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Effects of age and age-related hearing loss on the neural representation of speech cues

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Abstract

Objective: To examine the effects of aging and age-related hearing loss on the perception and neural representation of a time-varying speech cue.

Methods: P1, N1 and P2 cortical responses were recorded from younger and older normal-hearing adults, as well as older adults with age-related hearing loss. Synthetic speech tokens representing 10 ms increments along a /ba/–/pa/ voice-onset-time (VOT) continuum were used to evoke the responses. Each participant's ability to discriminate the speech tokens was also assessed.

Results: Compared with younger participants, older adults with and without hearing loss had more difficulty discriminating 10 ms VOT contrasts. In addition, both older groups elicited abnormal neural response patterns. There were no significant age-related findings for P1 latency; however, N1 latencies were prolonged for both older groups in response to stimuli with increased VOT durations. Also, P2 latencies were delayed for both older groups. The presence of age-related hearing loss resulted in a significant increase in N1 amplitude in response to voiceless stimuli.

Conclusions: Aging and age-related hearing loss alter temporal response properties in the central auditory system. Because both older groups had difficulty discriminating these same speech stimuli, we conclude that some of the perceptual difficulties described by older adults might be due to age-related changes regulating excitatory and inhibitory processes.

Significance: Some of the speech understanding difficulties expressed by elderly adults may be related to impaired temporal precision in the aging auditory system. This might explain why older adults frequently complain that wearing a hearing aid makes speech louder, but does not necessarily improve their ability to understand speech.

Keywords: Speech perception and aging; Aging and temporal processing; Auditory-evoked potentials; Aging and cortical potentials; Speech-evoked cortical potentials

1. Introduction

Older adults, with and without normal-hearing sensitivity, often have difficulty understanding speech (Marshall, 1981; Jerger et al., 1989, 1990; Humes, 1996). 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, it is frequently hypothesized that aging adversely affects the ability to process temporal cues (Dubno et al., 1984; Trainor and Trehub, 1989; Abel et al., 1990; Moore et al., 1992; Fitzgibbons and Gordon-Salant, 1994; Schneider et al., 1994; Snell, 1997). More specifically, it is speculated that: (1) temporal processing is dependent on the neural detection of time-varying acoustic cues and (2) impaired perception results from age-related factors affecting neural synchrony (Frisina and Frisina, 1997; Schneider and Pichora-Fuller, 2001).

Recent findings by Strouse et al. (1998) and Tremblay et al. (2002) support the notion that older adults have more difficulty processing time-varying cues, and perceptual difficulties might be related to factors affecting neural synchrony. For example, in the English language, the voiced stop consonant /b/ is distinguished from its voiceless counterpart /p/ based on a temporal cue called voice-onset-time (VOT). VOT is defined as the time interval between the release from the consonant stop closure and the onset of voicing (Lisker and Abramson, 1970). Both Strouse et al. (1998) and Tremblay et al. (2002) found that older

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adults, compared with younger adults, had difficulty discriminating short VOTs along a /ba/–/pa/ continuum. In addition, these same /ba/–/pa/ speech-sounds-evoked abnormal neural response patterns in older adults (Tremblay et al., 2002). That is, synchronous responses to the onset of the vowel were delayed in older adults.

Tremblay and colleagues used the N1–P2 complex to examine the neural representation of the VOT in younger and older adults. The N1 component of the N1–P2 complex is an onset response reflecting synchronous neural activation of structures in the thalamic-cortical segment of the central nervous system in response to acoustic change (Wolpaw and Penry, 1975; Naatanen and Picton, 1987; Woods, 1995). Moreover, N1 latency has been shown to reflect the onset of voicing along a VOT continuum (Kurtzberg, 1989; Steinschneider et al., 1999; Sharma et al., 2000; Tremblay et al., 2002). Because speech-evoked N1–P2 responses reflect spectral and temporal acoustic changes contained within the speech signal (Kaukoranta et al., 1987; Ostroff et al., 1998; Martin and Boothroyd, 1999), there has been a surge of interest in using speech-evoked N1 and P2 responses to assess the neural representation of time-varying speech cues in various populations with communication disorders. For instance, abnormal speech-evoked N1–P2 responses have been reported in people with impaired speech perception (e.g. simulated hearing loss, Martin et al., 1997; Whiting et al., 1998; Martin and Boothroyd, 1999; auditory neuropathy, Kraus et al., 2000; Rance et al., 2002 and children with auditory based learning problems, Cunningham et al., 2001; Purdy et al., 2002; Wible et al., 2002). In addition, the N1–P2 complex reflects central auditory plasticity associated with various types of auditory rehabilitation including cochlear implantation (Ponton et al., 2000; Purdy et al., 2001) and auditory training (Tremblay et al., 2001; Tremblay and Kraus, 2002; King et al., 2002).

When Tremblay et al. (2002) recorded N1–P2 responses in younger and older adults, N1 and P2 latencies were prolonged for older adults. Specifically, N1 latencies were prolonged for older listeners in response to stimuli with increased VOT durations. P2 latencies were delayed for all stimuli. Because participants in the Tremblay et al. (2002) and Strouse et al. (1998) studies had hearing thresholds that fell within normal limits, age-related differences were unrelated to audibility differences between the two groups. However, most aging adults experience age-related sensorineural hearing loss. The combination of aging and hearing loss likely exacerbates communication problems in at least two ways. First, peripheral hearing loss reduces the audibility of certain acoustic cues and the perceptual consequence is decreased speech intelligibility (Boothroyd, 1984). Second, animal research has shown that peripheral hearing loss alters spatial and temporal response properties throughout the central auditory system (Kitzes, 1984; Willott, 1986; Robertson and Irvine, 1989; Harrison et al., 1993; Rajan and Irvine, 1998; Irvine et al., 2001).

While much is known about the perceptual consequences of age-related hearing loss, less is known about the physiological effects of age and age-related hearing loss in the human central auditory system. From a neuroscientific perspective, this information is important because it helps define neural processes associated with aging and age-related hearing loss. From a clinical perspective, this information may identify a source of performance variability among people who wear hearing aids. Only 40–60% of hearing aid users report significant benefit from using their hearing aids (Humes, 2001). Because aging auditory systems have more difficulty processing temporal cues, making sounds louder through the use of a hearing aid might have a better outcome for younger than older users. Thus, information from the proposed experiment might help explain some of the performance variability experienced by hearing aid users, and could change the way we approach rehabilitating older hearing-impaired adults.

For these reasons, we use the N1–P2 complex to examine the effects of age and age-related hearing loss on the perception and neural detection of VOT. Three groups are examined: (1) young normal-hearing listeners, (2) older normal-hearing listeners and (3) older listeners with high-frequency sensorineural hearing loss. Because Tremblay et al. (2002) reported prolonged N1 and P2 latencies in older normal-hearing listeners, perhaps reflecting age-related changes in neural synchrony, we expect that older adults with hearing loss will show similar latency delays. Prolonged latencies and decreased amplitudes have also been reported in young adults with simulated (Martin et al., 1997; Whiting et al., 1998; Martin and Boothroyd, 1999) and organic hearing loss (Polen, 1984; Oates et al., 2002); therefore, we hypothesize that the presence of age-related hearing loss will add to the age-effects previously reported. That is, older adults with hearing loss will have more difficulty than younger and older normal-hearing groups perceiving VOT contrasts. Also, compared with both younger and older normal-hearing groups, the presence of age-related hearing loss will result in additional N1 and P2 latency delays as well as amplitude reductions.

2. Methods

2.1. Participants

Participants were 10 young normal-hearing (mean = 26.3 years; range = 19–32 years), 10 older normal-hearing (mean = 68.3 years; range = 61–79 years) and 10 older adults with age-related high-frequency hearing loss (mean = 71.2; range = 60–81 years). Audiometric thresholds for the right ear are shown in Fig. 1. To rule out any major age-related cognitive impairment, participants older than 65 years obtained a passing score of 24 or better on the mini mental status examination (Folstein et al., 1975). Participants described themselves as being in good health and denied any significant otologic or neurologic

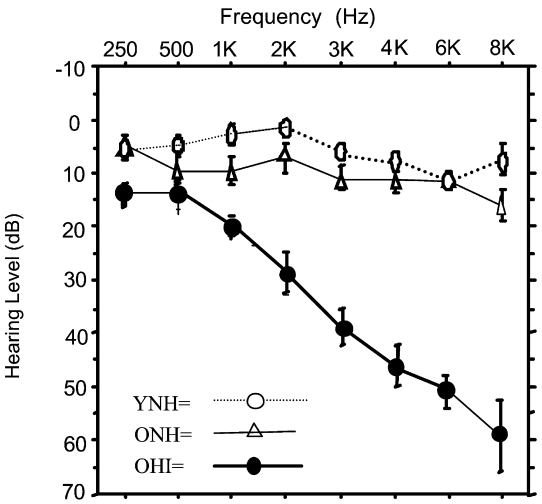

Fig. 1. Mean audiometric thresholds (±1 standard error) for the right ear are shown for the young normal-hearing (YNH), older normal-hearing (ONH) and older hearing-impaired (OHI) groups.

medical history. Each participant signed a consent form approved by the Human Subjects Review Committee.

2.2. Stimuli

Stimuli were synthesized tokens from a 7-step VOT /ba/–/pa/ continuum modeled after those by McClaskey et al. (1983). The 7 tokens ranged from 0 to 60 ms VOT in 10 ms VOT steps (Fig. 2). Stimuli were generated using a Klatt digital speech synthesizer (Klatt, 1980) and adjusted to within 1 dB peak of one another. Also, each stimulus was digitally adjusted to within 0.1 dB root mean squared (RMS) of one another using WaveMod software. Synthesized, rather than natural speech, tokens were necessary to systematically alter the VOT cue in 10 ms increments.

Formant transitions were 40 ms in duration. Starting frequencies for the formant transitions were: $F1 = 438$ Hz, $F2 = 1025$ Hz, $F3 = 2425$ Hz, $F4 = 3250$ and $F5 = 3700$. The fundamental frequency of the stimuli began at 120 Hz and then fell to 100 Hz during the steady-state portion of the vowel. To simulate a burst, a turbulent noise source (AF) 10 ms in duration and 60 dB in amplitude was added to the onset of the formant transition. The spectrum of the burst was 2500–4000 Hz. The steady-state portion of the stimuli consisted of the vowel /a/, which varied in duration relative to the VOT so that the overall duration for each stimulus remained constant at 180 ms. Formant frequency (F) and bandwidth (BW) values for this vowel 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.

2.3. Procedure

2.3.1. Behavioral measures

Stimuli were presented monaurally, to the right ear, at an intensity level of 74 dB peSPL. Subjects were familiarized with the 7 tokens (10 ms VOT increments) of the /ba/–/pa/ continuum. That is, each subject was asked to listen to the single presentations of the 7 tokens beginning with the 0 ms VOT stimulus and ending with the 60 ms VOT stimulus. This familiarization process was repeated a second time. Next, during the one-step AX (same–different) discrimination task, stimulus pairs that were either identical to each other, or differed by 10 ms of voicing were presented. Each listener was asked to determine if the two stimulus tokens presented were the same or different from each other and choose 'same' or 'different' on the computer screen in front of them. A total of 190 trials were presented across 10 blocks (19 trials per block). Each block consisted of 6 pairs of 'different' (e.g. 0–10, 10–20, 20–30, 30–40, 40–50, 50–60 ms VOT) stimuli. Each 'different' stimulus pair was presented twice. Each block also contained 7 'same' or 'catch' stimulus pairs (e.g. 0–0, 10–10, 20–20, 30–30, 40–40, 50–50, 60–60 ms VOT). Each 'catch' trial was presented once. Thus, each block was identical in that, is consisted of 19 trials, two presentations of each 'different'

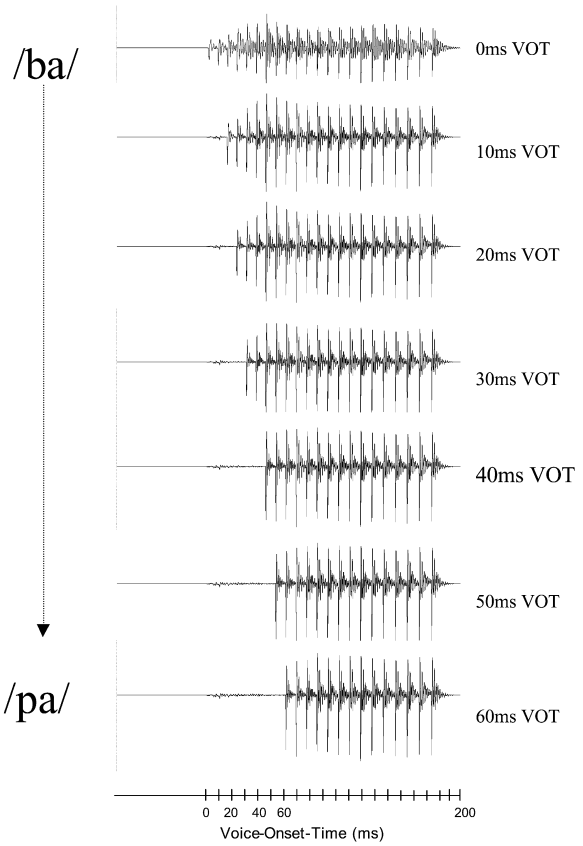

Fig. 2. Acoustic waveforms of each stimulus along the /ba/–/pa/ VOT continuum.

pair and one presentation of each 'same' pair. Order of stimulus pair presentation within each block was randomized. The inter-stimulus interval was 500 ms and all participants took breaks between blocks of trials.

If the participant correctly identified the two stimuli as being 'different', this response was scored as a 'hit'. When the participant responded that the stimuli were 'different' when in fact the two stimuli presented were the same, this response was considered a 'false alarm'. Using a same–different differencing model (MacMillan and Creelman, 1991), 'hit' and 'false alarm' rates were used to calculate *d*-prime (d') values. Participants did not receive any feedback indicating whether their response was correct or incorrect.

2.3.2. Electrophysiology measures

The N1–P2 complex was measured in response to the same 7 tokens used during behavioral testing. For example, the 0 ms VOT stimulus was presented 500 times in order to obtain a single 0 ms averaged response. Then an averaged response was obtained to the +10 ms VOT stimulus. This procedure continued until 7 averaged electrophysiologic responses were obtained. Stimulus order was randomized to prevent potential order effects.

Each stimulus was presented to the right ear at the same intensity level (74 dB peSPL) using the same ER-3A insert earphone worn during behavioral testing. The inter-stimulus interval was 910 ms. Recordings were conducted in a sound-treated booth with the subject sitting in a reclining chair. All participants watched a closed-captioned movie during electrophysiologic testing.

EEG activity was recorded from 32 silver–silver chloride electrodes using the International 10/20 system (Jasper, 1958). 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 ±80 μV were rejected off-line. A PC-based system controlled the timing of stimulus presentation and delivered an external trigger to the evoked potential system. Evoked responses were analog band-pass filtered on-line from 0.1 to 100 Hz (12 dB/octave roll off). Using a Neuroscan™ system, electroencephalogram (EEG) channels were amplified with a gain × 500, 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 ms pre-stimulus period and 500 ms post-stimulus time.

Neural responses were examined across all electrode sites to help identify peak latencies. In addition, grand mean waveforms provided a latency window to aid in response identification. Two research assistants used the following criteria to identify peaks: (1) each P1, N1 and P2 peak should be largest when measured from the fronto-central recording sites and smaller over the parietal region and (2) the polarity of each peak should invert over the mastoid and temporal sites (Vaughan and Ritter, 1970). Latency and amplitude measures were then recorded from electrode site Cz.

3. Results

3.1. Behavioral results

Older listeners, compared with younger listeners, had more difficulty discriminating 10 ms VOT contrasts (young normal-hearing group: mean $d' = 2.0$, standard error = 0.20; older normal-hearing: mean $d' = 1.4$, standard error = 0.14; older hearing-impaired: mean $d' = 0.94$, standard error = 0.15). A one-way repeated measures analysis of variance (ANOVA) revealed a significant age effect ($F = 10.57$, df = 2, $P < 0.001$). Post hoc tests indicate that younger adults performed significantly better than older adults with ($P < 0.001$) and without ($P < 0.05$) age-related hearing loss. Older adults with hearing loss performed more poorly than older adults with normal hearing ($P < 0.05$).

3.2. Electrophysiology results

3.2.1. Latency

Averaged responses recorded from electrode site Cz are shown for each age group in Fig. 3 (left). A repeated measures ANOVA performed on P1 latency, comparing age groups (younger, older listeners with and without hearing loss) and VOT (7 VOT stimulus conditions) revealed a significant main effect for VOT ($F = 17.48$, df = 6, $P < 0.0001$). That is, P1 latency increased with each increase in stimulus VOT. There was no significant effect for group ($F = 1.1$, df = 2, $P > 0.05$) and no group × VOT interaction ($F = 1.7$, df = 12, $P > 0.05$) suggesting increases in P1 latency occurred for all groups, regardless of age or hearing status.

Peak latency values of N1 and P2 for each group are plotted in Fig. 3 (right). A repeated measures ANOVA performed on N1 latency, comparing age groups (younger, older listeners with and without hearing loss) and VOT (7 VOT stimulus conditions) revealed a significant main effect for group ($F = 3.9$, df = 2, $P < 0.05$), a significant main effect for VOT ($F = 66.0$, df = 6, $P < 0.001$), as well as a significant group × VOT interaction ($F = 2.25$, df = 12, $P < 0.05$). In other words, N1 latencies increased with each increase in VOT. However, N1 latencies were prolonged for older listeners in response to certain VOT stimuli.

Post hoc tests were conducted to determine which stimuli evoked prolonged latencies in older adults. No significant latency differences ($P > 0.05$) were seen for the 0, 10 and 20 ms VOT stimuli when the onset of the vowel /a/ follows shortly after the onset of the consonant /b/. However, age-effects were seen when evoked by stimuli with increased

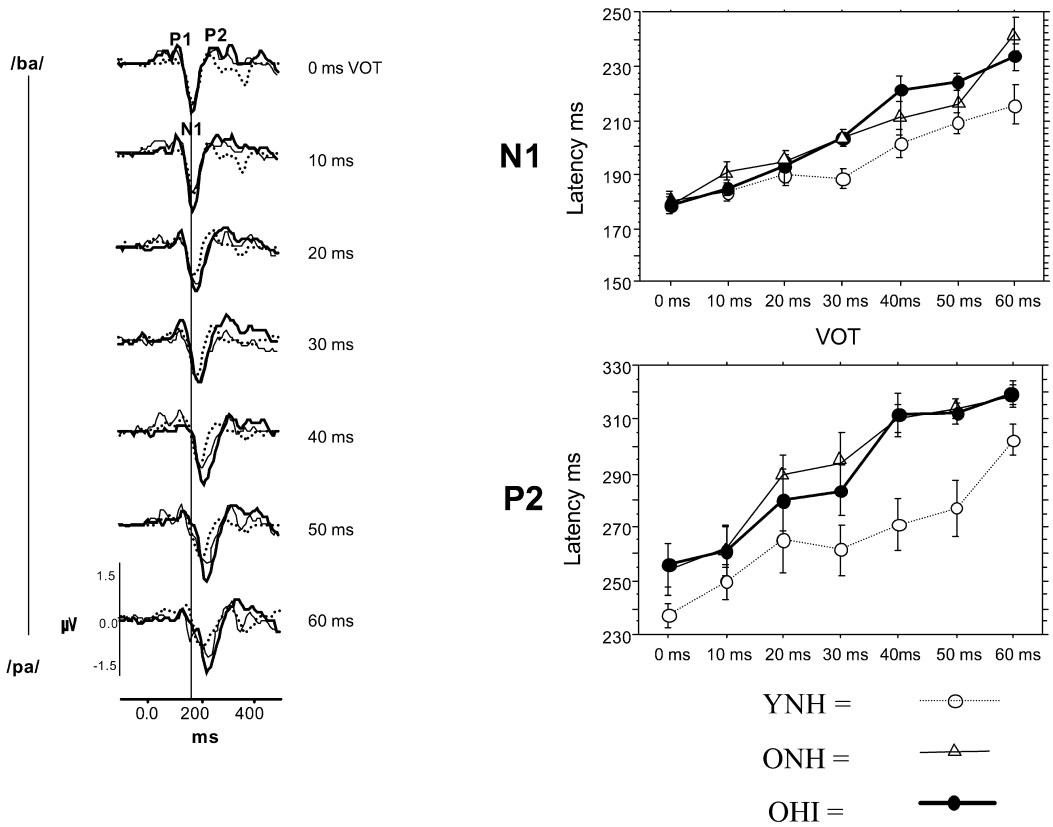

Fig. 3. (left) Group averaged waveforms for younger (YNH) and older normal-hearing listeners (ONH), as well as older adults with hearing loss (OHI) recorded from electrode Cz in response to each VOT stimulus. Peak latency values of N1 and P2 for each group are plotted on the (right).

VOTs (>20 ms VOT). Compared with the younger group, N1 latencies were prolonged for older listeners with hearing loss for the 30 ($P < 0.01$), 40 ($P < 0.05$), 50 ($P < 0.05$) and 60 ms ($P < 0.05$) VOT stimuli. N1 latencies were also delayed for the older group without hearing loss for the 30 ($P < 0.01$) and 60 ms ($P < 0.01$) VOT conditions. Although N1 latencies appear prolonged for older normal-hearing listeners in response to the 40 and 50 ms stimuli, these delays approached but did not reach statistical significance ($P > 0.05$). No significant latency differences were found between the two older groups (0 ($P > 0.05$), 10 ($P > 0.05$), 20 ($P > 0.05$), 30 ($P > 0.05$), 40 ($P > 0.05$), 50 ($P > 0.05$), 60 ms ($P > 0.05$)).

Similar to N1 latencies, P2 latencies increased with each increase in VOT ($F = 40.5$, df = 6, $P < 0.001$). Also, there was a significant main effect for age group ($F = 7.4$, df = 2, $P < 0.01$). Compared with younger listeners, P2 latencies were delayed for the older group with ($P < 0.001$) and without hearing loss ($P < 0.001$); however, P2 latencies for the two older groups were not significantly different ($P > 0.05$). There was no significant group × VOT interaction ($F = 1.04$, df = 12, $P > 0.05$) suggesting that age-related P2 latency delays were evident for all VOT conditions.

3.2.2. Amplitude

There were no significant findings for P1 amplitude (group: $F = 0.63$, df = 2, $P > 0.05$; VOT: $F = 0.25$, df = 6, $P > 0.05$; group × VOT: $F = 1.7$, df = 12, $P > 0.05$) or P2 amplitude (group: $F = 0.39$, df = 2, $P > 0.05$; VOT: $F = 0.56$, df = 6, $P > 0.05$; group × VOT: $F = 0.92$, df = 12, $P > 0.05$). Similarly, there were no main effects for N1 amplitude (group: $F = 2.4$, df = 2, $P > 0.05$; VOT: $F = 1.7$, df = 6, $P > 0.05$). However, there was a significant group × VOT interaction for N1 amplitude ($F = 1.9$, df = 12, $P < 0.05$). According to post hoc tests, compared with the younger normal-hearing group, N1 amplitude was larger for the older group with hearing loss for the 40 ($P < 0.05$), 50 ($P < 0.05$) and 60 ms ($P < 0.05$) VOT stimulus conditions. Increased N1 amplitude appears to be related to age-related hearing loss because amplitude responses were much larger for the older group with hearing loss compared with the normal-hearing older group (40 $P < 0.05$, 50 $P < 0.01$, 60 ms VOT, $P < 0.05$). As shown in Fig. 4, the age-related amplitude and latency differences reported at the Cz electrode site are also evident at multiple electrode sites.

3.2.3. Latency and amplitude summary

To summarize, all 3 groups showed systematic increases in P1, N1 and P2 latency with increases in VOT. In addition, compared to the younger group: (1) N1 responses were prolonged for both older groups in response to stimuli with longer VOT durations; (2) P2 was delayed for both older groups, regardless of stimulus condition and (3) in addition

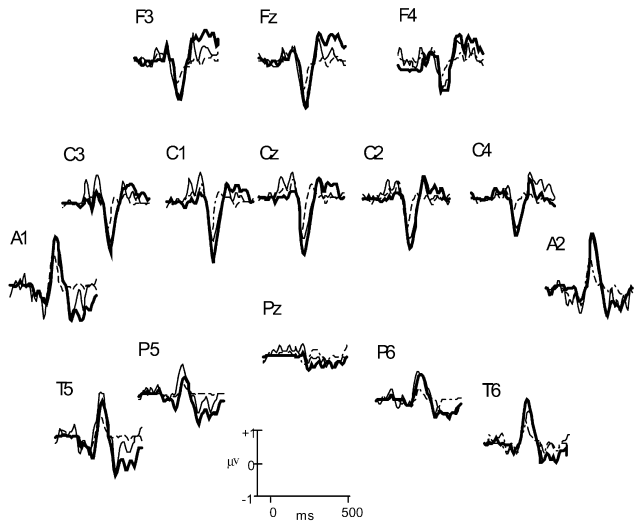

Fig. 4. Prolonged N1 and P2 latencies are seen at multiple electrode sites for both older groups (older normal-hearing, thin solid line; older hearing-impaired, thick solid line) in response to the 40 ms VOT stimulus.

to prolonged latencies, the presence of high-frequency age-related hearing loss resulted in larger N1 amplitudes when elicited by voiceless stimuli.

4. Discussion

4.1. Main findings

The present findings reinforce Strouse et al. (1998) and others (Moore et al., 1992; Fitzgibbons and Gordon-Salant, 1994; Schneider et al., 1994; Snell, 1997; Schneider and Pichora-Fuller, 2001) who report that older adults have more difficulty than younger adults perceiving temporal cues. In this experiment, older listeners (with or without hearing loss) had more difficulty than younger listeners discriminating 10 ms VOT contrasts. The presence of age-related hearing loss appears to compound the problem because older adults with hearing loss performed more poorly than older adults without hearing loss.

Electrophysiological findings suggest that age-related factors affecting excitatory and inhibitory processes might be responsible for some of the perceptual difficulties experienced by older adults because stimuli used during perceptual testing evoked neural response patterns that were different for younger and older adults. Specifically, N1 latencies were prolonged for both older groups in response to stimuli with increased VOT durations and P2 latencies were delayed for both older groups in response to all stimuli. In addition to latency delays, the presence of age-related hearing loss resulted in a significant increase in N1 amplitude in response to voiceless stimuli.

4.2. What do age-related differences in response latency and amplitude suggest about the aging brain?

4.2.1. N1 response – the effects of aging

At present, there are unanswered questions regarding the neural code underlying VOT perception. However, there is common agreement that VOT is partially represented by properties intrinsic to neurons in primary auditory cortex. Single and multiple unit studies in animal and human cortex have shown that temporal cues, such as silent gaps between two non-speech signals or two segments of a VOT speech signal are represented by synchronized responses of neuronal ensembles time-locked to both consonant release and voicing onset (Steinschneider et al., 1994, 1999; Eggermont, 1995, 1999, 2000, 2001; Phillips, 1999). Moreover, the combination of synaptic depression (excitation) and amount of after-hyperpolarization reflects a range of gap intervals (Eggermont, 1999, 2000).

Eggermont (2000) also demonstrated that the neural activation evoked by the initial portion of the stimulus (e.g. initial burst) modifies the ability to generate a response to the second stimulus segment (e.g. second burst) and proposed that forward masking may cause the effect of the initial burst on the second burst. Converging evidence from animal and human studies report prolonged physiological recovery from forward masking in older subjects using non-speech stimuli (Boettcher et al., 1996; Walton et al., 1999; Poth et al., 2001). Therefore, we question if similar forward masking age-effects are occurring here.

For example, as previously mentioned, the N1 response is an onset response. In this study, it is likely that the N1 for short VOTs consists of overlapping responses of two populations of units: one group responding to the onset consonant burst /b/ and the other to the onset of the vowel /a/. N1 latencies for short VOT durations (0 through 20 ms VOT) are similar for each group, likely because the N1 is dominated by the burst of the consonant. Because younger and older listeners exhibit N1 responses that are similar in latency when evoked by the 0 ms VOT stimuli, each group appears to be able to time-lock to the simultaneous onset of the consonant burst and voicing, suggesting that conduction delays are not different for younger and older adults. In other words, there do not appear to be age-related differences in excitation ability when the stimulus does not contain a gap, and the forward masking effect of the consonant burst is similar for all 3 groups. However, at increased VOT durations, N1 latencies are prolonged for both groups of older adults. 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 burst and onset of voicing. Perhaps younger systems recover more quickly than older systems, resulting in earlier N1 latencies for the younger group.

Prolonged N1 latencies in older adults might also reflect age-related synchrony differences to onset of the consonant burst. In other words, older auditory systems might be less

able to synchronize to the initial consonant burst, resulting in smaller neural responses to the onset of the burst but larger responses to the onset of voicing. In contrast, younger systems may be better at locking onto the initial onset of the consonant burst, resulting in a larger response to the burst and a smaller response to the onset of voicing. Fig. 5 shows response waveforms from 3 individuals in response to the 60 ms VOT syllable /pa/. Temporally overlapping responses to the burst and onset of voicing are seen and the onset to the burst is larger in the younger adult and smaller in older adults. While the N1 response is dominated by the burst in the younger individual, longer latency N1 responses are most prominent in the older individuals, thus resulting in N1 age-related latency differences. Even though the initial response is smaller in older adults, the amount of forward masking produced by that noise might be more disadvantageous for older auditory systems.

An alternative interpretation is that age-related refractory issues may be a factor. 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 literature (Papanicolaou et al., 1984) and might explain the N1 latency differences reported here. To answer this question, extended VOT durations (>60 ms VOT) could be used in future experiments to determine if age-related N1 latency differences still exist.

Finally, it is possible that the distribution of individual neuron firings to the onset of the vowel becomes broader with age, but with the same minimum latencies as younger adults. This would result in increased population-response latency for older adults. Thus, the loss in neural synchrony in response to the onset of voicing, shortly after the response to the burst, is more pronounced in older adults. This could result from stronger inhibition, but also stronger synaptic depression and slower recovery there from. Although this explanation could explain prolonged N1 latencies for older groups, it does not explain larger N1 amplitudes in older adults with hearing loss. Therefore, the effects of age-related hearing loss also need to be considered.

4.2.2. N1 response – the effects of age-related hearing loss

Let us assume that the N1 for short VOT durations consist of overlapping responses of two populations of units: one population with high characteristic frequencies (CFs) responds only to the burst and the other population with low CFs responds either to the burst or to the vowel onset, but not to both. Previous studies have shown N1 amplitude to be larger in response to low frequency rather than high-frequency signals (Picton et al., 1978). It is possible that the older population with hearing loss has few units with high enough CFs to respond to the burst (spectrum from 2500 to 4000), and thus most units respond to the vowel (specifically to the lower formants). Therefore, high-frequency hearing loss may act as a low-pass filter that may in turn elicit a larger N1 response. Then again, decreased rather than increased N1 amplitudes have been recorded in young adults with high-frequency hearing loss (Oates et al., 2002) and simulated high-frequency masked hearing loss (Martin et al., 1999); therefore, this line of interpretation does not fully explain the relationship between N1 amplitude and age-related hearing loss. Moreover, N1 amplitudes were larger only in response to stimuli with longer VOT durations; therefore, we question if amplitude differences reported here are related to inhibitory mechanisms regulating post-activation suppression following the onset response to the consonantal burst.

4.2.3. Does age-related hearing loss affect neural inhibition?

Numerous evoked potential studies report age-related impairments affecting neural inhibition (Milbrandt et al., 1996; Willott, 1999). According to Eggermont (2000), post-activation suppression signaling a gap after the onset response to the initial burst may involve feed-forward and feed-back inhibition via slowly or non-adapting neurons. Not only could age-related changes involving inhibitory circuits within primary auditory cortex explain delays to the onset of voicing, this may also explain the amplitude differences seen in older adults with high-frequency hearing loss. Numerous studies report increased response amplitudes among older adults, and these amplitude changes have been attributed to deficits in central inhibition (Pfefferbaum et al., 1979; Kelly-Ballweber and Dobie, 1984; Woods and Clayworth, 1986). Although these aging studies conclude that enhanced amplitudes are related to age-related inhibitory changes, many of the participants in these studies had high-frequency hearing loss. In the present study, increased amplitudes were seen only for the older group with hearing loss. Additionally, Oates et al. (2002) did not report increased N1 amplitudes in young adults with hearing loss. Collectively, these findings suggest that increased neural activity reflects changes in the central auditory system result from the combination of aging and hearing loss, and not necessarily aging or hearing loss independently.

4.2.4. Effects of age and age-related hearing loss on P2

To what degree N1 latency influences P2 latency is unclear because little is known about the P2 response. However, it is commonly accepted that N1 and P2 are distinct events, each reflecting several anatomic sources within auditory cortex and serving different function with respect to central auditory processing (Roth et al., 1976; Knight et al., 1980). Therefore, delayed P2 latencies may reflect age-related changes in the auditory system that are different than those reflected by N1. This notion is supported by the fact that older participants elicited prolonged P2

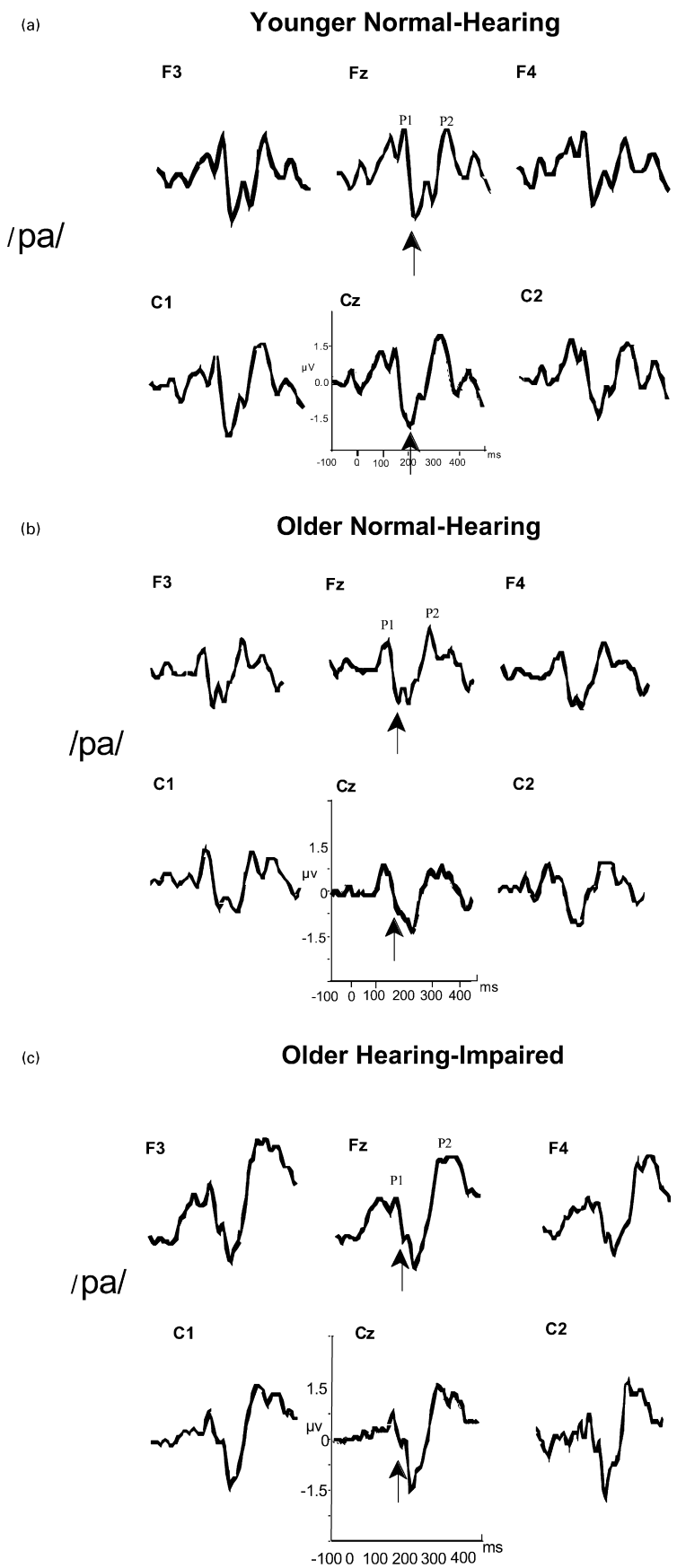

Fig. 5. Representative P1–N1–P2 waveforms recorded from 3 individuals in response to the 60 ms VOT /pa/ stimulus. Compared with both older listeners, responses to the consonant burst (marked by ↑) are larger for the younger listener.

latencies in response to the 0 ms VOT stimuli, when N1 latencies were comparable for both age groups.

Because P2 latencies are delayed in older adults, regardless of stimulus VOT, it could be concluded that prolonged P2 latencies are unrelated to the presence or absence of a stimulus gap. In which case, P2 latencies should be similar for younger and older listeners in response to simple stimuli such as tones. A comprehensive review of the literature suggests this to be the case. Summarizing the effects of aging on P2 is difficult because most studies recorded this obligatory response while simultaneously collecting discriminative responses such as the mismatch negativity (MMN) and P300 (Goodin et al., 1978; Pfefferbaum et al., 1980; Pekkonen et al., 1995a; Polich and Luckritz, 1995; Schroeder et al., 1995; Polich, 1997). Because the MMN and P300 can temporally overlap earlier obligatory responses, and because discriminative responses are often collected at faster stimulus presentation rates that habituate N1 and P2 responses, it is difficult to separate the effects of aging from age-related differences associated with stimulus recording parameters. However, studies that recorded 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). Therefore, prolonged P2 latencies might reflect impaired neural mechanisms responsible for detecting complex acoustic signals such as speech.

4.3. Speech perception and the aging brain

The present study suggests that the temporal properties of neural populations generating N1 and P2 responses change with age. This is not to say that aging only affects the temporal properties within auditory cortex. Numerous studies report anatomical and physiologic changes throughout central auditory system, including a decline in interhemispheric transfer of information, which may disrupt place and temporal coding in the aging system (Hansen and Reske-Nielsen, 1965; Willott et al., 1991; Pekkonen et al., 1995a,b; Bellis et al., 2000; Jerger et al., 2000; Bertoli et al., 2002). Furthermore, abnormal neural processing at the brainstem level has also been reported in populations with impaired speech understanding (Cunningham et al., 2001; King et al., 2002).

Likewise, we do not suggest that age-related deficiencies in speech perception are limited to the detection of VOT. 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; Sinex and McDonald, 1988; 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.

Finally, because the physiological responses were elicited using a passive paradigm that did not draw on memory and cognition, it can be argued that the age-related differences reported in this study are unrelated to cognitive ability. This is not to say that the mere presence of a neural response automatically brings about phonemic perception. Neural patterns representing VOT have been recorded both cortically and subcortically in animals that clearly do not possess language-specific capacities (Kuhl and Miller, 1978; Kuhl and Padden, 1982; Sinnott and Adams, 1987; Sinex et al., 1991; Eggermont, 1995; McGee et al., 1996). Ultimately, perception involves various cognitive processes that go beyond a single neural code. However, the N1–P2 complex does reflect underlying neural timing patterns believed to contribute to perception. For this reason, it is possible that abnormal neural response patterns may be one of many factors contributing to reduced speech understanding in older adults. Moreover, recent laboratory-based experiments have shown that impaired temporal processing can be improved through auditory training (Tremblay et al., 1997, 1998, 2001; Kraus, 2001; King et al., 2002). Therefore, older adults might improve their ability to understand speech through auditory training.

5. Conclusion

In conclusion, aging affects the ability to discriminate time-varying acoustic speech cues. Furthermore, aging affects temporal properties of auditory cortical responses resulting in delayed synchronous firing to the onset of voicing. Together, these brain and behavior measures suggest that some of the speech understanding difficulties expressed by elderly adults may be related to impaired temporal precision in the aging central auditory system. The problem appears to be compounded by the presence of significant high-frequency age-related hearing loss. This might explain why older adults frequently complain that wearing a hearing aid makes speech louder, but does not necessarily improve their ability to understand speech. Hearing aid helps to overcome audibility issues by increasing the intensity of sounds. However, a hearing aid cannot compensate for impaired temporal precision in the aging central auditory system. Because recent laboratory-based experiments have shown that temporal processing can be improved through auditory training (Tremblay et al., 1997, 1998, 2001; Kraus, 2001), older adults who wear hearing aid might improve their ability to understand speech through auditory training.

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