

Effects of aging on event-related brain potentials and reaction times in an auditory oddball task

VICENTE J. IRAGUI,^{a,c} MARTA KUTAS,^{a,b} MARK R. MITCHINER,^{a,b}
AND STEVEN A. HILLYARD^a

^a Department of Neurosciences, University of California–San Diego, La Jolla

^b Department of Cognitive Science, University of California–San Diego, La Jolla

^c Neurology Service, Veterans Affairs Medical Center, San Diego

Abstract

Auditory event-related potentials (ERPs) were recorded from 71 healthy individuals between 18 and 82 years of age during performance of a disjunctive reaction time task in an auditory oddball paradigm. The effects of aging on reaction times and on the latencies, amplitudes, and distributions of each of the main ERP components were examined. No significant slowing of the reaction times of the elderly subjects was observed in relation to the younger ones. The peak latencies of both the N1 and P2 components elicited by standard tones were slightly but significantly slowed with age. In the ERPs of target tones, the later, endogenous components (N2, P3, and SW) showed linear increases in latency as a function of age; the later the component, the longer the age-related delay. In general, aging was associated with less negativity (both N2 and SW) and more positivity (P3) over the anterior scalp, together with a smaller P3 and a more pronounced N2 over posterior scalp areas. Most of the effects observed in target ERPs were also evident in the difference waves derived from subtraction of the standard from the target ERPs, although the slope of the age-related latency increase of N2 was shallower and that of the P3 was steeper in the difference ERPs. These findings are discussed in relation to previous accounts of ERP changes with aging.

Descriptors: Event-related potentials, Reaction time, Aging, P3

Across a variety of experimental tasks, many of which have used reaction time (RT) measures of processing speed, young adults perform more quickly and accurately than healthy but older adults. Such differences in performance have generally been taken to reflect an age-related decrease in the speed and efficiency of cognitive processing (e.g., Birren & Schaie, 1985; Cerella, 1985; Salthouse, 1985). However, there is no consensus on the extent to which the slowed responsiveness observed with normal aging can be attributed to (a) an across-the-board decline in both peripheral and central nervous system functioning, (b) a pattern of dysfunction that differentially affects various perceptual and cognitive processes, or (c) a specific deficit in a critical mental operation or resource that is utilized in many tasks (see Salthouse, 1985). Despite the recent surge of interest

in the mechanisms of both normal aging and dementia, this question remains unresolved, in part because of a lack of experimental tools for teasing apart different cognitive operations.

The use of event-related potentials (ERPs) recorded from the scalp for the relatively precise temporal tracking of human perceptual and cognitive processes has been well documented (see Hillyard & Picton, 1987; Picton, 1988; Rohrbaugh, Parasuraman, & Johnson, 1990). Indeed, a central theme of ERP research in humans has been the identification of components associated with specific sensory, motor, and cognitive operations so as to delineate the flow of information and to distinguish among serial, parallel, and hierarchical processing modes. As a result, a number of components have been identified and linked to different mental processes, including early feature analysis and attentional gating (e.g., N1, Nd), sensory mismatch detection (e.g., mismatch negativity [MMN]), stimulus discrimination (e.g., Na, N2), completion of stimulus evaluation, cognitive closure, or context updating (P3b), and further analyses of stimulus significance (e.g., slow wave [SW]). Although no single ERP component has been linked to a specific cognitive operation unequivocally, there is generally good agreement as to the relative timings of the various components, the broad classes of sensory, motor, and cognitive processes associated with each component, and the antecedent conditions that are necessary for their elicitation.

This research was supported by grants from NINDS (NS 21604 and NS 17778) and NIA (AG08313) and by the Veterans Administration Research Service. M. Kutas was supported by a Research Career Development Award from NIH (MH00322).

We thank Drs. Robert Katzman and David Salmon for making subjects from the UCSD Alzheimer Disease Research Center registry available for this study and Ms. Barbara Reader and Ms. Lisa Russell for assistance with manuscript preparation.

Address requests for reprints to: Dr. Vicente Iragui, Department of Neurosciences (0608), University of California–San Diego, La Jolla, CA 92093-0608.

Several of these ERP components may be recorded in the so-called auditory *oddball* task, wherein subjects listen to a random (Bernoulli) sequence of tones of two frequencies and either count or push a button following each occurrence of the less probable (*oddball* or *target*) tone. The averaged ERPs elicited by the more probable or *standard* tones are characterized by evoked N1 and P2 components, whereas the ERPs elicited by the target tones contain these two exogenous components as well as a later sequence of endogenous components (e.g., N2, P3, and SW). The P3 seems to have a similar scalp distribution regardless of stimulus modality (Snyder, Hillyard, & Galambos, 1980; although see Johnson [1989a, 1989b] for a different position), but the N2 clearly has a modality-specific distribution; it is larger at the vertex for auditory stimuli and at preoccipital areas for visual stimuli (Simson, Vaughan, & Ritter, 1977). Furthermore, the latencies of N2 and P3 change with experimental manipulations of stimulus evaluation demands (Fitzgerald & Picton, 1983; Ritter, Simson, Vaughan, & Friedman, 1979). These observations suggest that N2 reflects stimulus categorization processes that are engaged during the decision-making process itself, whereas P3 may be more closely related to memory functions to prepare for stimulus processing of future trials (e.g., Donchin, Karis, Bashore, Coles, & Gratton, 1986).

Although early investigations had suggested a correlation between RT and P3 latency (e.g., Ritter, Simpson, & Vaughan, 1972; and see Donchin, Ritter, & McCallum, 1978), later studies revealed that the two measures can be dissociated and provide different and complementary estimates of mental chronometry (Kutas, McCarthy, & Donchin, 1977; Magliero, Bashore, Coles, & Donchin, 1984; McCarthy & Donchin, 1981). Whereas both RT and P3 latency measures are sensitive to changes in stimulus processing demands (i.e., encoding, recognition, and classification), the timing of P3 is relatively insensitive to demands of the response selection and execution process indexed by the overt response. Thus, manipulations of stimulus evaluation (varying target discriminability) and response selection (varying stimulus-response compatibility) have shown that P3 latency increases as stimulus discriminability becomes more difficult but is relatively unaffected by stimulus response incompatibility. In contrast, RT is affected significantly by both manipulations (Pfefferbaum, Ford, Johnson, Wenegrat, & Kopell, 1983; Pfefferbaum, Ford, Weller, & Kopell, 1985; Ragot, 1984). The relationship between P3 latency and RT also has been examined using the Sternberg memory-matching paradigm, a task that requires subjects to make a judgement on each trial about whether or not a probe stimulus belongs to a previously memorized set of items (Adam & Collins, 1978; McCallum, Curry, Cooper, Pocock, & Papakostopoulos, 1983; Roth, Ford, & Kopell, 1978). Generally, P3 latency has been found to increase as the size of the memory set increases, but the change is proportionally less than the associated increase in RT. These observations suggest that P3 is a more accurate measure of the serial comparison operation that underlies stimulus recognition than is the response latency. RT is presumably sensitive to additional processes of response mobilization and "double checking" that also vary with memory set size.

In the growing literature on the effects of increasing age on various components of the ERP, a significant segment is devoted to changes in the P3 component (for review, see Bashore, 1990; Bashore, Osman, & Heffley, 1989), particularly in two-tone auditory oddball tasks (Barrett, Neshige, & Shibasaki, 1987; Brown, Marsh, & LaRue, 1983; Goodin, Squires, Hen-

derson, & Starr, 1978; Kraiuhin, Gordon, Stanfield, Meares, & Howson, 1986; Patterson, Michalewski, & Starr, 1988; Picton, Stuss, Champagne, & Nelson, 1984; Polich, Howard, & Starr, 1985; Puce, Donnan, & Bladin, 1989; Squires, Chippendale, Wrege, Goodin, & Starr, 1980; Syndulko et al., 1982). The clear consensus of these studies is that the peak latency of the P3 is longer in elderly than in young adults. Differences of opinion arise, however, as to the exact nature of the function relating age to P3 latency. Most reports have described a linear increase of P3 latency with age across the adult life span, with slopes ranging from 0.91 to 1.85 ms/year (Barrett et al., 1987; Goodin et al., 1978; Patterson et al., 1988; Picton et al., 1984; Polich et al., 1985; Syndulko et al., 1982; see Table 5). However, Brown et al. (1983) reported that the relation between P3 latency and age is curvilinear rather than linear; they observed little change in P3 latencies in individuals younger than 45 years of age, but there was a striking, positively accelerating increase of 3.14 ms/year thereafter. An increase in P3 latency with age also has been observed in more complex auditory oddball tasks and in visual and somatosensory oddball tasks (Barrett et al., 1987; Beck, Swanson, & Dustman, 1980; Mullis, Holcomb, Diner, & Dykman, 1985; Pfefferbaum, Ford, Wenegrat, Roth, & Kopell, 1984; Picton et al., 1984).

There is also general agreement about the effects of aging on the latencies of the N1 and N2 components of the ERP elicited by target tones in oddball tasks. The N1 remains relatively invariant in latency across the life span (Barrett et al., 1987; Brown et al., 1983; Picton et al., 1984; Syndulko et al., 1982), whereas N2 latency increases, albeit to a lesser degree than does the P3 (Barrett et al., 1987; Goodin et al., 1978; Picton et al., 1984). There is less agreement about the relationship between age and the latencies of the target P2 and the standard N1 and P2. For example, although some investigators have found that target P2 latencies increase with age (Picton et al., 1984; Syndulko et al., 1982), others have observed no such change (Barrett et al., 1987; Brown et al., 1983). Similarly, Goodin et al. (1978) reported age-related increases in N1 and P2 latencies for standard tones, which Barrett et al. (1987) failed to observe. These apparent discrepancies in the reported effects of age on the different ERP components may be due, at least in part, to the use of different subject populations and sample sizes or different stimulus parameters and response requirements and/or differences in component measurement and analyses (see Fein & Turetsky, 1989).

Despite the large number of studies that have employed the auditory oddball task in assessing the effects of aging on the ERP, there are almost no studies in which each of the major ERP components of both standard and target tones were analyzed (see Table 5). The majority only reported effects on the N2 and P3 to targets. Notable exceptions are studies by Picton et al. (1984), who analyzed all of the components elicited by target tones, and those of Goodin et al. (1978) and Barrett et al. (1987), who analyzed the components elicited by standards. Moreover, among those who studied the N2 and P3, some based their conclusions on measurements derived from target minus standard difference waves (e.g., Goodin et al., 1978), whereas others measured the ERP of target tones (e.g., Barrett et al., 1987; Brown et al., 1983; Picton et al., 1984; Polich et al., 1985; Syndulko et al., 1982).

The majority of the ERP investigations of aging that have used the two-tone auditory oddball paradigm did not require an overt response to each target but rather assessed performance

via accuracy of subvocal mental counts of target tones (Brown et al., 1983; Goodin et al., 1978; Kraihin et al., 1986; Polich et al., 1985; Syndulko et al., 1982). Only Picton et al. (1984) compared age-related changes in ERPs and RT. In 24 subjects, they found neither an effect of age on RT nor a significant between-subject correlation of RT and P3 latency. In other paradigms, similar comparisons of the effects of age on P3 latency and RT have yielded inconsistent results. For example, Ford, Duncan-Johnson, Pfefferbaum, and Kopell (1982a), using a forced choice RT task in 10 young and 10 elderly individuals, found no age-related differences in RT and no significant correlation between RT and P3 latency. Utilizing a more complex three-tone discrimination RT task in 115 subjects, Pfefferbaum et al. (1984) found a significant age-related increase in P3 latency but not in RT. In contrast, Patterson et al. (1988), using a disjunctive RT task, observed parallel increases in P3 latency and RT in a group of 15 elderly individuals compared with 12 younger ones.

The present study was designed to compare ERPs and RTs in 71 healthy individuals between the ages of 18 and 82 years. Because one of the major goals was to collect normative data against which patient groups could be compared, a simple auditory oddball task rather than a more complex paradigm was chosen. Both early and late ERP components to standard and target tones were analyzed with RTs. To evaluate the effects of different measurement procedures, we compared the N2, P3, and SW components measured in the target ERPs with those taken on difference waves derived from subtraction of the standard from the target ERP. Moreover, we assessed the extent to which the observed effects of age on ERP latencies and RTs were described by linear rather than curvilinear functions.

Methods

Subjects

Seventy-one subjects (44 men, 27 women) participated in the study. There were 20 subjects ages 18–29, 8 in each decade between the ages of 30 and 59, 17 ages 60–69, and 10 between 70 and 82 years old. All were normal, healthy individuals with no history of neurological or psychiatric disease, hearing impairment, or drug/alcohol abuse and who were able to discriminate high- from low-frequency tones with ease. Young and middle-aged subjects, recruited from the University of California–San Diego (UCSD) campus, were screened on the basis of a brief questionnaire. Elderly subjects (>50 years) were recruited from the normal control population of the UCSD Alzheimer's Disease Research Center registry; these individuals lived independently and underwent medical and neurological exams and psychometric testing. All subjects scored better than 28 out of 30 points on the Mini-Mental State Exam (Folstein, Folstein, & McHuch, 1975).

Stimuli and Experimental Design

Subjects sat in a comfortable reclining chair in an electrically shielded room. During the experiment, they were instructed to fixate on a point located about 1 m in front of them, while keeping eye movements and blinks to a minimum.

The stimuli were 40-ms tone bursts with 5-ms rise and fall times presented binaurally at an intensity of 70 dB HL. The interstimulus interval was 1.5 s. Two tones of different frequencies were presented in random order: 80% of the tones were

1,000-Hz standards and 20% of the tones were 1,500-Hz targets. Subjects were instructed to press a button in response to each target tone as quickly and as accurately as possible. A practice run was used to ensure that all individuals understood this task. An experimental session consisted of four blocks of 220 trials each. The order of the responding hand was counter-balanced both within and across subjects.

Recording System

The electroencephalogram (EEG) was recorded using Ag/AgCl electrodes placed according to the 10–20 system (Jasper, 1958). Recordings were obtained from the left and right frontal (F3 and F4), central (C3 and C4), parietal (P3 and P4), occipital (O1 and O2), and posterior temporal (T5 and T6) sites, as well as from midline sites (Cz, Pz) and right mastoid (A2), each referred to the left mastoid (A1). Each scalp channel was rereferenced off-line to the average of the left and right mastoid recordings. The electrooculogram (EOG) was recorded in two ways: vertical eye movements and blinks were monitored with an electrode placed below the left eye (i.e., on the inferior orbital ridge), referred to the left mastoid; horizontal eye movements were monitored via a right to left bipolar montage at the external canthi.

The EEG and EOG were amplified by Grass P511 amplifiers with an 8-s time constant. The high-frequency half-amplitude cutoff was 300 Hz (–6 dB rolloff). The amplified signals were digitized on-line at a sampling rate of 256 Hz and stored on magnetic tape along with stimulus and response codes for subsequent averaging. The length of the sampling epoch was 1,000 ms.

Data Analyses

Separate ERP averages were obtained for all standard tones, all target tones, and correctly detected target tones. Each averaged waveform consisted of a 1,000-ms epoch including 100 ms prior to stimulus onset. Trials contaminated by eye blinks or movements, excessive muscle activity, or amplifier blocking were rejected by a computer algorithm prior to averaging (24.4% of the trials were eliminated because of artifacts in the young group, 24% in the middle-age group, and 23.9% in the elderly group).¹

ERPs were quantified by computer algorithms in terms of peak latencies and amplitudes of the maximum negative or positive deflections within specified latency ranges determined by visual inspection of the individual subject averages, measured relative to a 100-ms prestimulus baseline voltage. The N1 and P2 were measured in both standard and target tone ERPs. Because the N1 and P2 components were too small at temporal and occipital sites to yield reliable peak measurements, these waves were measured at frontal, central, and parietal electrodes only (a total of eight sites). N1 was identified as the most negative point between 50 and 150 ms poststimulus; P2 was identified as the maximum positive point between 125 and 230 ms.

¹The computer algorithm requires that thresholds be set by the experimenter for each subject's data; these values are in arbitrary units. The procedure thus involves the experimenter to determine the values associated with the smallest vertical and horizontal eye movements and to set thresholds for rejection in the vertical eye, horizontal eye, and frontal eye channels. Vertical eye movements are identified in large part by virtue of their polarity reversal across the eye (i.e., between electrode below the eye and frontal electrodes). Horizontal eye movements are considered those that deviate from a flat line.

The N2, P3, and SW were measured in target tone ERPs as well as in the difference waves derived from a point-by-point subtraction of the standard from the target ERPs at all scalp electrodes (a total of 12 sites). N2 was identified as the most negative point between 175 and 275 ms, and P3 as the maximum positive point between 250 and 500 ms. Because the peak of the SW could be identified consistently only in frontal recordings, its latency was taken as the most negative point between 375 and 900 ms at those sites. SW amplitude measurements at all the sites were taken at the average latency observed at F3 and F4 (F3/4). SW analyses were based on 52 subjects only because the SW peak could not be identified in the remaining 19 individuals.

Linear regressions performed included (a) age on the amplitudes and latencies of the different ERP components, (b) age on RT, (c) RT on the ERP latencies, and (d) N2, P3, and SW measures taken in target ERPs on those taken in difference ERPs. For N1, P2, N2, and P3, separate regressions were performed on measurements from Cz and Pz. Regressions on SW latency used the average value from F3 and F4 measurements. Because SW was negative over frontal sites and positive over posterior sites, separate regressions were carried out for amplitude measures from F3/4, Cz, and Pz.

To explore the possibility of a curvilinear relationship between age and the latencies of N2, P3, and SW, we attempted to fit the data with orthogonal polynomial regression equations (including degrees 1–3) in a stepwise manner (BMDP P5R).

Amplitude and latency values were subjected to repeated-measures analyses of variance (ANOVA) with three levels of age (18–39, 40–59, 60–82 years) as a between-group factor and two levels of stimulus type (target vs. standard), two levels of responding hand (right vs. left), two levels of gender, and electrode site (8 levels for N1 and P2; 12 levels for N2, P3, and SW) as within-subject factors. The probabilities reported for interactions are those obtained after Greenhouse-Geisser correction (Keselman & Rogan, 1980), and the epsilon correction factor (ECF) is given for all significant interactions. Significant condition by electrode site interactions were reassessed with repeated-measures ANOVAs of normalized amplitudes as outlined by McCarthy and Wood (1985). Only significant interactions on normalized amplitude measures are reported.

Results

Target Detection Accuracy

Age had a significant effect on the rate of target detection ($F[2, 67] = 5.61, p = .006$). The 60–82-year-old subjects detected significantly fewer targets (88.2%) than did the middle (96.6%) and younger (96.4%) age groups. Responding hand had no effect on accuracy rate, and there were no significant interactions between responding hand and age group.

Reaction Times

The mean RTs (and standard deviations) to target tones collapsed across dominant and nondominant hands were 371 (49) ms for the 18–39 year olds, 384 (71) ms for the 40–59 year olds, and 402 (82) ms for the 60–82 year olds. The slight increase in RTs with age was not statistically significant.

Event-Related Potentials

Grand average ERPs (Figure 1) show that standard tones evoked N1 and P2 components, whereas target tones elicited

Table 1. Linear Regressions of Age on ERP Component Amplitudes

Stimulus	Component (electrode site)	Intercept (μV)	Slope ($\mu V/\text{year}$)	SE	<i>r</i>	<i>p</i>
Standard	N1 (Cz)	−6.1	−0.00	2.5	−.01	n.s. ^b
	P2 (Cz)	3.9	0.02	2.3	.13	n.s.
Target	N1 (Cz)	−6.6	0.01	2.7	−.05	n.s.
	P2 (Cz)	1.5	0.03	3.8	.14	n.s.
	N2 (Cz)	−0.3	−0.08	4.8	−.29	*
	(Pz)	2.4	−0.08	3.6	−.38	**
	P3 (Cz)	10.8	−0.09	5.3	−.30	*
	(Pz)	14.4	−0.10	5.1	−.34	**
	SW ^a (F3/4)	−7.3	0.06	2.8	.38	**
	(Cz)	−0.1	−0.07	4.2	−.30	*
	(Pz)	6.4	−0.09	4.3	−.35	**
Difference waves	N2 (Cz)	−3.4	−0.09	5.3	−.31	**
	(Pz)	−0.1	−0.07	3.9	−.34	**
	P3 (Cz)	10.2	−0.08	5.3	−.29	*
	(Pz)	13.4	−0.08	4.9	−.31	**
	SW (F3/4)	−6.8	0.05	3.1	.27	*
	(Cz)	−0.3	−0.08	4.3	−.33	*
	(Pz)	6.0	−0.09	4.2	−.36	**

^aSW = slow wave. ^bn.s. = not significant ($p > .05$).

* $p < .05$; ** $p < .001$.

these exogenous components followed by N2, P3, and SW components. Because there were no abrupt discontinuities between adjacent age groups in any of the ERP measures (see Figures 3, 5), data were pooled into three groups (18–39, 40–59, 60–82 years) for the ANOVAs. Figure 1 shows the grand average ERPs for these three age groups, and Figure 2 shows the difference waves derived from a point-by-point subtraction of the standard tone ERPs from the target tone ERPs. These averaged ERPs were collapsed across responding hand. Because there were no differences between the averaged ERPs for all targets and those for correctly detected targets, analyses were based on the latter.

Age had an effect on a number of latency and amplitude measurements. Scatter plots of the relationships between age and the latencies of each of the main components (N1, P2, N2, P3, SW) of the ERPs elicited by target tones are shown in Figure 3. The results of linear regressions of age on the amplitudes and latencies of the different components are presented in Tables 1 and 2. Amplitude and latency measures for the various components in the three age groups are provided in Tables 3 and 4.

The N1 and P2 measures were taken from the ERPs for standard and target tones, whereas later components were measured in the target ERP and in difference waves (target minus standard). In general, N2, P3, and SW measures from the target ERPs and difference waves were highly correlated (across subjects). Correlation coefficients for N2, P3 (taken at Cz), and SW (taken at F3/4) were .81, .89, and .84 for latencies, and .91, .96, and .88 for amplitudes ($p < .0001$ for all measures), respectively.

N1 Component of Standard and Target Tones

Distribution. Across all ages, both the standard and target tones evoked an early negative component (N1) between 80 and

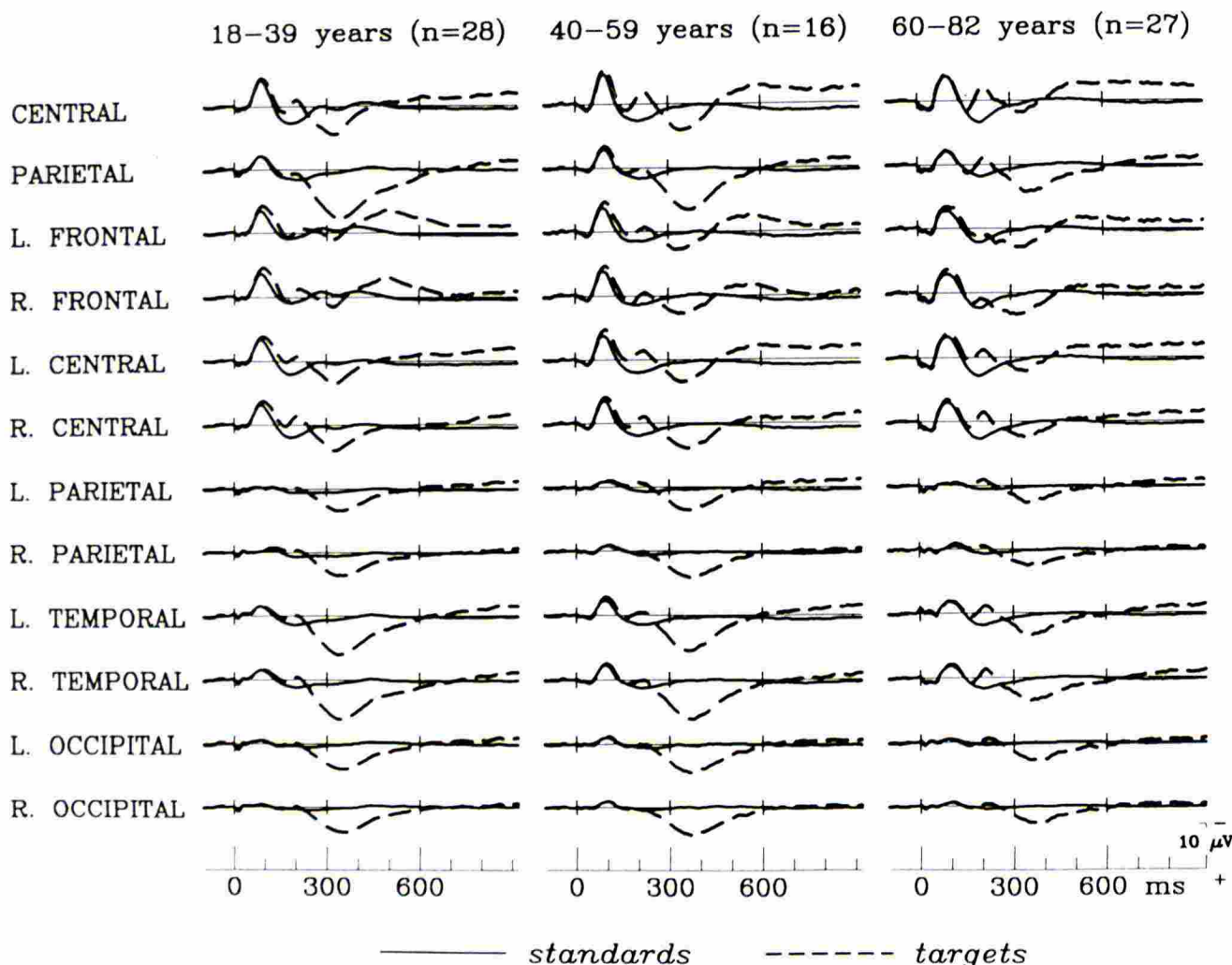


Figure 1. Grand average ERPs elicited by standard (1,000 Hz) and target (1,500 Hz) tones for the young (18–39 years), middle-aged (40–59 years), and elderly (60–82 years) groups. Negativity is up on this and all subsequent figures.

100 ms poststimulus onset with a frontocentral maximum. The largest N1 was observed at the vertex; laterally, N1 was large at the frontal and central sites, moderate at the parietal sites, and small at the temporal and occipital sites (main effect of electrode site, $F[7, 476] = 148.9$, $p < .0001$; ECF = 0.3401). There were no age-related changes in the distribution across the scalp of the N1 component of either standard or target tones.

Amplitude. Target tone N1s were approximately $1 \mu\text{V}$ larger than those evoked by standard tones (main effect of stimulus type, $F[1, 68] = 60.34$, $p < .0001$; ECF = 0.5219), and the difference was more pronounced at lateral than midline sites (Stimulus Type \times Electrode interaction, $F[7, 476] = 14.02$, $p < .0001$). There were no age-dependent effects on the amplitude of the N1 component elicited by either standard or target tones in either the ANOVAs or regression analyses (Table 1).

Latency. Target tone N1s had significantly longer latencies than did those evoked by standard tones (main effect of stimulus type, $F[1, 68] = 35.92$, $p < .0001$); this difference was more pronounced at lateral than midline sites (Stimulus Type \times Electrode interaction, $F[7, 476] = 3.33$, $p < .007$; ECF =

0.6796). Although the latency of the N1 for target tones was unaffected by age, the latency of the N1 for standard tones showed a small but significant (0.13–0.18 ms/year) increase with age at the midline sites (Table 2).

The latencies of N1s elicited by standard tones were not correlated with RTs across subjects. By contrast, the latencies of N1s for target tones were significantly correlated with RTs produced by the dominant hand ($r = .26$, $F[1, 66] = 4.63$, $p < .03$), but their correlation with RTs elicited by the non-dominant hand was not significant ($r = .21$, $F[1, 66] = 3.15$, $p < .077$).

P2 Component of Standard and Target Tones

Distribution. Across all ages, the ERPs of both standards and targets were characterized by a positive peak (P2) between 160 and 175 ms, which was largest at central sites.

Amplitude. Standard tone P2s were on the average $2 \mu\text{V}$ larger than those evoked by target tones (main effect of stimulus type, $F[1, 68] = 11.88$, $p < .001$), and the difference was more pronounced at central than frontal or parietal sites (Stim-

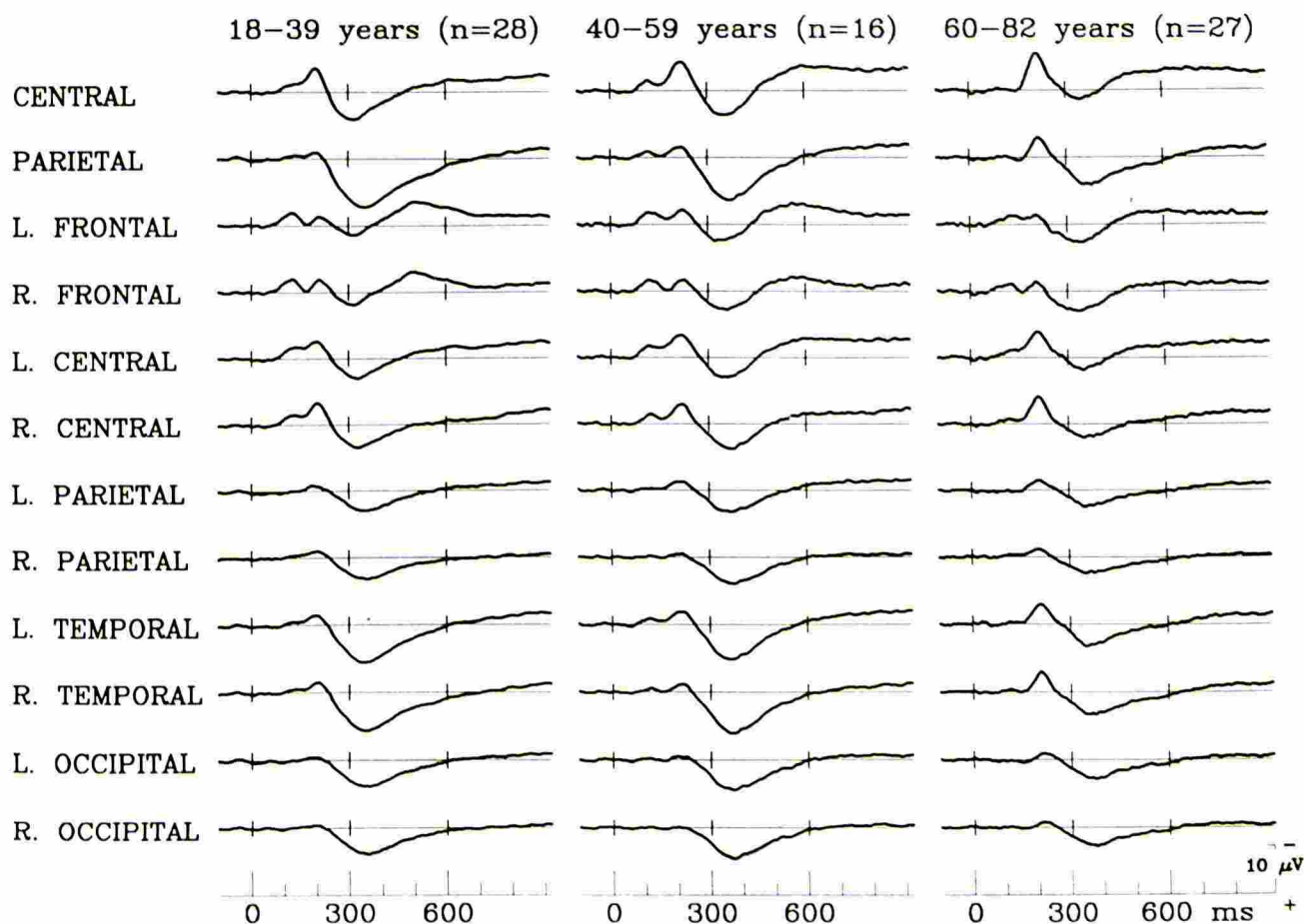


Figure 2. Grand average difference waveforms resulting from a point-by-point subtraction of the standard tone ERPs from the target tone ERPs for the young (18–39 years), middle-aged (40–59 years), and elderly (60–82 years) groups.

Table 2. Linear Regressions of Age on ERP Component Latencies

Stimulus	Component (electrode site)	Intercept (ms)	Slope (ms/year)	SE	r	p
Standard	N1 (Cz)	85	0.13	8	.30	*
	(Pz)	82	0.18	9	.36	**
	P2 (Cz)	163	0.55	22	.43	***
	(Pz)	161	0.59	28	.38	**
Target	N1 (Cz)	91	0.08	12	.12	n.s. ^b
	(Pz)	91	0.08	13	.12	n.s.
	P2 (Cz)	167	0.03	20	.03	n.s.
	(Pz)	171	0.03	24	.02	n.s.
	N2 (Cz)	198	0.49	29	.31	**
	(Pz)	188	0.58	25	.41	***
	P3 (Cz)	294	0.88	44	.36	**
	(Pz)	313	0.80	43	.33	*
Difference waves	SW ^a (F3/4)	463	1.37	53	.43	***
	N2 (Cz)	203	0.30	26	.21	n.s.
	(Pz)	192	0.47	24	.36	**
	P3 (Cz)	285	1.19	41	.49	***
	(Pz)	314	0.86	41	.38	**
	SW (F3/4)	479	1.37	52	.43	***

^aSW = slow wave. ^bn.s. = not significant ($p > .05$).

* $p < .05$; ** $p < .001$; *** $p < .0001$.

ulus Type \times Electrode interaction, $F[7, 476] = 19.55$, $p < .0001$; ECF = 0.5061). P2 amplitudes elicited by standard and target tones did not vary with age (Table 1).

Latency. Standard tone P2s had significantly longer latencies than did those evoked by target tones (main effect of stimulus type, $F[1, 68] = 49.16$, $p < .0001$). The latency of the P2 for standard tones at midline sites (Cz, Pz) increased significantly (0.55–0.59 ms/year) with age; the latency of the P2 evoked by target tones, however, was invariant with age (Table 2).

Like the target N1, target P2 latencies were correlated significantly with RTs of the dominant hand ($r = .25$, $F[1, 66] = 4.30$, $p < .04$); no such relation was observed for standard tone P2s for either hand.

Effects of Responding Hand on N2, P3, and SW Components

Based on previous work, the button pressing task was expected to elicit movement-related ERP activity, especially over the central areas (see McCallum, 1988). Therefore, before carrying out further planned analyses, we examined the effect of responding hand on N2, P3, and SW. Latency and amplitude measures were subjected to repeated-measures ANOVAs with three levels of age as a between-group factor and two levels of

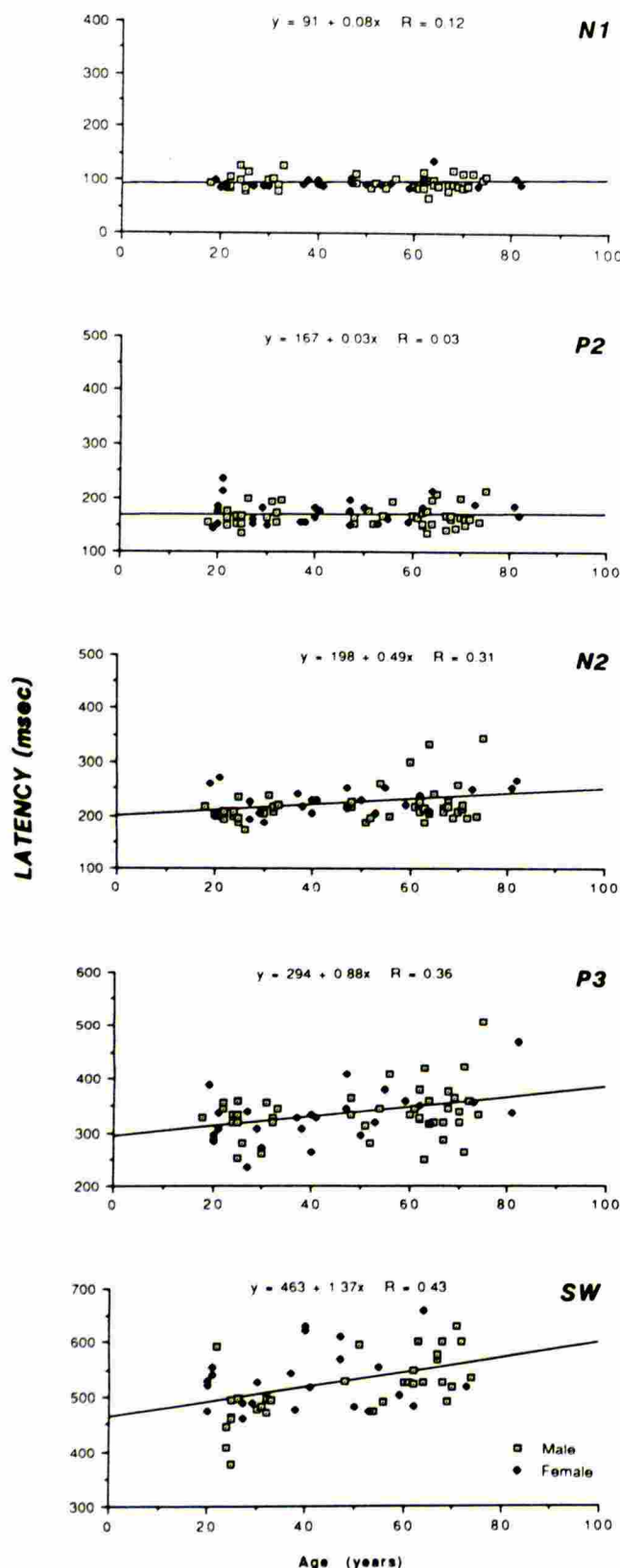


Figure 3. Scatter diagrams of the latencies of each of the main components of the ERPs elicited by target tones on age. Measures for N1, P2, N2, and P3 were taken from the vertex recording; the estimates for SW were based on the average of values measured at the two frontal (F3/4) sites for the 52 subjects who had a frontal SW peak. The two different symbols on each scatter plot indicate that individual's gender.

responding hand (right vs. left), and two electrode sites (right and left central) as within-group factors.

N2, P3, and SW amplitudes. The N2 component was larger contralateral to the responding hand (in target waves: $F[1, 68] = 46.59$, $p < .0001$; in difference waves: $F[1, 68] = 25.03$, $p < .0001$). The centrally negative SW also was larger contralateral to the responding hand in target waves ($F[1, 68] = 9.06$, $p < .004$) but not in the difference waves ($F[1, 68] = 3.19$, $p < .08$). In contrast, the P3 component was larger ipsilateral to the responding hand (in target waves: $F[1, 68] = 53.27$, $p < .0001$; in difference waves: $F[1, 68] = 50.92$, $p < .0001$).

N2 and P3 latencies. N2 latencies in target (but not difference) waves were significantly longer over the central site contralateral to the responding hand (225 vs. 218 ms; $F[1, 68] = 11.12$, $p < .002$). P3 latencies were unaffected by responding hand.

Because no significant interactions were observed between responding hand and age group for any of the ERP measures, all subsequent analyses were carried out on data pooled across responding hand.

N2 Component of Target and Difference ERPs

Amplitude and distribution. Following the P2 component, target tone ERPs were characterized by a negative component (N2) between 175 and 275 ms poststimulus, which was largest at central sites for all ages. In target waves, N2 became smaller anteriorly and larger centrally and posteriorly with advancing age (Age \times Electrode interaction, $F[22, 748] = 8.80$, $p < .0001$; ECF = 0.3140) (Figures 1, 4). However, the distribution across the scalp of the difference N2 was not significantly altered by age.

Amplitude. Linear regression analyses of the target and difference N2 amplitudes at midline sites revealed small but significant correlations with age with a slope of -0.07 to $-0.09 \mu\text{V}/\text{year}$ (see Table 1).

Latency. Linear regression analyses of the target N2 at midline sites revealed significant increases in latency with age of 0.5 – 0.6 ms/year (Table 2, Figure 3); the slope of this function was less steep in the difference waveforms and was statistically significant only at Pz. Polynomial regression analyses indicated no curvilinear trend. There was no significant correlation between mean N2 latencies and RTs.

P3 Component of Target and Difference ERPs

Distribution. Target tone ERPs were characterized by a positive component (P3) between 275 and 550 ms poststimulus. At all ages, the P3 was maximal at parietal sites. P3 amplitudes were relatively smaller over centroparietal sites and larger over frontal sites with advancing age (Age \times Electrode interaction for targets: $F[22, 748] = 4.91$, $p < .0002$; ECF = 0.2579; for difference ERPs: $F[22, 748] = 3.34$, $p < .004$; ECF = 0.2685). Thus, the P3 had a more equipotential distribution in the elderly than in the young (Figures 1, 2, 4).

Amplitude. Linear regression analyses of P3 amplitudes at midline sites revealed small but significant correlations with age with a slope of -0.08 to $-0.1 \mu\text{V}/\text{year}$ (Table 1).

Table 3. Peak Amplitudes^a (μV) of ERP Components in Different Age Groups

Stimulus	Component (electrode site) ^b	Age group (years)		
		18–39	40–59	60–82
Standard	N1 (Cz)	–6.05 (2.04)	–6.67 (2.99)	–5.94 (2.68)
	P2 (Cz)	4.37 (2.79)	4.37 (1.93)	5.07 (1.87)
Target	N1 (Cz)	–6.64 (2.03)	–8.08 (3.67)	–6.56 (2.64)
	P2 (Cz)	2.28 (3.05)	2.22 (2.70)	3.70 (4.90)
	N2 (Cz)	–2.26 (4.11)	–3.98 (4.17)	–5.46 (5.97)
	P3 (Pz)	11.87 (5.51)	10.19 (3.45)	7.54 (5.59)
	SW (F3/4) ^c	–5.24 (3.22)	–3.51 (2.30)	–3.25 (2.71)
	(Cz)	–1.91 (4.75)	–3.57 (3.40)	–5.15 (3.49)
	(Pz)	3.73 (5.28)	1.69 (3.73)	–0.11 (3.26)
Difference waves	N2 (Cz)	–5.70 (4.16)	–7.17 (4.39)	–9.60 (6.79)
	P3 (Pz)	11.24 (4.82)	9.78 (3.36)	7.48 (5.91)
	SW (F3/4)	–5.12 (3.47)	–4.16 (2.61)	–3.17 (3.14)
	(Cz)	–2.04 (4.92)	–4.85 (3.91)	–5.13 (3.45)
	(Pz)	3.60 (4.78)	0.84 (4.12)	0.09 (3.41)

^aMean (SD). ^bN1, P2, N2, and P3 were measured at the electrode of maximal amplitude.

^cSlow wave (SW) values are the averages of measurements at F3 and F4 (F3/4), and measures at Cz and Pz.

ANOVAs of both target and difference P3 amplitudes, including age group and gender as factors, revealed no significant main effects or interactions with gender. Nor were there significant correlations between mean P3 amplitudes at midline central and parietal sites and mean RTs.

Latency. A scatter plot of age versus P3 latency in the vertex recording is shown in Figures 3 and 5A. The correlation for the target ERP was 0.36, and the regression line had a slope of 0.88 ms/year over the entire adult age range; in difference waves, the correlation was 0.49 and the slope was 1.19 ms/year. However, closer scrutiny of Figure 5A suggests that there might be a discontinuity in the slope between 35 and 45 years of age. Following the work of Brown et al. (1983), we divided subjects into two groups—those below and those above the age of 45—and calculated the regression line for P3 latency on age for each

age group separately. The results (Figure 5B) indicate that the regression lines for these two age groups are different. Subjects above the age of 45 years showed a P3 latency increase with age of 0.91 ms/year in the target waves and 1.09 ms/year in difference waves. In contrast, P3 latency in subjects below 45 years actually decreased as a function of increasing age at a rate of 0.65 ms/year in the target ERP and 0.17 ms/year in difference waves, but neither of these slopes was significant.

We also examined the possibility of a curvilinear relationship between P3 latency and age by fitting orthogonal polynomial regression equations (including degrees 1–3) to the data in a stepwise manner. Only the first degree (linear) was significant; adding a curvilinear (quadratic) factor to the linear regression did not increase significantly the percent of variance accounted for (see Figure 5C).

There were no significant differences between subject corre-

Table 4. Latencies^a (ms) of ERP Components in Different Age Groups

Stimulus	Component ^b	Age group (years)		
		18–39	40–59	60–82
Standard	N1	88 (7)	92 (6)	94 (10)
	P2	176 (22)	192 (30)	200 (16)
Target	N1	94 (13)	95 (8)	97 (14)
	N2	168 (22)	168 (14)	170 (21)
	N2	211 (21)	220 (20)	231 (39)
	P3	317 (35)	342 (44)	350 (55)
	SW	490 (46)	548 (58)	553 (46)
Difference waves	N2	210 (19)	218 (16)	223 (35)
	P3	316 (34)	348 (32)	364 (54)
	SW	507 (38)	564 (56)	568 (57)

^aMean (SD). ^bSlow wave (SW) measurements are based on the average of F3 and F4 values. All other values reflect latencies measured at the vertex (Cz).

lations of mean P3 latencies and mean RTs for responses with either hand.

SW Component of Target and Difference ERPs

Distribution. Following the P3 component, target tone ERPs were characterized by a slow potential (between 400 and 700 ms), which was negative-going over the front of the head and positive-going over the back of the head (i.e., the slow wave).

Amplitude. The SW shifted its maximal negativity from frontal to central sites with increasing age and showed a reduction in positivity over posterior sites (Age \times Electrode interaction for target ERPs: $F[22, 748] = 4.71$, $p < .0001$; ECF = 0.4396; for difference ERPs: $F[22, 748] = 5.07$, $p < .0001$; ECF = 0.3951) (Figures 1, 2, 4). Linear regression analyses of these data revealed a significant age-related reduction in the frontal negativity of about 0.05–0.06 $\mu\text{V}/\text{year}$, whereas at central and parietal sites the negativity increased at about 0.07–0.09 $\mu\text{V}/\text{year}$ (see Table 1). We found no significant correlations between mean SW amplitudes at the frontal, central, or parietal sites and mean RTs with either hand.

Latency. A scatter plot of the relationship between age and SW latency at the frontal sites is shown in Figure 3. The peak latency of the SW in the difference ERPs at frontal sites increased with age from 507 ms for the youngest age group to 568 ms for the oldest group, and in the target ERPs this increase was from 490 ms to 553 ms (Table 4). This age-related increase was highly significant (Table 2), and there was no significant curvilinear trend.

Linear regression of mean RTs on mean SW across subjects revealed significant correlations (all $p < .05$) for both dominant (targets: $r = .41$; difference waves: $r = .32$) and nondominant (targets: $r = .32$; difference waves: $r = .34$) hands.

Discussion

The present results accord well with those in previous reports describing age-related increases in the latencies of the late endogenous components (e.g., N2, P3, SW) of the ERP elicited by target tones in an oddball task, with little or no change on the early exogenous components (e.g., N1 and P2). The age-related latency increase was steeper for components of longer latency, ranging from an increase of 0.5 ms/year for N2 to 1.37 ms/year for SW. For all component latencies in both the target and difference ERP waveforms, the effect of increasing age was better described by a linear than a curvilinear function.

Although the percentage of age-related variance accounted for by the linear regression for P3 latency was not significantly increased by adding a curvilinear factor, the curve did appear to steepen with advancing age. Separate regression analyses of age on P3 latency for each of two groups, one comprising individuals below 45 years of age and the other individuals above 45, revealed different patterns of effects. For the below-45 group, not only was the P3 latency-age correlation small and nonsignificant, but the slope of the regression line was negative. In contrast, the above-45 group showed a larger, significant correlation with a positive slope of 1.09 ms/year. Thus, even though the quadratic trend was not significant, there does seem to be some nonlinearity in the P3 latency-age function (see also Kraiuhin et al., 1986; Picton et al., 1984).

The slopes of the functions relating age and P3 latency in our subjects were slightly shallower than those that have been reported previously (see Table 5). Several factors may have contributed to this difference, and these may have important practical implications for the use of P3 latency data in clinical assessment (e.g., to distinguish demented from depressed and demented from normal individuals). Polich (1989) critically reviewed the literature on P3 and aging and suggested that variability in latency-age slopes across studies may be a consequence of differences in the tone intensities that were used; loud tones were presumed to be associated with shallow slopes and soft tones with steep slopes. However, the present results cannot be explained solely on the basis of stimulus intensity, because the intensities used were comparable to, or even lower than, those used by several other groups who reported steeper slopes (e.g., Brown et al., 1983; Kraiuhin et al., 1986; Picton et al., 1984).

The P3 latency-age slope has been reported as differing as a function of recording site (Patterson et al., 1988; Sydulko et al., 1982). In our data, the P3 latency-age slope was steeper at central than at parietal midline sites. In other reports, the slope at parietal sites was similar to or slightly shallower than that at central sites; the shallowest slopes were for frontal measures (Patterson et al., 1988; Sydulko et al., 1982). Another possible source of variability is whether P3 latency is measured in the target ERP waveform or the target minus standard difference wave. Although there have been no prior direct comparisons of measures in target and difference waves, the steepest slopes reported (Goodin et al., 1978) were based on difference waves measurements (see Table 5). In the present data set, the slope of the linear function relating P3 latency to age was steeper for measures derived from difference waves than for those from target ERPs, the correlation coefficient was higher, and the standard error lower. These differences between the target ERP and difference wave measures were not significant, however; therefore, either could be used for clinical applications.

The effects of age on the amplitudes and scalp distributions of N2, P3, and SW in our subjects were somewhat different from those previously reported. N2 increased significantly with age at central and parietal sites and (in target ERP measurements) decreased at frontal electrodes, whereas others have failed to observe N2 amplitude changes (Barrett et al., 1987; Brown et al., 1983; Picton et al., 1984; Smith, Michalewski, Brent, & Thompson, 1980). Also contrary to previous studies (Barrett et al., 1987; Picton et al., 1984), we found a gradual decrease of P3 amplitude with age at central and parietal sites along with a trend for an increase at frontal sites even though the subjects made manual responses to the target stimuli. Picton et al. (1984) observed that the vertex P3 recorded in a subvocal counting paradigm decreased in amplitude with age (a finding also reported by Brown et al. [1983], Goodin et al. [1978], Puce et al. [1989], Smith et al. [1980], and Sydulko et al. [1982]), whereas the P3 recorded in a RT paradigm did not. They suggested that negative motor potentials may have coincided with the P3 in the RT paradigm, thereby obscuring the age-related changes in the amplitude of P3. Although lateralized negative motor potentials could have accounted for the lower P3 and higher N2 and SW amplitudes observed in the hemisphere contralateral to the responding hand in the present study, age had no effect on these asymmetrical potentials. Thus, these effects probably were not responsible for concealing age-related P3 amplitude changes. The slopes of the age-related P3

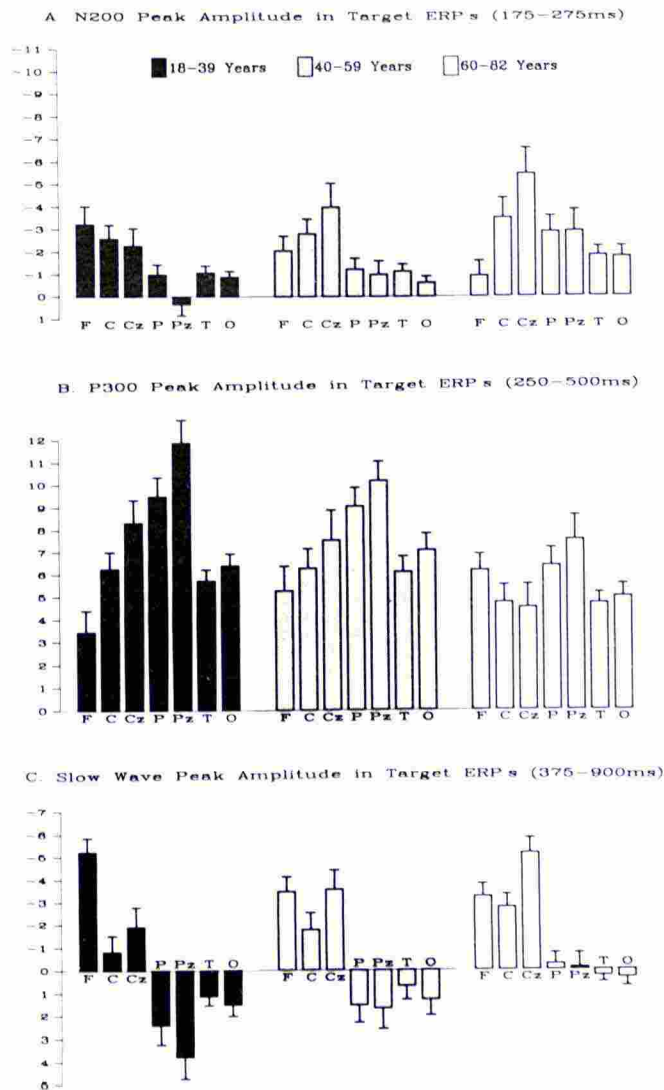


Figure 4. Bar graph of the peak amplitudes of the N2, P3, and SW components of the target ERP at each of the recording sites for the young (18-39 years), middle-aged (40-59 years), and elderly (60-82 years) subject groups. Measures at midline sites were taken at Cz and Pz. The other measures reflect the averages of amplitudes at the left and right frontal (F), central (C), parietal (P), temporal (T), and occipital (O) electrodes. Bars indicate mean amplitudes and lines indicate the standard deviations. The ordinate scale units are microvolts.

amplitude changes reported by Picton et al. (1984) were quite similar in the count ($-0.18 \mu\text{V}/\text{year}$) and RT ($-0.20 \mu\text{V}/\text{year}$) paradigms, but the standard error of the mean was much smaller in the count (0.04) than in the RT (7.56) paradigm; accordingly, the effect was not significant in the RT task. This problem of variability may have been due in part to the small number of subjects studied (24, in contrast to 72 studied in the count paradigm). In another study with a relatively small number of subjects ($n = 27$), Barrett et al. (1987) did not observe age-related P3 amplitude changes in either a count or an RT paradigm.

SW amplitude was significantly correlated with age at frontal, central, and parietal locations, replicating the observations of Pfefferbaum et al. (1984) and Smith et al. (1980). In contrast, Picton et al. (1984) found no significant age-related

changes in the amplitude of the centroparietal and only a small decrease in the frontal SW. In addition, they reported a shallower slope for the age-related latency increase in the SW than for the P3, whereas we found a steeper slope for the SW. However, Picton et al. (1984) used a more restrictive low-frequency cutoff (time constant of 1 s) than in the present study (8 s), and this may have attenuated age-related amplitude changes. The relatively short time constant also may have resulted in a phase shift of the waveform that could account for their finding of a reduced slope for the age-related latency increase in SW relative to that of P3, contrary to the present results.

The latencies of both N1 and P2 to standard stimuli increased with age; the rate of increase was higher for P2 than N1. These findings agree with those of Goodin et al. (1978), who used the same stimulus intensity in all subjects (as we did), but are at odds with reports of Pfefferbaum, Ford, Roth, Hopkins, and Kopell (1979), Pfefferbaum, Ford, Roth, and Kopell (1980b), and Pfefferbaum et al. (1984), who presented stimuli at an intensity relative to each individual's threshold. This procedural difference may account for the observed discrepancies. When stimuli of the same intensity are delivered to all subjects, an age-related hearing loss (prebycusis) could result in a decrease of effective stimulus intensity in the older subjects and, therefore, in a latency increase of the exogenous N1 and P2 potentials. Conversely, stimuli presented at an intensity relative to each individual's threshold may be associated with the presence of recruitment in elderly subjects, which could result in an increase of the effective stimulus intensity, thus confounding age-related latency changes.

The amplitudes of N1 and P2 were not affected by age. The differences in the amplitude of N1 elicited by standard and target tones observed in all age groups is probably related to the less refractory response to the rare high-pitched tones than to the frequent low-pitched tones. A higher noise level in the averaged ERPs of target tones (which were derived from fewer trials than were standard ERPs) may have played a role as well. However, the present comparisons across ages were not subject to such differences in refractoriness or in the number of trials averaged because the experimental design was identical for all subjects.

Our findings that the latencies of the late endogenous components increase with age and the early exogenous components change proportionately less, if at all, indicate that the cognitive processes are more affected by aging than are the sensory perceptual systems. Clearly, the large delays in the latencies of the N2, P3, and SW components cannot be accounted for solely by the modest latency increases in the N1 and P2 components. The latencies of the N2, P3, and SW show progressively greater delays as a function of age, which suggests that the mental operations they reflect are also differently affected by aging. Although the simplicity of the basic auditory oddball task used here makes it difficult to link the N2, P3, and SW with specific cognitive processes, the literature offers some clues. The consensus is that N2 latency reflects duration of stimulus evaluation comparison processes, and thus a delay in its latency with age indicates a decline in the speed of this mental operation. P3 latency, however, although clearly dependent upon stimulus evaluation processes, is generally viewed as a reflection of a subsequent stage that has been characterized in terms of decision closure or the updating of memory in aid of future actions (see Hillyard & Picton, 1987, for review). Thus, an increase in P3 latency with age may be indicative of a delay in such context up-

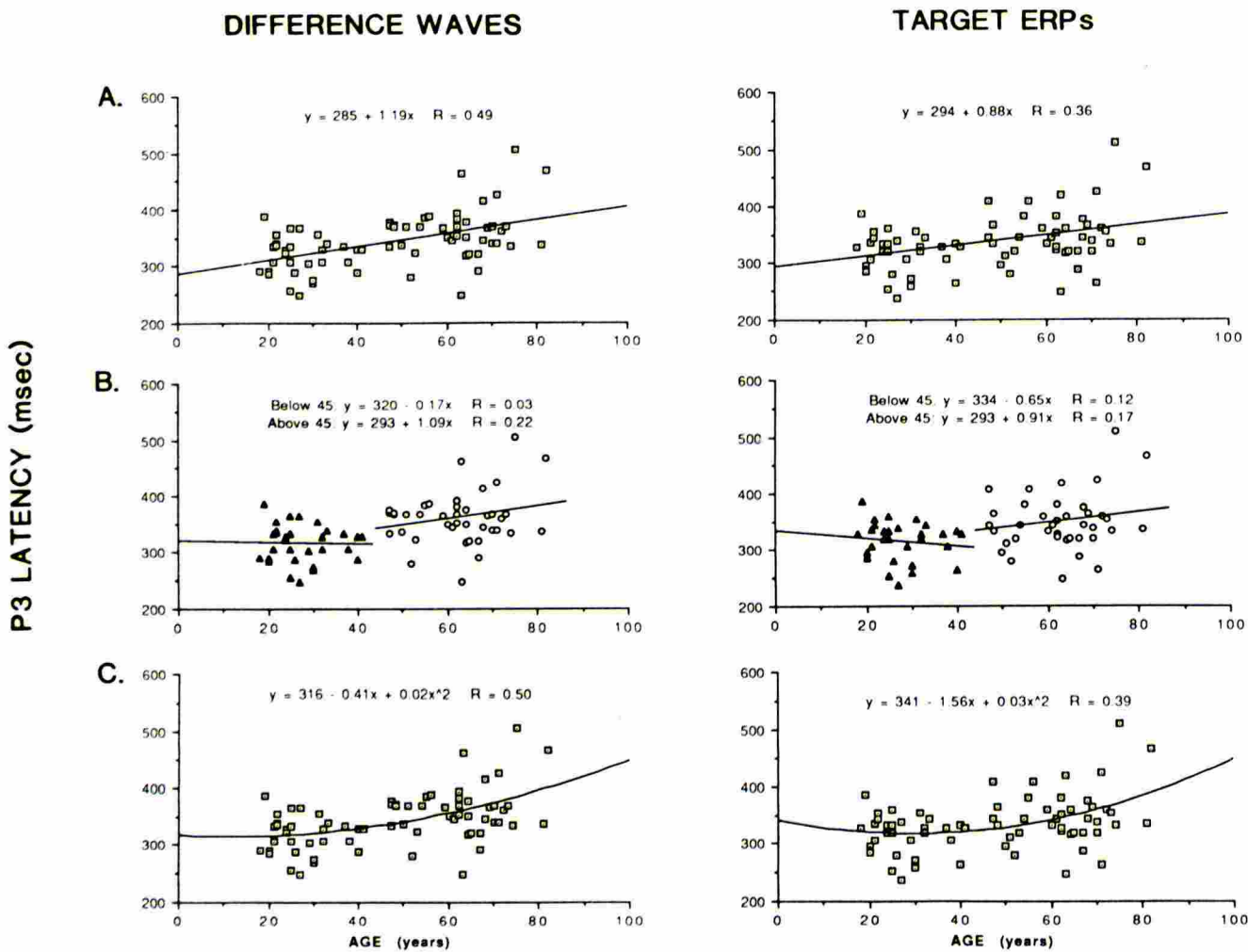


Figure 5. Scatter diagrams of target and difference ERPs depicting relationships of P3 latency measured at the vertex with age. A. Linear regression of P3 latency on age. B. Same data as in A with separate linear regressions of P3 latency on age for subjects below 45 years old and those above 45. C. Second degree polynomial regression of P3 latency on age for same data as in A.

Table 5. Slope of Linear Regressions of Age on ERP Latencies and Reaction Times (ms/year) for the Two-Tone Auditory Oddball Paradigm

Author	Subject age (years)	n	Task	Standard tones		Target tones					RT ^b
				N1	P2	N1	P2	N2	P3	SW ^a	
Goodin et al. (1978)	15-76	40	count	0.1	0.7			0.8 ^c	1.80 ^c		
Syndulko et al. (1982)	18-85	45	count			n.s. ^d	0.36		1.07		
Brown et al. (1983)	15-80	49	count			n.s.	n.s.		1.12		
Picton et al. (1984)	20-79	72	count			n.s.	0.25	0.65	1.36	0.89	
		24	button press						1.69		n.s.
Polich et al. (1985)	20-86	78	count						1.39		
Kraihuhin et al. (1986)	15-89	55	count						0.91		
Barrett et al. (1987)	20-78	27	count	n.s.	n.s.	n.s.	n.s.	n.s.	1.05		
		27	button press	n.s.	n.s.	n.s.	n.s.	0.83	1.21		
Patterson et al. (1988)	28-81	27	button press						1.0		
Puce et al. (1989)	16-52	24	count						1.34		
Present study	18-82	71	button press	0.13	0.55	n.s.	n.s.	0.48	0.88	1.37	n.s.
								0.47 ^c	1.19 ^c	1.37 ^c	

^aSW = slow wave. ^bRT = reaction time. ^cMeasurements done on difference waves; other measures were taken in target waves. ^dn.s. = not significant.

dating, which may underlie the increasing frailty of recognition and recall memory functions with age (Johnson, Pfefferbaum, & Kopell, 1985). The SW, however, has been related to the amount of processing required for a perceptual decision, and its amplitude seems to covary with the extent that such processing is warranted, either by the experimental demands or by the subject's lack of confidence in his sensory decision (Ruchkin, Munson, & Sutton, 1982; Ruchkin & Sutton, 1983).

Although there was a tendency for RTs to be longer in the elderly, we found no significant increase in RTs with advancing age in this simple auditory oddball task, in line with the report of Picton et al. (1984), who used a similar task, and with studies of Pfefferbaum et al. (1980b, 1984) and Ford and Pfefferbaum (1985), who employed a three-tone paradigm. The modest differences that were observed are consistent with observations that RT slowing with age is less pronounced in simple than in demanding tasks (Ford, Pfefferbaum, & Kopell, 1982b; Ford, Roth, Mohs, Hopkins, & Kopell, 1979; Marsh, 1975; Pfefferbaum, Ford, Roth, & Kopell, 1980a; Picton, Cerri, Champagne, Stuss, & Nelson, 1986; Pratt, Michalewski,

Patterson, & Starr, 1989; Strayer, Wickens, & Braune, 1987; also see Cerella, 1985, for review). The lack of a relationship between P3 latency and RT supports the view that these two measures reflect at least partially different sets of cognitive processes, where RT is sensitive to response selection and organization factors. The degree of dissociation between P3 latency and RT may then be attributed to postrecognition factors such as the complexity of response selection rules, response strategies, and speed-accuracy tradeoffs (Ford et al., 1979). The lack of significant age effect on RT and the increased error rate in our elderly subjects are consistent with findings of Ford and Pfefferbaum (1985), who also used a simple auditory discrimination task, and suggest that elderly subjects may adopt a strategy that favors speed over accuracy in simple tasks and thus minimize RT delays at the expense of an increased error rate. Future studies are needed to examine the effects of age on P3 latency and RT across tasks of increasing complexity. Such data will be useful for fractionating the contributions of peripheral and central mechanisms to the slowed processing accompanying aging.

REFERENCES

- Adam, N., & Collins, G. I. (1978). Late components of the visual evoked potential to search in short-term memory. *Electroencephalography and Clinical Neurophysiology*, 44, 147-156.
- Barrett, G., Neshige, R., & Shibasaki, H. (1987). Human auditory and somatosensory event-related potentials: Effects of response condition and age. *Electroencephalography and Clinical Neurophysiology*, 66, 409-419.
- Bashore, T. R., Jr. (1990). Age-related changes in mental processing revealed by analyses of event-related brain potentials. In J. W. Rohrbaugh, R. Parasuraman, & R. Johnson, Jr. (Eds.), *Event-related potentials of the brain* (pp. 242-275). New York: Oxford University Press.
- Bashore, T. R., Jr., Osman, A., & Heffley, E. F., III. (1989). Mental slowing in elderly persons: A cognitive psychophysiological analysis. *Psychology and Aging*, 4, 235-244.
- Beck, E. C., Swanson, C., & Dustman, R. E. (1980). Long latency components of the visually evoked potential in man. *Experimental Aging Research*, 6, 523-545.
- Birren, J. E., & Schaie, K. W. (Eds.). (1985). *Handbook of the psychology of aging*. New York: Van Nostrand Reinhold.
- Brown, W. S., Marsh, J. T., & LaRue, A. (1983). Exponential electrophysiology of aging: P3 latency. *Electroencephalography and Clinical Neurophysiology*, 55, 277-285.
- Cerella, J. (1985). Information processing rates in the elderly. *Psychological Bulletin*, 98, 67-83.
- Donchin, E., Karis, D., Bashore, T. R., Coles, M. D. H., & Gratton, G. (1986). Cognitive psychophysiology and human information processing. In M. G. H. Coles, E. Donchin, & S. W. Porges (Eds.), *Psychophysiology: Systems, processes, and applications* (pp. 244-267). New York: Guilford Press.
- Donchin, E., Ritter, W., & McCallum, W. C. (1978). Cognitive psychophysiology: The endogenous components of the ERP. In E. Callaway, P. Tueting, & S. H. Koslow (Eds.), *Event-related brain potentials in man* (pp. 349-441). New York: Academic Press.
- Fein, G., & Turetsky, B. (1989). P300 variability in normal elderly: Effects of paradigm and measurement technique. *Electroencephalography and Clinical Neurophysiology*, 72, 384-394.
- Fitzgerald, P. G., & Picton, T. W. (1983). Event-related potentials recorded during the discrimination of improbable stimuli. *Biological Psychology*, 17, 241-276.
- Folstein, M. L., Folstein, C. E., & McHuch, P. R. (1975). Mini-mental state. A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, 12, 189-198.
- Ford, J. M., Duncan-Johnson, C. C., Pfefferbaum, A., & Kopell, B. S. (1982a). Expectancy for events in old age: Stimulus sequence effects on P300 and reaction time. *Journal of Gerontology*, 37, 696-704.
- Ford, J. M., & Pfefferbaum, A. (1985). Age-related changes in event-related potentials. In P. Ackles, R. Jennings, & M. G. H. Coles (Eds.), *Advances in psychophysiology* (Vol. 1, pp. 301-339). Greenwich, CT: JAI Press.
- Ford, J. M., Pfefferbaum, A., & Kopell, B. S. (1982b). Effects of perceptual and cognitive difficulty on P3 and RT in young and old adults. *Electroencephalography and Clinical Neurophysiology*, 54, 311-321.
- Ford, J. M., Roth, W. T., Mohs, R. C., Hopkins, W. F., & Kopell, B. S. (1979). Event-related potentials recorded from young and old adults during a memory retrieval task. *Electroencephalography and Clinical Neurophysiology*, 47, 450-459.
- Goodin, D. S., Squires, K. C., Henderson, B. H., & Starr, A. (1978). Age-related variations in evoked potentials to auditory stimuli in normal human subjects. *Electroencephalography and Clinical Neurophysiology*, 44, 447-458.
- Hillyard, S. A., & Picton, T. W. (1987). Electrophysiology of cognition. In F. Plum (Ed.), *Handbook of physiology, Section 1, The nervous system* (Vol. V, pp. 519-584). Higher Functions of the Brain, Part II. Washington, DC: American Physiological Society.
- Jasper, H. H. (1958). The ten-twenty electrode system of the International Federation. *Electroencephalography and Clinical Neurophysiology*, 10, 371-375.
- Johnson, R., Jr. (1989a). Auditory and visual P300s in temporal lobectomy patients: Evidence for modality-dependent generators. *Psychophysiology*, 26, 633-650.
- Johnson, R., Jr. (1989b). Developmental evidence for modality-dependent P300 generators: A normative study. *Psychophysiology*, 26, 651-667.
- Johnson, R., Jr., Pfefferbaum, A., & Kopell, B. S. (1985). P300 and long-term memory: Latency predicts recognition performance. *Psychophysiology*, 22, 497-507.
- Keselman, H. J., & Rogan, J. C. (1980). Repeated measures *F* tests and psychophysiological research: Controlling the number of false positives. *Psychophysiology*, 17, 499-503.
- Kraihuin, C., Gordon, E., Stanfield, P., Meares, R., & Howson, A. (1986). P300 and the effects of aging: Relevance to the diagnosis of dementia. *Experimental Aging Research*, 12, 187-192.
- Kutas, M., McCarthy, G., & Donchin, E. (1977). Augmenting mental chronometry: The P300 as a measure of stimulus evaluation time. *Science*, 197, 792-795.
- Magliero, A., Bashore, T. T., Coles, M. G. H., & Donchin, E. (1984). On the dependence of P300 latency on stimulus evaluation processes. *Psychophysiology*, 21, 171-186.
- Marsh, G. R. (1975). Age differences in evoked potential correlates of a memory scanning process. *Experimental Aging Research*, 1, 3-16.
- McCallum, W. C. (1988). Potentials related to expectancy, preparation

- and motor activity. In T. W. Picton (Ed.), *Human event-related potentials. Handbook of electroencephalography and clinical neurophysiology (revised series)* (Vol. 3, pp. 427-541). Amsterdam: Elsevier.
- McCallum, W. C., Curry, S. H., Cooper, R., Pocock, P. V., & Papakostopoulos, D. (1983). Brain event-related potentials as indicators of early selective processes in auditory target localization. *Psychophysiology*, 20, 1-17.
- McCarthy, G., & Donchin, E. (1981). A metric for thought: A comparison of P300 latency and reaction time. *Science*, 211, 77-80.
- McCarthy, G., & Wood, C. C. (1985). Scalp distribution of event-related potentials: An ambiguity associated with analysis of variance models. *Electroencephalography and Clinical Neurophysiology*, 62, 203-208.
- Mullis, R. J., Holcomb, P. J., Diner, B. C., & Dykman, R. A. (1985). The effects of aging on the P3 component of the visual event-related potential. *Electroencephalography and Clinical Neurophysiology*, 62, 141-149.
- Patterson, J. V., Michalewski, H. J., & Starr, A. (1988). Latency variability of the components of auditory event-related potentials to infrequent stimuli in aging, Alzheimer-type dementia, and depression. *Electroencephalography and Clinical Neurophysiology*, 71, 450-460.
- Pfefferbaum, A., Ford, J., Johnson, R., Jr., Wenegrat, B., & Kopell, B. S. (1983). Manipulation of P3 latency: Speed vs. accuracy instructions. *Electroencephalography and Clinical Neurophysiology*, 55, 188-197.
- Pfefferbaum, A., Ford, J. M., Roth, W. T., Hopkins, W. F., & Kopell, B. S. (1979). Event-related potential changes in healthy aged females. *Electroencephalography and Clinical Neurophysiology*, 46, 81-86.
- Pfefferbaum, A., Ford, J. M., Roth, W. T., & Kopell, B. S. (1980a). Age differences in P3-reaction time associations. *Electroencephalography and Clinical Neurophysiology*, 49, 257-265.
- Pfefferbaum, A., Ford, J. M., Roth, W. T., & Kopell, B. S. (1980b). Age-related changes in auditory event-related potentials. *Electroencephalography and Clinical Neurophysiology*, 49, 266-276.
- Pfefferbaum, A., Ford, J. M., Weller, B. J., & Kopell, B. S. (1985). ERPs to response production and inhibition. *Electroencephalography and Clinical Neurophysiology*, 60, 423-434.
- Pfefferbaum, A., Ford, J. M., Wenegrat, B. G., Roth, W. T., & Kopell, B. S. (1984). Clinical application of the P3 component of event-related potentials. I. Normal aging. *Electroencephalography and Clinical Neurophysiology*, 59, 85-103.
- Picton, T. W. (Ed.). (1988). Human event-related potentials. In *Handbook of electroencephalography and clinical neurophysiology (revised series)* (Vol. 3, p. 541). Amsterdam: Elsevier.
- Picton, T. W., Cerri, A. M., Champagne, S. C., Stuss, D. T., & Nelson, R. F. (1986). The effects of age and task difficulty on the late positive component of the auditory evoked potential. In W. C. McCallum, R. Zappoli, & F. Denoth (Eds.), *Cerebral psychophysiology: Studies in event-related potentials. Electroencephalography and Clinical Neurophysiology* (Suppl. 38, pp. 130-131). Amsterdam: Elsevier.
- Picton, T. W., Stuss, D. T., Champagne, S. C., & Nelson, R. F. (1984). The effects of age on human event-related potentials. *Psychophysiology*, 21, 312-325.
- Polich, J. (1989, June). P300 in the evaluation of aging and dementia. Paper presented at the Aging and Dementia Symposium, Evoked Potential International Conference (EPIC) IX, Noordwijk, The Netherlands.
- Polich, J., Howard, L., & Starr, A. (1985). Effects of age on the P300 component of the event-related potential from auditory stimuli: Peak definition, variation, and measurement. *Journal of Gerontology*, 40, 721-726.
- Pratt, H., Michalewski, H. J., Patterson, J. V., & Starr, A. (1989). Brain potentials in a memory scanning task. II. Effects of aging on potentials to the probes. *Electroencephalography and Clinical Neurophysiology*, 72, 507-517.
- Puce, A., Donnan, G. A., & Bladin, P. F. (1989). Comparative effects of age on limbic and scalp P3. *Electroencephalography and Clinical Neurophysiology*, 74, 385-393.
- Ragot, R. (1984). Perceptual and motor space representation: An event-related potential study. *Psychophysiology*, 21, 159-170.
- Ritter, W., Simson, R., & Vaughan, H. G., Jr. (1972). Association cortex potentials and reaction time in auditory discrimination. *Electroencephalography and Clinical Neurophysiology*, 33, 547-555.
- Ritter, W., Simson, R., Vaughan, H. G., Jr., & Friedman, D. (1979). A brain event related to the making of a sensory discrimination. *Science*, 203, 1358-1361.
- Rohrbaugh, J., Parasuraman, R., & Johnson, R., Jr. (Eds.). (1990). *Event-related brain potentials*. New York: Oxford University Press.
- Roth, W. T., Ford, J. M., & Kopell, B. S. (1978). Long-latency evoked potentials and reaction time. *Psychophysiology*, 15, 17-23.
- Ruchkin, D. S., Munson, R., & Sutton, S. (1982). P300 and slow wave is a message consisting of two events. *Psychophysiology*, 19, 629-642.
- Ruchkin, D. S., & Sutton, S. (1983). Positive slow wave and P300: Association and disassociation. In A. W. K. Gaillard & W. Ritter (Eds.), *Tutorials in ERP research: Endogenous components* (pp. 233-250). Amsterdam: North-Holland.
- Salthouse, T. A. (1985). *A theory of cognitive aging*. Amsterdam: North-Holland.
- Simson, R., Vaughan, H. G., Jr., & Ritter, W. (1977). The scalp topography of potentials in auditory and visual discrimination tasks. *Electroencephalography and Clinical Neurophysiology*, 42, 528-535.
- Smith, D. B. D., Michalewski, H. J., Brent, G. A., & Thompson, L. W. (1980). Auditory averaged evoked potentials and aging: Factors of stimulus, task and topography. *Biological Psychology*, 11, 135-151.
- Snyder, E., Hillyard, S. A., & Galambos, R. (1980). Similarities and differences among the P3 waves to detected signals in three modalities. *Psychophysiology*, 17, 112-122.
- Squires, K. C., Chippendale, T. J., Wrege, K. S., Goodin, D. S., & Starr, A. (1980). Electrophysiological assessment of mental function in aging and dementia. In L. W. Poon (Ed.), *Aging in the 1980s: Selected contemporary issues in the psychology of aging* (pp. 125-134). American Psychological Association, Washington, DC.
- Strayer, D. L., Wickens, C. D., & Braune, R. (1987). Adult age differences in the speed and capacity of information processing: 2. An electrophysiological approach. *Psychology of Aging*, 2, 99-110.
- Syndulko, K., Hansch, E. C., Cohen, S. C., Pearce, J. W., Goldberg, Z., Montan, B., Tourtellotte, W. W., & Potvin, A. R. (1982). Long-latency event-related potentials in normal aging and dementia. In J. Courjon, F. Mauguire, & M. Revol (Eds.), *Clinical applications of evoked potentials in neurology* (pp. 279-285). New York: Raven Press.

(RECEIVED January 1, 1991; ACCEPTED October 7, 1991)

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.