The Effect of Prestimulus Alpha Activity on the P300

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ABSTRACT

Trials on which highly discrepant, auditory 'oddball' stimuli were presented were sorted into two bins on the basis of prestimulus alpha band RMS magnitude. The trial bins were then separately averaged to produce a 'high alpha' auditory ERP (event-related potential) and a 'low alpha' ERP for each subject. Study 1 found that larger amplitude P300s were obtained in the 'high alpha' ERP. No effect of alpha was found on the N100. Study 2 employed extra factors of stimulus intensity change (increases and decreases) and alpha measurement period (before and after the 'oddball' stimulus). It was found that P300 amplitude enhancement was independent of both stimulus intensity and the amount of alpha poststimulus. 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 the generation of both alpha and the P300.

DESCRIPTORS: Event-related potentials (ERPs), P300 component, Alpha activity, Neuronal inhibition and rebound.

Event-related potentials (ERPs) have become a major focus of human psychophysiological research. 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 would bear some relationship to the ongoing electroencephalogram (EEG). Given the overlap between the stimuli known to block alpha and those used in cognitive ERP research (i.e., salient, surprising, or informative stimuli), it would be worthwhile to consider a relationship between the alpha rhythm and 'cognitive' ERPs. Although some explanations of ERP phenomena are based on prestimulus states (Karlin, 1970; Näätänen, 1975), they identify the prestimulus states with signal-averaged waveforms such as the Contingent Negative Variation (CNV) or the readiness potential. However, it is possible that the relation between an ERP component and prestimulus activity may disappear as a consequence of the averaging process. Questions about the physiological basis or function of ERPs may remain unanswered if ERPs are regarded in isolation from their electrophysiological environment. If a link could be established between ERPs and the background EEG, new models of both could develop.

A relation between the alpha rhythm and an evoked late positive wave has been reported. Jasper (1936) noted that alpha blocking was associated with a marked slow positive swing during the first part of the blocked period. During the 1930s, it was debated whether blocking of the alpha rhythm could be due to endogenous psychological processes. Adrian and Matthews (1934) stated that patterned visual input produced desynchronization among occipital lobe neurons and that alpha blocking was more the result of visual stimulation than of general attention. On the other hand, Loomis, Harvey, and Hobart (1936) and Bagchi (1937) suggested that alpha blocking was not so much a function of visual input, but rather the expectation to see upon eye-opening. This controversy of several decades ago resembles the current debate over whether various components of the ERP are exogenous or endogenous (Donchin, Ritter, & McCallum, 1978).

Recently, Parvin, Torres, and Johnson (1980) digitally filtered single-trial ERPs into three band-
widths, 1–7 Hz (delta/theta), 7–14 Hz (alpha), and 14–25 Hz (beta). The filtered epochs were averaged to obtain ERPs 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 latency, but its peak was usually between 150 and 300 ms poststimulus. Parvin et al. also obtained plots of average power within the 1–7 Hz and 7–14 Hz bands over the poststimulus epoch. Power in the 1–7 Hz band increased up until 300 ms and then subsided. In the 7–14 Hz band, power decreased after stimulus presentation, mirror-imaging the increase in low frequency power. Jasper (1936), likewise, had observed a slow positive wave developing after the point of alpha blocking, i.e., an increase in low frequency power coincident with a decrease in alpha power poststimulus.

Basar, Basar-Eroglu, Rosen, and Schutt (1984) similarly averaged stimulus omission responses into separate bandwidths. They found that, in some subjects, the stimulus omission response was characterized by a phase shifting and amplitude enhancement of activity in the delta/theta range. Basar et al. 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 prestimulus aspect, Basar et al. labeled the entrained low frequency harmonic as P300. Pritchard, Brandt, Shappell, O'Dell, and Barratt (1985) examined correlations between P300 amplitude and prestimulus EEG power in the delta, theta, and alpha bands. The P300 to target stimuli correlated positively with prestimulus alpha power, whereas the correlation of P300 amplitude with prestimulus delta power was negative and with prestimulus theta it was zero. While Pritchard et al.'s correlations provide further evidence for a positive relationship between P300 and prestimulus alpha activity, they do not support Basar et al.'s (1984) hypothesis that the P300 is derived from entrained prestimulus delta activity.

In our laboratory, while averaging small numbers of trials to study ERPs to rare stimuli, we noticed the phenomenon exemplified by the tracings presented in Figure 1. This is a single-trial response of the background EEG to a 100-ms duration, 1000 Hz tone pip, which was a rare (10%), 90dB intensity tone embedded in a sequence of (90%) 80dB, 1000 Hz tones. As can be seen, the stimulus was preceded by a 20–40 microvolt (µV) alpha spindle. The alpha activity blocked after the last positive phase of the spindle around 300 ms poststimulus. The last two cycles of the alpha spindle also seem to be riding on a slow positive shift. We hypothesized that this positive shift is a facet of alpha blocking and that it might be time-locked to stimulus presentation so as to contribute to, or even be responsible for, the P300. Given our observations and the suggestions of a relationship between prestimulus alpha and a poststimulus slow positive wave in the literature, we decided to investigate the effect of prestimulus alpha activity upon the P300 elicited by an infrequently occurring stimulus. Specifically, trials that are likely to evoke a P300 would be sorted into separate averages as a function of the amount of prestimulus alpha band activity.

STUDY I
Method
Subjects
The subjects were 7 volunteer graduate students (4 women, 3 men, median age = 29 yrs) from Queens College in Flushing, New York. All reported no history of hearing loss. All subjects had participated in prior EEG experiments and 6 of the 7 were known to have well developed alpha rhythms with their eyes open.

Stimuli
The stimuli were 100-ms duration, 1000 Hz binaural tone pips delivered through headphones. Rise/fall times were .25 ms. The tone pips were generated by a Hewlett Packard 200 CDR audio-oscillator. They were gated through switches under the control of a Heathkit H8 microprocessor and presented in pairs separated by 1.5 s. An LED 4 ft in front of the subject served as a visual fixation point when illuminated. Trial presentation was manually initiated on the microprocessor with an intertrial interval between 5 and 15 s (mean = 11 s).
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Procedure

Each subject sat in a chair while listening to the tone pips. The 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 response speed was not important but that accuracy was. The LED went on 1 s prior to S1 and was turned off by the subject's key press. Feedback was provided by the brief re-illumination of the fixation light .5 s 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.

Prior to data collection, each subject's intensity difference detection threshold had been determined by a descending staircase procedure. S1 was always set to 70dB SPL. On a random half of the staircase trials, S2 was set to 70dB SPL. On the remaining half, S2 was set to 80dB SPL and then decreased by 1dB on each trial until the subject's first failure to report a greater-than-70dB S2 as different from S1. Thereafter, S2 was adjusted by .1dB steps across 'different' trials until the subject achieved a criterion of 3/4 trials correctly reported. This value was used as the difference threshold in the ERP data collection block.

The total number of trials for each subject during recording was 160. On a random 40% of the trials, both S1 and S2 were 'same,' i.e., set to 70dB SPL. On another random 40%, S2 was set to a higher intensity which equaled the subject's 75% correct difference detection threshold. On the remaining 20% (32 trials), S2 was set to 10dB above the threshold intensity increase. Subjects were informed that an occasionally "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' with the same key press as they did to the other S2s detected at a threshold increase. The only constraints on the above randomization were that none of the 32 +10dB intensity increase trials could follow another, or be one of the first 5 trials in a block.

EEG Recording

EEG activity was recorded with Beckman Ag-AgCl electrodes from central (Cz) and parietal (Pz) midline sites according to the 10-20 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. 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 (~3dB attenuation) of 30 Hz and an upper bandpass (~3dB attenuation) of .03 Hz. The amplified EEg, along with the EOG and an external synchronizing pulse 500 ms prior to the onset of S1 on each trial, were stored on a Sangamo eight-channel FM tape recorder.

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 the 500 ms immediately prior to S2. The normalized RMS was calculated from the standard deviation of the filtered input so as to eliminate contribution by DC offset throughout the digitized period.

For each subject, 8 of the 32 rare trials with no associated EOG activity were selected as having the highest filtered alpha band RMS values for 500 ms prior to S2 (high alpha). Another 8 rare trials free of EOG activity were selected as having the lowest filtered alpha band RMS 500 ms prior to S2 (low alpha). The unfiltered EEG at Cz and Pz for the above selected trials was digitized by the H8 microprocessor for a period from 200 ms prior to and 1000 ms after S2 at a rate of 77 samples/s (13-ms resolution) and then averaged separately into high and low prestimulus alpha ERPs.

Results

The grand mean ERPs (N = 7) elicited by the rare (20%) increases in stimulus intensity are displayed in Figure 2. Correct detection of the highly discrepant increase was 100% for all subjects. The ERPs associated with high and low prestimulus alpha are superimposed. The ERPs reveal greater positivity around 300 ms in the high prestimulus alpha con-

![Figure 2. Grand-mean waveforms of average ERPs to rare increased intensity auditory stimuli. The ERPs were averaged from separate high and low prestimulus alpha trial bins. Grand-mean alpha prestimulus RMS levels are indicated beneath their linetype identifiers.](image-url)
dition. At Cz, the enhanced positivity appears mostly
in the P300 peak; while at Pz, it appears distributed
throughout the epoch from 100 ms to roughly
800 ms poststimulus. There does seem to be a
smaller N100 peak in the high alpha ERP in com-
parison to the low alpha ERP at Cz. The N100 is
poorly developed at Pz for both high and low alpha
conditions. The difference between the grand-mean
alpha band RMS levels for the high and low alpha
sorted trials is on the order of 34 μV.

P300 amplitude scores were obtained for each
subject’s data by baseline-to-peak measurement of the
most positive point within the waveform from
250–500 ms poststimulus. N100 amplitudes were
obtained by measuring the most negative point from
75–150 ms poststimulus. The measurements for
both peaks were submitted to separate repeated
measures analyses of variance with two levels of a
prestimulus alpha factor (high vs. low), and two
levels of an electrode factor (Cz vs. Pz). The analysis
of variance on the P300 measure yielded a significant
main effect of prestimulus alpha (F(1/6) =
46.71, p = .0005), but there was no significant effect
of electrode or an interaction between prestimulus
alpha and electrode. The analysis of variance on
the N100 measure yielded a significant main effect
of electrode (F(1/6) = 13.32, p = .02), but there was
no significant main effect of prestimulus alpha or
an interaction between the alpha and electrode fac-
tors.

Since the rare stimulus ERP in Study 1 was elic-
ted by increases in stimulus intensity, the question
of whether the alpha-sensitive P300 was actually
endogenous might be raised. The purpose of our
second study was to replicate Study 1 with a rare
declined intensity stimulus condition to test if the
alpha/P300 relationship holds when the ERP is elic-
ted by stimulus discrepancy per se. In addition, a
trials sort by poststimulus alpha RMS was per-
formed to test for an interaction of alpha density ×
alpha measurement period. Specifically, this
would be whether larger P300s were associated
with high prestimulus alpha and also low poststimulus
alpha. Such an interaction would indicate that it
was alpha blocking, i.e., high prestimulus alpha fol-
lowed by low alpha poststimulus, that was associ-
ated with larger P300s.

STUDY 2

Method

Subjects

The subjects were 11 normally-hearing volunteer
graduate and undergraduate students from Queens
College in New York, with no previous experience in
EEG experiments. They were 7 women and 4 men with
a median age of 27 yrs.

Stimuli

The stimulus presentation apparatus was the same
as that described in Study 1.

Procedure

Prior to data collection, each subject’s intensity dif-
ference threshold was determined by separate descend-
ing staircases for intensity increases and decreases. For
the intensity decrease threshold, the staircase pro-
cedure was performed with S1 set to 80dB SPL and S2
set initially to 70dB SPL.
The total number of trials (S1-S2 pairs) for each
subject during EEG recording was 320. These were
presented in 4 blocks of 80 trials each. For 2 of the
blocks, the intensity increase discrimination as de-
scribed in Study 1 was employed. For the remaining
2 blocks, S1 was set to 80dB, while S2 was randomly
set to 80dB (40% of the trials) or to a lower intensity
(another 40%) which equaled the subject’s 75% correct
detection threshold as determined by the staircase for
intensity decrease. The 4 blocks were presented in a
counterbalanced ABBA or BAAB fashion across sub-
jects.

On the remaining 20% (32) of the trials in the 2
intensity increase blocks, S2 was set to 10dB above
the 75% correctly detected threshold increase. On the
remaining 20% (32) of the trials in the 2 intensity de-
crease blocks, S2 was set to 10dB below the 75% cor-
rectly detected threshold decrease. Subjects were in-
structed to respond to S2 by differential keypress as in
Study 1. The rare S2s were to be responded to as ‘dif-
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**EEG Recording**

With the exception of the inclusion of two extra alpha electrode channels, Fz and O2, EEG recording was identical to that described in Study 1.

**EEG and ERP Analysis**

The measurement of filtered EEG RMS from the Fz electrode was the same as that described in Study 1. High and low prestimulus alpha ERPs were averaged separately for rare intensity increase and decrease trials as in Study 1. In addition, a second selection was performed on the same 32 rare trials in each intensity change condition to obtain those with the 8 highest and 8 lowest filtered alpha band RMS values from 00-1000 ms after S2 (high alpha post-S2 and low alpha post-S2). These trials were then averaged separately into high and low poststimulus alpha ERPs for each intensity condition for each subject.

**Experimental Analysis**

The experimental design consisted of a 2 x 2 x 2 x 4 factorial experiment with repeated measures over all subjects. The four independent variables were: 1) two levels of Alpha Density (high and low normalized MS), 2) two levels of Stimulus Condition (100% above and 100% below discriminable threshold), 3) two levels of Alpha Measurement Epoch (500 ms prior to S2 and 00-1000 ms after S2), and 4) four levels of Electrode location (Fz, Cz, Pz, and Oz).

Principal Components Analysis (PCA) of the averaged waveforms along with Varimax rotation was performed. The variates for the PCA were 352 waveforms (11 subjects x 4 electrodes 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 orthogonality. The factor scores were then subjected to repeated measures analyses of variance. To correct for possible inflated degrees of freedom due to non-orthogonality of the electrode factor, the degrees of freedom associated with each F-test which included its factor were reduced according to the method of Jennings and Wood (1976).

**Results**

**ERP Waveforms**

The grand-mean ERPs elicited by rare stimulus intensity changes sorted as a function of high and low levels of prestimulus alpha band RMS are depicted in Figure 3. Correct detection of the highly discrepant increases and decreases was 100% for all subjects.

The effect of prestimulus alpha upon the intensity increase ERP is seen most clearly at Fz. Here the high prestimulus alpha waveform exhibits a P300 that is 6 µV larger than the corresponding P300 of the low prestimulus alpha ERP. From Cz to O2 there is only a small increment in positivity (2 to 3 µV) for the P300 in the high alpha condition. This enhancement seems to diminish toward the posterior area.

In the intensity decrease condition, the effect of high prestimulus alpha appears as an overall displacement of the waveform toward positivity starting as early as 150 ms. The enhanced positivity is maximal at Cz and Pz, while also visible at Fz and O2. This produces greater amplitude (4-6 µV) for the slow positive wave around 300-600 ms poststimulus in the high alpha condition.

The grand-mean ERPs associated with high and low levels of poststimulus alpha are depicted in Figure 4. In contrast to the prestimulus alpha sorted data, there appears to be no effect of alpha RMS level upon the waveforms of the poststimulus alpha sorted ERPs.

**Principal Components Analysis**

Initial PCAs were performed to determine a common factor structure in the waveforms of the pre- and poststimulus alpha sorted data. The rotated factor loadings based on the covariance matrix from the two separate data sets are depicted in Figure 5. It is evident that only the first two factors can be matched on the basis of waveform and order of extraction. Furthermore, these two factors accounted for 75% of the variance after rotation in both data sets.

Factor 1 would appear to be associated with the late tonic positivity in the original waveforms. Brief
Figure 4. Grand-mean ERPs from the poststimulus alpha sorted data. Solid bars indicate the duration of the 100-ms tone burst. Vertical hatchings are at 100-ms intervals.

Study 2 Factor Loadings

Figure 5. First four extracted factor loadings from the pre- and poststimulus alpha sorted data sets. Large vertical hatching indicates stimulus onset, smaller hatchings are at 200-ms intervals. Solid lines are factor loadings from the prestimulus sorted data set, dotted lines are factor loadings from the poststimulus sorted data set.

Figure 6. P300 factor scores from the combined pre- and poststimulus alpha sorted ERP data set, collapsed across stimulus intensity. ●●● high prestimulus alpha; ◀-▶ low prestimulus alpha; ▼▼▼ high poststimulus alpha; ◁ initialised low poststimulus alpha.

...and poststimulus alpha sorted data sets. The factor scores of the same two loadings from a PCA on the entire data set were then submitted to separate repeated measures analyses of variance. No significant main effect or interaction with the alpha density factor was found for the factor scores of the first component (Slow Wave). The ANOVA on the factor scores of the extracted P300 component yielded a significant interaction between alpha density and alpha measurement epoch, $F(1/10) = 6.18$, $p<.04$. As can be seen from the plot of the P300 factor scores from the combined PCA (Figure 6), the poststimulus alpha sorted P300 factor scores at Cz and Pz lie midway between the high and low prestimulus alpha sorted P300 factor scores. This is what would be obtained if poststimulus alpha sorting had no effect on the P300. It should also be pointed out that the factor of stimulus condition (intensity increase vs. intensity decrease) did not interact significantly with alpha density.

**ANOVA on Baseline-to-Peak Measurements**

The ANOVA on the P300 factor scores from the combined data set demonstrated an interaction between alpha density and alpha measurement epoch but not between alpha density and direction of intensity change. To reinforce these findings, the same analysis using a baseline-to-peak measurement of the P300 was performed. On the basis of the latency 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 ms.

The results of the ANOVA on the baseline-to-peak P300 also produced a significant interaction...
between alpha density and alpha measurement epoch, \( F(1/10) = 7.31, p \leq 0.03 \). Again, the stimulus intensity factor did not interact with alpha density. Figure 7 shows that the baseline-to-peak P300 is maximal at C\(_z\) and P\(_z\) and is enhanced by prestimulus alpha. The poststimulus alpha sorted P300 amplitudes no longer lie between the high and low alpha levels of the prestimulus data, as they did in the factor scores plot (Figure 6), but are now of the same magnitude as the prestimulus low alpha P300.

**Discussion**

Study 2 provides evidence that the positive relationship between prestimulus alpha density and P300 amplitude is independent of stimulus intensity. This is consistent with the view that alpha affects the endogenous or psychological processes responsible for the P300, rather than the exogenous or stimulus-driven processes that can also contribute to this component (Roth, Doyle, Pfefferbaum, & Kopell, 1980). Both the factor scores and baseline-to-peak amplitudes of the P300 component are increased by the presence of prestimulus alpha. In this regard, there is convergence of the two types of measures. However, the baseline-to-peak measure (Figure 7) does not show the 'dilution' of the alpha density effect for poststimulus sorted data as do the factor scores (Figure 6). It appears that the extracted factor scores provide a more definitive measure of the alpha effect on the P300.

Even more striking is the disagreement between the formal analyses and the appearance of the grand-mean waveforms. The grand-mean waveforms suggest that there is a difference in the topography of the prestimulus alpha effect as a function of intensity change (Figure 3). Prestimulus alpha appears to enhance the P300 of the increased intensity ERP primarily at the frontal electrode. The effect of alpha in the decreased intensity condition seems to be more at the centro-parietal electrodes and more upon Slow Wave rather than P300. However, the ANOVAs on the P300 factor scores and baseline-to-peak measures did not reveal a significant interaction between alpha density, intensity change, and electrode. Nor did the ANOVA on Slow Wave find any significant effect of alpha density. Therefore, it cannot be concluded that there is any difference in the prestimulus alpha effect as a function of stimulus intensity or that there is any other alpha sensitive event-related 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 prestimulus alpha has little bearing on the amount of alpha blocking or suppression poststimulus. However, as indicated in Table 1, the high alpha levels poststimulus did not approach those found prestimulus. This attests to the fact that alpha was effectively blocked by most of the rare stimulus presentations. Alternatively, it could be that the duration of P300 amplitude-related alpha suppression may not outlast the P300 itself—it might be over by 500 ms, and thus would not have been measured in our design.

**GENERAL DISCUSSION**

A positive relationship between P300 and the alpha rhythm might seem counter-intuitive. The P300 is regarded as an indicator of an engaged processing state, while alpha has been considered a hallmark of mental quiescence (Brown, 1970). One hypothesis that could reconcile this apparent discrepancy is that the enhancement of P300 by prestimulus alpha activity represents a rebound phenomenon. The psychological states usually associated with alpha and P300 may be mutually exclu-
sive, but the onset of one could be more pronounced if preceded by predominance of the other.

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 facilitative 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 to the cortex. Of crucial importance for the appearance of alpha activity in both structures is the intrathalamic spread of rhythmic activity. Andersen and Andersson postulated that a type of interneuron or nucleus exists that can act as a distributor of intrathalamic synchrony. A possible candidate for such a role is the nucleus reticularis of the thalamus. The nucleus reticularis consists of a thin sheet of cells surrounding the thalamus. It receives inputs from and sends efficients to specific and nonspecific thalamic nuclei. Scheibel and Scheibel (1966), in noting this arrangement, suggested that the nucleus reticularis would be well suited in synchronizing thalamic discharges. Physiological studies have shown that the nucleus reticularis exerts an inhibitory effect on thalamic neurons (Schlag & Waszak, 1970, 1971). Thus, it could act as a thalamic pacemaker by synchronizing the excitatory period of thalamic cell groups through widespread inhibition.

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). They 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 during alpha would produce a pronounced excitatory rebound in thalamic nuclei which would appear cortically as a larger P300. The inhibitory influence is cascaded from MRF to nucleus reticularis to thalamus. Therefore, there can be 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.

REFERENCES


Research Positions in Neuromagnetism Laboratory

Two positions are available at the University of Texas Medical Branch, for researchers with strong technical background to help operate newly established magnetencephalography laboratory dedicated to the exploration of sensory, motor, and cognitive brain function. The lab, in addition to EEG instrumentation, includes a 7-channel SQUID system supported by an HP processor (UNIX) and a magnetically shielded room. Experience in signal processing instrumentation and computer science are highly desirable. Salary range $20,000-$40,000 depending upon qualifications. Send CV, explicit description of skills and work experience, sample publications (if available), and names of three references no later than December 1 to: Andrew C. Papanicolaou, PhD, Division of Neurosurgery E-17, UTMB, Galveston, TX 77550.

Postdoctoral Training in Gastrointestinal Psychophysiology

Johns Hopkins Medical School is currently offering a postdoctoral training program in gastrointestinal psychophysiology. The program includes training by psychologists and gastroenterologists in physiological measurement, automated data acquisition and analysis, psychological assessment, and behavioral treatment of gastrointestinal disorders. Eighteen months, beginning January 1, 1988, $18,000/year. Candidate should hold a PhD with training in psychophysiology. Send CV and references to: W.E. Whitehead, Digestive Diseases, Francis Scott Key Medical Center, Baltimore, MD 21224. Applicants will be considered without regard to race, religion, sex, or national origin. The Johns Hopkins University is an equal opportunity/affirmative action employer.

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Faculty members on sabbatical leave who are interested in psychophysiology are invited to apply for a (non-paying) position in the Institute for Stress Management at U.S. International University. Send applications to: F.J. McGuigan, Director, Institute for Stress Management, U.S. International University, 10455 Pomerado Road, San Diego, CA 92131.
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Announcement

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