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AGE-RELATED VARIATIONS IN EVOKED POTENTIALS TO AUDITORY STIMULI IN NORMAL HUMAN SUBJECTS*

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The recent success of clinical investigators in using averaged evoked potentials to assess the integrity of primary sensory pathways (see Starr, in press for review) provides the impetus for further application of evoked potential techniques to other types of neurological disease. Our approach is to study the late (200–500 msec) event-related components, which are sensitive to psychological variables (Sutton et al. 1965; Tueting, in press), in neurological diseases that affect cognitive function.

Before these potentials can be used clinically, however, additional normative studies should be conducted. In particular, the vast majority of studies of the event-related components of the evoked potentials have used subjects from a restricted age group of young adults. Most neurological patients, however, belong to a substantially older adult population. Since there are numerous reports of age-related changes in stimulus-evoked cortical potentials with latencies in the range of 50–250 msec post-stimulus (Dustman and Beck 1969; Dustman et al. 1976; Kooi and Bagchi 1964; Lüders 1970; Shagass and Schwartz 1965; Shagass et al. 1972; Straumanis et al. 1965; Tamura et al. 1972), similar variations in the event-related potentials should be anticipated. Indeed, those few studies which have considered the effect of aging on event-related potentials, such as P300, have demonstrated significant effects on both peak amplitude

and latency (Brent et al. 1976; Marsh and Thompson 1972), the general trend being toward decreased amplitude and increased latency with increasing age. At the other end of the age spectrum there are many reports of maturational changes in the stimulus-evoked cortical components (Barnet et al. 1975; Buchsbaum et al. 1974; Callaway and Halliday 1973; Creutzfeldt and Kuhnt 1967; Dustman and Beck 1969; Dustman et al. 1976; Ohlrich and Barnet 1972) and Shelburne (1972, 1973) Karrer and Ivins (1976) and Courchesne (1977) have noted developmental changes in the P300 component.

It is thus apparent that age is a significant variable affecting both early and late cortical potentials and that systematic studies of these potentials covering the complete life-span must be conducted. This study tests a number of subjects of different ages to define age-related changes in various components of the cortical auditory-evoked potential waveform, and in particular those components related to cognitive processes.

Method

Forty-seven subjects (25 female, 22 male) with no known neurological problems, ranging in age from 6 to 76 years, were tested. Four of the subjects were familiar with the experimental design. Subjects were considered 'normal' if they were fully employed or attending school. When that criterion was not applicable, such as with the older, retired subjects, the subject's mental status was assessed using the 'Mini-Mental State' test

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(Folstein et al. 1975) and all who were included in the study scored 29 or 30 out of the 30 possible points. Two subjects were eliminated from the experiment due to low test scores and one was eliminated because of a psychiatric problem.

Four hundred binaural tone bursts (50 msec, 60 dB SL, 5 msec rise-fall times) were presented through earphone (TDH-39) at a rate of 1/1.5 sec in each run. Eighty-five percent (340) of the tones had a frequency of 1000 c/sec and 15% (60) had a frequency of 2000 c/sec. The stimulus sequence was random with the constraint that no two rare stimuli appeared in succession.

None of the subjects reported having hearing problems. All subjects were able to clearly hear the tone sequences and all reported that they had no difficulty distinguishing between high and low frequency tones.

After familiarizing the subject with the tone bursts, the pre-recorded stimulus sequence was presented twice with an intervening 5-min rest period. Before the first run, the subject was instructed to ignore the tones and to read a magazine of the subject's choice ('Ignore' condition). Before the second run ('Attend' condition), the subjects were instructed to keep a mental record of the rare, 2000 c/sec tones and to report the number at the end of the run. In both conditions the subjects were cautioned to refrain from movements especially those associated with counting in the Attend condition. All subjects were able to perform the task correctly. Correct performance was assumed when subjects counted within 2 or 3 of the actual number of rare tones. The errors which occurred were attributed by the subjects to confusions in counting ('losing the count') rather than to perceptual errors. There was no systematic variation in error rate with age.

Silver disc electrodes were affixed to the scalp at F_z, C_z, P_z, C₃, C₄, P₃ and P₄ with collodion and referred to linked mastoids. Additional electrodes were positioned superior and lateral to the right eye in order to monitor eye-related potentials. The EEG was amplified

10 000 times with a bandpass of 0.3–70 Hz. Evoked potentials were computer (TN-1500) averaged for 768 msec following stimulus onset.

Averaged evoked potential waveforms were computed separately for all of the rare and all of the frequent stimuli in each condition. In addition to the 4 basic-evoked potential waveforms (two tones in two separate conditions), 'difference' waveforms were constructed by digital subtraction of the evoked potentials in the Ignore condition from those for the corresponding stimulus and electrode site in the Attend condition.

Peak latencies and amplitudes of the N₁ and P₂ components were obtained from the evoked potential waveforms for the frequent stimulus in each attention condition. Peak latencies and amplitudes for the N₂ and P₃ components were obtained from the 'difference' waveforms. Latency values were obtained from the intersection of extrapolated lines from the ascending and descending slopes of each peak. (Alternative methods of picking peaks were tested and yielded equivalent results*). Amplitudes were measured as peak-to-peak voltages for N₁-P₂ and for N₂-P₃ rather than base-to-peak due to uncertainties in establishing a baseline voltage.

Results

The four vertex evoked potential waveforms for one subject are shown in Fig. 1. The difference waveforms for each tone (Attend minus Ignore) are also shown. As in previous reports (Picton et al. 1971; Simson et al. 1977; N. Squires et al. 1975, 1977; K. Squires et al. 1977; Tueting et al. 1971) the waveforms for the frequent tone are characterized by a negative-positive deflection (N1-P2), and the waveform for the rare tone in the

* An alternative method of identifying peak latencies consisted of taking the latency of the largest peak within a selected latency range, or, if two approximately equal amplitude peaks were found, taking the average of the two peak latencies.

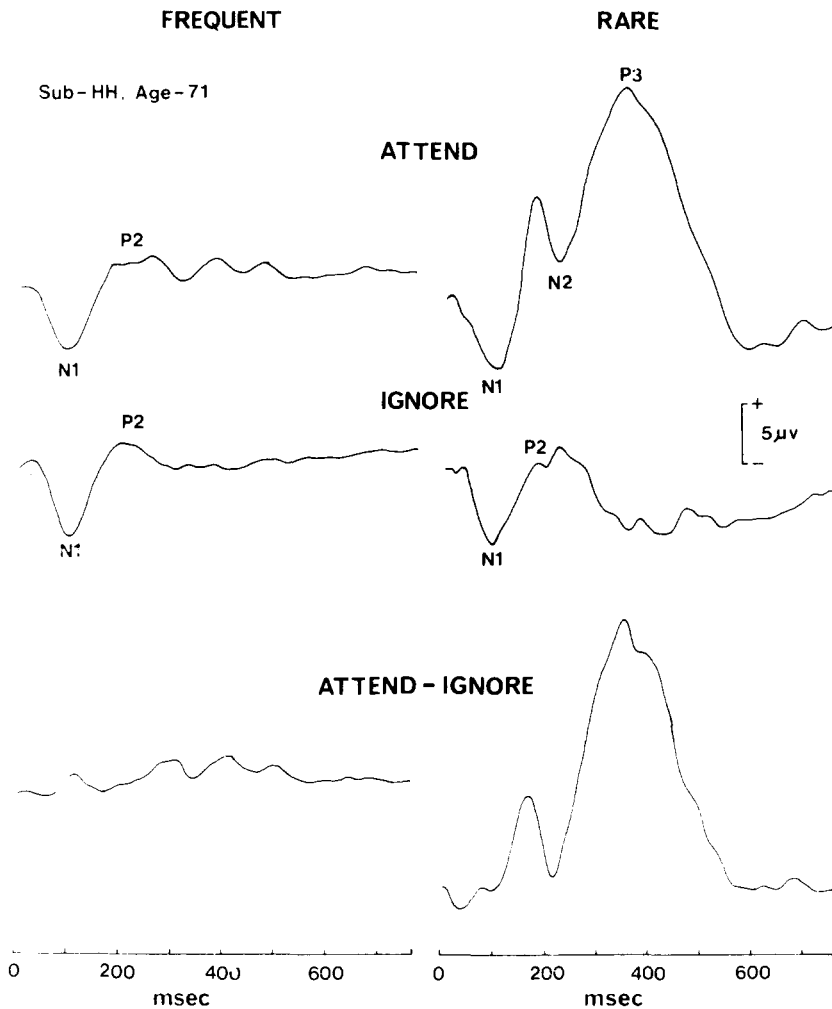


Fig. 1. Vertex evoked potentials for one subject for the rare and frequent tones in the two attention conditions (Attend and Ignore). Also shown are the 'difference' waveforms resulting from the subtraction of the Ignore waveform from the Attend waveform for each tone.

Attend condition by an additional large positive component, P3. In Fig. 2 the difference waveforms for the rare tone are shown for 6 subjects including the subject from Fig. 1. These waveforms show an additional prominent negative component, N2, preceding P3 (Picton et al. 1976; Ruchkin and Sutton in press; Simson et al. 1977). The difference waveforms for the frequent tone showed no such components (Fig. 1).

Event-related components — N2 and P3

Latency — adults

The latencies of N2 and P3 are plotted in Fig. 3 as a function of age. The shortest latencies were from subjects in their late teens and early twenties; latencies were longer for both the older and the younger subjects. Because of the biphasic nature of the latency-age function, the subject

population was divided into adult and child groups at age 15 for further analysis. A regression analysis for the adult population (40 subjects 15 years and older) showed that the latency of P3 significantly increased with age.

ATTEND - IGNORE

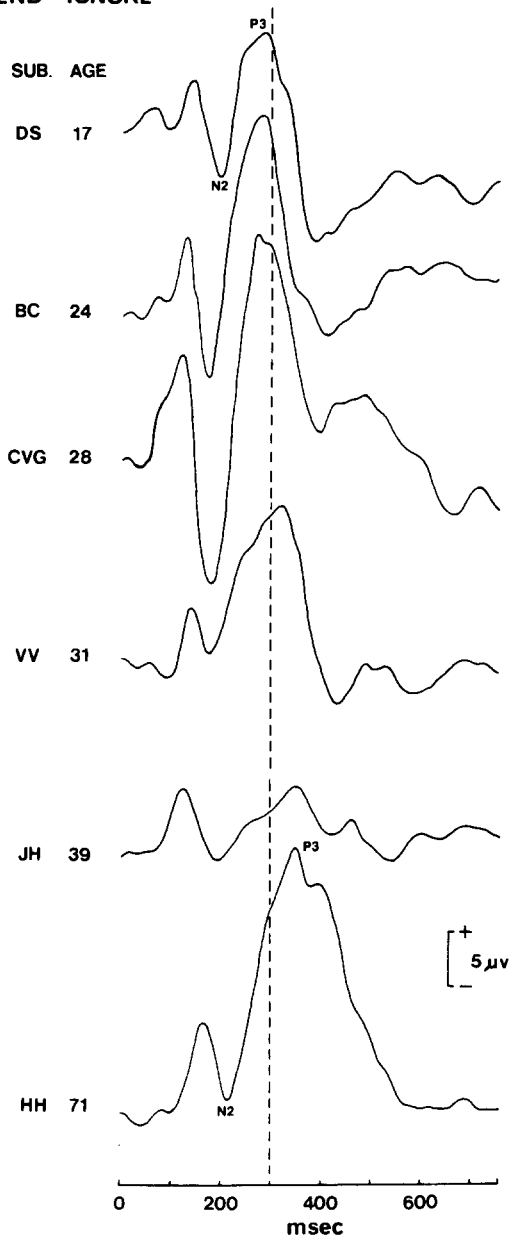


Fig. 2. Difference waveforms (vertex) for the rare tone are shown in order of increasing age for 6 subjects.

The rate from a regression analysis was 1.8 msec/year. At age 15 the latency for P3 calculated from the regression line (Fig. 3) was 294 msec and the standard error from the regression line was 21 msec (Table I). A similarly significant increase in latency with age was found for N2, although the rate of change (0.8 msec/year) (Fig. 3) was significantly less than for P3 ($P < 0.001$). At age 15 the calculated N2 latency was 199 msec and the standard error from the regression line was 15 msec (Table I).

Amplitude - adults

The adult N2-P3 amplitude decreased with age at the rate of $0.2 \mu\text{V}/\text{year}$ ($\rho = -0.313$, $P < 0.05$) from a calculated value of $17.7 \mu\text{V}$ at age 15. The standard error from the regression line was $7.9 \mu\text{V}$.

Scalp distribution - adults

The mean normalized amplitudes of N2-P3 (expressed as a percentage of the C_z amplitude for each subject) at F_z and P_z were 86 and 85%, respectively. At both F_z and P_z the N2-P3 amplitude decreased less rapidly with age than at C_z , yielding a more nearly equipotential voltage distribution for the older subjects. The F_z/C_z ratio had a correlation with age of 0.330 ($P < 0.05$) and for P_z/C_z it was 0.295 ($P < 0.10$). The ratio of N2-P3 amplitudes at F_z and P_z remained unchanged with age.

No systematic differences were found between the N2-P3 amplitudes at C_3 and C_4 or between P_3 and P_4 and no systematic age-related variation were found.

Latency - children

While the adult N2 and P3 latencies increased with age, the opposite effect was found for the 7 children between ages 6 and 15. The latency of N2 decreased with age at a rate of 12.3 msec/year ($\rho = -0.820$, $P < 0.02$) and P3 latency decreased with age by 18.4 msec/year ($\rho = -0.970$, $P < 0.001$) (Fig. 3).

Amplitude and scalp distribution - children

The N2-P3 amplitudes for children tended to increase with age, although the results were

not statistically significant, and tended to be smaller than the amplitudes expected by extrapolation from the adult data. The N2-

P3 scalp distribution did not differ significantly from that of the adults and did not vary significantly with age.

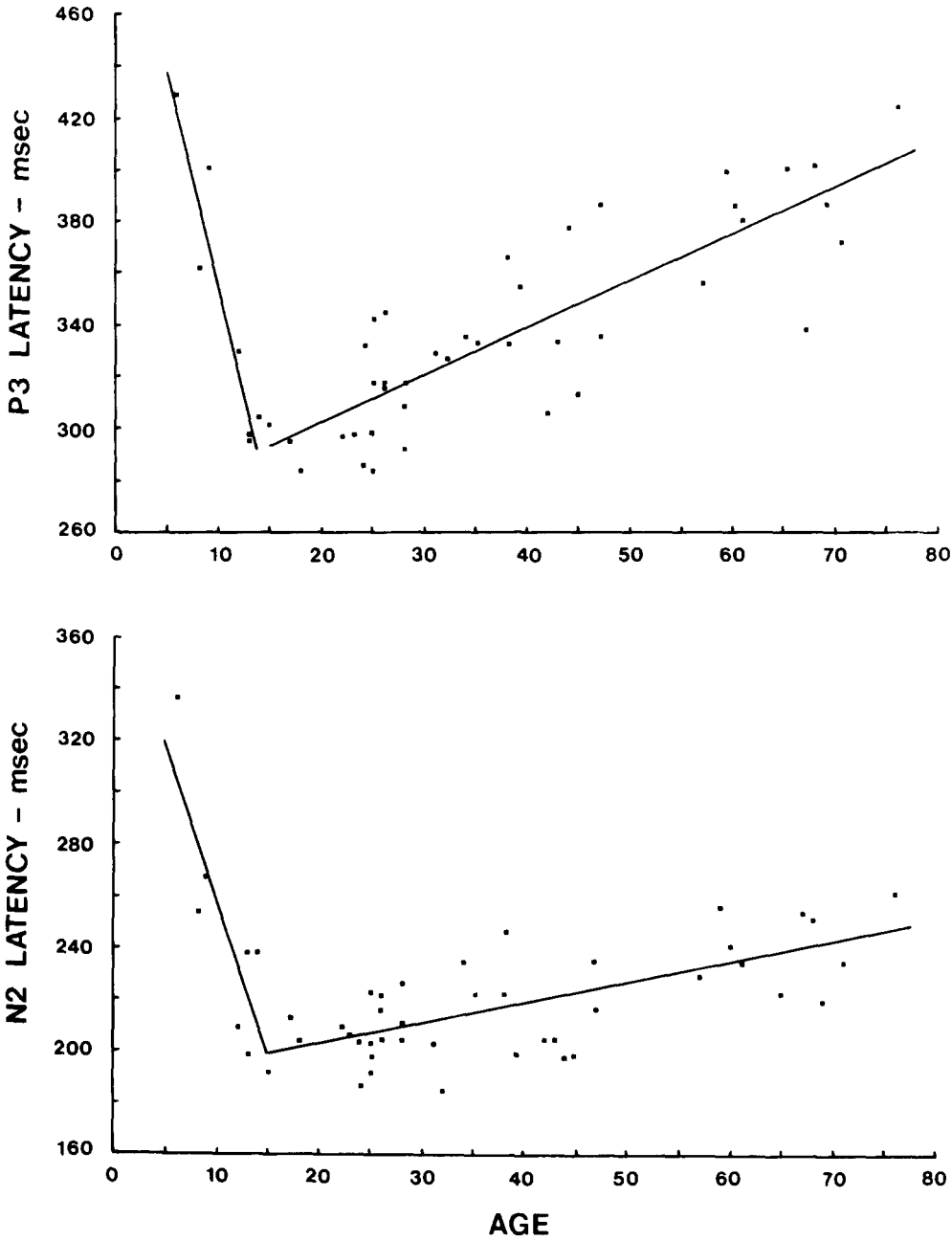


Fig. 3. Plots of N2 and P3 latency as a function of age. Also shown are the calculated regression lines for each component. The two regression lines for each component were calculated separately for subjects less than and greater than 15 years of age.

TABLE I

Summary of adult age-related variations in evoked potential component latencies

Component	Slope (msec/year)	Calculated latency at 15 years (msec)	Standard error from line (msec)	Correlation coefficient	Significance level
N1-Attend	0.1	94	8	0.288	$P < 0.1$
N1-Ignore	0.1	94	8	0.256	$P > 0.1$
P2-Attend	0.7	168	19	0.560	$P < 0.001$
P2-Ignore	0.7	163	19	0.545	$P < 0.001$
N2	0.8	199	15	0.691	$P < 0.001$
P3	1.8	294	21	0.829	$P < 0.001$

Stimulus-evoked components — N1 and P2

Latency — adults

The N1 and P2 peak latencies are plotted in Fig. 4 as a function of age. The adult P2

latency increased significantly at the rate of 0.7 msec/year in both conditions, from a calculated value of 163 msec at age 15 in the Ignore condition (Table I). The difference in slopes between the age-latency regression lines

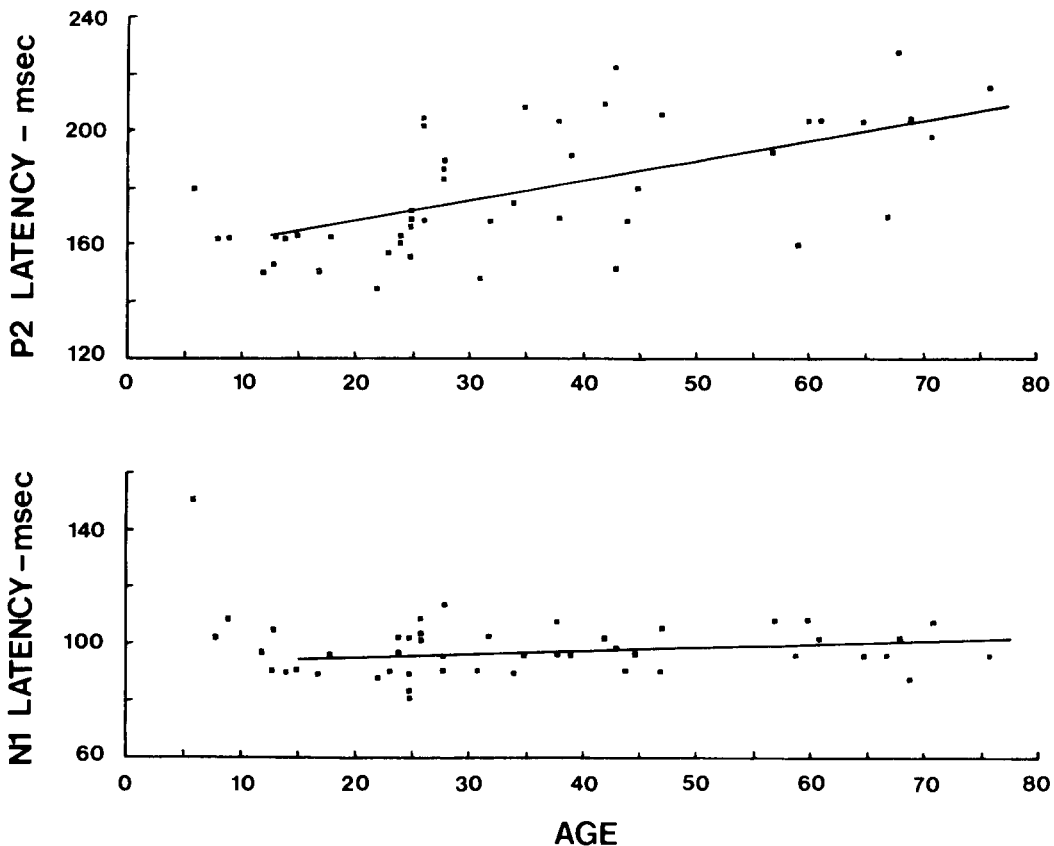


Fig. 4. Plots of N1 and P2 latency as a function of age. Also shown are the regression lines for each component calculated for subjects greater than 15 years of age.

for N1 and P2 was significant ($P < 0.001$).

Amplitude — adults

In both attention conditions the N1-P2 amplitude decreased with age at a rate of $0.2 \mu\text{V}/\text{year}$ (Ignore, $\rho = -0.417$, $P < 0.01$; Attend, $\rho = -0.420$, $P < 0.01$) from a value of $15.6 \mu\text{V}$ at age 15.

Scalp distribution — adults

As was the case for N2-P3, the N1-P2 scalp distribution varied slightly with age. The mean normalized N1-P2 amplitude for all adults was 85% at F_z and 67% at P_z . The normalized amplitudes with respect to C_z of both F_z and P_z increased with age ($P < 0.01$ at F_z , and $P < 0.10$ at P_z), yielding a more nearly equipotential distribution. The ratio of F_z/P_z amplitude did not vary significantly with age.

Amplitude and latency — children

The latencies of N1 and P2 for the whole group did not differ significantly from those predicted from the adult data (Fig. 4). It should be noted that while N1 and P2 latencies for the 6-year-old subject were the most deviant from the adult function these measurements may be unreliable due to the small amplitudes of these potentials.

As a group, the amplitude of N1-P2 for children tended to be smaller than predicted from the adult data and there was a non-significant trend to larger amplitudes with increasing age ($\rho = 0.493$).

With increasing age there was a striking change in the N1-P2 scalp distribution for the children from a predominantly parietal distribution to the centro-frontal distribution of adults ($P < 0.0001$). For the 3 subjects aged 8–12 (the 6-year-old was not included) the F_z/P_z ratio was 0.529, for the 3 subjects aged 12–15 it was 0.869 and for the adults aged 15–29 it was 1.328.

Discussion

The results of this study show that com-

ponents of the auditory evoked potential change in a systematic manner with age. This is particularly apparent for the event-related potentials, N2 and P3, which are sensitive to cognitive processes, here being associated only with rare attended tones. During childhood the latency of these components decreases rapidly with age (12.3 and 18.4 msec/year for N2 and P3, respectively) to their minimum values in the midteens. This is followed by a slower age-related increase in latency during adulthood (0.8 and 1.8 msec/year for N2 and P3, respectively). These latency shifts were associated with changes in N2-P3 amplitude such that increasing latencies were associated with decreasing amplitude and vice-versa. Similar, but less marked age-related changes were also found in the latencies of the stimulus-evoked components, N1 and P2. The effect of age on the amplitude of the N1 and P2 components was the same as that observed for N2 and P3.

The latency data reported here for the event-related components are consistent with the statements by Shelburne (1973) and Courchesne (1977) that P3 latencies for children are longer than for young adults. Brent et al. (1976) also reported an age-dependent increase in the latencies of N2 and P3 when comparing the data of two discrete groups of young (ages 18–33) and old (ages 65–80) adults. Their data differed somewhat from ours however, in that their N2 occurred later and had a larger shift over a comparable age range, while their P3 was comparable in latency to ours for young adults but shifted with age to a lesser extent. The amplitude changes in P3 found here are also consistent with the reports by Brent et al. (1976) for adults and Karrer and Ivins (1976) for children.

The scalp distributions reported here for the N2-P3 amplitude are also consistent with those reported by Courchesne (1977), who noted that for visual target stimuli the P3 scalp distributions did not differ for children and adults.

The age-related increase in P2 latency

and the decrease in N1-P2 amplitude with age in adults are also consistent with Brent et al. (1976). Schenkenberg (Dustman et al. 1976), however, found no such age-related changes for N1 and P2 (N3, P3 in his nomenclature). He did, however, report latency decreases and amplitude increases for N1 and P2 from childhood to adolescence, which agree with our findings.

A further conclusion that can be reached from these data, which span the adult age range rather than sampling from discrete groups as was done previously, is that the processes of aging, as they affect the auditory evoked potential exhibit a smooth time course. Over a 61-year age range the amplitude-versus-age and latency-versus-age functions for both the stimulus-evoked and event-related components were continuous and approximately linear.

As shown in Fig. 5, the slopes (in msec/year) of the adult age-related latency changes increased in proportion to the mean latency of each component ($P < 0.01$, $\rho = 0.991$). The linearity of this function suggests that the aging process is one of a continuous and

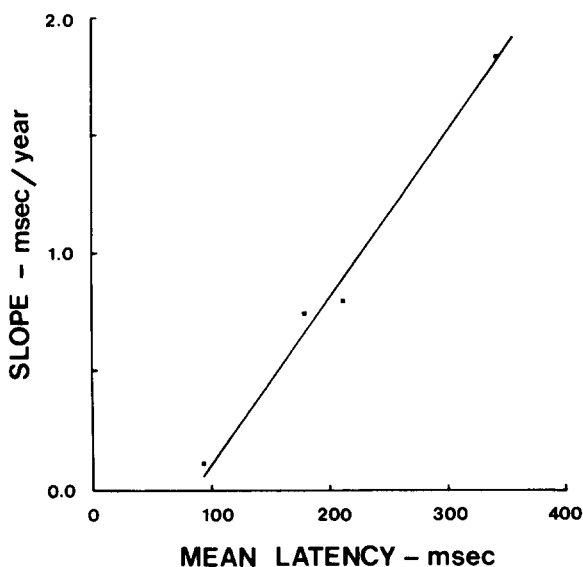


Fig. 5. Plot of the adult regression line slopes as a function of mean peak latency for the evoked potential components (N1, P2, N2, P3).

uniform slowing of neural transmission within those portions of the nervous system reflected in the auditory evoked potential. The first order rate constant for this process, evaluated from the slope of Fig. 5, is 0.007/year. Detailed descriptions of the neural sources underlying each component which would aid in determining the mechanisms responsible for this effect are unfortunately unavailable, however some tentative interpretations of the data in regard to aging mechanisms may be made.

A possible explanation is that the inferred decrease in rate of transmission is due to altered function within the neural populations involved and hence the input-output functions of each population. Decreases in the amount of both excitatory and inhibitory transmitters and/or their associated enzymes with age have been described, as have losses of both cells and dendrites (see Brady et al. 1975, for a review). The rates of decrease differ for the different transmitters and for the same transmitter in different parts of the nervous system, however, and cell and dendrite losses are also variable according to cell type and location. Thus it seems unlikely that such diverse factors would combine in such a way as to be primarily responsible for the apparently uniform process observed here.

Another possibility is that the decreased rate of transmission is due to a decrease in conduction velocity, as might be expected from an age-related alteration in myelination. It has been reported that the amount of central nervous system lipid decreases by 30% between ages 30 and 90 (Brady et al. 1975). Provided that the proportion of myelinated fibers remains reasonably constant and that the age-related lipid changes are not differential, a uniform slowing of neural transmission would result and latency shifts like those observed would be expected.

In contrast to the adult data, where age seemed to affect all components equally, the latency-age functions of the stimulus-evoked and event-related potentials in children differed in important ways. Except for the 6-year-

old, whose N1 and P2 latencies were considered unreliable, the N1 and P2 latencies were consistent with the values of the young adult population. The N2 and P3 latencies of the children, however, were markedly delayed relative to those for the young adults. While a number of interpretations are possible, this is consistent with an explanation in terms of variations in conduction velocity since myelination during maturation proceeds at greatly differing rates within the central nervous system reaching adult status in different parts of the brain at widely different ages (Brady et al. 1975).

A second line of evidence differentiating the maturational changes in the stimulus-evoked and event-related components comes from the scalp distribution data where the N2-P3 amplitude ratio between F_z and P_z was the same as that of adults, while the N1-P2 distribution for children showed a marked change from a parietal to a centro-frontal distribution with increasing age. The reason for the N1-P2 distributional change in maturing children remains obscure, however several possibilities might be advanced, including varying involvement of parietal and frontal cortex (that is, a shift in activity within the cortex), varying contributions of overlapping components (Donald 1976) and varying orientation of neural generators such as in the primary auditory cortex (Vaughan and Ritter 1970).

The relatively late maturation of N2 and P3 latencies may have functional significance if the latencies of the event-related components reflect the temporal course of decision-making by children. Correlations between P3 latency and decision latencies, as assessed by reaction time measures, have been reported on several occasions with adult subjects (Kutas et al. 1977; N. Squires et al., 1977; Ritter et al. 1972) and the age-related increases in N2 and P3 latencies for adults reported here are consistent with reported reaction time increases (Benton 1977). Moreover, choice reaction times for children are reported to decrease with age just as do the

latencies of the N2 and P3 components (Hohle 1967). It should be added that the magnitude of the reaction time shifts cannot be accounted for by shifts in the early components alone. The implication of this linkage of N2 and P3 latencies to decision latency is that there may be a sequential order of development in which the relatively high level decision processes associated with the N2 and P3 components mature later than the presumably lower level process associated with the earlier N1 and P2 components.

It is thus evident from the results of this study that age is a factor which should be carefully considered when attempting to reach conclusions from an evoked potential waveform. This is particularly true in a clinical situation where a majority of neurological patients are substantially older than the population of college age subjects on whose data most of evoked potential research is based. It should also be considered in basic experimental situations where subject ages often span a decade or more, especially when the late components are of principal interest. A considerable increase in the precision of latency relationships of the late components to cognitive processes might be achieved if subjects are selected from restricted age ranges.

On the other hand, the relatively small variations in peak latencies about the regression lines found in this study (15 msec for N2 and 21 msec for P3) indicate that once normal data for a given procedure have been collected, peak latency may be a very sensitive measure of maturation and normal or abnormal aging effects on cognitive processing.

Summary

Auditory evoked potentials were recorded from 47 subjects ranging in age from 6 to 76 years in order to assess the effects of maturation and aging on the evoked (N1 and P2) and event-related (N2 and P3) components. Because of clear differences in the effects of

age on the event-related components between children (less than 15 years of age) and adults the subjects were divided into two populations for analysis. For adults there was a systematic increase in the latency and decrease in amplitude of each component with age. Also the rate of the age-related increase in latency was proportional to the latency of the component. The scalp distributions of both the stimulus-evoked and event-related components were found to vary with age yielding a more nearly equipotential distribution for older subjects. For children the latencies of the event-related components decreased with age. The stimulus-evoked components had latencies which were not significantly different from those predicted from the adult data. In contrast to the adult data, age affected the scalp distributions of the stimulus-evoked components differently than the event-related components. These results suggest an aging process is reflected in the auditory evoked potential which is not the simple inverse of maturational processes.

Résumé

Variations suivant l'âge des potentiels évoqués a des stimulations auditives chez les sujets normaux

Les potentiels évoqués auditifs ont été enregistrés chez 47 sujets dont l'âge varie de 6 à 76 ans afin de mesurer les effets de la maturation et du vieillissement sur les composantes évoquées (N1 et P2) et des composantes liées aux événements (N2 et P3). En raison de différences nettes des effets de l'âge sur les composantes liées aux événements entre les enfants (moins de 15 ans) et les adultes, les sujets ont été divisés en deux populations pour cette analyse. En ce qui concerne les adultes, on note une augmentation systématique de la latence et une diminution d'amplitude de chaque composante avec l'âge. En outre, la vitesse de l'augmentation de latence liées à l'âge s'avère proportionnelle à

la latence de la composante. Les distributions sur le scalp des composantes évoquées par la stimulation et des composantes liées aux événements varient avec l'âge, aboutissant à une distribution presque équipotentielle chez les sujets plus âgés. En ce qui concerne les enfants cependant, les latences des composantes liées aux événements diminuent avec l'âge. Les composantes évoquées par la stimulation ont des latences qui ne sont pas significativement différentes de celles que l'on peut prédire à partir des données obtenues chez l'adulte. Contrairement aux données observées chez l'adulte, l'âge affecte les distributions sur le scalp des composantes évoquées par la stimulation de façon différente des composantes liées aux événements. Ces résultats suggèrent qu'un processus de vieillissement se reflète dans le potentiel évoqué auditif, mais qu'il n'est pas la simple image inversée du processus de maturation.

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