P300 Topography of Amplitude/Latency Correlations

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Summary: The correlational association from 19 electrode sites between peak amplitude and latency for the P3(00) event-related brain potential (ERP) for n=80 homogeneous subjects was assessed using a simple auditory discrimination task. The correlation strength varied systematically across scalp topography in different ways for the various ERP components. For the target stimuli, P3 amplitude and latency were negatively correlated and most tightly coupled over the frontal-central and right medial/lateral recording sites. In contrast, the N1 produced negative correlations that were strongest over the left and right central/lateral locations; P2 demonstrated a positive correlation that was strongest frontally and centrally; N2 demonstrated a positive correlations that was strongest over the central and parietal sites. ERPs from the standard stimuli produced generally similar patterns for the P3 and P2 components, with only weak or no reliable effects observed for the N1 and N2 potentials. Taken together, the findings suggest that analysis of amplitude/latency correlational relationships can provide information about ERP component generation. Theoretical implications are discussed.

Key words: Event-related potentials (ERPs); P3(00); Amplitude; Latency; Correlations; Topography.

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Introduction

The P3(00) component often is elicited with a simple discrimination task. This procedure has been dubbed the "oddball" paradigm, since two stimuli are presented in a random series such that one of them occurs relatively infrequently - i.e., the oddball. The auditory version of this task uses two different tones, inter-stimulus intervals of one to three seconds, and the target stimulus occurring less frequently than the non-target or standard stimulus (e.g., probabilities of .20 and .80, respectively). The subject is required to distinguish between the two tones by responding to the target (e.g., mentally counting, pressing a button, etc.) and not responding to the standard. This task has been used to study a wide variety of information processing issues (e.g., Duncan-Johnson and Donchin 1977; Polich 1987, 1989ab, 1990; Squires et al. 1976; Verleger and Berg 1991; Woodward et al. 1991) and has been the paradigm most often employed when applied/clinical data are acquired.

The P3 is measured by quantifying its amplitude (size) and latency (timing). Amplitude (μV) is defined as the voltage difference between a prestimulus baseline and the largest positive-going peak of the ERP waveform within a latency range (e.g., 250-400 ms, although the range can vary depending on subject characteristics, stimulus modality, task conditions, etc.). Latency (ms) is defined as the time from stimulus onset to the point of maximum positive amplitude within the latency window. In addition, P3 scalp distribution is

defined as the change in component amplitude across the midline recording sites from the Fz (frontal), Cz (central), and Pz (parietal) locations. Scalp distribution effects for both amplitude and latency are of considerable import, since variation in P3 measures from the manipulation of task or subject variables has been used to infer information about the underlying neural generators (Johnson 1993; Polich and Heine 1996).

However, despite the importance of amplitude and latency values for assessing scalp distribution trends, the possible association between these two dependent measures has not been well characterized (Fabiani et al. 1987; Michaelewski et al. 1986; Segalowitz and Barnes 1993). In a study designed to assess normative P3 variation, Polich (1986) employed an auditory discrimination paradigm to elicit ERPs in a large, homogeneous sample (N=100) and found that P3 amplitude and latency were correlated negatively at the Fz, Cz, and Pz electrode sites—results that imply that individuals who produce large components do so relatively quickly compared to individuals who produce smaller components. A second study of two independent samples (n=72 and n=88) obtained similar results, except the strongest P3 amplitude/latency correlations occurred over the central and frontal recording sites, even when subjects repeated the same task or engaged in more difficult auditory discriminations (Polich 1992). Taken together, the findings from both reports suggest that the correlation between P3 amplitude and latency may be an inherent property of this component.

If this assertion is accurate, then the topography of P3 amplitude/latency correlations could index component scalp distribution differences that may reflect the size and/or orientation of the underlying neurophysiological generator(s) (Johnson 1989, 1993; Polich and Squire 1993; Verleger et al. 1994). The theoretical rational for this hypothesis stems from the assumption that a correlational association between P3 amplitude and peak timing taken across individuals indexes the relative inter-subject variation for component generation "strength" at a specific scalp location: Components that attain peak amplitude relatively quickly are assumed to be generated at that position more robustly than potentials that are not as large and/or that take longer to reach their peak latency. Systematic variation in the strength of these P3 amplitude/latency correlational associations over the scalp therefore might help localize the neural events underlying normal and abnormal ERP individual differences (Polich and Martin 1992; Polich et al. 1983, 1986, 1990). Alternatively, an absence of any systematic correlational associations over the scalp would imply that individual variability for P3 size and timing is unrelated to ERP generation across subjects.

The present study was conducted to determine which of these outcomes would obtain given the previous midline studies by assessing amplitude/latency correlational relationships for a large, homogeneous sample with 19 scalp electrodes. The ERP data were obtained from a previous study, the analytical details of which are reported elsewhere (Alexander et al. 1994). However, extended analysis of these results resulted in the evolution of a novel method for displaying correlational relationships. Because previous findings from just the midline electrodes hinted at a systematic relationship across scalp locations for P3 amplitude/latency correlation, this new technique was applied to determine if systematic P3 amplitude/latency correlational changes could be observed over the entire scalp.

Materials and Methods

Subjects

A total of 80 young adult males (mean age = 22.6, SD=1.8 yrs) were paid for their participation and were recruited through advertisements. All provided informed consent, reported normal hearing, no personal or familial neurological or psychiatric problems, and were screened for alcohol/drug use with a questionnaire. All subjects were right-handed as determined by a self-report questionnaire, derived from standard assessment methods (Bryden 1977).

Recording conditions and procedure

EEG activity was recorded at 19 electrode sites using an electrode cap (FP1/2, F3/4, C3/4, P3/4, F7/8, T7/8, P7/8, O1/2, Fz, Cz, Pz), referred to the nose with a forehead ground and impedance maintained at 5kΩ or less. Electro-ocular (EOG) activity was monitored by two electrodes placed at the outer canthus and above the left eye. The filter bandpass was .02-50 Hz (3 dB down, 6 dB octave/slope). The EEG was digitized at 3.9 ms/point for 1500 ms, with a 187 ms prestimulus baseline. The same computer was used to average the ERP data on-line, control stimulus presentation, and perform artifact rejection. Trials on which the EEG or EOG exceeded ±73.3 μV were rejected automatically.

ERPs were elicited with 400 auditory stimuli presented binaurally and consisting of 600 Hz and 1600 Hz tones presented at 60 dB SPL (10 ms r/f, 60 ms plateau), with an inter-stimulus interval of 1.5 s. The target tone occurred randomly with a probability of 0.125. Half the subjects had the 600 Hz tone as the target, and half had the 1600 Hz as the target. Presentation of the stimuli was concluded when 25 target and 75 standard artifact-free stimulus trials were acquired. Subjects were instructed to press a key pad with a forefinger as rapidly

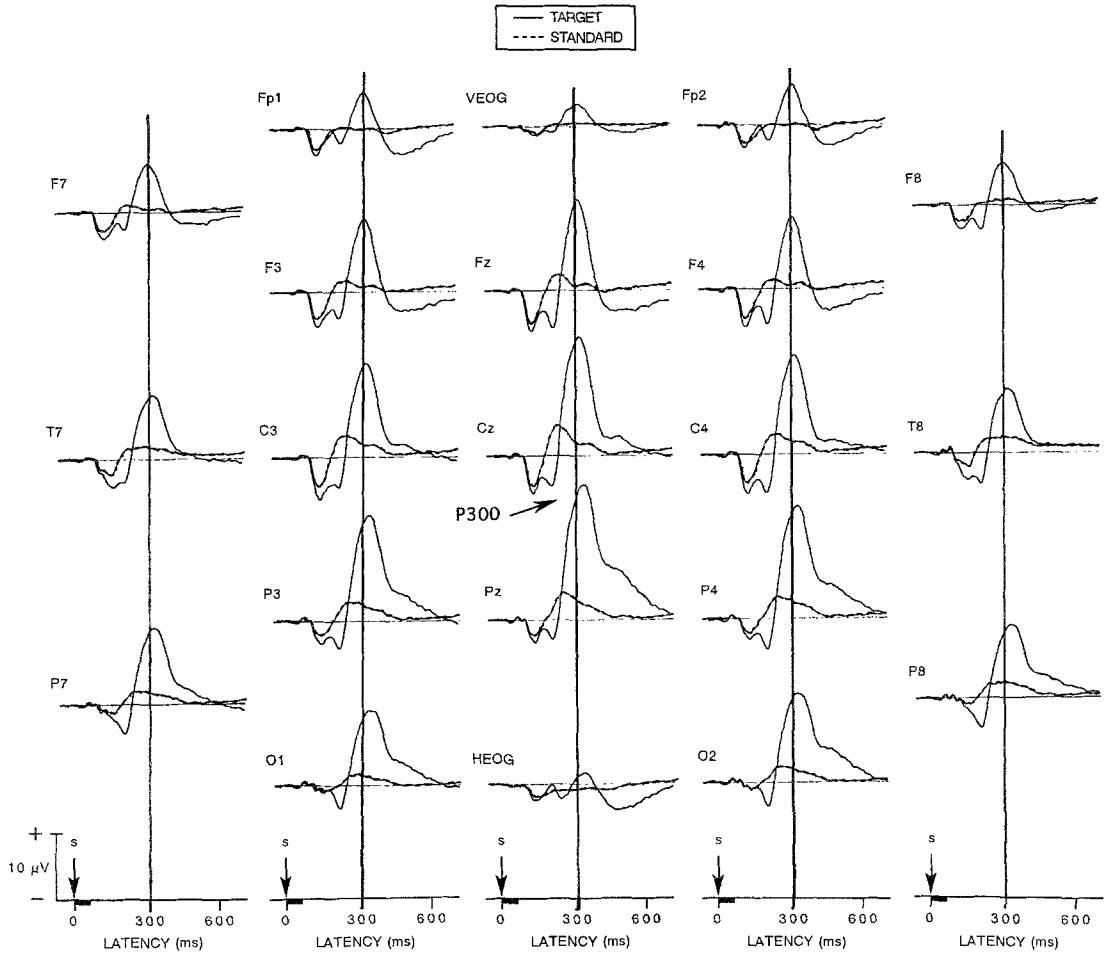

Figure 1. Grand averages for the target and standard stimuli from each electrode site (n=80). The horizontal line indicates the 300 ms latency point. Note that P300 components from the frontal recording sites generally occurred earlier and were morphologically sharper than those from the more posterior recording sites.

as possible whenever a target tone was detected and to refrain from responding to the standard tone. Response hand was counterbalanced across subjects.

Component measurement

Waveforms from each electrode for each condition were analyzed in the same fashion: Amplitudes and latencies of the N1,P2, N2, and P3 components at each electrode site were identified initially with a computer-assisted scoring program and confirmed visually. This procedure identified each component as the most negative or positive potential within the latency windows of 60-170, 100-275, 160-300, and 250-500 ms, respectively. Amplitude was measured relative to the prestimulus baseline, with peak latency defined as the time point of maximum positive or negative amplitude within the latency window. The ERP data were analyzed after signal averaging as described above, with no other filtering or smoothing algorithms applied.

Results

Performance analysis

Task performance was virtually perfect with fewer than 2% of the target trials missed across subjects. Mean response time was 379 ms. The grand average (n=80) ERP waveforms for the target and standard stimuli from each electrode position are illustrated in figure 1. Detailed analyses of the component scalp distribution, latency, and other data have been presented elsewhere and will not be considered here (Alexander et al. 1994).

Correlational analyses

The vertical line passing through each waveform in figure 1 demarcates the 300 ms latency time point. Note that P3 components from the front of the scalp generally peak earlier and are more sharply defined than those recorded from the back of the scalp. The correlation coefficients (Pearson's r) between the amplitude and

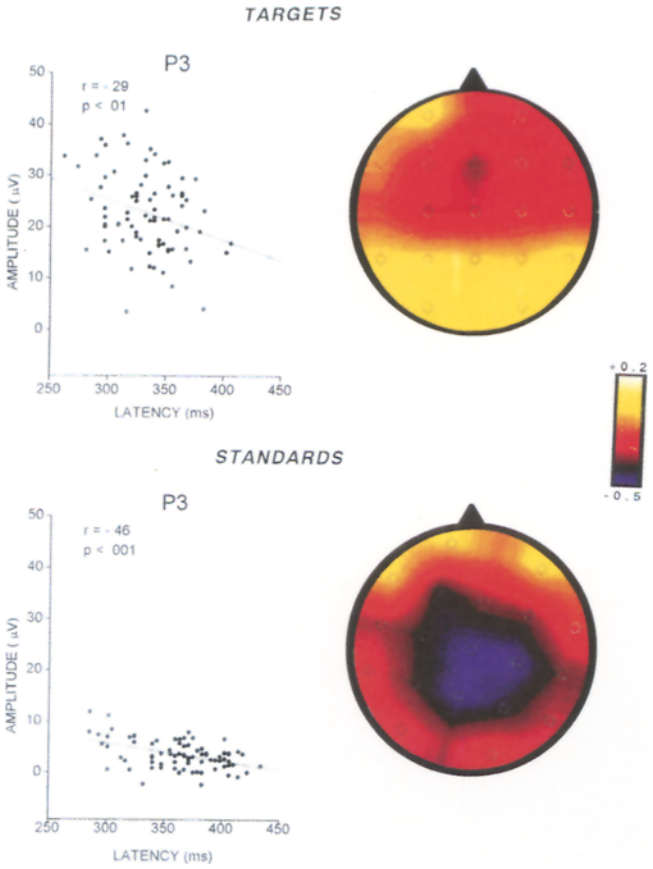

Figure 2. Left: Scattergrams of P300 amplitude and latency (Cz) for all subjects (n=80) from the target (top) and standard (bottom) stimuli. Right: Correlation coefficients for each electrode site plotted by linear interpolation of correlation values among individual sites. Statistical significance levels for each correlation are: r =.36, p<.05 and r=.42, p<.01 (df=78, 2-tail, Bonferonni corrected probabilities).

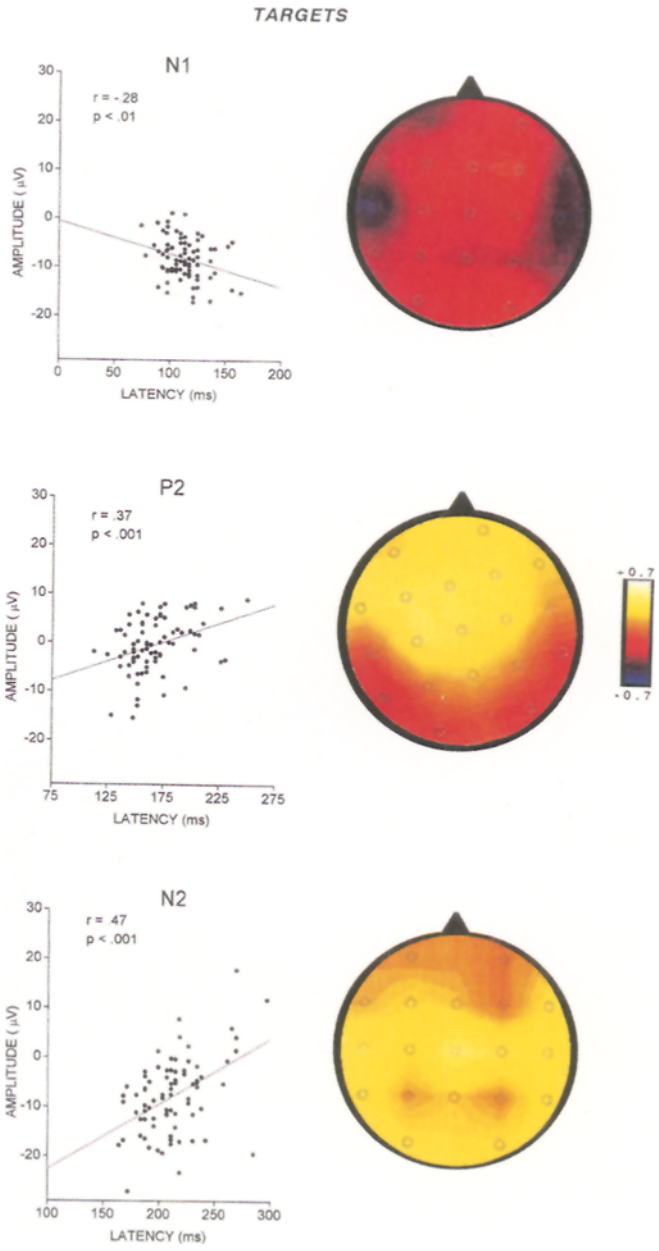

Figure 3. Left: Scattergrams of N1, P2, and N2 amplitude and latency (Cz) for all subjects (n=80) from the target stimuli. Right: Correlation coefficients for each component and electrode site plotted by linear interpolation of r values among individual sites. Statistical significance levels for each correlation are: r = .36, p<.05 and r = .42, p<.01 (df=78, 2-tail, Bonferonni corrected probabilities).

latency values from each electrode site across the n=80 subjects were computed for each component from each task condition. Figure 2 presents the correlational data for the P3 data from the target and standard stimuli. The scattergrams (left side) illustrate the general relationship between P3 amplitude and latency and were obtained from the Cz electrode site. The color topography maps (right side) illustrate the correlational strength at each of the 19 active scalp electrode sites, with the inter-electrode values obtained through linear interpolation. Figure 3 presents the target stimulus N1, P2, and N2 results; figure 4 presents the standard stimulus N1, P2, and N2 results in a similar manner. The correlational scales on the figure for the P3 component (+.20 to -.50) are different than for those used on the figures for the N1, P2, and N2 components (+.70 to -.70).

Statistical significance patterns

The statistical reliability of the correlational values should be considered in several ways. First, for the purposes of the present study the overall pattern of correlational variation is more critical than the specific numerical value obtained at a particular electrode site.

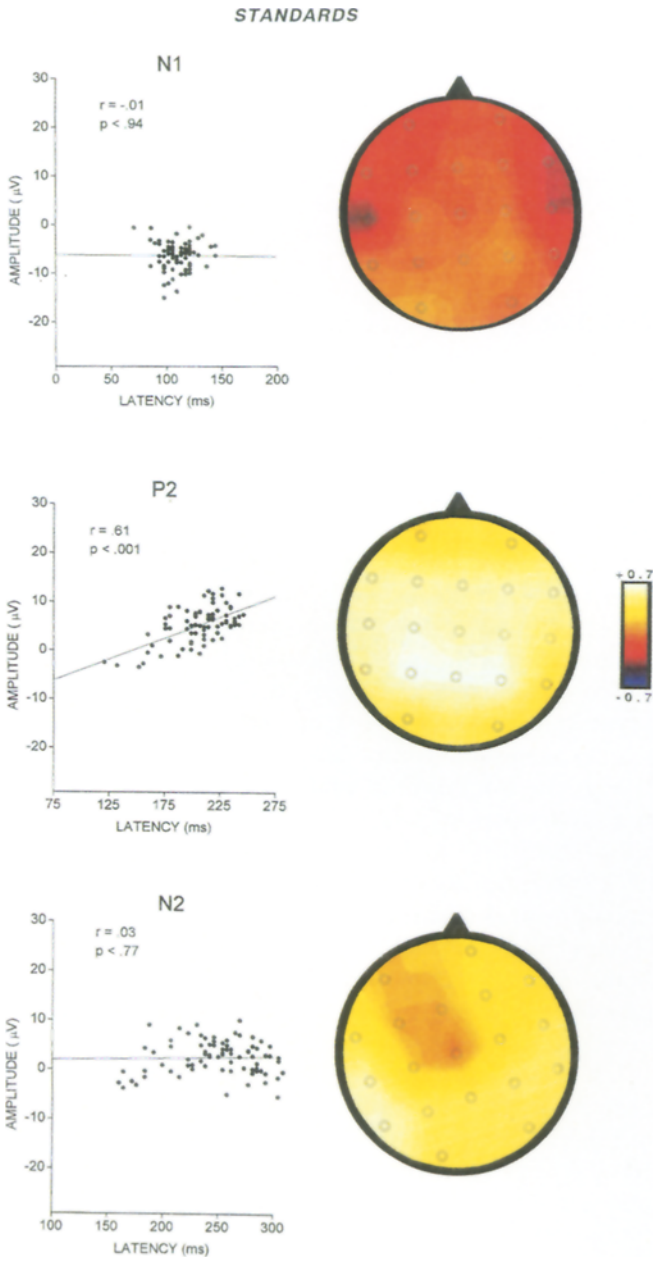

Figure 4. Left: Scattergrams of N1, P2, and N2 amplitude and latency (Cz) for all subjects (n=80) from the standard stimuli. Right: Correlation coefficients for each component and electrode site plotted by linear interpolation of r values among individual sites. Statistical significance levels for each correlation are: $r = .36$, $p<.05$ and $r = .42$, $p<.01$ (df=78, 2-tail, Bonferonni corrected probabilities).

As outlined above, systematic changes in the strength of the correlation values over the scalp are hypothesized to reflect systematic inter-individual variation for the amplitude/latency association, which can be considered as a measure of the relative neural generator potency at that specific scalp location. Thus, how the correlation pattern varies across the scalp is more important than the specific correlational value itself.

Second, each individual electrode location can be viewed as producing an amplitude/latency correlation that, even though it may be physically near the surrounding electrodes, is essentially independent in that it reflects activity at only one electrode location. Put another way, if only one electrode was assayed at any point on the scalp using a two-tailed test (df=78) of the correlation's strength would be evaluated with significance levels of: $r=.22$, $p<.05$ and $r=.28$, $p<.01$. However, since the majority of the correlations are significant using these criteria, it is the overall pattern of values that should be used to assess the underlying neural activity variation.

Third, because separate correlation coefficients were computed for 19 electrode sites that were all located at the same positions for each subject, the relationships among adjacent electrode sites also can be considered as inter-dependent rather than independent since the effects of neural activation at a particular locus is not restricted to a few centimeters' distance at the scalp. In this view, the significance levels should therefore take into account the multiple assessments performed, even though most of the inter-electrode spatial relationships are not adjacent or even close to one another. A robust approach to this issue employs Bonferroni corrections for multiple *t*-test comparisons (i.e., the basis for evaluating correlational significance), so that the significance levels corrected for 19 multiple comparisons using a two-tailed (df=78) test are: $r = .36$, $p<.05$ and $r=.42$, $p<.01$. These values represent a statistically very conservative estimate of the significance level so the pattern of correlational changes across electrode locations should be evaluated using these values.

P3 component (figure 2)

For the target stimulus, the P3 amplitude/latency negative correlations were strongest over the frontal/central electrodes, with a marked area increase in correlational strength observed over the right frontal hemisphere. It is noteworthy too that even though P3 amplitude is largest over the parietal areas, the strongest association between component amplitude and peak latency is distributed frontally (see figure 1). Assuming that the amplitude/latency correlations reflect the strength and timing of component generation, this pattern suggests that at least some portion of the P3 component is produced initially near the frontal recording sites such that the correlational patterns may index alerting processes that are engaged to effect subsequent task execution. The weaker amplitude/latency association observed at the parietal electrode locations could reflect posterior cortical activity stemming from attentional re-

source allocation—a processing event that could easily add appreciable amplitude/latency variability across subjects to produce the observed decrease in correlation strength over this part of the scalp. For the standard stimulus, a similar but somewhat stronger pattern of correlational effects was observed, with the most robust amplitude/latency associations again found at the frontal/central electrodes. However, because the subject was not required to respond to the standard, this correlational pattern indicates similar initial processing as that engaged by the target stimulus but without any additional process incurred since no response or subsequent processing was required. Thus, the different patterns of correlational strengths for the target compared to standard stimulus events may reflect the activation of different neural substrates that differentially respond to the stimulus-defined processing requirements.

N1, P2, N2 components (figures 3 and 4)

The N1, P2, and N2 components produced different patterns for the target compared to standard stimulus conditions. This is not unexpected, given the likely effects of the relatively large P3 component present for the target trials, whereas only a minimal corresponding component was observed for the standard stimuli (see figure 1).

N1 amplitude/latency correlations were strongest for the target stimuli and distributed such that the maximum correlations were observed for the lateral/temporal electrodes—i.e., over the loci associated with the generation of the N1 potential. A similar pattern was obtained for the N1 components from the standard stimuli, although the associations between amplitude and latency values were weaker they still reflected activity of the same lateral/temporal neural sources. The N1 amplitude/latency differences between the two stimulus types may originate from attentional processing garnered by the target stimuli compared to the relative lack of any cognitive operations surrounding standard stimulus processing.

P2 amplitude/latency correlations were generally positive across the scalp, with the strongest associations obtained over the central recording sites for both the target and standard stimuli. Indeed, the correlational relationships were quite strong for the data from the standard stimuli, which demonstrated a greater central/parietal distribution compared to the target stimuli. Because the standard stimulus results were stronger than those obtained from the target stimuli, it is likely that this outcome reflects the P2 component's sensitivity to the sensory rather than cognitive aspects of the task situation.

N2 amplitude/latency correlations were stronger for the target compared to standard stimuli, although the distributional patterns for both stimulus types were similar. However, because the N2 is closely related to the P3 component, it is likely that the correlational associations found for the target stimuli occurred primarily because N2 amplitude and latency were affected by generation of the P3 component. The lack of similar effects for the standard stimulus correlations supports this view.

Discussion

The correlational scalp patterns indicate that P3 amplitude and latency are negatively correlated across electrodes in the same fashion as observed previously for the midline electrodes (Polich 1986, 1992). Although the exact cause of this association is uncertain, it is reasonable to suppose that the inter-subject variation that contributes to P3 amplitude/latency correlational strength at particular electrode locations reflects the amount of neural activity underlying that site-at least for the auditory stimuli and a button press discrimination task used here. The correlation patterns observed across the scalp for the target stimuli suggest that an initial attention-related activity is engaged for the target stimuli, which appears to originate from right-frontal sources. This asymmetry may occur because greater alpha band power is found for right- compared to left-frontal areas (Davidson 1988, 1992; Tomarken et al. 1992), and variation in background alpha power is correlated positively with P3 amplitude (Basar et al. 1984; Intriligator and Polich 1994, 1995; Jasiukaitis and Hakerem 1988). Further, this finding is in agreement with previously reported P3 amplitude hemispheric asymmetries that are larger over right- compared to left-frontal/central locations (Alexander et al. 1995, 1996; Holinger et al. 1992; Naumann et al. 1992; Polich and Heine 1996). Thus, it is not unreasonable to conclude that the patterns of consistent amplitude/latency correlations may index an initial right frontal neural activation that is related to subsequent P3 component generation across the scalp.

A major theoretical interpretation of the P3 posits that it reflects a developing representation within short-term memory (Donchin and Coles 1988; Donchin et al. 1986; Polich and Kok 1995). This hypothesis is supported by human lesion studies, which suggest that P3 voltage patterns observed at the scalp originate from multiple neural loci that include temporal-parietal cortex (Johnson, 1989; Knight et al. 1989; Verleger et al. 1994; Yamaguchi and Knight 1991). In addition, however, particularly alerting stimuli will elicit an earlier subcomponent, dubbed the "P3a" (Courchesne et al. 1975; Katayama and Polich, 1997; Polich 1988; Squires et al. 1975), which is largest over frontal/central scalp locations and reflects initial signal evaluation (Ford et al. 1976; Knight 1984). Presentation of a target stimulus in an oddball task may

therefore elicit a P3a component initially and, when subsequent attentional resource and memory operations are engaged, the parietal maximum canonical P3b (Knight 1990; Polich and Squire 1993). Thus, it is likely that at least two different but related sets of neural activities comprise the "P3" component: an early frontal source and a later parietal locus.

Given this background, the frontal-central P3 amplitude/latency correlations observed in the present study may be reflecting the neurocognitive operations underlying the fundamental discrimination process required in the oddball paradigm. The somewhat larger negative correlations over the right frontal hemisphere electrode locations may stem in part from neural activity related to the processing of the incoming signal in a manner similar to effects observed using positron emission tomography (Posner and Petersen 1990). Discriminating the target from a standard stimulus could initiate right frontal engagement, because such processing requires the consistent application of attentional focus-a major attribute of frontal lobe function (Pardo et al. 1991; Posner 1992). If true, then the P3 amplitude/latency correlational associations observed here may be considered as a scalp signature of this neurocognitive system. The decrease in P3 correlational strength over the parietal areas and target/standard correlational pattern differences would occur because of subsequent subject-variable (temporal/parietal) neural activity for the target stimulus only, so that the different correlational patterns for the two stimulus types supports this view. Thus, P3 amplitude/latency correlations and their scalp distribution patterns may be a relatively more informative index of the theoretical mechanisms underlying component generation than independent assessments of each variable taken separately.

References

Alexander, J.E., Polich, J., Bloom, F.E., Bauer, L.O., Kuperman, S., Rohrbaugh, J., Morzorati, S., O'Connor, S.J., Porjesz, B. and Begleiter, H. P300 from an auditory oddball task: Inter-laboratory consistency. Int. J. Psychophysiol., 1994, 17: 35-46.

Alexander, J.E., Porjesz, B., Bauer, L.O., Kuperman, S., Morzorati, S., O'Connor, S.J., Rohrbaugh, J., Begleiter, H. and Polich, J. P300 hemispheric amplitude asymmetries from a visual oddball task. Psychophysiology, 1995, 32: 467-475.

Alexander, J.E., Bauer, L.O., Kuperman, S., Morzorati, S., O'Connor, S.J., Rohrbaugh, J., Porjesz, B., Begleiter, H. and Polich, J. Hemispheric differences for P300 amplitude from an auditory oddball task. Int. J. Psychophysiol., 1996, 21: 189-196.

Basar, E., Basar-Eroglu, C., Rosen, R and Schütt, A. A new approach to endogenous event-related potentials in man: relation between EEG and P300-wave. Int. J. Neurosci., 1984, 24: 1-21.

Bryden, M.P. Measuring handedness with questionnaires. Neuropsychologia, 1977, 15: 617-624.

Courchesne, E., Hillyard, S. and Galambos, R. Stimulus novelty, task relevance, and the visual evoked potential in man. Electroenceph. Clin. Neurophysiol., 1975, 39: 131-143.

Davidson, R.J. EEG measures of cerebral asymmetry: Conceptual and methodological issues. Int. J. Neurosci., 1988, 39: 71-89.

Davidson, R.J. Emotion and affective style: Hemispheric substrates. Psychol. Sci., 1992, 3: 39-43.

Donchin, E. and Coles, M.G.H. Is the P300 component a manifestation of context updating? Brain Behav.l Sci., 1988, 11: 357-374.

Donchin, E., Karis, D., Bashore, T.R., Coles, M.G.H. and Gratton. Cognitive psychophysiology and human information processing. In: M.G.H. Coles, E. Donchin and S.W. Porges (Eds.), Psychophysiology: systems processes, and applications. New York: The Guilford Press, 1986: 244-267.

Duncan-Johnson, C.C. and Donchin, E. On quantifying surprise: The variation of event-related potentials with subjective probability. Psychophysiology, 1977, 14: 456-467.

Fabiani, M., Gratton, G., Karis, D. and Donchin, E. The definition, identification, and reliability of measurement of the P300 component of the event-related brain potential. In: P.K. Ackles, J.R. Jennings and M.G.H. Coles (Eds.), Advances in Psychophysiology, Vol 2. Greenwich, CT: JAI Press Inc., 1987: 1-78.

Ford, J., Roth, W.T. and Kopell, B. Auditory evoked potentials to unpredictable shifts in pitch. Psychophysiology, 1976, 13: 32-39.

Holinger, D., Faux, S., Shenton, M., Sokol, N., Seidman, L., Green, A. and McCarley, R. Reversed temporal region asymmetries of P300 topography in left and right-handed schizophrenic subjects. Electroenceph. Clin. Neurophysiol., 1992, 84: 532-537.

Intriligator, J. and Polich, J. On the relationship between background EEG and the P300 event-related potential. Biol. Psychol., 1994, 37: 207-218.

Intriligator, J. and Polich, J. On the relationship between EEG and P300 variability. Int. J. Psychophysiol., 1995, 20: 59-74.

Jasiukaitis, P. and Hakerem, G. The effect of prestimulus alpha activity on P300. Psychophysiology, 1988, 25: 157-165.

Johnson, R. Auditory and visual P300s in temporal lobectomy patients: Evidence for modality-dependent generators. Psychophysiology, 1989, 26: 633-650.

Johnson, R. On the neural generators of the P300 component of the event-related potential. Psychophysiology, 1993, 30: 90-97.

Katayama, J. and Polich, J. Stimulus context determines P3a and P3b. Psychophysiology, in press 1997.

Knight, R.T. Decreased response to novel stimuli after prefrontal lesions in man. Electroenceph. Clin. Neurophysiol., 1984, 59: 9-20.

Knight, R.T. Neural mechanisms of event-related potentials from human lesion studies. In: J. Rohrbaugh, R. Parasuraman and R. Johnson (Eds.), Event-Related Brain Potentials: Basic Issues and Applications. New York: Oxford University Press, 1990: 3-18.

Knight, R.T., Scabini, D., Woods, D. and Clayworth, C. Contributions of temporal-parietal junction to the human auditory P300. Brain Res., 1989, 502: 109-116.

Michaelewski, H.J., Prasher, D.K. and Starr, A. Latency variability and temporal interrelationships of the auditory event-related potentials (N1, P2, N2, and P3) in normal subjects. Electroenceph. Clin. Neurophysiol., 1986, 65: 59-71.

Naumann, E., Huber, C., Maier, S., Plihal, W., Wustmans, A., Diedrich, O. and Bartussek, D. The scalp topography of P300 in the visual and auditory modalities: A comparison of three normalization methods and control of statistical type II error. Electroenceph. Clin. Neurophysiol., 1992, 83: 254-264.

Pardo, J.V., Fox, P. and Raichle, M. Localization of human system for sustained attention by positron emission tomography. Nature, 1991, 349: 61-64.

Picton, T.W. The P300 wave of the human event-related potential. J. Clin. Neurophysiol., 1992, 9: 456-479.

Polich, J. Normal variation of P300 from auditory stimuli. Electroenceph. Clin. Neurophysiol., 1986, 65: 236-240.

Polich, J. Task difficulty, probability, and inter-stimulus interval as determinants of P300 from auditory stimuli. Electroenceph. Clin. Neurophysiol., 1987, 68, 311-320.

Polich, J. Bifurcated P300 peaks: P3a and P3b revisited? J. Clin. Neurophysiol., 1988, 5: 287-294.

Polich, J. Frequency, intensity, and duration as determinants of P300 from auditory stimuli. J. Clin. Neurophysiol., 1989a, 6: 277-286.

Polich, J. Habituation of P300 from auditory stimuli. Psychobiology, 1989b, 17: 19-28.

Polich, J. P300, probability, and inter-stimulus interval. Psychophysiology, 1990, 27: 396-403.

Polich, J. On the correlation between P300 amplitude and latency. Bull. Psychon. Soc., 1992, 30: 5-8.

Polich, J. and Heine, M.R.D. P300 topography and modality effects from a single-stimulus paradigm. Psychophysiology, 1996, 33: 747-752.

Polich, J. and Kok, A. Cognitive and biological determinants of P300: An integrative review. Biol. Psychol., 1995, 37: 207-218.

Polich, J. and Martin, S. P300, cognitive capability, and personality: A correlational study of university undergraduates. Pers. Ind. Diff., 1992, 3: 533-543.

Polich, J. and Squire, L. P300 from amnesiac patients with bilateral hippocampal lesion. Electroenceph. Clin. Neurophysiol., 1993, 86: 408-417.

Polich, J., Howard, L. and Starr, A. P300 latency correlates with digit span. Psychophysiology, 1983, 20: 665-669.

Polich, J., Ladish, C. and Burns, T. Normal variation of P300 in children: Age, memory span, and head size. Int. J. Psychophysiol., 1990, 9: 237-248.

Polich, J., Ehlers, C.L., Otis, S., Mandell, A.J. and Bloom, F.E. P300 latency reflects the degree of cognitive decline in dementing illness. Electroenceph. Clin. Neurophysiol., 1986, 63: 138-144.

Posner, M.I. Attention as a cognitive and neural system. Cur. Dir. Psychol. Sci., 1992, 1: 11-14.

Posner, M.I. and Petersen, S.E. The attention system of the human brain. Ann. Rev. Psychol., 1990, 13: 25-42.

Segalowitz, S. and Barnes, K. The reliability of ERP components in the auditory oddball paradigm. Psychophysiology, 1993, 30: 451-459.

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

Squires, N.K., Squires, K.C. and Hillyard, S. Two varieties of long-latency positive waves evoked by unpredictable auditory stimuli in man. Electroenceph. Clin. Neurophysiol., 1975, 38, 387-401.

Tomarken, A.J., Davidson, R.J., Wheeler, R. and Kinney, L. Psychometric properties of resting anterior EEG asymmetry: Temporal stability and internal consistency. Psychophysiology, 1992, 29: 576-592.

Verleger, R., Heide, W., Butt, C. and Kömpf, D. Reduction of P3b in patients with temporo-parietal lesions. Cog. Brain Res., 1994, 2: 102-116.

Woodward, S., Brown, W., Marsh, J. and Dawson, M. Probing the time-course of the auditory oddball P3 with secondary reaction time. Psychophysiology, 1991, 28: 609-618.

Yamaguchi, S. and Knight, R. Age effects on the P300 to novel somatosensory stimuli. Electroenceph. Clin. Neurophysiol., 1991, 78: 297-301.