Title

Effects of age on the P300 component of the event-related potential from auditory stimuli: peak definition, variation, and measurement.

Journal

Journal of gerontology, 40(6)

ISSN

0022-1422

Authors

Polich, J
Howard, L
Starr, A

Publication Date

1985-11-01

DOI

10.1093/geronj/40.6.721

Journal of Gerontology
1985, Vol. 40, No. 6, 721-726

Effects of Age on the P300 Component of the Event-Related Potential from Auditory Stimuli: Peak Definition, Variation, and Measurement[1]

John Polich,[2] Lawrence Howard,[3] and Arnold Starr[4]

An auditory "oddball" paradigm was used to elicit the P300 component of the event-related brain potential (ERP) from a large sample of young (5 to 15 years) and older (20 to 86 years) persons. Distinct P3a and P3b subcomponents of the P300 were observed within individuals and across trial blocks. Age affected P300 latency in a similar fashion for both subcomponents with latency increasing about 65 msec between 20 and 70 years. P300 latency variability also was found to increase somewhat with advanced age. These results confirmed previous age-related ERP changes and extended them to the P3a and P3b subcomponents.

Key Words: Event-related brain potential, P300, P3a, P3b, Life span, Children, Aging, Variability

THE P300 component of the auditory event-related brain potential (ERP) is a large (5 to 15 μV), positive-going waveform that occurs with a modal latency of about 300 ms in normal young adults. Although the neurophysiology underlying the P300 is still being explored (Halgren et al., 1980; Okada et al., 1983), the cognitive events associated with its generation have received considerable attention (Donchin, 1981; Pritchard, 1981). Because the P300 component is thought to reflect the cognitive events of stimulus classification, its latency has been used to evaluate mental function in normal aging (Brown et al., 1983; Ford et al., 1982; Goodin et al., 1978; Pfefferbaum et al., 1984a; Picton et al., 1984; Smith et al., 1980; Syndulko et al., 1982) and in patients with neurological and psychiatric disorders (Brown et al., 1982; Goodin et al., 1978; Hansch et al., 1982; Pfefferbaum et al., 1984b; Squires et al., 1980). Thus, P300 latency is becoming an important and widespread clinical tool for the assessment of human cognition because suspected impairment can be compared with normative latency values to quantify objectively the amount of dysfunction.

A typical procedure used in these investigations involves the presentation of two different signals, one less frequently than the other. The subject keeps a mental count or presses a button to the infrequent event to provide a behavioral index of stimulus discrimination. This "oddball" paradigm reliably produces the P300 component and often has been used to study factors that affect its amplitude (Duncan-Johnson & Donchin, 1977). The latency of the P300 has also been examined in a variety of complex task situations usually involving visual stimuli (McCarthy & Donchin, 1981; Polich et al., 1983b). Relatively few studies have been performed using a simple auditory stimulus paradigm in which variations in waveform morphology (e.g., the P3a and P3b subcomponents, Snyder & Hillyard, 1976; Squires et al., 1975) and normative latency data have been reported (Ford et al., 1982; Polich et al., 1983a). Because subject age is a major factor affecting the latency of many sensory evoked potentials (Polich & Starr, 1984), the effects of maturation and age on P300 latency have been investigated with the general finding of increasing latency as the adult ages (Brown et al., 1983; Goodin et al., 1978; Pfefferbaum et al.,

[1]The authors wish to thank Beverly Henderson for help in collecting portions of these data and Gary Carter of the Nicolet Instrument Company for providing the equipment. This research was supported by National Institutes of Health grant NS 11876. Address requests for reprints to John Polich, Division of Neurology, Scripps Clinic and Research Foundation, 10666 N. Torrey Pines Road, La Jolla, CA 92037.

[2]Department of Neurology, California College of Medicine, University of California, Irvine, Irvine, CA 92717.

[3]School of Social Sciences, University of California, Irvine, Irvine, CA 92717.

[4]Departments of Neurology and Psychobiology, University of California, Irvine, Irvine, CA 92717.

1984a). It is not clear, however, from the published reports how waveform morphology (viz., observation of multiple peaks for the P300) varied across subjects or how reliable a single measure of peak latency might be with respect to determining the normative aging effects on P300 latency. Given the possible psychological distinctions between the P3a as reflecting an initital orienting response and the P3b as indicating a stimulus-based categorization process (Donchin, 1981; Squires et al., 1975), these two subcomponents may be affected differentially by age. Thus the present study was undertaken to determine the relationship between age, P3a and P3b latency, and measurement reliability for a large normative sample of young and adult humans.

METHODS

Participants. — A total of 104 volunteers who ranged in age from 5 to 86 years were obtained from the university community. Approximately equal numbers of each sex within the age decade were assessed. The mean ages and number of each age group are presented in the top portion of Table 1 (persons over the age of 70 were grouped together). All participants were attending school, employed, or leading an active retirement at the time of testing.

Stimuli and procedure. — ERPs were elicited by presenting participants with a series of binaural tones at 60 dB SPL and with a 9.9 ms rise/fall and 50 ms plateau time. The tones were presented in a random sequence with a 2,000 Hz tone (target) occurring 20% of the time and the 1,000 Hz tone (standard) occurring 80% of the time at a rate of 1.1 per second. Participants were instructed to keep a silent count of the number of high tones. Stimuli were presented until a total of 200 artifact-free trials were recorded. Then a second block of 200 trials was obtained to insure replication of morphological structure and to facilitate component identification.

Recording conditions. — Electroencephalographic activity (EEG) was recorded from the vertex (Cz electrode site in the 10 to 20 system) and referred to linked mastoids with a forehead ground. The filter bandpass was 1 to 30 Hz (3 dB down, 12 dB/octave slope). Although not optimal, the relative latency of the P300 generally will be unaffected with this bandpass range, even though its amplitude will be reduced compared with longer time constants (cf. Duncan-Johnson & Donchin, 1979; Polich et al., 1983a). The EEG was digitized at 3 ms/point for 768 ms and averaged on line by a Nicolet CA-1000 that also controlled the stimulus presentation and automatic artifact rejection of the averaged channel. Trials on which the EEG exceeded ± 45 μV were rejected automatically and not included in the averaged waveform. Hence, only trials uncontaminated by eye blinks, eye movements, and muscle contractions that usually produce much larger voltage fluctuations (typically greater than 100 μV) were recorded. The separate averages for the rare and frequent stimulus tones

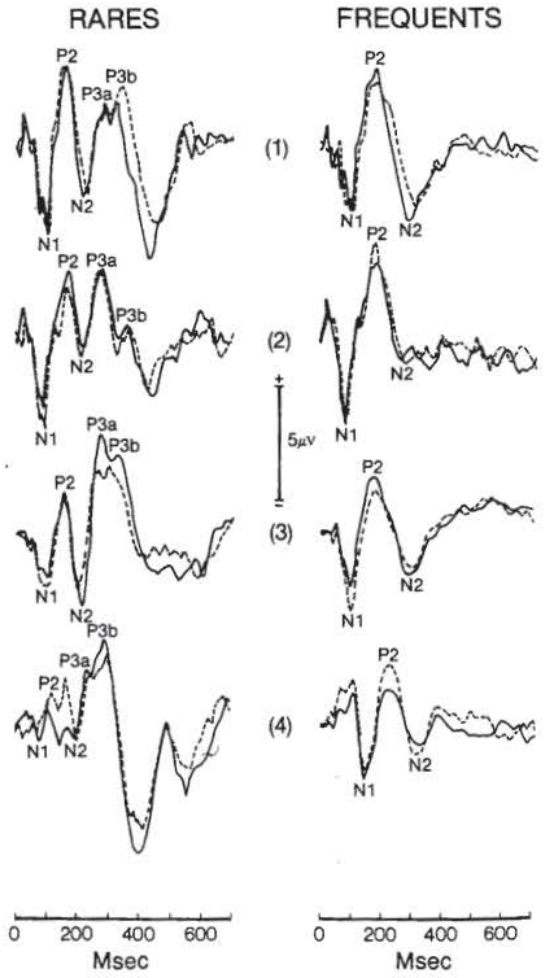

Figure 1. Waveforms obtained to the rare (20%) and frequent (80%) tones in an auditory oddball paradigm. Solid line represents waveforms collected from the first trial block; the dashed line represents waveforms collected from the second trial block (see text). Note the variability across participants in latency of the P300 components (No. 1: 61-year-old woman; No. 2: 72-year-old man; No. 3: 67-year-old man; No. 4: 13-year-old boy).

were plotted and the latencies of the components were defined at their peak or trough. Quantitative assessment of component amplitudes were not obtained, although the relative peak sizes were noted.

RESULTS

Count performance was perfect or missing the correct count by one or two for over 95% of the participants. Several of the younger children made a few more errors. Examples of representative waveforms from four participants obtained for the rare (target) and frequent (standard) stimuli are presented in Figure 1. These potentials agree in morphology and latency range with previously reported components employing an auditory count task. In most individuals the P300 component displayed two distinct subcomponents. Although only one electrode site could be recorded because of equipment limitations, the latency characteristics of these subcomponents displayed a pattern typical of previous reports. Thus, the earlier one was labelled P3a and the latter P3b, with the latency ranges 220 to 350 ms and 280 to 500 ms, respectively, used to define their peaks (Snyder & Hillyard, 1976; Squires et al., 1975). Because a complete scalp distribution could not be obtained and because of the wide variation in peak latencies of the P3a and P3b subcomponents across participants, the following procedures also were adopted: (a) only participants who had a clear bifurcated positive peak after the N200 component in both the first and second trial blocks were counted as producing a consistent P3a and P3b (see Figure 1); (b) the first peak was taken as the P3a and the second as the P3b if each peak latency fell within the critical ranges; (c) if only a single peak was observed on only one or both of the trials (i.e., each subcomponent was not obtained in both trial blocks), the largest positivity within the P3b latency window was labelled a P3b and no P3a was counted; (d) the mean latency for each subcomponent taken over both trial blocks then was computed as that person's P3a and P3b latency. This identification system promoted consistent and reliable measurement of the two subcomponents because individual latencies were systematically assessed across both trial blocks.

As is shown in Figure 1, participants produced clearly defined P300 components that replicated well from trial to trial. An identifiable P3a subcomponent was produced on both trial blocks by 82% of the participants. The mean latency from the two trial blocks for each subcomponent is plotted as a function of age in Figure 2. The mean latencies and standard deviations for the subcomponents are presented in Table 1. In addition to the subcomponent

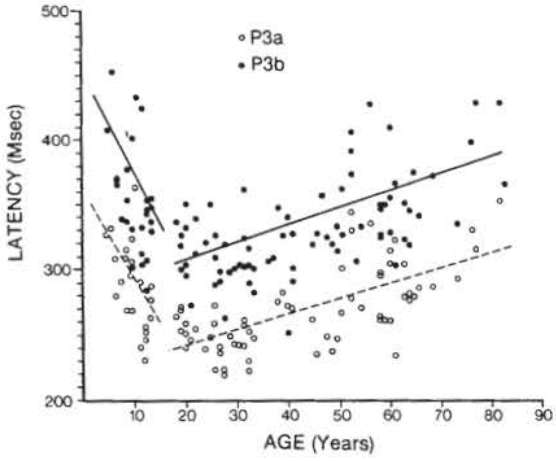

Figure 2. Mean latency of the P3a and P3b subcomponents obtained from two successive repetitions of an auditory oddball paradigm plotted as function of participant age. Dashed and solid lines represent regression lines computed for the P3a and P3b components, respectively.

Table 1. Mean Latency (ms), Standard Deviation (*SD*) and Number of Subjects (*n*) Demonstrating a P3a and P3b Subcomponent for each Age Group

Age groups	Mean age	P3a			P3b			P3MAX		
		n	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
0-9	7.4	9	311	20.8	9	385	36.2	9	355	25.7
10-19	12.2	17	285	35.4	19	356	44.1	19	322	29.1
20-29	23.8	18	253	19.9	19	319	24.7	19	287	37.9
30-39	34.2	10	248	12.0	13	315	19.7	13	302	37.1
40-49	43.3	7	275	17.3	10	327	31.2	10	315	36.3
50-59	54.0	10	292	39.7	12	362	40.8	12	312	40.8
60-69	64.0	16	289	23.9	16	355	26.4	16	329	48.9
70-90	81.0	6	325	26.0	6	402	36.6	6	387	54.9

Note. The sample sizes for P3b and P3MAX reflect the total number tested.

measurements, a P3MAX latency also was defined as a mean latency of the larger amplitude subcomponent obtained from each trial block for each person. This was derived from both later peaks in 52% of the participants (e.g., Figure 1, Subjects 1 and 4), from both early peaks in 35% of the participants (Subject 2), and from the mean of the early and later peak in 13% of the participants (Subject 3). The P3MAX latency measurement was developed in order to provide a means of comparison between the present and previous aging studies where the P3a and P3b subcomponents were not reported. The mean latency obtained from the P3MAX procedure is plotted as a function of age in Figure 3. The regression equations for each subcomponent and the P3MAX measurement were computed separately for participants within the ages 0 to 15 years and participants within the ages 16 to 90 years in order to compare the present results with previous data (Brown et al., 1983; Goodin et al., 1978). The resulting equations along with the correlation coefficients for each measurement are presented in Table 2. The regression lines for each measurement are plotted in the appropriate figure with the standard deviations for P3MAX latency also plotted at the mean age for the decade group.

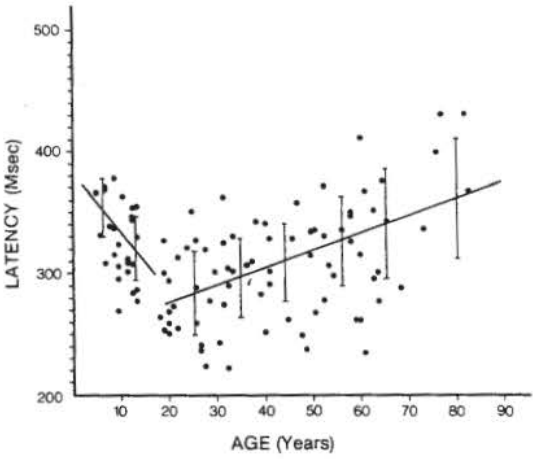

Figure 3. Mean latency for the P3MAX latency measure as a function of age. Each point represents the mean latency to the larger amplitude subcomponent of the P300 (P3a or P3b) taken from two successive blocks of trials. Regression lines are plotted separately for young and older participants with vertical lines plotted at the mean age of each group indicating the standard deviation.

The subcomponents of the P300 varied systematically with age in a similar, although not identical, fashion. The P3a demonstrated somewhat less variability and a smaller rate of latency increase over age than did the P3b for both the younger and adult participants (see Table 1 and Figure 2), although in neither group were the two slopes significantly different, $t(48) = 0.34$, $p > .05$, and $t(140) = 0.52$, $p > .05$, for younger and adult groups, respectively. In general, the variability associated with each measure decreases with maturation and increases with age, although these effects were not pronounced. The P3MAX latency measurement technique yielded results highly similar (1.4 ms per year increase for adults) to previous aging reports using a large sample of adults. The P3MAX mean latency for the adults was 313 ms, which is a prototypical value for the adult auditory P300 ERP component compared with 274 ms and 340 ms for the P3a and P3b. In order to evaluate the P3MAX measurement procedure, a summary of the present and previous P300 latency results from adult aging studies using a similar auditory paradigm is presented in Table 3. The P300 measurement techniques of previous studies typically used a latency window to specify a range in which the maximum positive voltage occurred. This point defined the P300 peak and therefore its latency. Multiple positive peaks have been ignored and either the single most positive value or an interpolated point picked by hand have been used (e.g., Brown et al.,

Table 2. Regression Analysis for the P3a, P3b, and P3MAX Latency Measures in Figures 2 and 3 Computed Separately for the Younger and Older Participants

Age group	Component	N	Ms/Year	Intercept	r
0-15	P3a	25	-6.6	365	-.541**
	P3b	27	-7.8	449	-.473*
	P3MAX	27	-4.5	381	-.375
16-90	P3a	67	1.14	223	.636***
	P3b	77	1.29	281	.607***
	P3MAX	77	1.39	249	.514***

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

Table 3. P300 Latency and Adult Aging Effects Comparing the P3MAX Measurement Technique with Results from Similar Studies

Study	N	r	Ms/year
Polich et al. (Present study)	77	.51	1.4
Picton et al. (1984)	72	.55	1.4
Brown et al. (1983)	49	.65	1.1
Syndulko et al. (1982)	45	.68	1.1
Goodin et al. (1978)	40	.81	1.8

Note. All studies used a two-tone auditory discrimination (oddball) count task; subject ages ranged from 18 to 90 years. Stimuli were typically 1,000 Hz and 2,000 Hz tones presented at 60 to 90 dB SPL with 10 to 20% target tone probabilities. See text for a discussion of the various peak detection and latency measurement procedures.

1983; Goodin et al., 1978). With the replicated trial technique of the present study and consistent observation of multiple P300 subcomponents, the P3MAX approach to peak latency measurement appears to result in accurate individual latency measures and yields results quite similar to the other measurement strategies.

The latency variability remains stable up to 50 years of age; then it appears to increase somewhat with advanced age. Although the sample size of the present study was relatively large, an accurate assessment of just how this variability might change for very elderly adults remains to be determined. It is noteworthy, however, that P300 latency as defined by the P3a and P3b subcomponents and by the P3MAX technique tends to become slightly more variable for older (over 50 years) adults.

Discussion

The increase in P300 latency with age for the adults and the decrease in latency for children with continued development corroborates the effects obtained previously with a similar paradigm (Goodin et al., 1978). The rate of increase in latency as defined by the P3b subcomponent (1.3 ms per year) and P3MAX technique agrees quite well with previous studies (Brown et al., 1983; Picton et al., 1984; Pfefferbaum et al., 1984; Syndulko et al., 1982) but is somewhat less than initial measurements (Goodin et al., 1978) — most likely resulting from differences in task difficulty (see Squires et al., 1980). The present study also revealed that subcomponents of the P300 demonstrate statistically similar increases in latency with age, although the slope of the earlier P3a does not appear to rise as sharply as the later P3b. This similarity suggests that if different processes are responsible for generating the P3a and P3b, age affects them in a similar fashion. Slightly more variability was observed for the various P300 subcomponent latency measures as adult age increased. With advanced age this variability was enhanced somewhat, perhaps reflecting the wide variation often observed in cognitive function of elderly adults.

The extent of P300 latency variability is an important consideration when this ERP component is applied for the evaluation of cognitive function. Previous aging studies using auditory stimuli (Brown et al., 1983; Goodin et al., 1978; Pfefferbaum et al., 1984a; Picton et al., 1984) have indicated that the standard deviation of P300 latency is on the order of 20 to 40 ms. Because a clinical definition of abnormality often is based on deviation from a mean population value by two to three standard deviations, an understanding of P300 variance is important for determining the limits of normal variation. Factors such as participant age, repeated testing, task difficulty (Goodin et al., 1983) and memory capacity (Polich et al., 1983a) may all play a role in determining an individual's P300 latency.

In general, the difficulties surrounding component identification arising from individual differences in waveform morphology and latency variability are present across a wide range of participant ages and variations in stimulus parameters. Although it is premature to advocate a single measurement technique, the present results suggest several guidelines for establishing normative P300 latency data. First, waveform replication over several trials will assure that ERP responses are obtained rather than artifactual ones. This methodology has not been implemented in most experimental studies even though it is a common procedure in clinical settings. Second, a consistent rule for measuring the peak latency of the P300 should be established. Although many participants demonstrate P3a and P3b subcomponents, not everyone shows this pattern or produces a reliable bifurcation of the major positive peak. Thus, measurement of the P300 may become problematic, especially for the increased morphological and latency variability found with aged and clinical populations.

Several alternatives are possible: (a) Measurement of the most positive-going portion of the waveform within a pre-established latency window yields latency measures for a single peak (e.g., Pfefferbaum et al., 1984a; Picton et al., 1984; Polich et al., 1983b); (b) extrapolation of the initial rise in positivity and the final negative shift to determine an estimate of the projected component peak has been used to circumvent the multiple-peak issue altogether (e.g., Brown et al., 1983; Goodin et

al., 1978; Squires et al., 1980); (c) establishment of separate normative values for the early (P3a) and later (P3b) P300 subcomponents, as in the present study, will provide specific peak identification data because each subcomponent may be reflecting different cognitive events (cf. Polich et al., 1983a; Squires et al., 1975); (d) use of multiple measurement replications and taking an average of the maximum voltage peak (i.e., a P3MAX approach) also appears to provide consistent and reliable individual latency data. Each of these procedures attempts to define individual P300 latency in a slightly different way because no single procedure has been established. As suggested by the summary presented in Table 3, however, if large sample sizes are employed with similar stimulus parameters, the various peak measurement procedures tend to yield similar results.

References

Brown, W. S., Marsh, J. T., & LaRue, A. (1982). Event-related potentials in psychiatry: Differentiating depression and dementia in the elderly. *Bulletin of the Los Angeles Neurological Societies, 47*, 91–107.

Brown, W. S., Marsh, J. T., & LaRue, A. (1983). Exponential electrophysiological aging: P300 latency. *Electroencephalography and Clinical Neurophysiology, 55*, 227-285.

Donchin, E. (1981). Surprise! . . . Surprise? *Psychophysiology, 18*, 493–513.

Duncan-Johnson, C. C., & Donchin, E. (1977). On quantifying surprise: The variation in event-related potentials with subjective probability. *Psychophysiology, 14*, 456–467.

Ford, J. M., Duncan-Johnson, C. C., Pfefferbaum, A., & Kopell, B. (1982). Expectancy for events in old age: Stimulus sequence effects on P300 and reaction time. *Journal of Gerontology, 37*, 696–704.

Goodin, D., Squires, K., & Starr, A. (1978). Long-latency event-related components of the auditory evoked potential in dementia. *Brain, 101*, 635–648.

Goodin, D., Squires, K., & Starr, A. (1983). Variations in early and late event-related components of the auditory evoked potential with task difficulty. *Electroencephalography and Clinical Neurophysiology, 55*, 680–686.

Goodin, D., Squires, K., Henderson, P., & Starr, A. (1978). Age-related variations in evoked potentials to auditory stimuli and normal human subjects. *Electroencephalography and Clinical Neurophysiology, 44*, 447–458.

Halgren, E., Squires, N., Wilson, C., Rohrbaugh, J., Bab, T., & Crandall, P. (1980). Endogenous potentials generated in the human hippocampal formation and amygdala by infrequent events. *Science, 210*, 803–805.

Hansch, E. C., Syndulko, K., Cohen, S. N., Goldberg, Z. I., Potvin, A. R., & Tourtellotte, W. W. (1982). Cognition in Parkinson's disease: An event-related potential perspective. *Annals of Neurology, 11*, 599–607.

McCarthy, G., & Donchin, F. (1981). A metric for thought: A comparison of P300 latency and reaction time. *Science, 211*, 77–80.

Okada, Y. C., Kaufman, L., & Williamson, S. J. (1983). The hippocampal formation as a source of the slow endogenous potentials. *Electroencephalography and Clinical Neurophysiology, 55*, 417–426.

Pfefferbaum, A., Ford, J. M., Wenegrat, B. G., Roth, W. T., & Kopell, B. S. (1984a). Clinical applications of P3: I. Normal aging *Electroencephalography and Clinical Neurophysiology, 59*, 85–103.

Pfefferbaum, A., Wenegrat, B. G., Ford, J. M., Roth, J. M., Roth, W. T., & Kopell, B. S. (1984b). Clinical application of the P3 component of event-related potentials. II. Dementing, depression, and schizophrenia. *Electroencephalography and Clinical Neurophysiology, 59*, 104–124.

Picton, T. W., Stuss, D. T., Champagne, S. C., & Nelson, R. F. (1984). The effects of age on human event-related potentials. *Psychophysiology, 21*, 312–326.

Polich, J., & Starr, A. (1984). Evoked potentials in aging. In: M. L. Albert (Ed.), *Clinical Neurology of Aging*. Oxford University Press, New York.

Polich, J., Howard, L. A., & Starr, A. (1983a). P300 latency correlates with digit span. *Psychophysiology, 20*, 665–669.

Polich, J., McCarthy, G., Wang, W., & Donchin, E. (1983b). When words collide: Interactions of orthographic and phonological factors during word processing. *Biological Psychology, 16*, 155–180.

Pritchard, W. S. (1981). Psychophysiology of P300. *Psychological Bulletin, 89*, 506–540.

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*, 1980, *11*, 135–151.

Snyder, E., & Hillyard, S. A. (1976). Long-latency evoked potentials to irrelevant, deviant stimuli. *Behavioral Biology 16*, 319–331.

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 1980's: Selected contemporary issues in the psychology of aging*. American Psychological Association, Washington, DC.

Squires, N. K., Squires, K. C., & Hillyard, S. A. (1975). Two varieties of long-latency positive waves evoked by unpredictable auditory stimuli in man. *Electroencephalography and Clinical Neurophysiology, 38*, 387–401.

Syndulko, K., Hansch, E. C., Cohen, S. N., 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. Courjan, F. Mauguiere, and M. Revol (Eds.), *Clinical applications of evoked potentials in neurology*. Raven Press, New York.