Age effects on the P300 to novel somatosensory stimuli

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Summary Event-related brain potentials (ERPs) to somatosensory task-relevant targets and task-irrelevant novel (tactile and shock) stimuli were studied in 30 subjects between the ages of 18 and 79. Target and novel P300 latencies increased linearly with age at comparable rates. P300 amplitudes and scalp topographies also changed with age. P300 amplitudes remained constant at frontal sites and decreased at central and parietal sites for both target and novel stimuli with increasing age.

The current results extend the age-related novel P300 changes reported in the auditory and visual modalities to the somatosensory system. The age-related amplitude reduction at posterior scalp sites supports independent contributions of frontal and posterior association cortex to P300 generation.

Key words: Aging; Novelty P300; Target P300; Somatosensory stimulation

Two types of P300 event-related brain potential (ERP) have been described in the visual (Courchesne et al. 1975; Beck et al. 1980), auditory (Squires et al. 1975; Knight 1984) and somatosensory modalities (Yamaguchi and Knight 1991). Task-relevant correctly detected stimuli generate a parietal maximal P300 (target P300). Non-target, deviant stimuli requiring no overt behavioral response generate an earlier latency P300 (novelty P300) with a more fronto-central distribution.

Target P300 generation requires a cooperative subject able to produce an organized behavioral response and has been studied in patients with memory and other cognitive disturbances (Goodin et al. 1978b). Novelty P300 generation requires limited subject cooperation and may provide a valuable index of orienting or automatic attention capacity in patients with impaired cooperation or motor capacity.

Numerous studies have assessed the effect of age on the target P300 (Goodin et al. 1978a; Pfefferbaum et al. 1984; Picton et al. 1984). However, only a few reports have examined chronological changes in the novelty P300. Visual (Snyder and Hillyard 1979; Beck et al. 1980) and auditory novelty P300 studies (Knight 1987) report latency prolongation with or without accompanying amplitude reduction with aging. There are no reports of aging effects on the somatosensory novelty P300. The current study was undertaken to examine the effect of aging on the somatosensory P300 generated by task-irrelevant novel stimuli. In addition to parametric data, age effects on target and novelty P300 may contribute to understanding the neural mechanisms underlying P300 generation.

Methods

Subjects

Thirty subjects (18 males, 12 females) ranging in age from 18 to 79 participated in the study. The subjects were divided into 3 groups according to age; a young group (18–29 years, mean 22.1 ± 2.8 years, n = 10), a middle-aged group (30–49 years, mean 39.1 ± 5.8 years, n = 10) and an older group (50–79 years, mean 63.9 ± 9.2 years, n = 10). All subjects were healthy and intellectually active and had no history of neurological or psychological hospitalization.

Procedures

The stimuli consisted of mechanical taps to the digits and electric shocks to the wrist using the methods previously described (Yamaguchi and Knight 1991). The experiment consisted of 4 blocks. Two blocks (first and fourth block) were assigned to the right hand and another two blocks (second and third block) were given to the left hand with a counter-balanced design across subjects. Each block consisted of 500 stimuli delivered at a rate of 1/sec. Of these stimuli, 76% were mechani-
caldi tactile stimuli to the second finger (standards), 12% were mechanical tactile stimuli to the fifth finger (targets), 6% were mechanical tactile stimuli to the third or fourth finger (tactile novels), and 6% were electric shock stimuli to the median nerve (shock novels). Target and novel stimuli were interspersed at random in the sequence. The subject was instructed to press a button with the thumb of the non-stimulated hand as accurately and quickly as possible upon detection of tactile stimuli to the fifth finger.

**Recording system**

Brain electrical activity was recorded using Ag/AgCl electrodes placed at 15 scalp locations (Fpz, F3, Fz, F4, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6 and Oz) based on the 10–20 system and below the left eye, all referenced to linked earlobes. Electrode impedances were kept below 5 kΩ. The EEG was amplified (bandpass 0.1–100 c/sec), digitized (250 Hz/channel) and stored on magnetic tape for off-line analysis by a PDP 11/73 computer. The averaging epoch was 1024 msec, including 200 msec of prestimulus baseline. Individual trials with excessive muscle activities or eye blinks were excluded. Amplitudes of all components were measured relative to the 200 msec prestimulus baseline. Latency was tabulated relative to stimulus delivery. The P300 component was defined as the most positive peak occurring in a post-stimulus window of 250–500 msec. The window was determined from inspection of individual averages and group superaverages. The potentials preceding P300 (i.e., P190, N2) were not subjected to further analysis because of substantial component overlap.

**Statistical analysis**

Regression analysis was performed between age and the P300 latency or amplitude. Data from Pz (target P300) and Cz (tactile and shock novelty P300) were used in the regression analysis.

Data were subjected to repeated measures analysis of variance. For analysis of age effects on P300 scalp distribution, P300 amplitude was normalized within each subject for each stimulus type and multivariate analysis (MANOVA) of repeated measures performed (SYSTAT Inc. 1987).

**Results**

The ERPs evoked by standards, targets, tactile novels and shock novels are illustrated in Fig. 1, which show the grand averaged wave forms for the 3 age groups. P190 ERPs were recorded to standards, targets and tactile novels. P300 ERPs were also generated by targets.

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**Fig. 1.** ERP grand average wave forms at Fz, Cz and Pz to the standard, target, tactile novel and shock novel stimuli for the young, middle-aged and old group. The grand averages were made across 10 subjects in each group.
tactile novels and shock novels. The negative slow wave was recorded to targets predominantly over frontal sites.

Latency

P300 latencies to targets, tactile novels and shock novels increased with age (targets, 1.28 msec/year, \( r = 0.63, P < 0.001 \); tactile novels, 1.72 msec/year, \( r = 0.65, P < 0.001 \); shock novels, 0.97 msec/year, \( r = 0.54, P < 0.01 \), see Fig. 2). There were no significant differences in the slopes of the 3 P300 types. An additional test for curvilinearity in the relationship between the P3 latency and age was performed and no evidence of curvilinearity was found. An increase in latency variability associated with aging was observed only for the tactile novel P300 (see Fig. 2).

Amplitude

The target P300 amplitude decreased by 0.18 \( \mu V \)/year (\( r = 0.54, P < 0.01 \)), a rate comparable to the shock novel P300 (0.16 \( \mu V \)/year, \( r = 0.41, P < 0.05 \)). P300 amplitude for tactile novels also decreased with age (0.11 \( \mu V \)/year, \( r = 0.45, P < 0.05 \)). The slopes of these regression lines were not significantly different.

Topography

Targets generated a parietal maximal P300 whereas tactile and shock novels generated central maximal P300s for all age groups. P300 scalp distributions were significantly affected by age (MANOVA, group \( \times \) electrode site interaction, targets, \( F (28, 26) = 2.33, P < 0.05 \); tactile novels, \( F (28, 26) = 1.92, P < 0.05 \); shock novels, \( F (28, 26) = 2.76, P < 0.05 \)). Amplitudes remained constant at Fz and decreased at Cz and Pz with advancing age for both target and novel P300s (at Pz, targets, \( F (2, 27) = 6.43, P < 0.01 \); tactile novels, \( F (2, 27) = 4.45, P < 0.05 \); shock novels, \( F (2, 27) = 6.16, P < 0.01 \), see Fig. 1).

Behavior

There were no significant age-related differences in the mean reaction time to correctly detected targets (young = 450 ± 60 msec, middle-aged = 441 ± 52 msec, old = 436 ± 57 msec, \( F (2, 27) = 0.14, \) n.s.). There was also no significant correlation between target P300 latency and reaction time (\( r = -0.15, \) n.s.). Percent correct target hit and false alarm rate were not different among the 3 age groups (correct target hit, young = 98 ± 2%, middle-aged = 98 ± 1%, old = 97 ± 3%, \( F (2, 27) = 1.04, \) n.s.; false alarm, young = 1.2 ± 1.3%, middle-aged = 2.6 ± 1.9%, old = 2.0 ± 1.8%, \( F (2, 27) = 1.6, \) n.s.).

Discussion

Aging has comparable effects on novelty and target somatosensory P300s. Somatosensory target P300 latency prolongation (1.28 msec/year) was within the range of previously reported values (1.01 msec/year in Barrett et al. 1987, 1.92 msec/year in Picton et al. 1984). These rates are comparable to the values reported in the auditory and visual modalities (1-2

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![Fig. 2. Plots of P300 latency and amplitude for targets, tactile novels and shock novels as a function of age. Data were obtained from Pz electrode for target P300 and from Cz electrode for tactile and shock novel P300.](image)
The somatosensory novelty P300 latency was also linearly prolonged with age. Age effects on novelty P300 latency (0.97 msec/year) were similar to those found for the target P300 latency and were consistent with prior visual (0.8 msec/year) and auditory (1.13 msec/year) P300 studies (Beck et al. 1980; Knight 1987).

Target P300 amplitudes inversely correlated with age. Most aging studies have reported modality-independent reduction in target P300 amplitude ranging from −0.15 to −0.28 μV/year. The value obtained in the current study (−0.18 μV/year) is consistent with Picton’s somatosensory data (−0.18 μV/year; Picton et al. 1984), although another recent study did not show age-related reduction of somatosensory target P300 amplitude (Barrett et al. 1987).

Comparable age-related decreases in novelty P300 amplitude were observed (−0.16 μV/year). Auditory novelty P300 was reported to show a larger age-related amplitude decrease (−0.38 μV/year) than target auditory P300 (−0.23 μV/year; Knight 1987). In the visual modality neither target nor novelty P300 amplitude was affected by age (Beck et al. 1980). This suggests that age effects on novelty P300 amplitude may be modality-dependent. However, differences in the degree of stimulus novelty may have contributed to modality-related differences in P300 amplitude. The P300 amplitude evoked by the shock novels was consistently larger than that evoked by the tactile novels in all age groups. The degree of stimulus novelty or the magnitude of the deviation relative to standard stimuli has been reported to contribute to the novelty P300 amplitude (Roth et al. 1982).

Topographical differences between somatosensory target and novelty P300s replicate a prior study (Yamaguchi and Knight 1991) and are consistent with findings in the auditory and visual modalities (Knight 1984; Courchesne et al. 1975). The lower probability of the novel stimuli may contribute to the frontal distribution of the novelty P300 (Campbell et al. 1979). The stimulus scenario employed in the present study is different from that used in Courchesne’s study in terms of categorization of novel stimuli (Courchesne 1978). The stimulus scenario employed for the novelty P300 also has characteristics similar to that which generates a centrally distributed non-target P300 (Pfefferbaum et al. 1984).

Aging had significant effects on the topographical distribution of both types of somatosensory P300. Several studies have reported changes in P300 scalp distribution with age. Pfefferbaum et al. (1984) and Smith et al. (1980) noted that P300 became more uniformly distributed from Pz to Fz in the older subjects in contrast to the parietal distribution found in the young subjects. They attributed this change to age-related decreases in overlapping components such as the frontal slow waves.

We found that the target and novelty P300s did not decrease at frontal sites. Since SW amplitude decreased at frontal sites with age, the lack of decrease in target P300 amplitude at frontal sites may be explained by loss of an overlapping negative SW. However, no age-related decrease in the frontal P300 was observed for novel stimuli which generated no SW. Conversely, both target and novelty P300 decreased significantly at posterior sites.

Lesion studies and intracranial recording support independent contributions of frontal and posterior association cortex to P300 generation (Knight 1984; Wood and McCarthy 1985; Knight et al. 1989). The differential effects of aging on the frontal and parietal P300 provide further support for distinct contributions from frontal and posterior association cortex to P300 generation.

Reaction times to correctly detected stimuli did not change with age. Similar findings were reported in a simple auditory discrimination task (Pfefferbaum et al. 1984; Picton et al. 1984). The lack of a relationship between P300 latency and reaction time supports the notion that these two measurements index different cognitive processes associated with perceptual and response organization systems.

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References


