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Short communication

P300 differences between sinistrals and dextrals

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Abstract

The P3(00) event-related potential (ERP) was elicited in 20 left- and 20 normal right-handed young adult male subjects using a simple visual stimulus discrimination task. For left-compared to right-handed subjects, P3 amplitude was larger at anterior electrode sites for the target stimuli and larger overall for the novel visual stimuli; P3 latency was shorter for left-compared to right-handers for the target stimuli. The N1, P2, and N2 components demonstrated similar handedness effects. The relationships of ERP amplitude and handedness to anatomical variables and cognitive factors are discussed.

Keywords: P300; Event-related potential (ERP); Handedness

Despite a general impression that the P3(00) event-related potential (ERP) is symmetrical in amplitude about the midline [4], asymmetries for this brain potential have been found under task conditions that encourage differential hemisphere-specific cognitive processing (e.g. [12]). Several studies also have found that P3 amplitude in normal subjects is greater over the right compared to left hemisphere even when simple stimulus discrimination paradigms are used [1,14]. An important variable in this context is subject hand preference, although the relationship of handedness to cerebral lateralization is complex, convoluted, and rife with contrary findings (cf. [2]). Initial anatomical studies found cerebral size asymmetries for left- compared to right-handed subjects (e.g. [6,13]), but subsequent brain imaging reports have not demonstrated consistent strong hemispheric size differences related to handedness or familial sinistrality (cf. [3,11]). More recently, corpus callosal size has been related to handedness: left-handed men have been found to have larger callosal areas compared to right-handers (cf. [7,19,20]). These findings imply that the anatomical basis for subject handedness underline the functional differences between left- and right-handed groups (e.g. [16]).

Since P3 amplitude has been found to be asymmetric across the hemispheres in right-handers and because the anatomical sources of subject handedness can affect fundamental cognitive mechanisms, it is not unreasonable to suppose that handedness also may alter the P3 and other ERP components. To assess this possibility, a visual oddball discrimination task was employed in which stimuli consisted of infrequently presented targets, frequently presented standards, and infrequently presented 'novel' patterns that were designed to engage stimulus discrimination in the absence of an overt response.

Different groups of 20 left- and 20 right-handed, normal young adult males (M = 22.6, S.D. = 1.8 years) served as subjects. Handedness was evaluated with a series of six unimanual tasks from a handedness questionnaire, with the addition of items that assessed for familial sinistrality. Left-handedness was defined as using the left-hand only for a minimum of four of the six tasks (M = 5.6, S.D. = 0.6); right-handedness was defined as showing only right (i.e. no left-hand) preferences for all questions (M = 0.0, S.D. = 0.0). The mean number of left-handed family members reported by the left-handed subjects was 1.4 (S.D. = 0.68, mode = 1); the mean number of left-handed family members reported by the right-handed subjects was 0.0 (S.D. = 0.0, mode = 0). All subjects reported an absence of psychiatric or neurologic problems, were screened for alcohol/drug use, and remunerated for their participation.

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), referred to the

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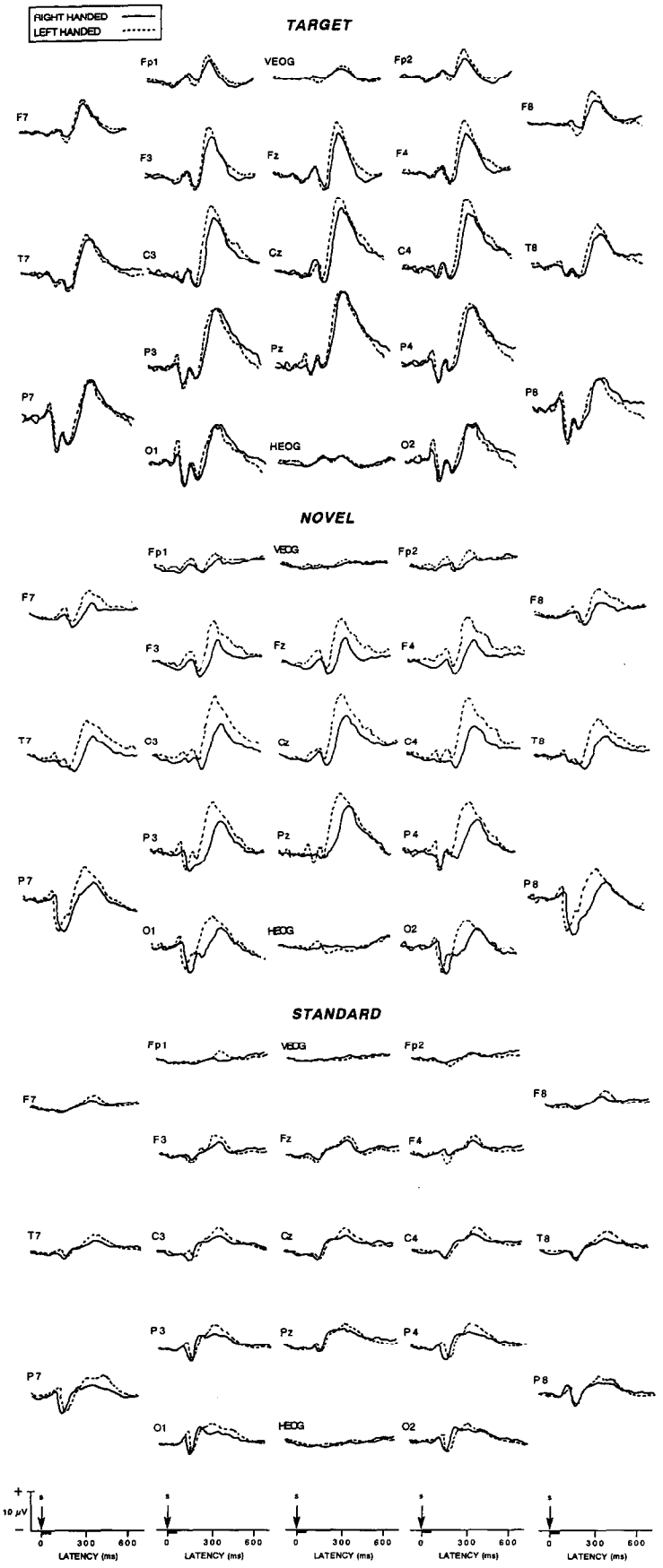
RIGHT HANDED
LEFT HANDED
TARGET
NOVEL
STANDARD
Fp1
VEOG
Fp2
F7
F3
Fz
F4
F8
T7
C3
Cz
C4
T8
P3
Pz
P4
P7
P8
O1
HEOG
O2
10 μV
0
300
600
LATENCY (ms)

nose, with a forehead ground and impedances at 5 kΩ or less. Electro-ocular (EOG) activity was assessed with two channels referred to the nose; one electrode placed at the outer canthus of the left eye and the second electrode placed on the forehead above the eye to monitor horizontal and vertical eye movements. 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 1500 ms, with a 187 ms prestimulus baseline. ERP data were averaged on-line; trials on which the EEG or EEG exceeded ± 73.3 μV 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 non-repeating colored geometric shapes (e.g. blue hexagons, red pentagons, green triangles, etc.) 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 0.125; the standard stimuli occurred with a probability of 0.75. 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 within each handedness group. Stimulus presentation was concluded when 25 target, 25 novel, and 150 standard artifact-free ERP trials were acquired.

All analyses of variance employed Greenhouse-Geisser corrections to the degrees of freedom. Task performance was nearly perfect for both groups, with the total number of errors (misses and false alarms) for left-handers = 0.4% and right-handers = 0.6%. Mean response time for the target stimuli for left-handers = 448 ms (S.D. = 57.6) and right-handers = 465 ms (S.D. = 65.1), with no reliable difference found ($t < 1$, $P > 0.30$). Waveforms for the target, novel, and standard stimuli were assessed visually and individually for each subject, with the amplitudes and latencies of the N1, P2, N2, and P3 components identified at each electrode site by locating the most positive or negative component within the latency windows of 200–300, 250–350, 300–400, and 350–600 ms, respectively. Amplitude was measured relative to the mean of the prestimulus baseline, and peak latency was defined as the time point of maximum positive or negative amplitude within the latency window. Statistical analyses were performed such that the same anterior-to-posterior locations (frontal, central, parietal) were used for the lateral (F7/8, T7/8, P7/8), medial (F3/4, C3/4, P3/4), and central (Fz, Cz, Pz) electrode locations (preliminary analyses indicated no reliable handedness or hemispheric effects from the Fp1/2 and O1/2 electrode sites, and these will not be considered further).

The grand average ERP waveforms from each handedness group for the target, novel, and standard stimuli at each electrode position are illustrated in Fig. 1. The mean P3 amplitude and latency values as a function of lateral electrode position and stimulus type are presented in Fig. 2. Separate three-factor (handedness group $\times$ anterior-to-posterior electrode $\times$ lateral electrode location) analyses of variance were performed on the amplitude and latency data obtained from each of the stimulus types for each component. Because the anterior-to-posterior and lateral electrode factors produced consistent main effects in the typical directions for each component, these variables will receive comment only if the handedness factor yielded statistically reliable interactions.

Target stimulus P3 amplitude was larger for left-handers across the anterior locations compared to right-handers who demonstrated larger component amplitudes at the posterior locations, as indicated by the significant interaction between the handedness group and anterior-to-posterior electrode factors, $F_{2,76} = 4.0$, $P < 0.05$. Target stimulus P3 latency was shorter for the left-compared to the right-handed group, $F_{1,38} = 6.5$, $P < 0.02$. Novel stimulus P3 amplitude was larger overall for the left-compared to right-handed group, $F_{1,38} = 4.5$, $P < 0.05$. This handedness difference was somewhat stronger over the left hemisphere and produced a significant interaction between handedness group and lateral electrode position, $F_{4,152} = 4.0$, $P < 0.01$. Standard stimulus P3 latency was shorter for left-handers compared to right-handers at the central and medial locations but longer at the extreme lateral sites to produce a significant hand group by lateral location interaction, $F_{4,152} = 4.6$, $P < 0.002$.

N1 amplitude was larger for the left-handed compared to right-handed subjects at the central recording sites, whereas the reverse generally was observed at the more lateral sites. These effects produced significant interactions between the handedness and lateral electrode factors for the target and novel stimuli, $F_{4,152} = 3.4$, $P < 0.03$ and $F_{4,152} = 3.2$, $P < 0.05$, respectively. N1 latency from novel stimuli demonstrated a significant interaction between handedness group and lateral electrode site, $F_{4,152} = 5.0$, $P < 0.01$. P2 amplitude from the novel stimuli was smaller for the left-compared to right-handers and more so at the central and over the left hemisphere to yield a complex interaction between handedness, anterior-to-posterior electrode, and lateral location factors, $F_{8,308} = 5.0$, $P < 0.001$. P2 latency from the novel stimuli was shorter overall for the left-compared to the right-handed group over the right

Fig. 1. Grand average event-related potentials from the target, novel, and standard stimuli for the left- and right-handed subjects ($n = 20$/handedness group).

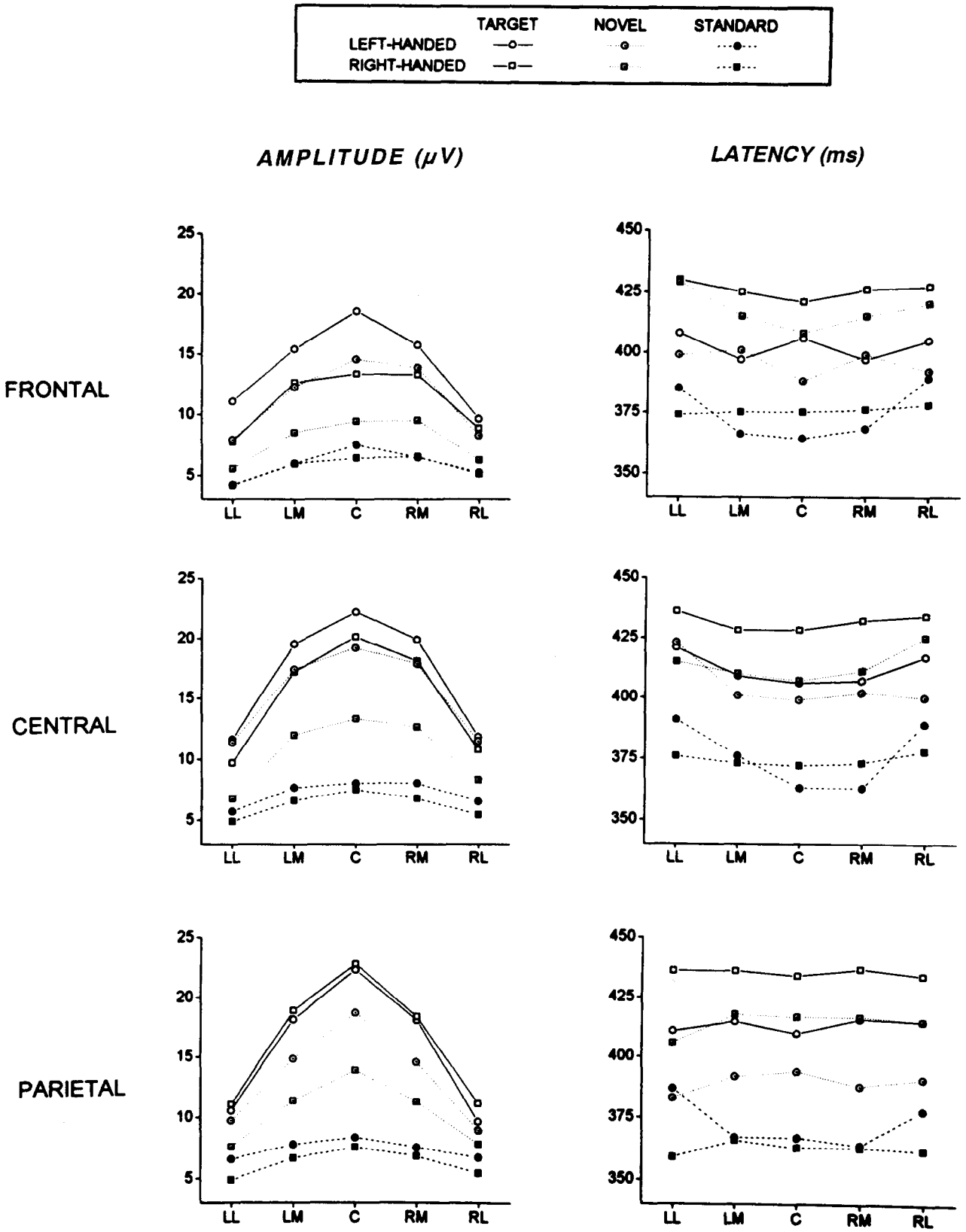

Fig. 2. Mean P3 amplitude and latency from the target, novel, and standard stimuli as a function of lateral and anterior-to-posterior electrode sites for the left- and right-handed subjects (LL = left lateral, LM = left medial, C = central, RM = right medial, RL = right lateral).

hemisphere to yield a significant interaction between handedness group and lateral electrode location, $F_{4,152} = 6.7$, $P < 0.001$; P2 latency from the standard stimuli was shorter for left-compared to right-handed subjects, $P_{1,38} = 4.8$, $P < 0.05$. N2 amplitude did not produce any statistically robust handedness results, but N2 latency was shorter overall for left-compared to right-handers for all three stimulus types ($P < 0.05$ in each case).

P3 amplitude was significantly larger for left-handed compared to right-handed subjects for the infrequently presented target stimuli over anterior and central electrode locations, and larger overall for left-handers compared to right-handers. P3 latency also was generally shorter for the left- compared to right-handed subjects for the target stimuli. The sources of these ERP handedness differences are unknown. However, given the previously reported anatomical differences between left- and right-handed individuals with respect to corpus callosal size [7,19,20], it is not unreasonable to suppose that ERP amplitudes (and latencies) might be affected by group variation in neural mass for the strongly left- and right-handed male subjects employed in the present study. Furthermore, the P3 amplitude handedness differences were most reliable for the target stimulus – a result that suggests that stimulus and task processing requirements contribute to these handedness effects. If the size of the callosal connection does affect the communication efficiency between the hemispheres, variation in P3 values between handedness (callosal) groups may be reflected by differential handedness group information processing capabilities.

A major theoretical interpretation of the P3 posits that this ERP component reflects a developing representation within short-term memory [5]. This hypothesis is supported by the results from human lesion studies, which have suggested that multiple neural generators – possible originating from portions of temporal-parietal cortex – are involved in P3 production [10]. In addition, alerting stimuli will elicit a P3a subcomponent that is of maximum amplitude over frontal/central electrode sites [cf. [8]]. When the stimulus is processed subsequently in memory, the central/parietal canonical P3b is generated [9,17]. Given this background, it may be that the frontal-central P3 amplitude differences observed for the left- and right-handed subject groups of the present study are indexing neurocognitive operations underlying the fundamental discrimination process required in the oddball paradigm. Assuming callosal connections are active during oddball task processing, discriminating the target from a novel or standard stimulus could initiate frontal engagement, because such a process requires the consistent application of attentional focus – a major attribute of frontal lobe function [15,18]. The lack of strong handedness results for the novel and standard stimuli, which do not require an attentionally driven response, and the generally similar amplitude and latency handedness patterns obtained for the other components are consistent with this view.

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