Alpha-Cz	15	10
Low Alpha-Pz	16	7

Appendix B

Study 1: ANOVA on Baseline-to-Peak N100 Measure

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Alpha	34	1	34	3.4	.1144
Error	60	6	10		
Electrode	93	1	93	13.3	.0107
Error	42	6	7		
Alpha x Electrode	10	1	10	1.5	.2621
Error	40	6	7		

	<u>Cell Means (uV)</u>	<u>Standard Deviation</u>
High Alpha-Cz	-8	4
High Alpha-Pz	-5	2
Low Alpha-Cz	-11	4
Low Alpha-Pz	-6	3

Appendix C

 Study 2: ANOVA on the Factor Scores for Component 1
 (Slow Wave) of the Pre-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Intensity Change	184	1	184	1.9	.1926
Error	943	10	94		
Alpha	70	1	70	.9	.3526
Error	739	10	74		
Electrode	180	3	60	1.9	.1585
Error	969	30	32		
I x A	141	1	141	2.2	.1645
Error	628	10	63		
I x E	52	3	17	1.1	.3527
Error	464	30	15		
A x E	7	3	2	.2	.8679
Error	289	30	10		
I x A x E	2	3	0.7	.06	.9737
Error	362	30	12		

Appendix D

Study 2: ANOVA on the Factor Scores for Component 1
(Slow Wave) of the Post-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Intensity Change	466	1	466	6.6	.0281
Error	708	10	71		
Alpha	27	1	27	.6	.4348
Error	406	10	41		
Electrode	213	3	71	2.6	.0738
Error	843	30	28		
I x A	0.5	1	0.5	.01	.9071
Error	364	10	36		
I x E	6	3	2	.3	.8427
Error	231	30	8		
A x E	9	3	3	.4	.7667
Error	245	30	8		
I x A x E	31	3	10	1.6	.2063
Error	192	30	6		

Appendix E

Study 2: ANOVA on the Factor Scores for Component 2
(P300) of the Pre-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Intensity Change	3481	1	3481	30	.0003
Error	1173	10	117		
Alpha	390	1	390	5.5	.0415
Error	714	10	71		
Electrode	1343	3	448	12.4	.0001
Error	1083	30	36		
I x A	7	1	7	.06	.8115
Error	1222	10	122		
I x E	258	3	86	4.6	.0091
Error	560	30	19		
A x E	69	3	23	2.3	.1008
Error	304	30	10		
I x A x E	43	3	14	1.5	.2427
Error	293	30	10		

 Appendix F

 Study 2: ANOVA on the Factor Scores for Component 2
 (P300) of the Post-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Intensity Change	2679	1	2679	31	.0002
Error	871	10	87		
Alpha	33	1	33	0.7	.4325
Error	495	10	49		
Electrode	2034	3	678	18	.0001
Error	1111	30	37		
I x A	2	1	2	0.02	.8858
Error	735	10	73		
I x E	166	3	55	3.2	.0373
Error	520	17			
A x E	6	3	2	0.3	.8533
Error	236	30	8		
I x A x E	20	3	7	0.6	.6407
Error	350	30	12		

Appendix G

Study 2: ANOVA on the Factor Scores for Component 3
(N400) of the Pre-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Intensity Change	1017	1	1017	11	.0083
Error	945	10	94		
Alpha	1	1	1	.02	.8804
Error	525	10	52		
Electrode	1487	3	496	30	.0001
Error	499	30	17		
I x A	63	1	63	3	.1403
Error	244	10	24		
I x E	130	3	43	4	.0138
Error	312	30	10		
A x E	61	3	20	3	.0319
Error	183	30	6		
I x A x E	30	3	10	2	.1643
Error	166	30	5		

Appendix H

Study 2: ANOVA on the Factor Scores for Component 4
(N100) of the Pre-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Intensity Change	13	1	13	.3	.6041
Error	451	10	45		
Alpha	4	1	4	.1	.7535
Error	401	10	40		
Electrode	899	3	300	20	.0001
Error	444	30	15		
I x A	147	1	147	3	.1225
Error	518	10	52		
I x E	13	3	4	0.4	.7335
Error	296	30	10		
A x E	2	3	0.6	.08	.9693
Error	243	30	8		
I x A x E	20	3	7	1	.2449
Error	138	30	5		

Appendix I

Study 2: ANOVA on the Factor Scores for Component 3
(N100) of the Post-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
Intensity Change	44	1	44	.4	.5342
Error	1058	10	106		
Alpha	0.1	1	0.1	.00	.9657
Error	450	10	45		
Electrode	337	3	112	5	.0079
Error	709	30	24		
I x A	112	1	112	3	.1238
Error	396	10	40		
I x E	2	3	0.7	0.1	.9499
Error	191	30	6		
A x E	6	3	2	0.2	.9143
Error	356	30	12		
I x A x E	44	3	15	2	.1915
Error	262	30	9		

Appendix J

Study 2: ANOVA on the Factor Scores for Component 2 (P300)
of the Combined Pre- and Post-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
RMS Epoch	4	1	4	0.1	.7224
Error	314	10	31		
Intensity	3960	1	3960	25	.0005
Error	1592	10	159		
Alpha	267	1	267	8	.0204
Error	353	10	35		
Electrode	350	1	350	9	.0134
Error	389	10	39		
R x I	0.4	1	0.4	0.02	.8869
Error	178	10	18		
I x A	32	1	32	0.4	.5279
Error	751	10	75		
R x E	0.8	1	0.8	0.4	.5372
Error	20	10	2		
I x E	33	1	33	4	.0877
Error	93	10	9		
A x E	7	1	7	2	.1596
Error	32	10	3		
A x R	180	1	180	6	.0323
Error	291	10	29		
R x I x A	9	1	9	0.1	.7176
Error	624	10	62		
R x I x E	0.6	1	0.6	0.3	.6225
Error	23	10	2		
R x A x E	2	1	2	0.2	.6979
Error	111	10	11		
I x A x E	12	1	12	1	.3075
Error	106	10	11		
R x I x A x E	8	1	8	1	.3476
Error	80	10	8		

Appendix K

Study 2: ANOVA on Baseline-to-Peak Measurements of P300
in the Combined Pre- and Post-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
RMS Epoch	284	1	284	14	.0034
Error	196	10	20		
Intensity	280	1	280	4	.0809
Error	743	10	74		
Alpha	622	1	622	10	.0090
Error	597	10	60		
Electrode	3298	3	1099	19	.0001
Error	1744	30	58		
R x I	49	1	49	2	.2279
Error	300	10	30		
I x A	5	1	5	0.1	.7495
Error	511	10	51		
R x E	33	3	11	2	.2082
Error	189	30	6		
I x E	27	3	9	0.4	.7480
Error	662	30	22		
A x E	35	3	12	1	.3488
Error	315	30	10		
A x R	219	1	219	7	.0222
Error	300	10	30		
R x I x A	32	1	32	0.8	.3778
Error	375	10	37		
R x I x E	8	3	3	0.8	.4806
Error	100	30	3		
R x A x E	9	3	3	0.2	.8044
Error	523	30	17		
I x A x E	50	3	16	1	.2613
Error	343	30	11		
R x I x A x E	20	3	6	0.7	.5056
Error	292	30	10		

Appendix L

Study 2: ANOVA on Baseline-to-Peak Measurements of P150
in the Combined Pre- and Post-Stimulus Alpha Sorted Data

<u>Source</u>	<u>Sum of Squares</u>	<u>df</u>	<u>Mean Squares</u>	<u>F</u>	<u>p</u>
RMS Epoch	49	1	49	1	.2634
Error	353	10	35		
Intensity	1156	1	1156	6	.0394
Error	2061	10	206		
Electrode	811	3	270	9	.0011
Error	868	30	29		
Alpha	0.5	1	0.5	0.02	.8959
Error	309	10	31		
R x I	7	1	7	0.1	.7275
Error	553	10	55		
I x A	169	1	169	4	.0862
Error	467	10	47		
R x E	4	3	1	0.1	.9584
Error	369	30	12		
I x E	43	3	14	0.7	.5356
Error	575	30	19		
A x E	4	3	1	0.2	.9235
Error	245	30	8		
A x R	21	1	21	0.8	.3943
Error	265	10	26		
R x I x A	88	1	88	1	.3056
Error	755	10	75		
R x I x E	4	3	1	0.2	.9250
Error	292	30	10		
R x A x E	2	3	0.6	0.08	.9702
Error	211	30	7		
I x A x E	3	3	1	0.1	.9532
Error	318	30	11		
R x I x A x E	12	3	4	0.5	.6708
Error	236	30	8		

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