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Speech Evoked Cortical Potentials: Effects of Age and Stimulus Presentation Rate

Kelly L. Tremblay*
Curtis Billings*
Neeru Rohila*

Abstract

We examined the effects of stimulus complexity and stimulus presentation rate in ten younger and ten older normal-hearing adults. A 1 kHz tone burst as well as a speech syllable were used to elicit the N1-P2 complex. Three different interstimulus intervals (ISI) were used (510, 910, and 1510 msec). When stimuli were presented at the medium presentation rate (910 msec ISI), N1 and P2 latencies were prolonged for older listeners in response to the speech stimulus but not the tone stimulus. These age effects were absent when stimuli were presented at a slower rate (1510 msec ISI). Results from this study suggest that rapidly occurring stimulus onsets, either within a stimulus or between stimuli, result in prolonged N1 and P2 responses in older adults. This is especially true when processing complex stimuli such as speech. One potential explanation for this age effect might be age-related refractory differences in younger and older auditory systems. Refractory issues might in turn affect synchronized neural activity underlying the perception of critical time-varying speech cues and may partially explain some of the difficulties older people experience understanding speech.

Key words: Auditory evoked potentials aging, cortical evoked potentials aging, N1-P2 complex and aging, speech understanding and aging

Abbreviations: CAEPs = cortical auditory evoked potentials; ISI = interstimulus interval, VOT = voice-onset-time

Sumario

Examinamos los efectos de la complejidad y de la tasa de presentación del estímulo en diez adultos normo-oyentes jóvenes y en diez más viejos. Un burst tonal de 1 kHz y una sílaba de lenguaje fueron utilizadas para despertar el complejo N1-P2. Se usaron tres diferentes intervalos inter-estímulo (ISI) (510, 910 y 1510 mseg). Cuando los estímulos fueron aplicados a la tasa media de presentación (ISI de 910 mseg), las latencias de N1 y de P2 resultaron prolongadas en respuestas a los estímulos de lenguaje en los sujetos más viejos, pero no para los estímulos tonales. Este efecto de la edad estuvo ausente cuando los estímulos fueron presentados a una tasa más lenta de estimulación (ISI de 1510 mseg). Los resultados de este estudio sugieren que iniciaciones rápidas del estímulo, ya sea dentro del estímulo o entre estímulos, generan en los adultos más viejos, respuestas N1 y P2 prolongadas. Esto ocurre especialmente cuando se procesan estímulos complejos como el lenguaje. Una explicación potencial para este efecto de la edad podría ser las

*Department of Speech and Hearing Sciences, University of Washington, Seattle, WA

Reprint requests: Kelly L. Tremblay, 1417 NE 42nd Street, Seattle, WA 98105; Phone: 206-616-2479; Fax: 206-543-1093; E-mail: tremblay@u.washington.edu

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diferencias refractarias relacionadas con la edad que existen en los sistemas auditivos más jóvenes y más viejos. Estos asuntos de refractariedad pueden, a su vez, afectar la actividad neural sincronizada, involucrada en la percepción de claves críticas para el lenguaje que varían en el tiempo, y puede explicar parcialmente algunas de las dificultades que la gente mayor experimenta para entender el lenguaje.

Palabras Clave: Envejecimiento de los potenciales evocados auditivos, envejecimiento de los potenciales evocados corticales, envejecimiento y el complejo N1-P2, envejecimiento y comprensión del lenguaje

Abreviaturas: CAEP = potenciales evocados auditivos corticales; ISI = intervalo inter-estímulo; VOT = tiempo de iniciación de la voz

Speech evoked P1-N1-P2 cortical auditory evoked potentials (CAEPs) are frequently used to study the neural representation of speech-sounds in populations with impaired speech understanding. The underlying assumption is that speech perception is dependent on the neural detection of time-varying spectral and temporal cues contained in the speech signal. The P1-N1-P2 complex reflects the neural detection of time-varying acoustic cues. Because abnormal P1-N1-P2 response patterns have been reported in children and adults with varying types of speech perception impairments, there is a current surge of interest in learning more about this brain-behavior relationship (Gravel et al, 1989; Kurtzberg, 1989; Gravel and Stapells, 1993; Klein et al, 1995; Kraus, 2001; Rance et al, 2002).

We recently used the P1-N1-P2 complex to examine the neural representation of speech-sounds in older adults with and without hearing loss (Tremblay et al, 2002a, 2002b). Older adults often have difficulty understanding speech (Hayes, 1985; Lutman et al, 1987; Jerger et al, 1989, 1990). They frequently complain: "I can hear you, but I can't understand you." Because speech is a complex signal, composed of multiple time-varying acoustic cues, numerous investigators have hypothesized that aging adversely affects the ability to process temporal cues. More specifically, it is speculated that impaired temporal processing results from age-related factors affecting neural synchrony (Frisina and Frisina, 1997; Schneider and Pichora-Fuller, 2001).

Recent findings support the notion that older adults have more difficulty processing time-varying cues (Strouse et al, 1998; Tremblay et al, 2002a). For example, in the English language, one of the acoustic cues that distinguishes the voiced stop consonant /b/ from its voiceless counterpart /p/ is voice-onset-time (VOT). VOT is defined as the time interval between the release from the consonant stop closure and the onset of voicing (Abramson and Lisker, 1970). Both Strouse et al (1998) and Tremblay et al (2002a) found that older adults, compared with younger adults, had difficulty discriminating short VOTs along a /ba/-/pa/ continuum. In addition, Tremblay et al found that the same /ba/-/pa/ speech-sounds used during perceptual testing evoked abnormally prolonged N1 and P2 latencies in older adults.

Response latencies reflect neural conduction time; therefore, prolonged N1 and P2 latencies in older adults suggest age-related delays in synchronous firing among neural populations generating N1 and P2 responses. However, of particular interest was the fact that Tremblay et al found that younger and older listeners exhibited N1 responses that were similar in latency when evoked by a 0 msec /ba/ VOT stimulus. Therefore, both age groups were able to time-lock to the simultaneous onset of the consonant burst and voicing. However, as VOT increased, age-related differences in N1 emerged. Specifically, N1 latencies in the older group lagged N1 latencies for the younger group. These findings suggest that older auditory systems are less able to time-lock to the onset of voicing when there is a gap between the onset of the consonant and the onset of the voiced vowel.

Animal and human studies have shown that VOT is represented by synchronized responses of neuronal ensembles time-locked

to both consonant release and voicing onset (Eggermont, 1995, 2001; Steinschneider et al, 1999). If it is assumed that some of the neurons that responded to the onset of the consonant are the same neurons that fire in response to the onset of voicing, then delayed N1 responses to the onset of voicing might reflect slower recovery processes from the initial response to the consonant burst. That is, there might be age-related within-stimulus refractory differences with neurons in older auditory systems requiring longer periods of time to recover from the initial period of excitation before they are able to respond to the onset of voicing.

Another finding was that P2 latencies were prolonged regardless of VOT (Tremblay et al, 2002a). Because little is known about the P2 response, the significance of P2 latency delays is unclear. However, it is commonly accepted that N1 and P2 are distinct events, each reflecting several anatomic sources within auditory cortex (Roth et al, 1976; Knight et al, 1980). Therefore, it could be argued that prolonged P2 latencies reflect age-related changes in the auditory system that are not specific to speech-sound processing. In fact, it could be argued that the N1 and P2 latency delays reported in our previous studies occur in older adults regardless of the stimulus complexity and do not reflect age-related neural processing differences specific to speech. Put differently, if the latency delays reported by Tremblay et al (2002a) are simply the result of aging and not specific to speech processing, then we would expect to see delayed N1 and P2 latencies in response to a simpler stimulus that does not contain the VOT cue. To examine this possibility, a literature review was conducted to determine if prolonged N1 and P2 latencies have been reported in older adults in response to simpler stimuli such as tones.

While some studies report age-related N1 or P2 latency delays in response to tones (Pfefferbaum et al, 1980; Papanicolaou et al, 1984; Picton et al, 1984; Schroeder et al, 1995), others report no age-related latency differences (Laffont et al, 1989; Woods, 1992; Polich, 1997; Boutros et al, 2000). Conflicting results might be due to methodological differences. First, many studies do not report or control for differences in hearing sensitivity in younger and older listeners (Papanicolaou et al, 1984; Bahramali et al, 1999; Boutros et al, 2000). Others compensate for age-related hearing loss by presenting stimuli at sensation levels equal to younger normal hearing controls (Laffont et al, 1989; Woods, 1992; Pekkonen et al, 1995b). While effective in equating stimulus audibility, this method does not take into account possible central changes due to peripheral hearing loss (Harrison et al, 1993; Rajan et al, 1993; Schwaber et al, 1993; Willott, 1996). Second, few have examined the effects of aging on the N1-P2 complex recorded in isolation (Spink et al, 1979; Papanicolaou et al, 1984; Laffont et al, 1989; Boutros et al, 2000). Instead, most aging studies elicited the N1-P2 complex in the context of discriminative event-related potentials such as the P300 or MMN (Pfefferbaum et al, 1980; Picton et al, 1984; Woods, 1992; Schroeder et al, 1995; Polich, 1997; Bahramali et al, 1999). Because the MMN and P300 can temporally overlap earlier obligatory responses, and because discriminative responses are often collected at faster stimulus presentation rates (possibly affecting N1 and P2 responses), it is difficult to separate the effects of aging from age-related differences associated with stimulus recording parameters. Nevertheless, studies that did record N1 and P2 responses separately from discriminative responses, using simple stimuli such as tones, report no age-related delays in N1 or P2 latency (Spink et al, 1979; Laffont et al, 1989; Boutros et al, 2000). Therefore, N1 and P2 latency delays reported by Tremblay et al (2002a) might reflect age-related differences in the way complex time-varying acoustic signals, such as speech, are processed.

Finally, if there is reason to think that older adults have more difficulties processing time-varying acoustic cues, perhaps due to within-stimulus refractory issues, then there is also reason to question if age-related differences might be related to between-stimulus refractory issues such as stimulus presentation rate. In short, little is known about age and rate effects using speech stimuli. That is, it is critical that we understand age and stimulus effects before using CAEPs to assess aging adults with disabilities. Because most hearing-impaired people are elderly, the contribution of aging needs to be examined. For example, N1 and P2 responses are also being used to examine age-related disorders such as dementia (Schroeder et al, 1995). Without clearly understanding the contribution of aging, erroneous conclusions

that reflect age-related differences in auditory function (rather than the disorder in question) could be made.

Therefore, in this study we examine the effects of stimulus complexity and stimulus presentation rate in younger and older normal-hearing adults. Younger and older listeners were compared using a tone burst, matched in duration and intensity to the speech syllable, to elicit the N1-P2 complex. Three different interstimulus intervals (ISI) were used: (1) a medium presentation rate equal to that used by Tremblay et al (910 msec ISI), (2) a faster rate (510 msec ISI), and (3) a slower rate (1510 msec ISI). Two questions were asked: (1) Are N1 and P2 latencies delayed in older adults when elicited by a tone presented at the same rate used in our previous study (910 msec ISI)? (2) Are age-related latency differences present at slower stimulus presentation rates?

METHODS

Subjects

Participants were 20 normal-hearing, right-handed adults. "Normal hearing" was defined as pure-tone sensitivity better than or equal to 25 dB HL for octave frequencies between 250 and 8000 Hz in addition to interoctave frequencies (3000 and 6000 Hz). Audiometric thresholds are shown in Figure 1. All subjects had normal immittance results, defined as peak pressure between -150 and +50 daPa, ear canal volume between .6 and 1.5 cc, and admittance between .3 and 1.4 ml (ASHA, 1990).

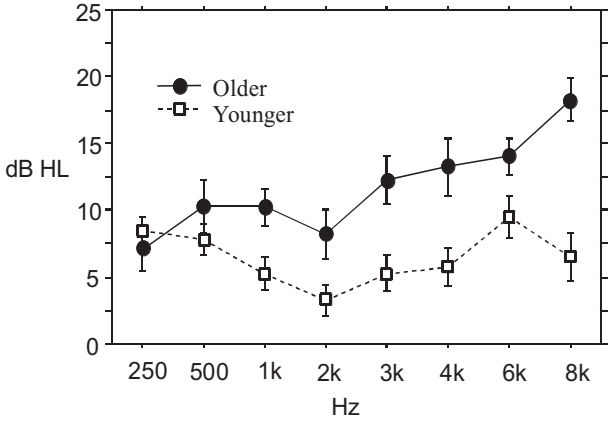

Figure 1. Mean audiometric thresholds for the right ear (1 standard error of the mean). Thresholds for older adults are shown using solid lines. Younger subjects are represented with dotted lines.

Subjects were divided into two groups according to age (10 subjects in each group). The younger group had an age range of 21–33 years, and the older group had an age range of 63–79 years. To rule out any major cognitive impairment, all older subjects obtained a passing score of 24 or better on the Mini Mental Status Examination (Folstein et al, 1975).

Stimuli

A 1 kHz tone and the speech syllable /pa/ were both sampled at a rate of 10 kHz. Tone and speech stimuli were equal in duration (180 msec) and were equated in intensity. Rise/fall times for both stimuli were 20 msec. The /pa/ stimulus was the same Klatt (1980) synthesized signal used in our previous aging study (Tremblay et al, 2002a). The formant (F) and bandwidth (BW) values were: F1 = 700 Hz, BW1 = 90 Hz; F2 = 1200 Hz, BW2 = 90 Hz; F3 = 2600 Hz, BW3 = 130 Hz; F4 = 3300 Hz, BW4 = 400 Hz; F5 = 3700 Hz, BW5 = 500 Hz. The fundamental frequency of the stimulus began at 120 Hz and fell to 100 Hz during the steady-state portion of the vowel. The formant transition was 40 msec in duration. To simulate a burst, a turbulent noise source 10 msec in duration and 60 dB in amplitude was added. The spectrum of the burst was centered around 2500 Hz to 4000 Hz.

Procedure

The N1-P2 complex was measured in response to two stimulus conditions and three interstimulus intervals. In other words, the 1 kHz tone was presented at the three different presentation rates (510, 910, and 1510 msec ISI). Similarly, the /pa/ speech syllable was presented at 510, 910, and 1510 msec ISI. Therefore, a total of six sets of N1-P2 responses were recorded for each subject. In each condition, the stimulus was presented 300 times to obtain the averaged response for the given stimulus and rate. Stimulus and rate order were randomized to prevent potential order effects. A five-minute break (no auditory stimulation) was given between each stimulus block.

All stimuli were presented to the right ear at 74 dB SPL using an Etymotic Research 3A insert earphone. Recordings were obtained while the subject was seated in a reclining chair in a sound-treated booth. During testing the subjects watched a closed-captioned movie of their choice. All subjects were instructed to ignore the stimuli delivered to their right ear, while watching the movie.

Electroencephalographic activity was recorded using the Neuroscan™ Quik-Cap. Because the N1-P2 complex is traditionally recorded and reported from the midline, for the purposes of this study, latency and amplitude measurements were analyzed from electrode Cz. Additional channels were used to define scalp topography and peak latency and amplitude. A nose electrode served as the reference and a forehead electrode as ground. Eye blink activity was monitored using electrodes located on the superior and outer canthus of one eye. Epochs with artifact measuring in excess of ±70 microvolts were rejected off-line. The remaining sweeps were prestimulus baselined, then referred to a common reference. Evoked responses were analog bandpass filtered on-line from .1 to 100 Hz (12 dB/octave roll off). Using a Neuroscan™ 32 channel recording system, EEG activity was amplified using a gain of x500 and converted using an analog-to-digital rate of 1 kHz. Responses were then filtered off-line from 1.0 Hz (high-pass filter, 24 dB/octave) to 40 Hz (low-pass filter, 24 dB/octave). The recording window included a 100 msec prestimulus and 500 msec poststimulus period. A PC-based system controlled the timing of stimulus presentation and delivered an external trigger to the evoked potential system.

RESULTS

Are N1 and P2 Latencies Delayed in Older Adults When Elicited by a Tone or Speech Stimulus?

Figure 2 shows the speech evoked N1-P2 complex recorded from younger and older listeners using the stimulus presentation rate (910 msec ISI) as Tremblay et al (2002a). Figure 3 shows N1-P2 waveforms elicited by the tone stimulus. Unpaired t-tests were used to determine if there were age-related latency differences for N1 and P2 responses. For the speech stimulus, significant latency differences were found for N1 ($t = 2.38$, $df = 18$, $p = .03$) and P2 ($t = 2.46$, $df = 18$, $p = .02$). No significant latency differences were found for the tone condition (N1: $t = 1.32$, $df = 18$, $p = .20$; P2: $t = 1.26$, $df = 18$, $p = .22$). In other words, N1 and P2 latencies were prolonged for older listeners, compared with younger listeners, in response to the speech but not the tone stimulus. Age-related differences observed at electrode Cz are also evident at surrounding recording sites (Figure 4).

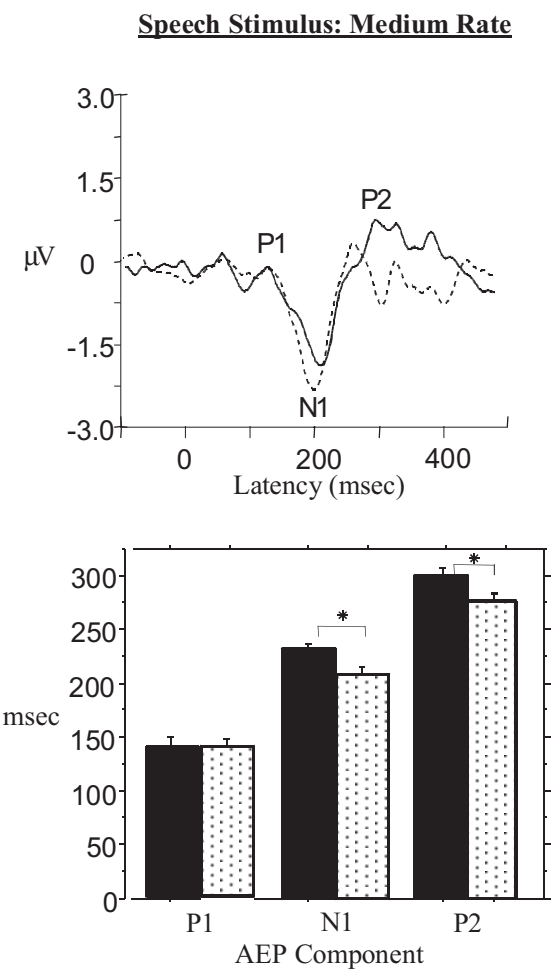

Figure 2. *Top*: Grand mean N1-P2 waveforms recorded from electrode site Cz for 10 older (solid line) and 10 younger subjects (dotted line) in response to speech stimuli presented at the medium presentation rate (910 msec ISI). *Bottom*: Latency values are presented for each AEP component. A significant difference between groups (* = $p < .05$) is seen for N1 and P2.

Are Age-Related Differences Present at Slower Stimulus Presentation Rates?

Latency

Speech Stimulus. Figure 5 shows grand mean N1-P2 waveforms as well as mean latency values for older and younger subjects in response to the speech stimulus. Separate 2 x 3 repeated measure ANOVAs comparing age (younger versus older listeners) and stimulus presentation rate (510, 910, and 1510 msec ISIs) were completed for each AEP component (P1, N1, and P2).

No significant main effects or interactions were obtained for the P1 response (age [F = .03, *df* = 1, p = .87], rate [F = 1.67, *df* = 2, p = .20], age x rate [F = .64, *df* = 2, p = .53]). However, significant effects were found for the N1 response (age [F = 10.00, *df* = 1, p = .006], rate [F = 7.91, *df* = 2, p = .007], age x rate interaction [F = 3.79, *df* = 2, p = .03]). That is, N1 latencies were prolonged for older

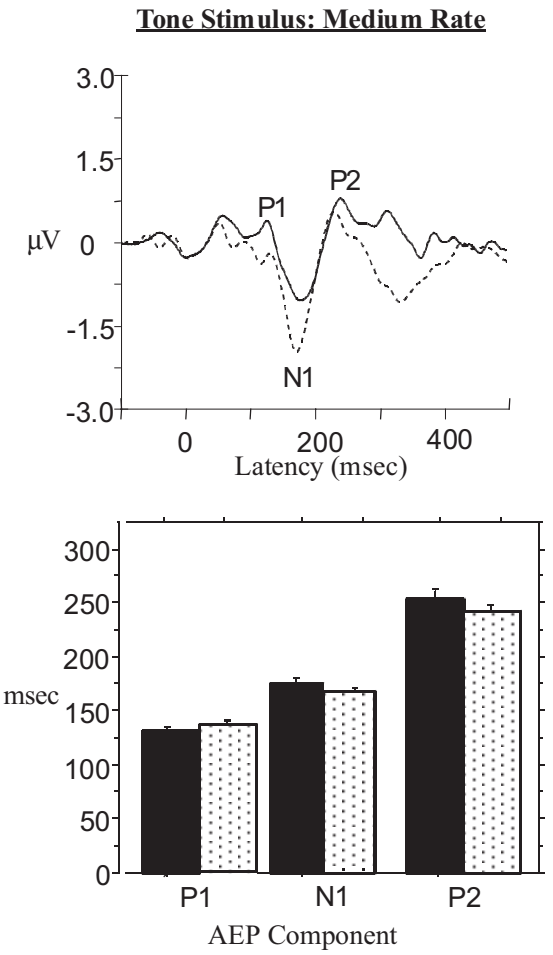

Figure 3. *Top*: Grand mean N1-P2 waveforms recorded from electrode site Cz for 10 older (solid line) and 10 younger subjects (dotted line) in response to tones presented at the medium presentation rate (910 msec ISI). *Bottom*: Latency values are presented for each AEP component. No significant group differences were found.

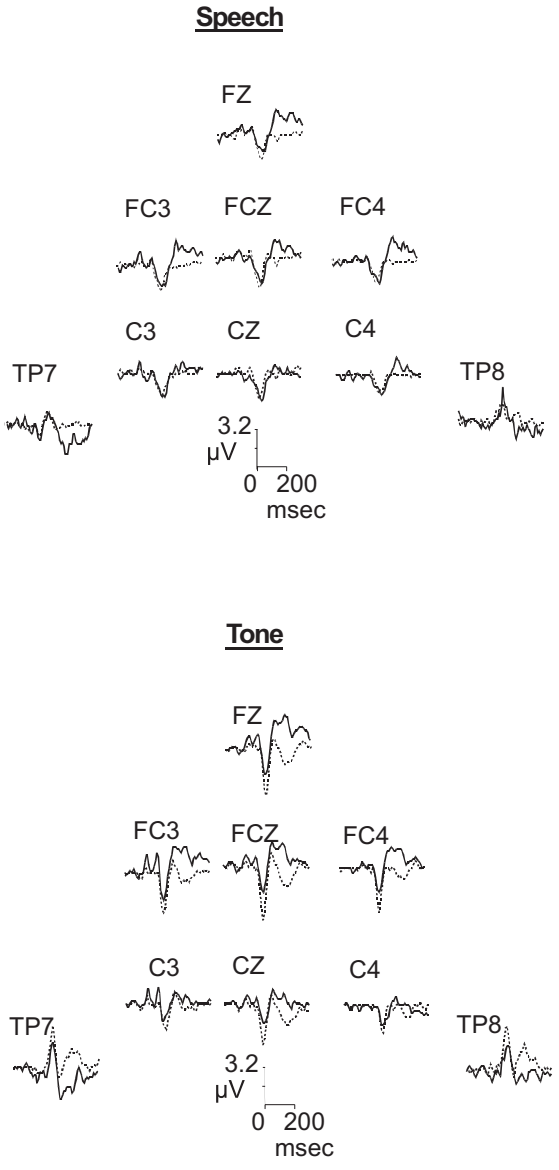

Figure 4. Age-related differences observed at electrode Cz are also evident at surrounding recording sites. N1 and P2 latencies are prolonged for older adults (solid line) in response to the speech stimulus but not the tone stimulus.

adults but only at specific stimulus presentation rates. Post hoc Scheffe analyses revealed no significant difference between groups at the slow rate (p = .44), but significant differences did exist for medium (p = .028) and fast stimulus presentation rates (p = .007). In other words, N1 latencies for older subjects were significantly longer than latencies for younger subjects at medium and fast rates.

A significant main effect for age (F = 4.67, *df* = 1, p = .04) was seen for P2 latencies. However, there was no significant effect for rate (F = .87, *df* = 2, p = .43) or age x rate interaction (F = .36, *df* = 2, p = .70). Therefore, P2 latencies were delayed for the older group in all rate conditions.

Tone Stimulus. Figure 6 shows grand mean N1-P2 waveforms as well as mean latency values for older and younger subjects in response to the tone stimulus. Repeated measure ANOVAs were used to compare P1, N1, and P2 components elicited by tones. No significant main effects or interactions were obtained for the P1 response (age [F = 1.14, *df* = 1, p = .30], rate [F = 1.66, *df* = 2, p = .21], age x rate [F = .80, *df* = 2, p = .46]).

N1 latency analyses revealed a main effect for age (F = 8.34, *df* = 2, p = .01) as well as an age x rate interaction (F = 4.54, *df* = 2, p = .02). There was no main effect for rate (F = 2.65, *df* = 2, p = .09). Therefore, N1 latencies were prolonged for older adults but only at specific stimulus presentation rates. Post hoc Scheffe tests indicated no significant N1 latency differences between age groups for the slow (p = .11) and medium rates (p = .21), but differences did exist for the fast rate (p = .003). For the P2 response, there were no significant main effects for age (F = 3.97, *df* = 1, p = .06) or rate (F = .60, *df* = 2, p = .56), and no significant age x rate interaction (F = 2.11, *df* = 2, p = .14).

Amplitude

Speech Stimulus. Because the N1 response has been shown to decrease in response amplitude with increased stimulus presentation rate (Davis et al, 1966), repeated measure ANOVAs were also used to compare age and rate effects on P1, N1, and

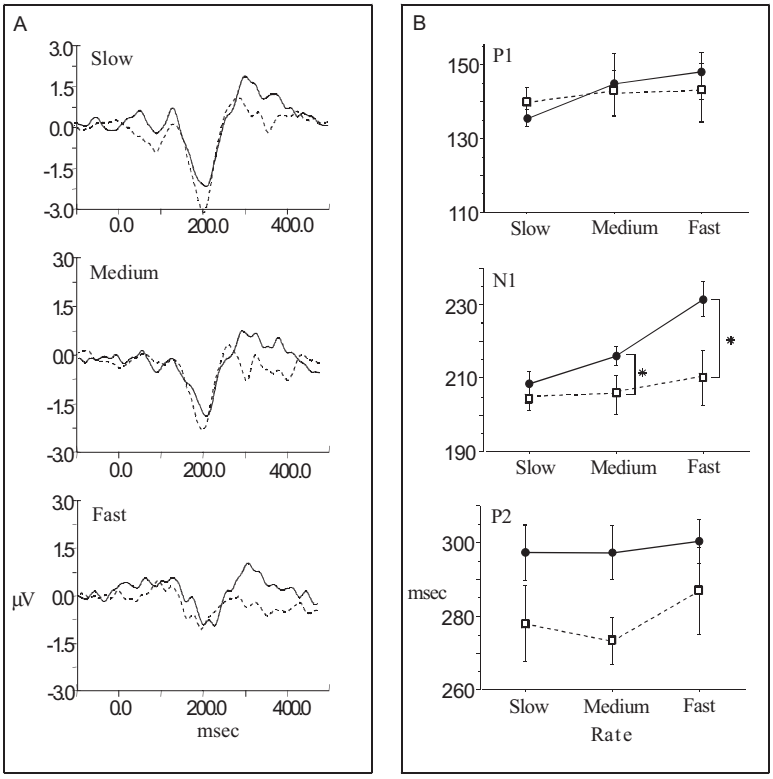

Figure 5. (A) Grand mean N1-P2 waveforms for 10 older (solid line) and 10 younger subjects (dotted line) in response to speech stimuli presented at slow, medium, and fast rates (1510, 910, 510 msec ISI). (B) Latency values (+/-1 standard error) for P1, N1, and P2 components at three stimulus presentation rates. A significant difference between groups (* = p < .05) is seen for N1 at medium and fast rates.

P2 amplitude. No significant main effects or interactions were obtained for the P1 response (age [F = .85 , *df* = 1 , p = .37], rate [F = 2.13, *df* = 2, p = .13], age x rate [F = 1.21, *df* = 2, p = .31]). N1 amplitude decreased as stimulus presentation rate increased [F = 16.7, *df* = 2, p = <.0001); however, there was no significant main effect for age (F = 2.57, *df* = 1, p = .13) and no age x rate interaction (F = 1.30, *df* = 2, p = .29). Therefore, N1 amplitude decreased with increased presentation rate regardless of age. Similar findings were observed for P2 amplitude. As rate increased, P2 amplitude decreased (F = 4.10, *df* = 2, p = .03), but there was no main effect for age (F = 2.04, *df* = 1, p = .17) and no significant age x rate interaction (F = .23, *df* = 2, p = .79).

Tone Stimulus. P1 amplitude was significantly larger for older compared with younger adults (age: F = 4.36, *df* = 1, p = .05), but there was no significant main effect for rate (F = 1.05, *df* = 2, p = .19), and no significant age x rate interaction (F = .32, *df* = 2, p = .73). Significant main effects were also found for N1 amplitude age (F = 6.17, *df* = 1, p = .02) and stimulus presentation rate (F = 12.71, *df* = 2, p < .0001), confirming that N1 amplitude was smaller for older adults and N1 amplitude decreased as stimulus presentation rate increased. No age x rate interaction was found (F = .16, *df* = 2, p = .85). P2 amplitude also decreased with increasing stimulus presentation rate (F = 4.68, *df* = 2, p = .02); however, there was no main effect for age (F = .015, *df* = 1, p = .90) and no significant age x rate interaction (F = .14, *df* = 2, p = .87).

Results Summary

To summarize, when stimuli are presented at the same ISI (910 msec) used by Tremblay et al (2002a), N1 and P2 latencies are prolonged for older listeners in response to the speech stimulus but not the tone stimulus. While age-related delays were present for both stimuli at the faster rate, these age effects are absent when presented at slower stimulus presentation rates.

DISCUSSION

N1 and P2 latencies are prolonged for older adults when elicited by a speech

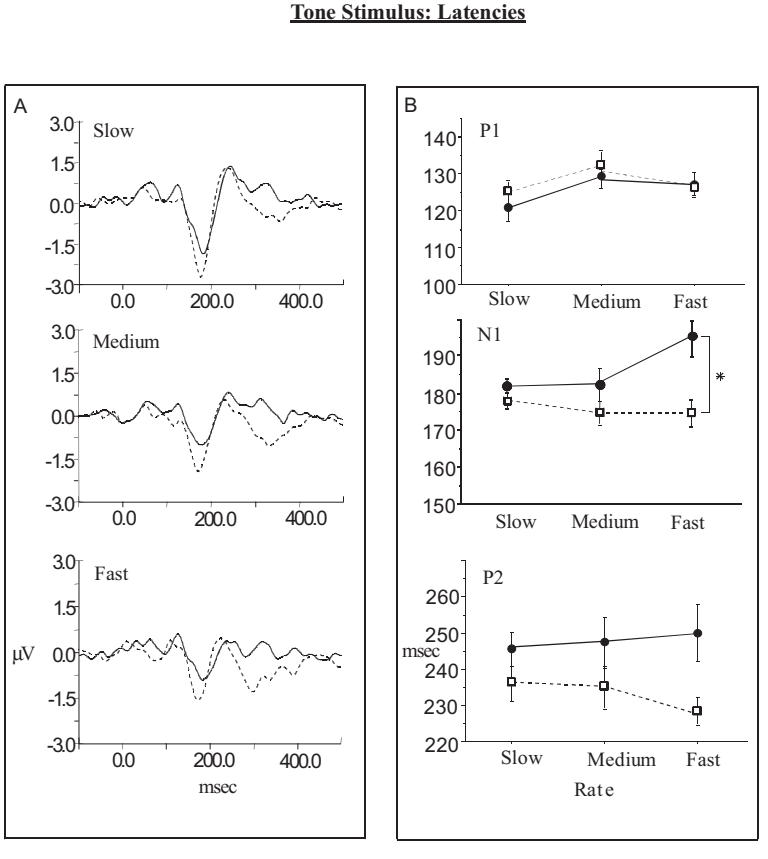

Figure 6. (A) Grand mean N1-P2 waveforms for 10 older (solid line) and 10 younger subjects (dotted line) in response to the tone stimulus presented at slow, medium, and fast rates (1510, 910, 510 msec ISI). (B) Latency values (+/-1 standard error) P1, N1, and P2 components at each stimulus presentation rate. A significant difference between groups (* = p < .05) was found for N1 at the fastest rate.

stimulus using an interstimulus interval of 910 msec. These results are consistent with our previous study (Tremblay et al, 2002a) and might be suggestive of impaired neural synchrony to the onset of the voiced vowel following the consonant burst. Furthermore, age-related differences in neural synchrony might account for some of the perceptual difficulties older people experience. This speculation is made because abnormal N1-P2 responses have been reported in populations with impaired speech understanding (e.g., simulated hearing loss [Martin et al, 1997; Martin et al, 1999], sensorineural hearing loss [Oates et al, 2002], and auditory neuropathy [Kraus, 2001]).

However, it was also possible that delayed N1 and P2 responses were merely a result of age-related delays in neural conduction time that are not specific to the time-varying cues contained in speech, and therefore unrelated to speech understanding. To determine if N1 and P2 latency delays were evident in older listeners, regardless of stimulus complexity, the current study also compared age groups using a steady-state tone that did not contain the time-varying VOT cue. Consistent with previous studies (Spink et al, 1979; Laffont et al, 1989; Boutros et al, 2000), N1 latencies were not prolonged in older adults when elicited by a tone presented at the medium rate, indicating delays were speech related.

If it is assumed that some of the neurons that responded to the onset of the consonant are the same neurons that fire in response to the onset of voicing, then delayed N1 responses to the onset of voicing could reflect slower recovery processes from the initial response to the consonant burst. That is, older auditory systems may require a longer period of time to recover from the initial excitation before neurons are able to fire again, in response to the onset of voicing. Age-related refractory differences have been reported in the literature (Papanicolaou et al, 1984) and might be occurring here.

If there is reason to think that older adults have more difficulties processing time-varying acoustic cues, perhaps due to within-stimulus refractory issues, then there is also reason to question if age-related differences might be related to between-stimulus refractory issues such as stimulus presentation rate. Therefore, stimulus presentation rate was also examined. While N1 latency increased with increasing stimulus presentation rate for the speech stimulus, no significant rate effect was seen for the tone condition. Interestingly, there was an age x rate interaction in both stimulus conditions indicating that prolonged N1 latencies in older adults were absent at the slower presentation rate. Therefore, prolonged N1 latencies observed in response to the speech stimulus presented at the medium rate were not seen in older adults when the stimulus presentation rate was slow. That is, no significant differences in N1 latency were found between younger and older subjects when speech or tones were presented at the slowest stimulus presentation rate (1510 msec ISI). In contrast, age effects are most obvious at increased stimulus presentation rates. From a neuroscience perspective, these results might suggest that older adults may require more time than younger adults to recover from the previous stimulus presentation. This is especially true when processing complex stimuli such as speech. From a clinical perspective, these findings might relate to why older adults frequently complain that people speak too quickly. Numerous studies have shown age-related difficulties perceiving time-compressed speech stimuli (Wingfield et al, 1985; Gordon-Salant and Fitzgibbons, 2001).

P2 latencies were prolonged for the older group in response to the speech stimulus regardless of presentation rate. Although P2 also appears to be prolonged in the tone condition; statistically, this delay approached but did not reach significance. Therefore, unlike the N1 response, stimulus presentation rate does not appear to be a contributing factor, but stimulus complexity does. Even though prolonged P2 responses are consistent with our earlier studies (Tremblay et al, 2002a), the significance of this age effect remains unclear. To date, little is known about the P2 response or its possible relation to speech-sound processing.

In summary, results from this study suggest rapidly occurring stimulus onsets, either within a stimulus or between stimuli, result in prolonged N1 responses in older adults. This is especially true when processing complex stimuli such as speech. One potential explanation of this age effect might be age-related refractory differences in younger and older auditory systems. Specifically, older auditory systems may require a longer period of time than younger systems to recover from

the initial excitation before neurons are able to fire again. These refractory issues may in turn affect synchronized neural activity of critical time-varying speech cues and may partially explain some of the difficulties older people experience understanding speech.

Although these effects were measured using cortical evoked responses, this is not to say that aging only affects the temporal properties within auditory cortex. Numerous studies report anatomical and physiologic changes throughout the central auditory system that might affect speech-sound processing (Hansen and Reske-Nielsen, 1965; Scheibel et al, 1975; Willott, 1991; Jerger, 1995; Pekkonen et al, 1995a; Pekkonen et al, 1995b; Willott, 1999; Bellis et al, 2000; Jerger, 2000; Greenwald and Jerger, 2001; Bertoli et al, 2002). For example, the auditory brainstem response (ABR) has been used to study age-related differences in neural recovery time. Using a forward-masking paradigm, where the time between a masker and the following potential-evoking tone burst is varied, Walton et al (1999) found that older adults exhibited greater latency shifts than younger adults at short forward-masker intervals. Similar effects might be occurring here.

Likewise, we are not suggesting that age-related deficiencies in speech perception are limited to the detection of voice-onset-time. Speech perception is dependent on multiple spectral, temporal, and intensity cues not examined in this study. For example, speech parameters such as first formant onset frequency are also important for discriminating voiced from unvoiced stop consonants (Liberman et al, 1958; McClaskey et al, 1983; Soli, 1983; Treisman et al, 1995). Similarly, higher order semantic and contextual cues contribute to speech understanding. Therefore, the present findings represent only a fraction of the age-related changes that may be associated with impaired speech understanding in the elderly.

CONCLUSION

Results from this study suggest rapidly occurring stimulus onsets, either within a stimulus or between stimuli, result in prolonged N1 and P2 responses in older adults. One potential explanation of this age effect might be age-related refractory differences in younger and older auditory systems. Specifically, older auditory systems might require a longer period of time than younger systems to recover from the initial excitation before neurons are able to fire again. These refractory issues may in turn affect synchronized neural activity of critical time-varying speech cues and may partially explain some of the difficulties older people experience understanding speech.

Although speech-evoked potentials are being used to examine populations with communication disorders, little is known about patient variables (age) and stimulus variables (ISI). This is especially important when studying the hearing impaired because most of these people are elderly, and erroneous conclusions could be made. For example, if the N1-P2 complex is used to compare the neural representation of speech stimuli in two different groups of cochlear implant users, it is important to recognize that latency differences might reflect age differences between or within the groups rather than factors related to the cochlear implant. In these situations, it is necessary to control for age, or use an appropriately slow stimulus presentation rate to minimize stimulus rate effects.

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