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Effects of aging on event-related brain potentials (ERPs) in a visual detection task

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Summary Event-related brain potentials (ERPs) were recorded from 74 subjects (45 men) between 18 and 82 years of age in a simple visual detection task. On each trial the subject reported the location of a triangular flash of light presented briefly 20° laterally to the left or right visual field or to both fields simultaneously. ERPs to targets exhibited a similar morphology including Pl, Nl, P2, N2, and P3 components across all age groups. The principal effects of advancing age were (1) a marked reduction in amplitude of the posterior P1 component (75-150 latency) together with an amplitude increase of an anterior positivity at the same latency; (2) an increase in amplitude of the P3 component that was most prominent over frontal scalp areas; and (3) a linear increase in P3 peak latency. These results extend the findings of age-related changes in P3 peak latency and distribution to a non-oddball task in the visual modality and raise the possibility that short-latency ERPs may index changes in visual attention in the elderly.

Keywords: Event-related potentials; Visual detection task; Age-related effects; P3; Late positive complex

In a variety of paradigms that require subjects to discriminate and categorize stimuli, the event-related brain potentials (ERPs) triggered by these sensory decisions are characterized by a positive-going potential between about 300 and 800 msec after stimulus onset that is maximal over the parieto-central scalp (for reviews see Pritchard 1981; Donchin and Coles 1988). ERP components with these characteristics have been variously labeled the P300, P3, P3b or late positive complex (LPC), and over the years many proposals have been put forward regarding the cognitive processes associated with this late positivity. These include the updating of current working memory toward the betterment of future performance (Donchin and Coles 1988), resolution of uncertainty (Sutton et al. 1965), cognitive closure (Verleger 1988), and accessing of information necessary for successful task performance from a long-term memory store (Johnson 1993) among others. To date, however, no single psychological construct appears to account for all the available data. In spite of this uncertainty, however, it is noteworthy that the P3 component has proven to be highly informative in studies by cognitive psychologists and neurologists alike (e.g., Squires et al. 1975; K.C. Squires et al. 1980; Parasuraman 1990; Wickens 1990).

A growing literature documents the use of P3 latency as a measure of mental chronometry, that is, to track the normal time course of the processes central to decision-making (Van der Molen et al. 1991). Important in this regard are studies showing that the latency of the P3 component reflects the time it takes to evaluate and categorize stimulus events and is relatively independent from the time required to select and execute a response (Kutas et al. 1977; McCarthy and Donchin 1981; Callaway 1983; Ritter et al. 1983; Magliero et al. 1984; Mulder et al. 1984; Pfefferbaum et al. 1986). Thus, while the peak of the P3 component becomes progressively longer in latency with more difficult categorizations, unlike the RT it does not vary significantly with changes in speed/ accuracy trade-offs (Kutas et al. 1977; Pfefferbaum et al. 1983; Coles et al. 1985).

Because of its relationships with the timing of cognitive operations, it has been suggested that P3 might have a place in clinical neurology, specifically in tracking mental deterioration during various dementias and demyelinating diseases as well as the efficacy of various treatment regimes (Knight 1990). In support of this possibility, studies from several laboratories have documented an amplitude diminution and a marked latency prolongation of the P3 in many, albeit not all, demented patients with Alzheimer's disease, Parkinson's disease, and Huntington's disease (Goodin et al. 1978b, 1983; Hansch et al. 1982; Syndulko et al. 1982). Al-

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though P3 latency may not be a definitive marker for dementia, it may still prove to be an important aid to the neurologist faced with diagnosing as demented a patient who may merely be suffering from a severe depression; depression per se does not reportedly prolong P3 latencies more than 2 S.D. beyond the norm on the average (Gordon et al. 1986; Patterson et al. 1988). [1]

The need for a normative database against which neurological patients can be compared has engendered a large number of studies of P3 latency change across the lifespan. The results of these studies are in broad agreement in certain respects, but are at odds in others. For instance, there is general consensus that the peak latency of the P3 increases systematically with advancing age, although there is a continuing disagreement as to whether the function is linear or curvilinear (for reviews see Polich and Starr 1984; Ford and Pfefferbaum 1985; Miller et al. 1987; Bashore 1990; Polich 1991; Iragui et al. 1993). The results for P3 amplitude are less consistent in that amplitudes were found to be unaffected by age in some cases or to exhibit reductions only at certain recording sites (Picton et al. 1984; Polich 1991).

The majority of these normative studies have examined the P3s elicited by target stimuli in "oddball" tasks or variants thereof primarily in the auditory modality from very few recording sites. [2] In the basic oddball paradigm, subjects are presented with two classes of stimuli in a random, Bernoulli sequence. The target or "oddball" stimuli occur unexpectedly with an a priori probability (5-25%) that is lower than that of the standard or non-target stimulus class. The subject's task is to push a button in response to all target stimuli or to keep a running mental count of the number of targets for verbal report upon completion of each experimental run.

Only a handful of studies have investigated the effects of aging on auditory ERPs in a non-oddball task or in the visual or somatosensory modalities in oddball or other tasks (Snyder and Hillyard 1979; Polich 1991; Yamaguchi and Knight 1991b). The results tend to be fairly similar to those for the auditory oddball P3 - reduced amplitudes and prolonged latencies for older persons. Nonetheless, insofar as age-related changes in P3 amplitude and latency are to be interpreted within a cognitive framework and used on a regular basis in the clinic, it is important to know how specific the results are to either the task or the input modality.

Complicating the interpretation of age-related effects even further is the new conceptualization of the scalp-recorded P3 component as a reflection of the summation of the activity of several distinct neural generators from various areas. Analyses of scalp topography data indicate that distinct combinations of neural generators contribute to the P3 activity elicited by different combinations of experimental variables (see Johnson 1993). Likewise, data from intracranial recordings, neuromagnetic recordings, and from the scalps of patients with various cortical lesions have all converged on the view that multiple brain regions including the frontal lobe (McCarthy and Wood 1987), the auditory association cortex in the temporal-parietal junction (Knight et al. 1988), medial temporal lobe structures (Halgren et al. 1980; McCarthy et al. 1989) probably contribute to the scalp-recorded P300. The need for studies of age effects on P3 that go beyond the auditory oddball paradigm is heightened by recent studies that implicate different neural generators for the auditory, visual and somatosensory P3s elicited in highly similar oddball tasks (Johnson and Fedio 1987; Johnson 1989a,b; Knight 1990; Yamaguchi and Knight 1991a).

If the P3 wave is indeed associated with information processing operations that differ according to stimulus modality and task demands, then caution would be warranted in its use and interpretation for both experimental and clinical purposes. In particular the question of what mental operation or set of operations, if any, are slower in the normal elderly than in younger subjects could not be answered unless we have a more complete understanding of the functional significance of the various positive potentials elicited in the P3 latency range across different tasks and input modalities.

To this end we recorded visual ERPs from a relatively large group of men and women (N = 74) ranging in age from 20 to 80 years in a simple visual detection task [3] that all subjects could perform easily. Visual ERPs were collected in response to triangles flashed briefly at 20° eccentricity to either the left visual field, right visual field, or both fields simultaneously, following an auditory warning signal. Thus, our aim in this study was to examine age-related effects on the late positive complex (LPC) elicited in a visual detection task that differed from a standard oddball paradigm. The task chosen was simple enough to be performed by demented patients as well as patients with various cortical and subcortical lesions.

[1] Note that there are mixed findings on the relation between P3 latency, aging and dementia (e.g., Kraiuhin et al. 1990).

[2] Recordings are often restricted to the midline central sites (Fz, Cz, and Pz), an understandable trend for clinical purposes, although with the advance of electrode caps the length of the application procedure for 12-16 locations has effectively been halved.

[3] Many of these subjects (N = 71) had also previously participated in an auditory oddball task (Iragui et al. 1993).

Methods

Subjects

Seventy-four subjects (45 men, 29 women) participated in the study. There were 20 subjects aged 18-29, 8 between 40 and 49, 9 between 50 and 59, 20 aged 60-69, and 9 between 70 and 82 years. All were healthy, functioning individuals without a history of neurological or psychiatric disease, or visual impairment. Young and middle-aged subjects were recruited from the UCSD campus. Subjects older than 50 years of age were recruited from the normal control population of the UCSD Alzheimer's Research Center registry. They were living independently, scored better than 28 out of 30 points in the Mini-Mental State exam (Folstein et al. 1975) and had normal or corrected-to-normal visual acuity of 20/30 or better. Only partial data on educational level were available due to incomplete records. The mean number of years of education for 21 (out of 28) of the subjects in the young group (aged 18-39) was 17.4. The mean for 12 (out of 17) of the subjects in the middle-aged group (aged 40-59) was 16.6 years and the mean for 19 (out of 29) of the subjects in the oldest group (aged 60-82) was 14.2.

Stimuli

The stimuli consisted of triangular white flashes subtending 2° of visual angle in height, with a luminance of 0.7 log FL. Stimuli were presented on a 22 inch video monitor covered by a black screen except at the sites of stimulus presentation. In the center of the screen was a 1° white dot designated as the fixation point. The triangles were presented 20° laterally to this fixation point in random order to either the left visual field, right visual field or, to both visual fields simultaneously with a 100 msec duration.

Procedures

The subject sat in a comfortable reclining chair in an electrically shielded room. During the experiment they were instructed to fixate the dot in the center of the screen at a viewing distance of 120 cm and to blink as infrequently as possible. Each trial was initiated by the experimenter. A trial consisted of a 1000 Hz, 100 msec warning tone followed 800 msec later by a triangle flash. The triangle was flashed with equal probability (0.33) to the left visual field (LVF), right visual field (RVF), or both visual fields (BVF) simultaneously. Two seconds after onset of stimulus presentation, a 250 Hz tone prompted the subject to indicate the location of the flash by saying "right," "left," or "both." A practice run was given to ensure that each subject understood and was able to perform the task. A total of 300 trials were presented in separate blocks of 100 trials each. The electroencephalogram (EEG) and electrooculogram (EOG) were monitored on-line, and subjects were informed if eye movements occurred and were encouraged to maintain fixation on the center of the screen.

EEG recording

The EEG was recorded using Ag/AgCl electrodes from central (Cz) and parietal (Pz) midline sites, from homologous right and left hemisphere locations over frontal (F3 and F4), central (C3 and C4), parietal (P3 and P4), occipital (O1 and O2), and posterior temporal (T5 and T6) regions as well as from the right mastoid (A2), each referred to a left mastoid electrode (A1). Vertical eye movements and blinks were monitored with an electrode on the left inferior orbital ridge, referred to the left mastoid. Horizontal eye movements were recorded bipolarly between electrodes placed on the two external canthi.

The EEG signals were amplified with a recording bandpass of 0.02-100 Hz (-6 dB). The amplified EEG and EOG signals were digitized on-line at a sampling rate of 250 Hz over a 1024 msec epoch, beginning 100 msec prior to stimulus onset. Digitized signals were coded and stored on magnetic tape for off-line averaging and analysis.

Data analysis

ERPs were averaged separately for triangles flashed to the RVF, LVF, and BVF. Trials contaminated by eye blinks or movements, excessive muscle activity, or amplifier blocking were rejected by a computer algorithm during averaging; approximately 5-10% of the trials were lost due to such artifacts for all age groups. Averaged ERPs from all scalp sites were referred to an off-line average of the two mastoid recordings. The ERPs were quantified by computer in terms of peak measures (latency and amplitude of the maximum negative or positive deflection within a specified time window), and mean amplitude measures over a specified time interval, all referred to a baseline voltage averaged over a 100 msec interval preceding stimulus onset.

The P1 component was identified as the maximum positive peak between 75 and 150 msec, the N1 as the maximum negative peak between 125 and 200 msec, and the peak of the late positive component (LPC) as the maximum positive peak between 300 and 700 msec. Mean amplitude measures within these same windows were also taken. The P2 and N2 components were found to be too unreliable to measure.

Amplitude and latency values were subjected to a mixed design analysis of variance (ANOVA) using 3 levels of age (18-39, 40-59, and 60-82 years) as the between-subjects variable, and visual field (LVF, RVF, and BVF) and electrode site as the within-subjects factors. Repeated measures with greater than 1 degree of freedom were evaluated with P values adjusted

using the Greenhouse-Geisser epsilon correction factor. Significant condition by electrode site interactions were reassessed with repeated-measures ANOVAs of the amplitudes normalized as outlined by McCarthy and Wood (1985). Only those interactions that were significant after normalization are reported.

Linear regressions were performed on the latencies and amplitudes of the LPC, with age as the independent measure. Tests for curvilinearity of LPC latency/age and amplitude/age functions using polynomial (second and third order) regression were also performed. A significance level of $P < 0.01$ was adopted for all statistical comparisons.

Results

Grand average ERPs from the 74 subjects divided into young, middle-aged, and elderly subgroups are

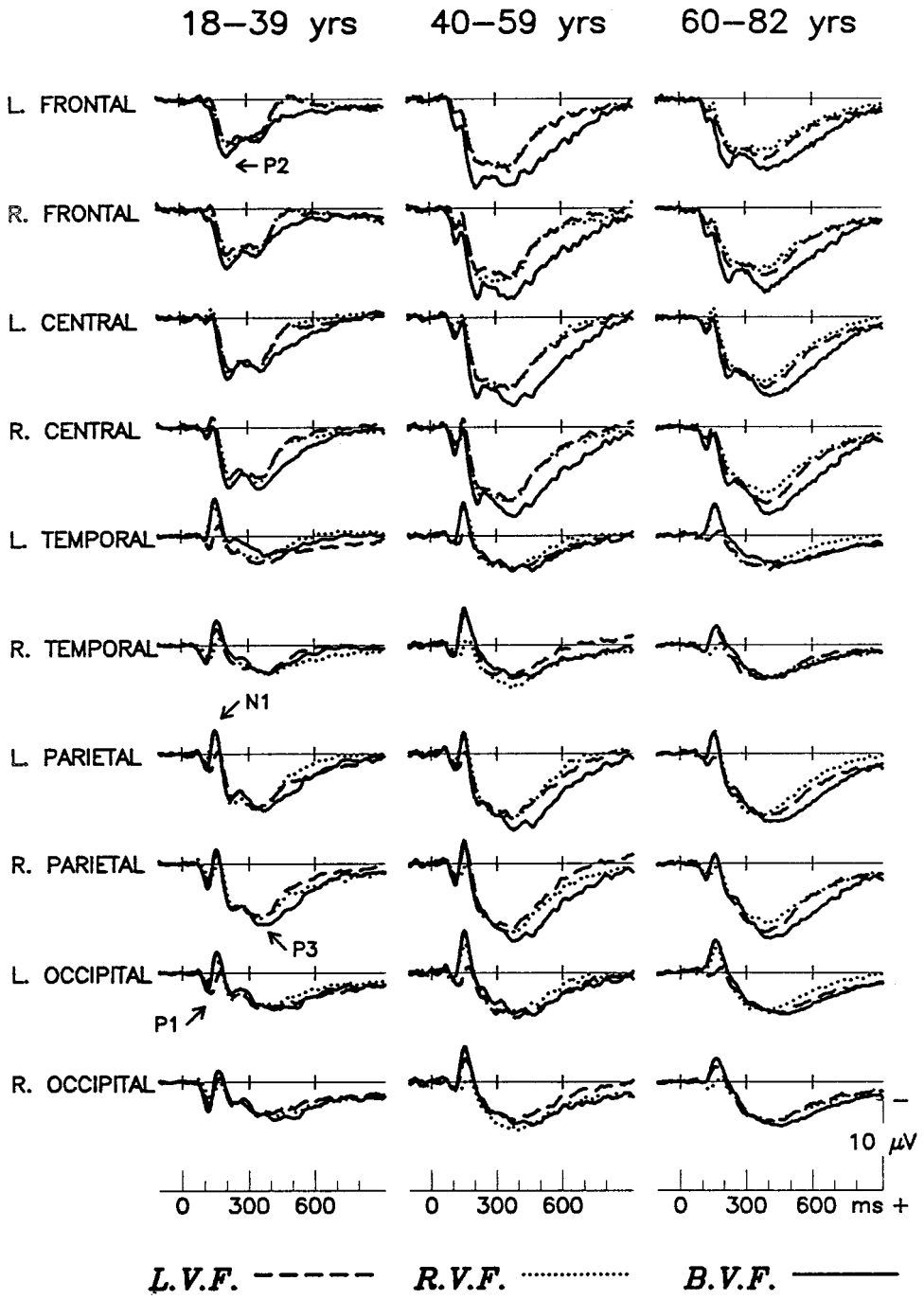

Fig. 1. Comparison of the grand average ERPs from all scalp sites for all three visual field conditions for young (18-39 years), middle-aged (40-59 years), and elderly (60-82 years) subject groups.

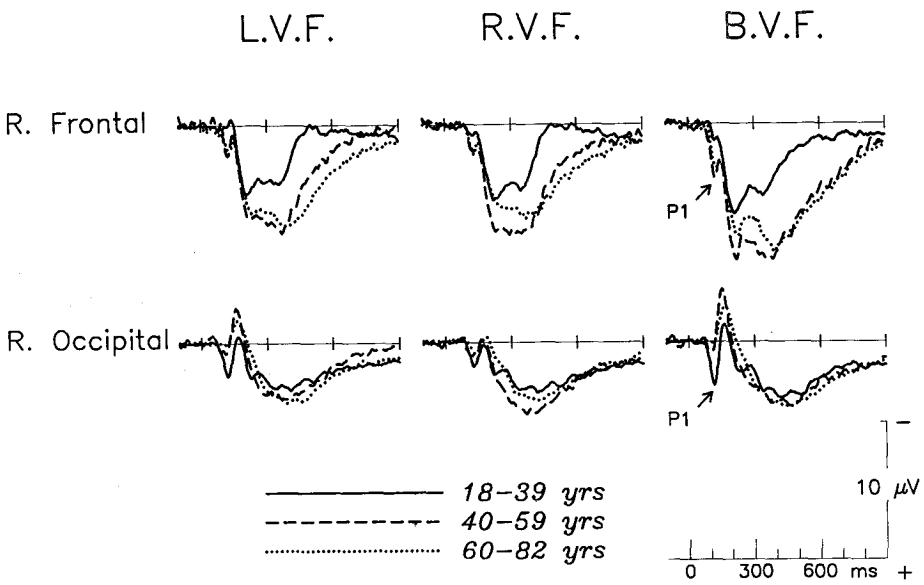

Fig. 2. Comparison of the grand average ERPs from the 3 age groups at frontal and occipital recording sites from the right hemisphere in the 3 visual field conditions, separately. Note that the P1 is larger over frontal sites for older subjects but larger over occipital sites for the younger subjects. Likewise, the LPC is much larger for the older than younger subjects over frontal sites and only slightly larger at the occipital site. The occipital N1 is smaller in the younger than older subjects.

shown superimposed for the triangle detections in the LVF, RVF and simultaneously in BVF, respectively (Fig. 1). For all ages, the averaged ERPs showed a similar morphology that included P1, Nl, P2, N2 and P3 components (N2 was often difficult to identify).

P1 component

The first robust component in the visual ERPs to a triangle flash was the P1, a positive deflection over the latency range 75-150 msec that was larger over occipital sites in the younger subject group. The P1 was slightly larger in response to bilateral than unilateral flashes (for peak amplitude, main effect of visual field, $F(2, 42) = 16.83$, $P < 0.0001$, epsilon = 0.98). For unilateral presentations, the P1 peaked earlier over the hemisphere contralateral to the visual field of presentation *(contralateral:* 108 versus *ipsilateral:* 122 msec; for peak latency, visual field by hemisphere interaction, $F(2, 142) = 78.12$, $P < 0.0001$, epsilon = 0.92). For bilateral presentations, P1 peak latency was earlier over the left (111 msec) than right (116 msec) (main effect of hemisphere, $F(1, 71) = 10.53$, $P < 0.0018$).

Neither the overall amplitude nor the latency of the P1 component is altered by age. However, there is a striking age-related change in the anterior-posterior distribution of the P1 component across the scalp (age by electrode interaction, for peak amplitude, $F(22, 781) = 7.11$, $P < 0.0001$, epsilon = 0.29; for mean amplitude, $F(22, 781) = 7.57$, $P < 0.0001$, epsilon = 0.26). Specifically, as shown in Fig. 2, the P1 was maximal occipitally for young subjects, whereas for middle-aged and older subjects, it was maximal over fronto-central regions. At the parietal and temporal sites the P1 was similar in amplitude across the 3 age groups (see Fig. 3).

N1 component

The N1 component ranged in latency between 125 and 200 msec and was largest at occipital and temporal scalp sites. The N1 was significantly larger over the hemisphere contralateral to the visual field of triangle presentation (visual field by hemisphere, $F(2, 142) = 151.16$, $P < 0.00001$, epsilon = 0.77). [4] Similarly, the N1 had an earlier peak latency over the hemisphere contralateral to the visual field of triangle presentation (visual field by hemisphere, $F(2, 142) = 13.04$, $P < 0.0001$, epsilon = 0.93).

With age, the measured N1 component increased in amplitude at certain posterior sites, including T6, O1, and O2 (for peak amplitudes, age by electrode $F(12, 426) = 3.81$, $P < 0.0001$, epsilon = 0.64; for mean amplitudes, age by electrode $F(12, 426) = 5.79$, $P < 0.0001$, epsilon = 0.61). Part of this increase may be a consequence of the reduced amplitude of the preceding and partially overlapping P1 component in the older group (e.g., see Fig. 2, right occipital tracings). There was no effect of age on the latency of the N1 component.

Late positive complex (LPC)

Following the N1 component was a complex of negative and positive potentials, including the P2, N2,

[4] The same results are obtained when the mean amplitude measure is analyzed (visual field by hemisphere, $F(2, 142) = 111.07$, $P < 0.0001$, epsilon = 0.73; for normalized data, $F(2, 142) = 93.19$, $P < 0.0001$, epsilon = 0.66).

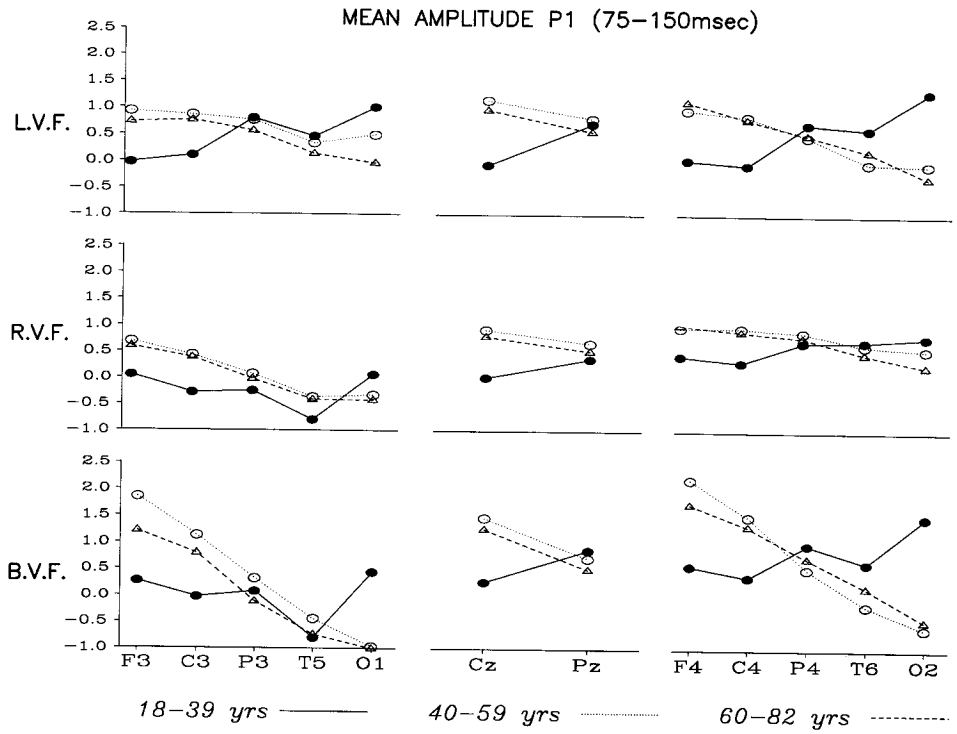

Fig. 3. Linegraphs comparing the distributions of the mean amplitudes (in μV) of the P1 component (measured between 75 and 150 msec post stimulus) for the young, middle-aged and elderly subgroups in each of the visual field conditions, separately. Note the change in the distribution of the P1 with age.

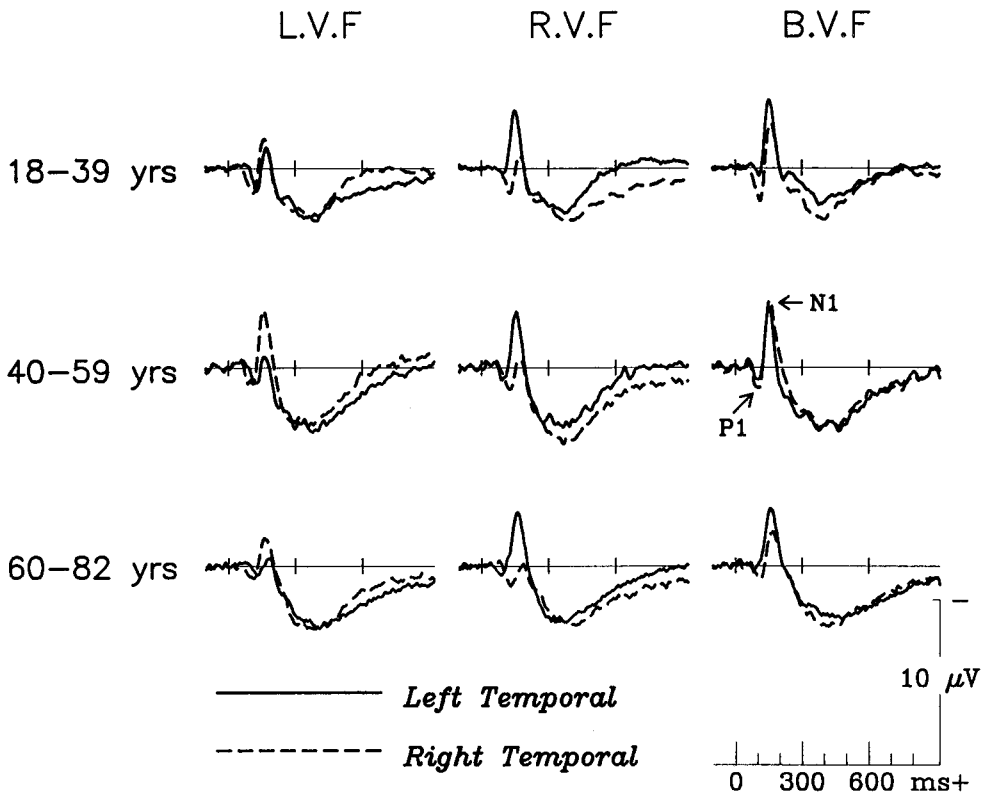

Fig. 4. Comparison of the grand average ERPs recorded from the left versus right temporal sites for the 3 age groups for all 3 visual field conditions, separately. Note that the N1 is larger over the hemisphere contralateral to the visual field of presentation while the LPC from 500 to 700 msec is larger over the hemisphere ipsilateral to the visual field of presentation.

and P3 components. Both the P2 and N2 were difficult to measure and in many cases were only visible as small inflections on the larger late positive component. This large late positivity consists of the P3 component with perhaps some overlapping slow wave activity; as these two were difficult to dissociate in this particular experiment, they are collectively referred to as the late positive complex (LPC). The LPC generally peaked at 300-500 msec and had a broad scalp distribution maximal at midline central and parietal sites in the younger subjects.

The LPC was larger and more prolonged and peaked later following bilateral than unilateral stimulus presentations (for peak amplitude between 300 and 700 msec, main effect of visual field, F (2, 142) = 22.82, P < 0.0001, epsilon = 0.93; for peak latency, main effect of visual field, F (2, 192) = 20.60, P < 0.0001, epsilon = 0.92). By and large, the LPC was bilaterally symmetrical or slightly larger over the right hemisphere except at the temporal sites where it was slightly larger over the hemisphere ipsilateral to the field of stimulation (for mean amplitude 300-700 msec, visual field by anterior/posterior by hemisphere, F (8, 568) = 15.76, P < 0.0001, epsilon = 0.47) (see Fig. 4).

In response to all visual field presentations, the LPC was slightly larger in amplitude and decidedly more frontal in the middle-aged and older as opposed to younger subjects (for mean amplitude 300-700 msec, main effect of age, F (2, 71) = 16.31, P < 0.0001; age by electrode, F (22, 781) = 10.38, P < 0.0001). While the LPC was overall larger with age, the age-related difference was more pronounced for bilateral than unilateral presentations (age by visual field, F (4, 142) = 4.08, P < 0.005), especially at the frontal sites (age by visual field by electrode, F (44, 1562) = 2.42, P < 0.001, epsilon = 0.27) (see Fig. 5).

The relationships between age and LPC amplitude were also characterized in terms of regression analyses. Separate regressions of age on LPC amplitude were performed on mean amplitudes measured at midline parietal (Pz) and frontal (averaged across F3 and F4) sites for the 3 visual field conditions separately. The scatter diagrams and regressions of age on LPC amplitude at the frontal and parietal sites in the bilateral presentation condition are shown in Fig. 6 (top and middle). There was no significant correlation between age and LPC amplitude at parietal sites (LVF: r = 0.31, RVF: r = 0.22, BVF: r = 0.19) whereas the frontal LPC increased in amplitude by about 0.07-0.09 μV/year in all visual field conditions (LVF: r = 0.62, RVF: r = 0.48, BVF: r = 0.52; all P < 0.01).

As can be seen in Fig. 2, the peak latency of the P3

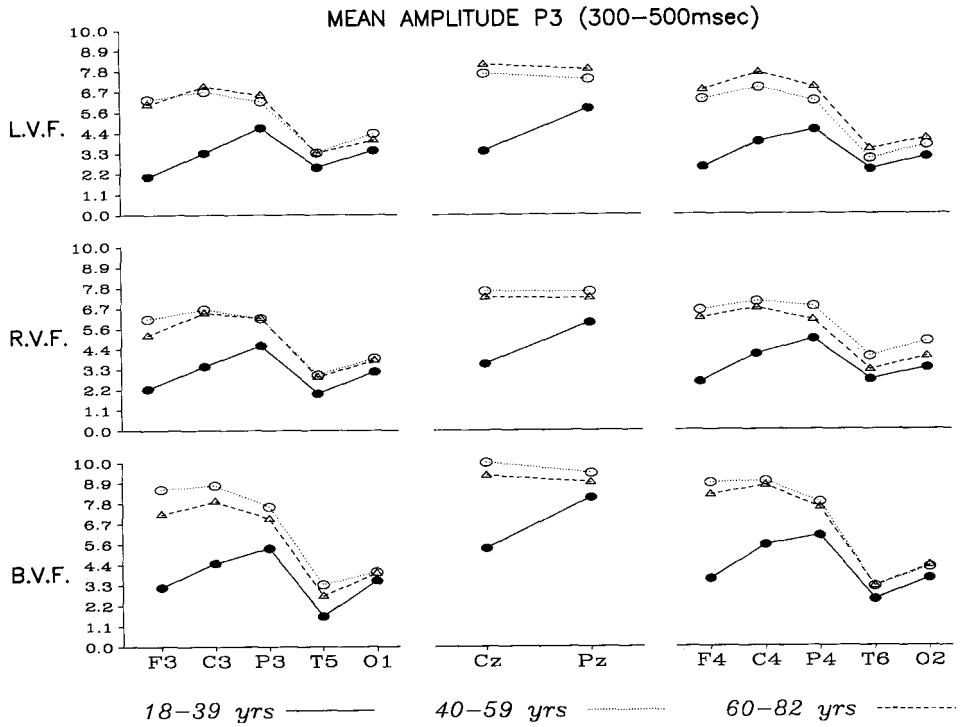

Fig. 5. Linegraphs comparing the distributions of the mean amplitudes of the LPCs (measured between 300 and 500 msec post stimulus) for the young, middle-aged and elderly subgroups in each of the visual field conditions, separately. Note the change in the distribution of the LPC with age; no significant age-related changes at the occipital and temporal sites together with large differences at frontal and central sites and moderate effects at parietal sites. These observations hold equally for midline, left hemisphere and right hemisphere recordings.

component of the LPC becomes progressively delayed with increasing age. The linear nature of the relationship between P3 peak latency (measured at the vertex) and age is illustrated in the scatter diagrams for each of the visual field conditions in Fig. 7. The age-peak latency correlations ranged between 0.52 and 0.65 (all $P < 0.01$) and the slopes between 1.4 and 1.7 msec/year for the midline vertex and parietal sites for unilateral and bilateral visual field flashes.

As some previous reports have suggested that the function relating age and P3 latency is curvilinear, the data were also fit using an orthogonal regression equation (including degrees 1 through 3) in a stepwise manner. Only the first degree (i.e., linear) proved to be significant, that is, adding a curvilinear (quadratic or cubic) factor to the linear equation did not significantly alter the percent variance accounted for.

Discussion

To summarize the principal findings of this study, unilateral and bilateral flashes were found to elicit ERPs with similar wave forms in young, middle-aged and elderly subjects. Both the P1 and LPC were larger following bilateral than unilateral flashes. For unilateral flashes, both the P1 and LPC were larger over the hemisphere ipsilateral to the visual field of presentation, while the N1 was larger over the contralateral hemisphere. Of these components, only the LPC showed an age-related increase in latency, and none showed an age-related decline in amplitude. Finally, both the P1 and the LPC become markedly more frontal in scalp distribution with advancing age. These and other aspects of the results are considered in detail below.

Visual field effects

There were slight differences in the amplitudes and lateral asymmetries of some of the ERP components as a function of visual field of presentation; all of these are consistent with previous findings. For example, both the P1 component and LPC were larger in response to bilateral than unilateral visual presentations, although in neither case was the component in the bilaterally elicited ERP as large as the sum of the components in the two unilateral ERPs. Kutas et al. (1988) noted a similar non-additivity of the LPCs elicited by unilateral visual flashes of the subject's first name embedded in a list of other names of equal length presented one at a time (compared to the ERP elicited by bilateral presentations of the same target name). Also, in line with previous reports, we found that the LPC to lateralized visual presentations showed a slight amplitude asymmetry favoring the hemisphere ipsilateral to the field of stimulus presentation (Hillyard and Muente 1984; Kutas et al. 1988; Heinze et al. 1990; Mangun and Hillyard 1990). Most importantly for present concerns, none of these visual field effects interacted with age.

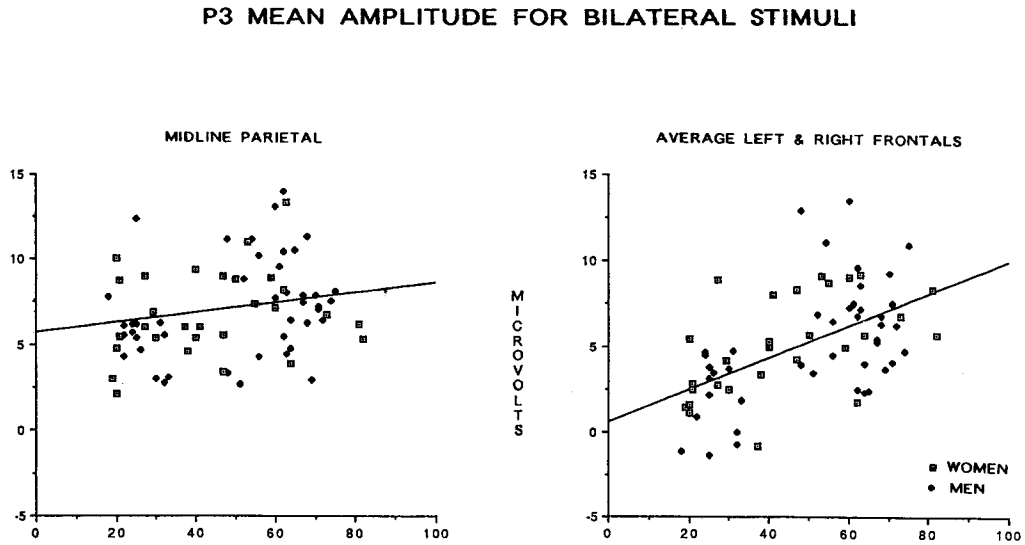

Fig. 6. Top: scatter diagram of the linear regression of age on LPC amplitude based on the average of two frontal sites (F3 and F4). Note the significant correlation $r = 0.55$, with a slope = 0.095 μV/year. Middle: scatter diagram of the linear regression of age on LPC amplitude at the midline parietal site (Pz). Note the lack of a significant correlation ($r = 0.19$). Bottom: for both regressions, the ERP measure is mean amplitude between 300 and 700 msec taken for the bilateral visual presentation condition.

P3 latency effects

As expected the latency of the centro-parietal P3 component was found to be significantly prolonged with age; this pattern held equally for men and women. Moreover, both the size of the correlations (r = 0.5-0.6) and the slope of the regressions (1.4-1.7 msec/year) of age on P3 latency fall within the range of previously reported values (Ford and Pfefferbaum 1980, 1985; Polich and Starr 1984; Miller et al. 1987; Bashore 1990; Polich 1991). Finally, we obtained no evidence of any curvilinearity in the regression line for P3 peak latency. [5] Our results thus further extend the generality of the age-related slowing of the P3 component to non-oddball paradigms. These findings are consistent with numerous reports of a slowing of some aspects of central processing with age (Bashore 1989; Bashore et al. 1989).

While age-related delays in P3 latency have been widely reported in numerous subject populations, it is possible that the individuals in the different age groups in this study varied along dimensions besides chronological age and that such factors could have affected the ERP measure in some cases. Certainly so-called cohort effects (i.e., effects of non-maturational variables) such as those due to differences in health and medical care trends, social class, nutrition habits, exposure to environmental toxins, prevailing social customs, type and average number of years of formal education) have been found to affect performance on some cognitive tasks (Schaie 1965, 1972; Bayles and Kaszniak 1987) and thus could have some effect on cognitive ERP components. Of possible concern in the present study are the minor differences in educational level among the younger, middle-aged and older subject groups (17.4, 16.6, and 14.2 years, respectively). While it would have been preferable if the groups had been matched perfectly on number of years of education, given the secular trends for increased college attendance, it is not clear whether such matching would in fact equate the groups precisely for intellectual ability or achievement. In any case, it should be noted that the number of years of education of our oldest group is well above the national average of 12 for individuals above 65 (American Association of Retired Persons 1984). Moreover, we think that it is unlikely that these small intergroup differences of 2-3 years in post-secondary educational attainment could have accounted for age-related differences in latency or other ERP parameters that we or others with a similar confound have obtained (see Friedman et al. 1993). Finally, given the data showing that exposure to complex experience can generate new synapses in cerebral cortex even in

[5] Note that this regression analysis does not include data from children. The youngest subject in this study was eighteen.

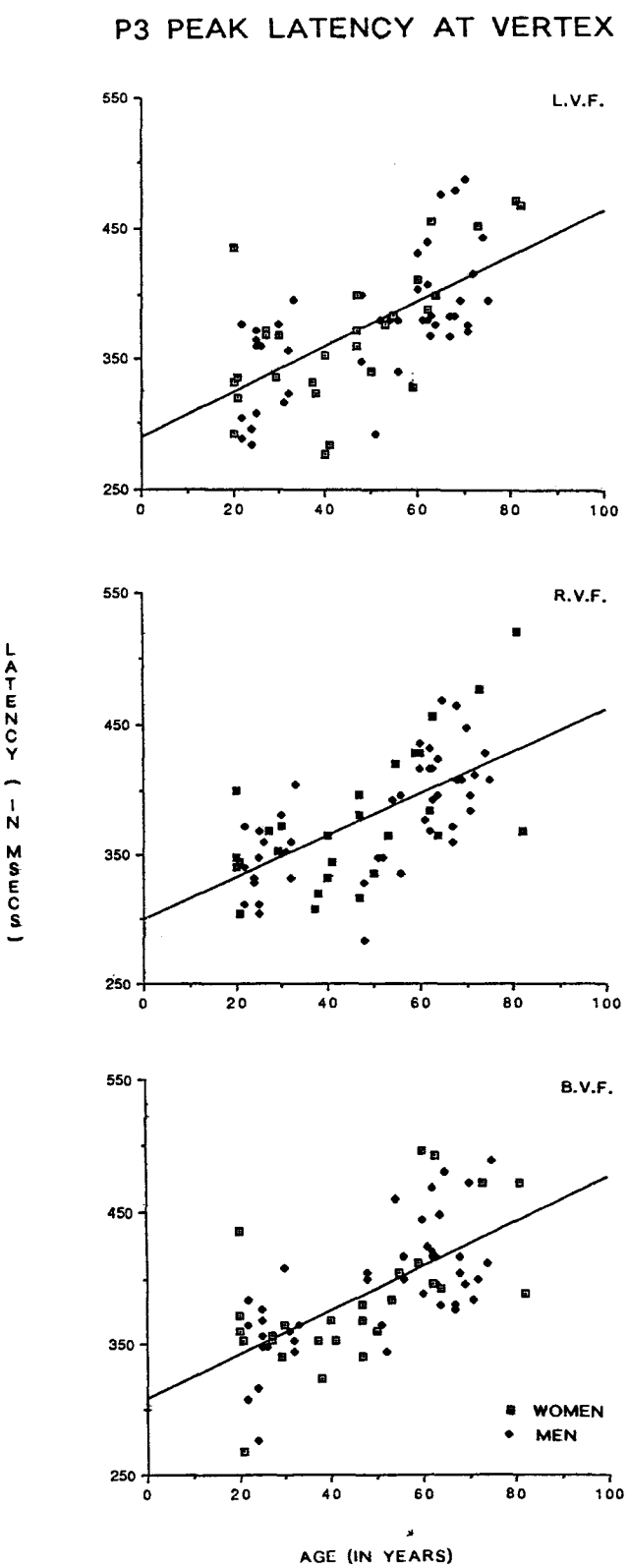

Fig. 7. Scatter diagrams of the linear regression of age on the peak latency of the P3 component at midline central site (Cz) for left (left part), right (middle part) and bilateral (right part) visual field presentations. Note the significant correlations (r = 0.65), reflecting an increase of about 1.6 msec/year. The regression equations for LVF: y =1.75x+289, r = 0.65; RVF: y =1.62x+299, r = 0.66; BVF: y = 1.68x+308, r = 0.66.

old age (Connor et al. 1980; Black et al. 1991), we suggest that an important variable for future studies of aging may be the amount of mental stimulation and/or physical exercise the middle-aged and elderly individuals are experiencing in their present time of li [6]

LPC amplitude and scalp distribution effects

Somewhat at odds with our expectations was the finding that the amplitude of the LPC did not decrease with age but was overall slightly larger in the older subjects. Although previous reports have observed that the P3 distribution becomes more frontal (and thereby more equipotential) with increasing age (Pfefferbaum et al. 1980a, 1984; Smith et al. 1980; Looren de Jong et al. 1988; Friedman et al. 1993), this pattern has not always been found (Picton et al. 1984). In fact, now consensus has emerged on the locus of these age-related distributional changes. For instance, Pfefferbaum et al. (1984) reported a significant age-related increase in P3 amplitude but only at the midline frontal site, yielding a more equipotential distribution of P3 across the scalp in elderly subjects. Picton et al. (1984) observed an age-related decrease in P3 amplitude but only at the vertex; moreover, this central decrease was *not* accompanied by any age-related change in the frontal-to-parietal P3 amplitude ratio. The present results were characterized by significant age-related increases in LPC amplitude not only at frontal sites (like Pfefferbaum et al. 1984 and Smith et al. 1980) and central sites but also at the parietal sites, unlike any previous reports. In fact, only at temporal and occipital sites were there no age-related differences in LPC amplitude.

Several different hypotheses have been offered to account for these various patterns of topographical changes in P3 with age. For example, Picton et al. (1984) suggested that age-related changes in the vertex P3 in Pfefferbaum's data might have been obscured by the overlap of negative movement-related potentials engendered by the responses to the target stimuli. Since the present task did not require an immediate manual response, and we did observe age-related changes in vertex P3, the present data are consistent with some explanation along these lines; however, it should be noted that we observed an increase, not a decrease, in vertex P3. Clearly, a much stronger test of Picton's proposal would be to compare directly the P3s in a single experimental situation with and without a motor response requirement.[7]

A different component overlap hypothesis was proposed to account for the increase in the measured amplitude of the P3 over frontal sites (Pfefferbaum et al. 1984). On this view, the increased frontality of the P3 with aging was a spurious consequence of a reduction in the amplitude of an overlapping, frontally maximal slow wave (SW); in other words, the measured P3 was larger frontally because the concomitant SW activity generated by elderly brains was smaller (i.e., less negative). Again, without additional experimental manipulations that dissociate the P3 and SW, this hypothesis is difficult to evaluate. Complicating this separation is the recent proliferation in the number and diversity of slow wave phenomena (Ruchkin et al. 1992); no longer can we assume that there is only one SW that is negative over frontal sites, small at the vertex and maximally positive over posterior sites as in the original report by Squires et al. (1975). As summarized by Ritter and Ruchkin (1992), "it makes no sense to refer to the slow wave phenomenon as the slow wave . . . there is a variety of slow waves, with different types of slow waves associated with different types of task demands" (p. 26).

It has been proposed that variations in SW amplitude are related to increased task demands in the perceptual domain or to various working memory operations, in particular for more sustained cognitive processing (Ritter and Ruchkin 1992). It is hard to determine whether these operations would be engaged in the simple visual detection task used here, and it is not apparent that SW activity contributed to the present target ERPs. Moreover, Picton et al. insist that the "reduced SW account" does not provide a satisfactory explanation of the age-related decrease in P3 at the vertex. Instead they raised the possibility that the P3 is comprised of two subcomponents one of which is central in focus and sensitive to aging; they hinted at a correspondence between these two subcomponents and the P3a/P3b division proposed by Squires et al. (1975). It would indeed be important to determine the relation between the fronto/centrally distributed positivity that is increased with age and other positive frontal components such as P3a or "novelty" P3 (Courchesne et al. 1975). Clearly, an explanation of the change in P3 scalp distribution with age in terms of reduction in the amplitude of an overlapping SW versus an increase in activity of a P3 generator with a fronto-central maximum would have very different implications for the interpretation of the changes with age, since these components are associated with distinct constellations of information processing operations as outlined above.

P1 scalp distribution and aging

A striking and unexpected finding of the present study was the marked change in the scalp distribution of the sensory evoked P1 component (measured be-

[6] Our current studies on aging are focussed on elderly adults from continuing education classes.

[7] We are currently running a variant of this triangle detection task wherein subjects are required to make a reaction time response to occasional target flashes.

tween 75 and 150 msec post stimulus) with age. For the young subjects, the P1 was largest in amplitude at the occipital sites whereas for the older subjects it was largest at frontal sites. To our knowledge this result has not been previously reported.

The occipitally distributed P1 wave has been studied extensively (mainly in younger subjects) in spatial attention tasks in which subjects were instructed or cued to attend to stimuli in one visual half-field and ignore stimuli in the opposite field (Eason 1981; Hillyard and Muente 1984; Neville and Lawson 1987; Rugg et al. 1987; Mangun and Hillyard 1988, 1990, 1991; Heinze et al. 1990). The general finding has been that stimuli presented to the attended location elicit enlarged P1 (and N1) components in relation to stimuli presented outside the focus of attention. On the basis of scalp distribution and current density mappings, the neural generators of the occipital P1 wave have been localized in the ventral-lateral extrastriate visual cortex (Brodmann's areas 18/19) (Mangun and Hillyard 1991; Mangun et al. 1993). Accordingly, the amplitude modulation of the P1 by spatially focussed attention has been interpreted as a sign of a sensory gain control of visual information passing from primary visual cortex to higher cortical areas (Mangun et al. 1993).

In the absence of detailed scalp mapping and source localization evidence, it is difficult to interpret the age-related diminution of the posterior P1 and the concurrent enlargement of an anterior positivity in the same latency range observed in the present study. A number of possible scenarios could account for such an effect. For example, since the P1 is a sensory evoked component, it might be sensitive to age-related changes in peripheral ocular factors (such as the density of the intraocular media); however, it is unclear why such factors would alter the component's scalp distribution rather than simply decreasing its amplitude or increasing its latency. A second possibility may be an age-related decline in activity of the cortical generator system of the posterior P1 that is parallelled by the emergence of an anatomically distinct anterior generator. A third possibility is that the neural generator of the posterior P1 may be altered in its cortical geometry through cerebral atrophy or selective cell loss such that its equivalent dipole shifts in orientation. A fourth possible explanation would be that the anterior P1 is increased in amplitude due to an age-related decrease in the size of the overlapping anterior N1 component. Indeed, Snyder and Hillyard (1979) reported a substantial decrease in the amplitude of an anterior N1 component with aging, which could result in an increase in the measured P1 wave anteriorly. Whatever the mechanism of this change in P1 topography, it raises the possibility that deficiencies or alterations in visual-spatial attention may accompany normal aging. Such an effect would be consistent with evidence supporting age-related decrements in visual search (Plude and Hoyer 1985) and a reduced efficiency of inhibitory mechanisms used to suppress irrelevant sensory inputs in the elderly (McDowd and Oseas-Kreger 1990; Hartman and Hasher 1991; Hasher et al. 1991). Dustman and his colleagues have proposed that age-dependent changes in intensity-amplitude functions of visual evoked potentials are due in large part to reduced monoamine efficiency and cell loss specifically in those frontal association regions believed to exert inhibitory control over other brain regions (e.g., Dustman and Shearer 1987).

Finally, although it would be premature to attempt to inter-relate the myriad neuroanatomical, neurochemical, metabolic and electrophysiological consequences of aging, certain overall patterns are becoming evident. One in particular is the finding that many of the functional distinctions between young and old brain seem to occur along the anterior-posterior axis, with the frontal lobe being disproportionately implicated in the aging process. For example, there is considerable regional variability in the overall 6% reduction in brain volume observed in the brains of octogenarians, with as large as 17% reduction in the frontal cortex. Similarly, the widening of cortical sulci and narrowing of gyri that typifies the aging brain is most prominent in the frontal and parasagittal regions. Likewise, in those cases where aging has been accompanied by a reduction in cerebral blood flow, the observed reductions are usually largest in the anterior cortex. Finally, Duara et al. (1983, 1984) found that, although the overall cerebral metabolic rate for glucose consumption does not differentiate the brain of young and elderly subjects, under conditions of minimal external stimulation, the number of statistically significant correlations between different brain areas does; the young-old differences were most pronounced within the parietal lobe and between the parietal and frontal regions. In the long run, we must relate these differences in various brain measures to age-related changes in ERPs along the anterior-posterior axis to yield an accurate picture of the aging brain and the minds they subserve.

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