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P300 hemispheric amplitude asymmetries from a visual oddball task

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Abstract

The P3(00) event-related potential (ERP) was elicited in 80 normal, right-handed male subjects using a simple visual discrimination task, with electroencephalographic (EEG) activity recorded at 19 electrodes. P3 amplitude was larger over the right than over the left hemisphere electrode sites primarily at anteromedial locations (F3/4, C3/4) for target, novel, and standard stimuli. The N1, P2, and N2 components also demonstrated hemispheric asymmetries. The strongest P3 hemispheric asymmetries for all stimuli were observed at anterior locations, suggesting a frontal right hemisphere localization for initial stimulus processing, although target stimuli produced larger P3 amplitudes at parietal locations than did novel stimuli. The relationships of hemispheric asymmetries to anatomical variables, background EEG activity, and neurocognitive factors are discussed.

Descriptors: Event-related potentials, P3(00), Hemispheric differences

Hemispheric differences are observed readily using behavioral techniques with auditory (Ivry & Lebby, 1993; Kimura, 1993), visual (Hellige, 1993; Polich, 1993; Polich & Morgan, 1994; Sergent, 1991), and tactile (O'Boyle, van Wyhe-Lawler, & Miller, 1987; Reitan, Wolfson, & Hom, 1992) stimuli. Similar approaches have been employed using electroencephalographic (EEG) measures, with consistent laterality effects obtained in a wide variety of studies (e.g., Alexander & Sufka, 1993; Davidson, 1992; Davidson, Chapman, Chapman, & Henriques, 1990; Gevins et al., 1979; Tomarken, Davidson, Wheeler, & Doss, 1992). Further, despite a general impression that cognitive event-related potentials (ERPs) are fairly symmetrical in amplitude about the midline (Donchin, Kutas, & McCarthy, 1977), hemispheric asymmetries for the P3(00) also have been found under task conditions that encourage differential cerebral processing (e.g., Gevins et al., 1983; Kok & Rooyakkers, 1986; Rugg & Beaumont, 1978; Schweinberger & Sommer, 1991; Tenke, Bruder, Towey, Leite, & Sidtis, 1993; van de Vijver, Kok, Bakker, & Bouma, 1984). Thus, reliable hemispheric differences can be found with behavioral and electrophysiological measures.

The advent of multielectrode recording and topographical mapping techniques has prompted a reassessment of hemispheric ERP differences. In particular, several recent studies employing a relatively large number of electrodes have demonstrated that P3 amplitude in normal subjects is greater over the right than over the left cerebral hemisphere when a simple auditory oddball paradigm is used to elicit the ERPs, that is, hemispheric asymmetries are found in the absence of lateralized stimulus or task influences (Alexander et al., 1994; Holinger et al., 1992; Karniski & Blair, 1989; Naumann et al., 1992). Additional studies on schizophrenic patients (and normal controls) have yielded similar effects (Faux et al., 1993; McCarley et al., 1992; Morstyn, Duffy, & McCarley, 1993) and suggest that P3 amplitude from simple auditory discrimination tasks may be asymmetric

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across the hemispheres. However, although such hemispheric asymmetries have been observed they are not completely consistent in their strength or location. The sources of these discrepancies are not clear but may be related to the nonhomogeneous samples that are compromised by a lack of control over variables that can affect laterality differences (e.g., left/right handedness, male/female subjects, press/count tasks, etc.), the relatively small samples that are typically employed (e.g., $n = 10$–20), and the use of linked ears or mastoids as a reference (this method does not appear to affect asymmetry magnitudes appreciably, but it has caused some controversy in this area; cf. Andino et al., 1990; Faux et al., 1990; Nunez, 1981; Senulis & Davidson, 1989). When taken together in this context, previous studies suggest a possible P3 hemispheric difference for nonlateralized stimulus presentations and simple tasks, but the nature of these effects is still uncertain.

The present study was conducted to ascertain whether P3 amplitude laterality asymmetries are reliable and to assess systematically the role of stimulus and task parameters on hemispheric effects. A simple visual oddball discrimination task was employed in which stimuli consisted of an infrequently presented target, infrequently presented novel patterns, and frequently presented standards. Subjects were instructed to respond only to the target stimuli and not to the other stimulus types. The infrequent novel stimuli were presented to engage the discrimination mechanism in the absence of a response requirement but with the same stimulus probability as the target stimulus in a manner used previously (Courchesne, 1978; Courchesne, Courchesne, & Hillyard, 1978; Friedman, Simpson, & Hamberger, 1993; Pfefferbaum, Ford, Roth, & Kopell, 1980). Although presentation of an infrequent nontarget stimulus can alter P3 scalp distribution and peak latency in some populations (Fein & Turetsky, 1989; Turetsky, Raz, & Fein, 1988), this approach was adopted in the present study to examine whether a response to an infrequent stimulus contributes to P3 hemispheric differences. If, as suggested by auditory studies, the P3 component is larger over the right than over the left frontal regions, then similar lateralized amplitude effects should be obtained for visual stimuli, with the influence of stimulus type, probability, and response requirements assayed directly.

Methods

Subjects

A total of 80 young adult right-handed males (age: $M = 22.6$ years, $SD = 1.8$ years) served as subjects for pecuniary remuneration. Only males were used to maximize the likelihood of obtaining cognition-based hemispheric differences (Halpern, 1992). All subjects reported an absence of psychiatric or neurologic problems and were screened for alcohol and drug use. Handedness was evaluated by self-report of dominant hand, foot, and eye use in addition to direct observation of writing. All subjects had normal or corrected-to-normal vision.

Recording Conditions and Procedure

EEG activity was recorded monopolarly using an electrode cap at 19 electrode sites (Fp1/2, F3/4, C3/4, P3/4, F7/8, T7/8, P7/8, O1/2, Fz, Cz, Pz; see Scharbrough et al., 1990) referred to the nose, with a forehead ground and impedances maintained at 5 kΩ or less. The electrooculogram (EOG) was assessed with two channels referred to the nose. One electrode was placed at the outer canthus of the left eye to measure vertical eye movement, and the second electrode was located on the forehead to monitor horizontal eye movement. The filter bandpass was 0.02–50 Hz (3 dB down, 6 dB octave/slope). The EEG was digitized at 3.9 ms/point for 1,500 ms, with a 187-ms prestimulus baseline. ERP data were averaged on line with the same computer used to control the stimulus presentation and artifact rejection. Trials in which the EEG or EOG exceeded $\pm 73.3\ \mu V$ were rejected automatically.

ERPs were elicited with 280 stimuli presented on a computer monitor for a duration of 60 ms, with an interstimulus interval of 1.6 s. The target stimulus was a white "X" (4×4 cm, $2.9° \times 2.9°$), novel stimuli (5×5 cm, $3.6° \times 3.6°$) consisted of nonrepeating colored geometric shapes (e.g., blue hexagons, red pentagons, green triangles) arranged in variegated patterns, and the standard stimulus was a white square (4×4 cm, $2.9° \times 2.9°$). All stimuli were viewed from a distance of 110 cm, with low level, diffuse ambient lighting provided by a ceiling fixture. The target and novel stimuli each occurred with a probability of .125; the standard stimuli occurred with a probability of .75. Subjects were instructed to focus on a dot located in the center of the monitor, to press a key pad with their forefinger whenever a target stimulus was detected, and to refrain from responding when the novel or standard stimuli occurred. Response hand was counterbalanced across subjects. Stimulus presentation was concluded when 25 target, 25 novel, and 150 standard artifact-free ERP trials were acquired; time on task ranged from 7 to 10 min.

Component Measurement and Analyses

Waveforms for the target, novel, and standard stimuli were assessed visually and individually for each subject to identify amplitudes and latencies of the P1, N1, P2, N2, and P3 components at each electrode site by locating the most positive or negative component within the latency windows of 150–250, 200–300, 250–350, 300–400, and 350–600 ms, respectively. Amplitude was measured relative to the mean of the prestimulus baseline, with peak latency defined as the time point of maximum positive or negative amplitude within the latency window.

Results

All analyses of variance (ANOVAs) employed Greenhouse-Geisser corrections to the degrees of freedom; only probability values from corrected degrees of freedom are reported. Task performance was nearly perfect, with the total number of errors (misses and false alarms) at 0.4% across subjects. Mean response time for the target stimuli for all subjects was 440 ms ($SD = 57.6$, range = 318–559 ms).

The grand average ERP waveforms for the target, novel, and standard stimuli at each electrode position are presented in Figure 1. The effects of stimulus type on P3 component values from the midline electrodes (Fz, Cz, Pz) were straightforward. The P3 scalp distributions were similar, albeit not identical, and the amplitude decreased in magnitude for the target, novel, and standard stimuli, respectively. P3 latency decreased slightly from the frontal to parietal electrodes and became shorter for the target, novel, and standard stimuli, respectively. Similarly, the typical amplitude, latency, and scalp distribution results were obtained for the other components. Because the focus of the present study is on hemispheric asymmetries, data from the midline electrode sites will not be considered further.

To assess within-hemisphere medial/lateral electrode differences, the left lateral (F7, T7, P7), left medial (F3, T3, P3), right

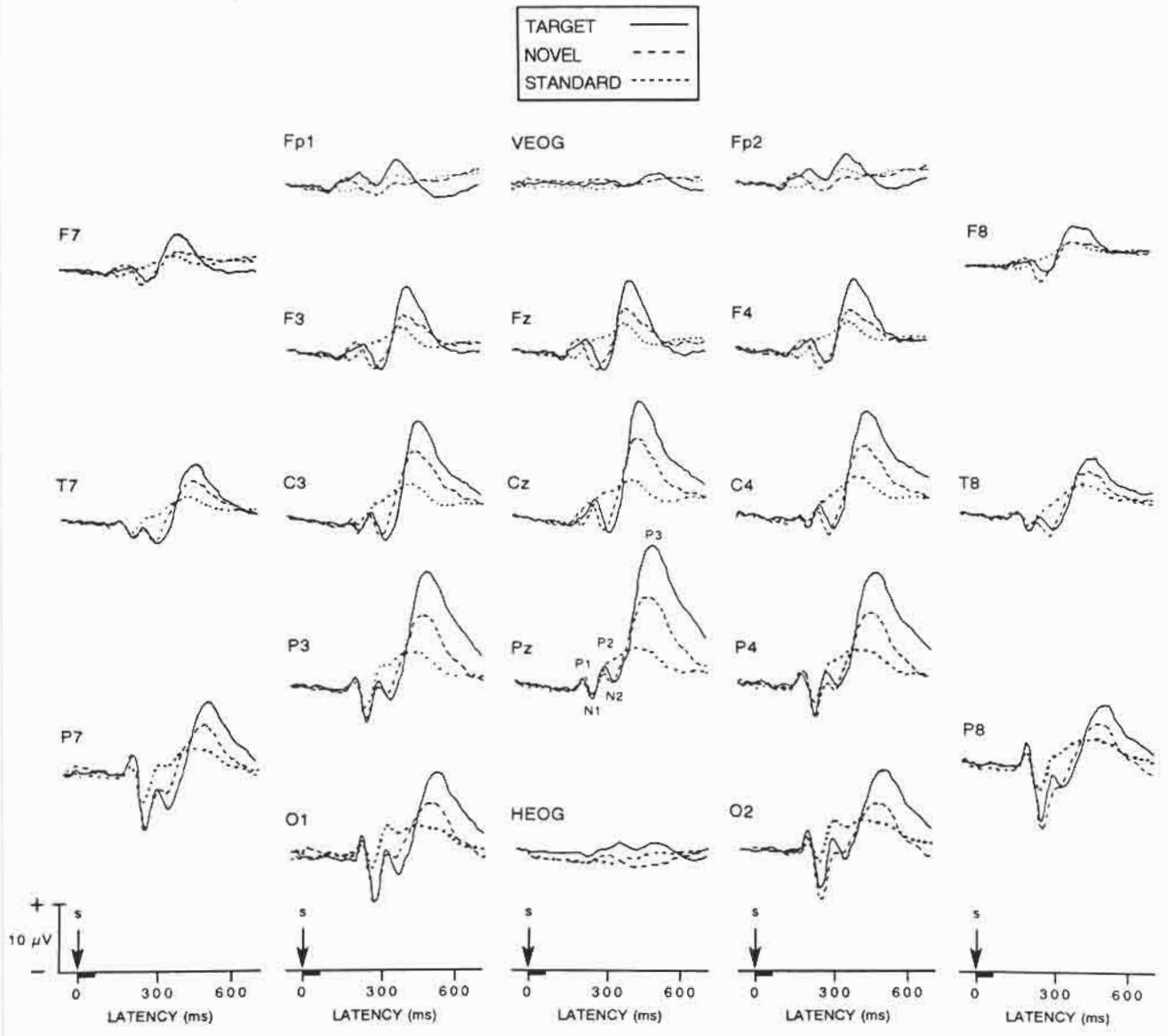

Figure 1. Grand average event-related potentials from the target, novel, and standard stimuli for each electrode recording site ($n = 80$).

lateral (F8, T8, P8), and right medial (F4, T4, P4) electrode locations were assessed (preliminary analyses indicated no reliable hemispheric effects from the Fp1/2 and O1/2 electrode sites, and these will not be considered further). Assessment of P3 peak latency revealed no reliable P3 hemispheric differences and only a few inconsistent differences for the other components. Because virtually all of the latency data demonstrated no hemispheric asymmetries of consequence, these analyses are presented in an abbreviated manner (see Table 1).

The mean P3 amplitudes from the target, novel, and standard stimuli recorded over each hemisphere and medial/lateral location as a function of the frontal, central, and parietal electrode positions are illustrated in Figure 2. An initial four-variable (Stimulus Type × Hemisphere × Medial/Lateral Location × Frontal-to-Parietal Electrode Site) ANOVA was performed on the P3 amplitude data for the target and novel stimuli. These analyses produced several complex Stimulus Type × Medial/Lateral Location interactions ($p < .01$ in all cases): (a) hemisphere interacted with electrode site, and there was a three-way interaction among hemisphere, stimulus type, and frontal-to-anterior electrode placements; (b) the P3 amplitude difference between target and novel stimuli was larger at medial than at lateral electrode locations; and (c) the medial electrode locations yielded larger amplitudes overall than did the lateral electrode locations. Because the primary hemispheric effects of interest differed somewhat among stimulus types and between the medial and lateral electrode locations, separate two-variable (Hemisphere × Frontal-to-Parietal Electrode Site) ANOVAs were performed on the amplitude data from each stimulus type for the medial and lateral locations. The significant effects are summarized in Table 1. Because the frontal-to-parietal electrode variable produced consistent main effects in the typical direction, this variable will receive comment only when the hemisphere and electrode variables yielded reliable statistical interactions.

Table 1. *Summary of F Ratios (and ε Corrections) ANOVAs Performed on Component Amplitudes and Latencies*

		Amplitude					Latency				
Variable	*df*	P1	N1	P2	N2	P3	P1	N1	P2	N2	P3
Target stimuli											
Medial											
Hemisphere	1, 79	8.0** (1.000)		7.8* (1.000)	15.2** (1.000)						
Electrode	2, 158	81.0*** (0.6636)	70.8*** (0.5776)		8.8*** (0.5930)	116.7*** (0.5886)		28.0*** (0.7059)	3.9* (0.6554)	15.5*** (0.7618)	25.2*** (0.8399)
H × E	2, 158				6.3** (0.8550)	26.8*** (0.8633)					
Lateral											
Hemisphere	1, 79	5.2* (1.000)		4.3* (1.000)	18.9*** (1.000)						
Electrode	2, 158	123.8*** (0.6564)	185.4*** (0.5493)	21.8*** (0.5642)	21.1*** (0.5445)	47.0*** (0.6062)	3.5* (0.9093)	23.3*** (0.7714)		34.5*** (0.8043)	18.9*** (0.8566)
H × E	2, 158	3.6* (0.7684)		4.1* (0.7596)	12.8*** (0.7180)	15.5*** (0.8239)			3.9* (0.8873)		
Novel stimuli											
Medial											
Hemisphere	1, 79				4.2 (1.000)						
Electrode	2, 158	62.5*** (0.6686)	48.2*** (0.5720)	4.2* (0.5693)	17.0*** (0.5840)	90.7*** (0.5861)	8.6*** (0.9236)	37.0*** (0.8634)	25.9*** (0.6783)	9.7*** (0.7031)	
H × E	2, 158	3.7* (0.8746)			3.7* (0.8730)	3.4* (0.7946)					
Lateral											
Hemisphere	1, 79	5.8* (1.000)						4.4* (1.000)			
Electrode	2, 158	100.8*** (0.6162)	137.1*** (0.5579)	35.5*** (0.5463)		43.7*** (0.5843)		42.0*** (0.9085)	29.1 (0.8327)	15.7 (0.7046)	
H × E	2, 158	7.4*** (0.7253)	5.6* (0.6975)		3.6*** (0.7029)	3.7* (0.8319)					
Standard stimuli											
Medial											
Hemisphere	1, 79	13.0*** (1.000)		6.0* (1.000)	9.8*** (1.000)	12.2*** (1.000)					
Electrode	2, 158	54.3*** (0.8263)	18.9*** (0.5574)	51.0*** (0.6605)	42.8*** (0.6851)	47.8*** (0.6350)					4.1* (0.7345)
H × E	2, 158										
Lateral											
Hemisphere	1, 79	37.9*** (1.000)		10.9** (1.000)	30.3*** (1.000)	26.9*** (1.000)		4.9** (1.000)		4.7* (1.000)	
Electrode	2, 158	118.1*** (0.5859)	64.2*** (0.5420)	28.2*** (0.5657)	13.5*** (0.5680)	42.5*** (0.6367)			3.8* (0.8600)		5.5** (0.7683)
H × E	2, 158	5.2* (0.7413)				4.5* (0.7455)					

*$p < .05$. **$p < .01$. ***$p < .001$.

P3 component. The primary P3 amplitude hemispheric effects originated from the interaction between hemisphere and frontal-to-parietal electrode. The strongest of these asymmetries were obtained for the target stimuli; the novel stimuli demonstrated relatively weak interactive patterns. In contrast, the standard stimuli yielded larger right than left hemisphere P3 components, and there was an interaction between hemisphere and frontal-to-parietal electrode for the lateral recording sites. No reliable hemispheric effects for P3 latency were obtained.

To assess the strength of the laterality/electrode interaction effects directly, simple main effects tests were performed by evaluating each pair of lateral electrodes separately (Table 2). The results of these tests for the P3 component suggest that the majority of the significant Right-Greater-Than-Left Hemisphere × Electrode interactions for the P3 component were produced at the frontal and central sites by the target and standard stimulus conditions, although a significant left-greater-than-right hemisphere effect was observed at the parietal electrode sites. More important, however, is that the statistically conservative nature of these tests implies that the observed hemispheric differences are reliable, at least for the specific loci indicated.

P1, N1, P2, and N2 components. The mean P1, N1, P2, and N2 component amplitudes from the target, novel, and standard stimuli for each lateral location are plotted as a function of frontal-to-parietal electrode site in Figure 3. Significant hemi-

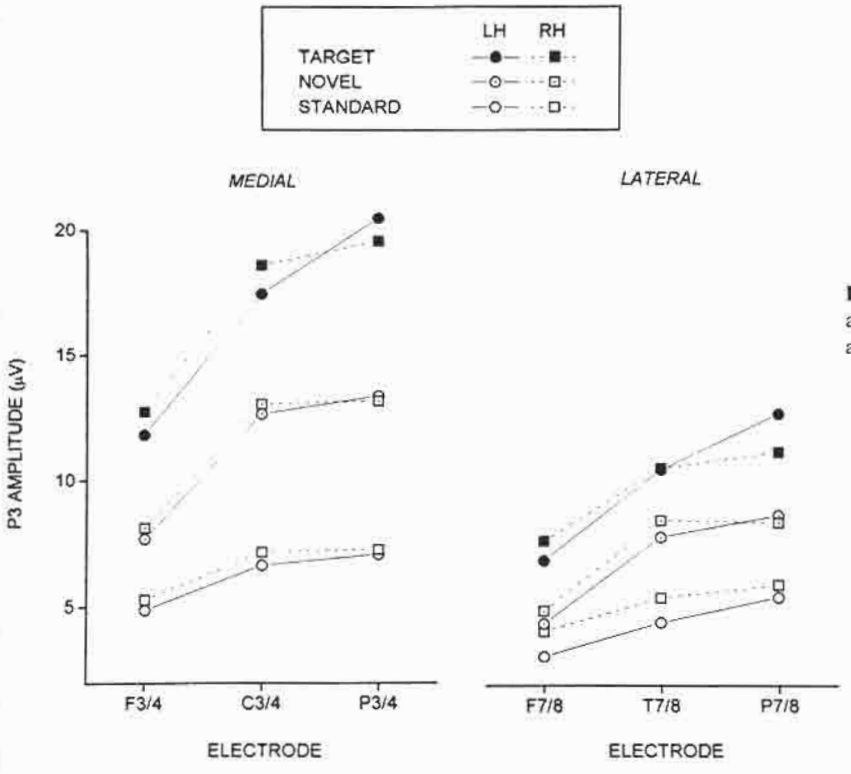

Figure 2. Mean P3 amplitude from the target, novel, and standard stimuli at the medial and lateral locations as a function of anterior-to-posterior electrode sites.

Table 2. *Summary of Simple Main Effects (F Ratios) for Electrode Site for Component Amplitudes and Latencies*

	Amplitude					Latency				
Electrode site[a]	P1	N1	P2	N2	P3	P1	N1	P2	N2	P3
Target stimuli										
Medial										
F3/4					8.8*	4.1*				
C3/4	3.8*			6.7*	14.5***					
P3/4	6.5**		7.5**	16.9***	9.1**					
Lateral										
F7/8										
T7/8				4.5*						
P7/8	8.2**		7.3**	30.1***	10.6**					
Novel Stimuli										
Medial										
F3/4				5.6*						
C3/4										
P3/4	4.1*									
Lateral										
F7/8										
T7/8										
P7/8	16.2***	6.1*								
Standard Stimuli										
Medial										
F3/4					5.7*					
C3/4	5.8*			5.1*	5.9*					
P3/4	8.6*			5.2*						
Lateral										
F7/8	6.7**			7.6**	13.6***	5.5*	9.7**			
T7/8	8.6**			9.7**	9.3**		5.4*			
P7/8	40.3***		8.1**	18.0***						

[a] df = 1,152 to 1,232. Denominator is variable across individual tests because df values are based on the specific mean squared error value when repeated measures simple effects are calculated.
*$p < .05$. **$p < .01$. ***$p < .001$.

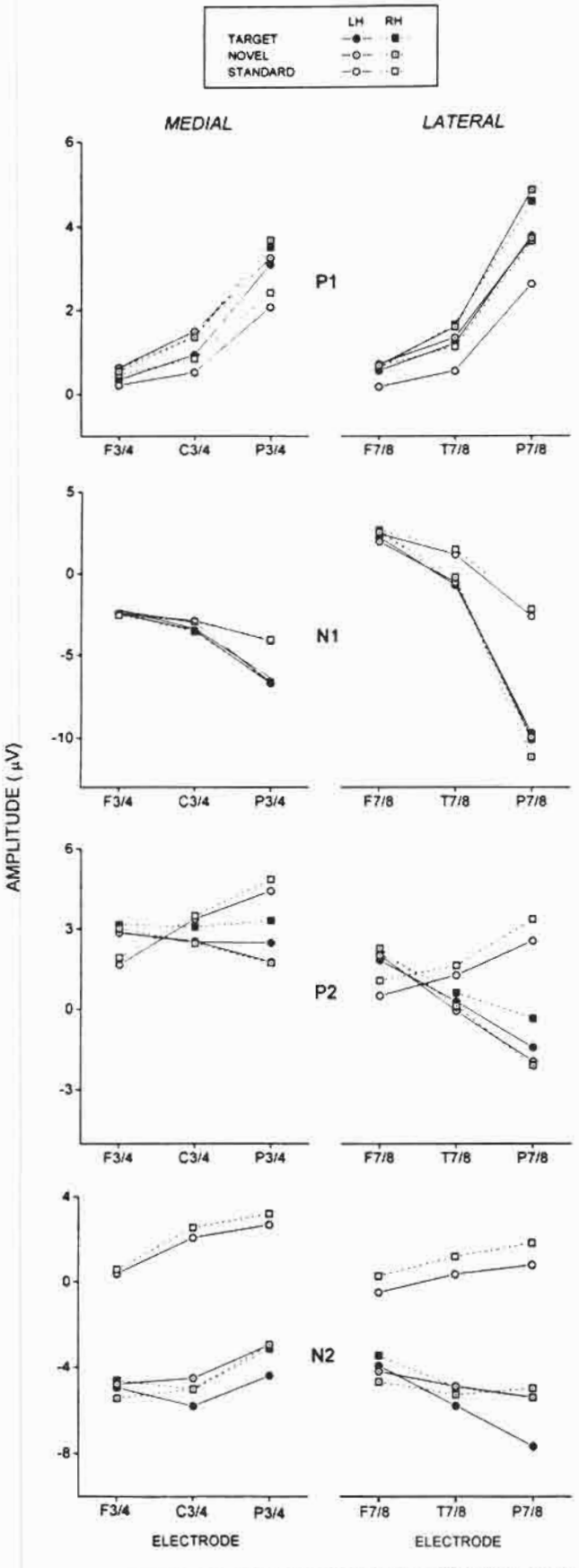

Figure 3. Mean P1, N1, P2, and N2 amplitudes from the target, novel, and standard stimuli at the medial and lateral locations as a function of the anterior-to-posterior electrode sites.

spheric main effects were obtained for the P1, P2, and N2 components; right hemisphere amplitudes were larger than left hemisphere amplitudes primarily for the target and standard stimuli. The interactions between hemisphere and frontal-to-parietal electrode sites were in the direction opposite of those for the P3 component, with larger hemispheric asymmetries observed toward the parietal recording locations. The only hemispheric difference for the N1 component occurred for the novel stimuli at the lateral electrode locations. The hemispheric latency differences observed for the N1 and N2 components stemmed from shorter latencies for the right than the left electrode sites. Specific comparisons between homologous lateral electrodes from the simple main effects analyses are presented in Table 2. Right hemisphere superiorities were obtained for ERP components other than the P3, most often with the maximum difference obtained over the parietal locations for both lateral and medial electrode sites.

Discussion

P3 amplitude from a visual oddball paradigm was consistently larger over the right hemisphere frontal and central electrode sites for target, novel, and standard stimuli waveforms; these findings are similar to those of P3 studies that used auditory stimuli (Alexander et al., 1995; Holinger et al., 1992; Karniski & Blair, 1989; Naumann et al., 1992). The magnitude of the P3 right-greater-than-left hemispheric amplitude asymmetry at frontal and central electrode locations for target stimuli was 1–2+ μV, as was the left-greater-than-right difference at the parietal electrode locations; both outcomes were statistically robust for the homogeneous sample of right-handed male subjects assessed in the present study. Because right hemisphere amplitude asymmetries were observed for each of the stimulus types, these lateralized P3 amplitude differences appeared to be relatively unaffected by stimulus probability or response task variables. Although the underlying sources for these P3 amplitude asymmetry effects are still uncertain, these effects might originate from fundamental structural, electrophysiological, or cognitive differences between the two hemispheres.

P3 Hemispheric Differences

Structural effects. Structural differences in brain morphology, skull thickness, and cranial irregularities may have contributed to the P3 hemispheric amplitude variations observed; such factors can affect ERP amplitude. For example, plagiocephaly or the underlying brain configuration associated with lateralized cranial deformities consists of a counterclockwise torque in which the left occipital pole flattens and the right frontal locations are larger and often protrude forward (Daniel, Myslobodsky, Ingraham, Coppola, & Weinberger, 1989; Simpson & David, 1986). The resulting hemispheric difference in neural mass may therefore produce electrophysiological asymmetries by redirecting current flow through the skull, such that larger amplitudes are recorded over locations that contain more cellular volume and/or where the skull is thinner (cf. Ford et al., 1994; Pfefferbaum & Rosenbloom, 1989). Although this explanation is plausible, measurable plagiocephaly occurs only in about 10% of the population (Binnie, Dekkerm, Smit, & Van der Linden, 1982). Furthermore, only a slight and apparently unstable relationship between skull thickness and occipital EEG alpha asymmetry has been reported (Myslobodsky, Coppola, &

Weinberger, 1991), with strong indications that plagiocephaly affects the occipital recording sites more than it affects the symmetrical, central, parietal, and temporal recording locations (Chui & Damasio, 1980; Myslobodsky et al., 1989). Hence, the lateralized effects found for P3 amplitude at the frontal and central locations are most likely unrelated to structural differences in skull and brain morphology.

EEG and ERP relationships. A more appealing explanation for the overall ERP lateralized amplitude effects stems from EEG hemispheric asymmetries, because greater right hemisphere alpha-band EEG power has been observed often (cf. Davidson, 1988; Tomarken, Davidson, Wheeler, & Kinney, 1992; Wieneke, Deinena, Spoelstra, Strom Van Leeuwen, & Versteeg, 1980). Variation in background alpha power was correlated positively with P3 amplitude from midline recording locations in several studies (Başar, Başar-Eroglu, Rosen, & Schutt, 1984; Intriligator & Polich, 1994; Jasiukaitis & Hakerem, 1988), although the relationships between EEG power and the P1, N1, P2, and N2 component are less consistent (Intriligator & Polich, in press). More important, the most consistent P3 amplitude hemispheric effects in the present study were obtained over the frontal and central recording sites, locations where EEG alpha power asymmetries are readily observed (Davidson, 1988, 1992). Given these electrophysiological similarities and the possible association between EEG alpha power and P3 amplitude, the implication arises that lateral asymmetries for alpha power may underlie the observed increases in P3 amplitude over the right hemisphere for the frontal and central electrode sites. association between variation in EEG power from specific bands and ERP component amplitudes is not yet clear. Positive correlations between EEG power and P3 amplitude have been reported for the delta, theta, and alpha bands (Polich & Luckritz, 1994; Spencer & Polich, 1992), frequencies that also appear related to slow wave activity (Ruchkin, Johnson, Mahaffey, & Sutton, 1988; Ruchkin & Sutton, 1983). Moreover, the right-greater-than-left P3 hemispheric asymmetries of the present study were observed at the frontal and central electrodes, locations where P3 amplitude is typically small, with significant left-greater-than-right asymmetries observed at the parietal electrode where P3 amplitude and alpha power are largest. Thus, EEG contributions to P3 and other ERP amplitude asymmetries may not be the only source of these effects.

Cognitive contributions. The strongest interaction between hemisphere and frontal-to-parietal electrode sites for P3 amplitude was found for the target stimuli (see Figure 2, Table 1), suggesting that neurocognitive influences also may underlie these hemispheric differences. A major theoretical interpretation of the P3 posits that this ERP component reflects a developing representation within short-term memory (Donchin & Coles, 1988; Donchin, Karis, Bashore, Coles, & Gratton, 1986). This hypothesis is supported by the results from human lesion studies, which have suggested that multiple neural generators, most likely originating from portions of the temporal-parietal cortex, are involved in P3 production (Johnson, 1989; Knight, Scabini, Woods, & Clayworth, 1989; Yamaguchi & Knight, 1991). In addition, very surprising or alerting stimuli will elicit a P3a subcomponent that is of maximum amplitude over frontal/central electrode sites (cf. Courchesne, Hillyard, & Galambos, 1975; Polich, 1988; Squires, Squires, & Hillyard, 1975), reflects initial signal evaluation, appears to originate from the frontal lobe, and readily habituates (Ford, Roth, & Kopell, 1976; Knight, 1984; Roth, 1973). When the stimulus is processed subsequently in memory, the central/parietal canonical P3b is generated (Knight, 1990; Polich & Squire, 1993). Taken together, these findings imply that at least two different ERPs comprise the "P3" component and that these different subcomponents have distinct neuroanatomical loci (Johnson, 1993; Picton, 1992).

Given this background, it is not unreasonable to suppose that the frontal–central P3 amplitude asymmetries of the present study may be reflecting the neurocognitive operations underlying the fundamental discrimination process required in the oddball paradigm. In this view, the observed right hemisphere P3 amplitude superiorities 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 & Petersen, 1990). Discriminating the target from a novel or standard stimulus could initiate right frontal engagement, because such a process requires the consistent application of attentional focus, a major attribute of frontal lobe function (Pardo, Fox, & Raichle, 1991; Posner, 1992). The reason for the reversal of these effects at the parietal location is not clear, but this reversal may stem from localized decision processes that govern response production. The decrease in the parietal hemispheric effects across stimulus types (target, novel, standard) is consonant with this view, because each stimulus type is likely to evoke different propensities to respond. The lack of similar patterns for the other components is also consistent with this approach. Thus, P3 amplitude asymmetries may be a relatively more precise manifestation of context updating processes than are midline recordings because the P3 recordings localize at least the initial processing response to the right frontal lobe areas, perhaps with task decision processes occurring in the left parietal lobe.

Other ERP Asymmetries

The P1, N1, P2, and N2 components also showed hemispheric asymmetry in patterns similar but not identical to the P3 effects: greater right hemisphere positive-going amplitudes and shorter latencies relative to left hemisphere locations. However, in contrast to the P3 component these hemispheric amplitude differences from the other components were typically largest at the central/posterior locations, a finding that implies that their hemispheric differences may stem from cognitive influences unrelated to those governing P3 generation. Additional studies that manipulate variables affecting these potentials directly are needed to specify the source of their lateralization patterns more clearly.

Summary

P3 amplitude elicited with a simple visual oddball discrimination task was larger over the right than the left frontal and central hemisphere electrode sites. The sources of these lateralized amplitude effects are not yet clear, but these effects may originate from asymmetric electrophysiological and neurocognitive processes governing attentional operations on incoming stimuli. Assessment of individuals for both EEG and ERP measures with other discrimination tasks is needed to delineate the genesis of these effects.

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