

RESEARCH ARTICLE

Cortical auditory temporal processing abilities in elderly listeners

Aseel Al-Meqbel^{1*}, Catherine McMahon²

¹- Department of Hearing and Speech Sciences, Faculty of Allied Health Sciences, Health Sciences Center, Kuwait University, Kuwait, Kuwait

²- Linguistics Department, Faculty of Human Sciences, Macquarie University, The Hearing Cooperative Research Centre, Sydney, Australia

Received: 1 Sep 2014, Revised: 7 Dec 2014, Accepted: 12 Jan 2015, Published: 25 Mar 2015

Abstract

Background and Aim: This study investigated whether objective temporal processing paradigms including voice-onset-time, speech-in-noise, and amplitude modulated-broad-band noise (AM-BBN) are sensitive to disrupted temporal processing in elderly listeners with normal hearing (age-related-temporal processing deficit).

Methods: We evaluated 15 adults aged 64–80 years using behavioural measures of temporal processing temporal modulation transfer function (TMTF) and speech perception. Cortical auditory evoked potentials (CAEPs) were elicited by three temporal paradigms presented in the sound field at 65 dB SPL: (1) naturally produced stop consonant-vowel (CV) syllables /da/-/ta/ and /ba/-/pa/; (2) speech-in-noise stimuli using the speech sound /da/ with varying signal-to-noise ratios (SNRs); and (3) 16 Hz amplitude-modulated (AM) BBN presented in two conditions: (i) alone (representing a temporally modulated stimulus) and (ii) following an unmodulated BBN (representing a temporal change in the stimulus) using four modulation depths.

Results: Findings demonstrated a statistically

significant mean difference in n1 latency ($p < 0.05$) between normally hearing elderly and young adult listeners in all paradigms. Compared with young adult participants, n1 latency of the CAEP was always prolonged for elderly participants.

Conclusion: The three developed temporal processing paradigms are sensitive to disrupted temporal processing in elderly participants, and n1 latency may serve as a reliable objective measure of the efficiency of auditory temporal processing. The aging process affects temporal representations of the acoustic stimulus and reduces the ability to detect temporal cues, evidenced by abnormal n1 latency.

Keywords: Temporal processing; voice-onset-time; speech-in-noise; amplitude-modulated; n1 latency; elderly

Introduction

Impaired speech perception is one of the major consequences of the aging process [1]. Both peripheral hearing loss and disruptions in auditory temporal processing can contribute to the deterioration of speech perception in elderly listeners with and without hearing loss [2,3]. That is, aging adversely affects the ability to process temporal cues of speech and non-speech stimuli [4,5,6], more specifically affecting the neural synchrony [1,7]. Results from studies of age-related changes in speech processing have

*Corresponding author: Department of Hearing and Speech Sciences, Faculty of Allied Health Sciences, Health Sciences Center, Kuwait University, Sulaibikat, 90805, Kuwait. Tel: +965249833412, E-mail: aseel.m@hsc.edu.kw

shown that many elderly people may have substantial limitations in their ability to process timing of the acoustical signal especially under acoustically complex conditions such as a noisy environment [8,9]. In aged listeners, including those with normal hearing, degraded temporal processing has been directly linked to speech perception difficulties [6,10].

Psychoacoustically, numerous studies have demonstrated an impairment of auditory temporal processing in healthy elderly individuals with or without hearing loss using several different temporal processing paradigms, such as gap detection (GD), which measures a listener's ability to detect brief gaps in noise [11]. Elderly listeners have been found to have more difficulties than younger adult listeners in detecting short gaps within noise, with researchers reporting larger GD thresholds for elderly listeners than young listeners [2,4,5,10,12,13,14]. Furthermore, age-related difference in GD appears to be independent of peripheral hearing loss because performance in GD is not correlated with pure tone hearing thresholds [9,15].

More importantly it should be noted that sensorineural hearing loss (SNHL) is a well-documented consequence of the aging process [16,17]. As an independent factor, SNHL influences several measures of auditory temporal processing, including GD [11,18] and amplitude modulation [19]. To minimize and control the SNHL factor, some investigators have studied GD performance in elderly listeners with normal audiometric thresholds. For example, using participants with normal hearing, He et al. [20] found that elderly listeners had larger GD thresholds than young listeners. Other studies [2,5,9,13] have obtained similar findings.

Another psychoacoustic paradigm for measuring temporal processing is the amplitude modulation detection test [21], which evaluates the listener's ability to detect periodic fluctuations in the envelope of a noise signal that has sinusoidal amplitude modulated at various rates. For this temporal processing paradigm, listeners with better temporal

processing are able to detect smaller depth of amplitude modulation. Takahashi and Bacon evaluated the temporal processing ability of elderly listeners in three decades age groups (50s, 60s, and 70s) and compared it with that of young normal-hearing listeners (21-33years) using amplitude modulation frequencies ranged from 2 to 1024 Hz. Generally, the elderly listeners were observed to be less sensitive to amplitude modulation than the younger [21]. The 70s age group had the poorest AM thresholds (-12dB) compared with the younger (-27.3dB) and the other two older groups, 50s (-18.0dB) and 60s (-16.0dB). With greater age, sensitivity to detect amplitude modulation decreased, thus suggesting a gradual deterioration of temporal processing with the aging process [21].

Moreover, Strouse et al. [2] found that older adults have more difficulty than younger in identifying and discriminating short voice-onset-time (VOT) durations along a /ba-/pa/ continuum. The authors suggested that older adult listeners are at a disadvantage when trying to perceive temporal changes in the acoustic waveforms that compose conversational speech. Evidence from the above psychoacoustic studies demonstrates a deficit in temporal processing ability in elderly listeners, with such deficits not necessarily associated with peripheral hearing loss. However, one disadvantage of using behavioural psychoacoustic measures is that they may be affected by participants' attention level, concentration, and motivation to perform the task, as well as the response criteria used [22,23]. In order to reduce and eliminate the contribution of these confounding variables, some investigators have used electrophysiological measures such as cortical auditory evoked potential (CAEP) to evaluate and compare temporal processing in elderly and young participants. In particular, Tremblay et al. [24,25] evaluated temporal processing using the VOT (/ba-/pa/) continuum from 0 ms to 60 ms in 10 normally hearing young adults (19-32 years) and 10 older adults (61-79 years). They found that elderly adults had more difficulty than younger adults in discriminating short

VOTs along a /ba-/pa/ continuum, and that n1 latencies for the older group were delayed. At 0 ms stimulus both younger and older groups had n1 peaks with similar latency; that is, there were no differences when the speech token did not contain a gap of silence. On the other hand, n1 latencies were delayed for the older group at increased VOT duration. As a result, Tremblay et al. suggested that the auditory system in the aged population is 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. They suggested that the prolonged n1 latencies of the CAEP in elderly individuals might indicate a disrupted neural synchrony to the onset of the speech stimuli and thereby account for some of the perceptual difficulties observed in the elderly [24]. In other words, the aging process changes the temporal properties of the auditory cortical responses, resulting in delayed synchrony firing to the onset of voicing. Those studies by Tremblay et al. supported the hypothesis that elderly listeners with and without hearing loss have temporal processing disruptions and have more difficulty processing time-varying cues [25]. Bearing in mind these electrophysiological results using a single temporal processing paradigm such as VOT, the aim of this study was to use a variety of temporal processing paradigms to provide greater information regarding auditory temporal processing ability at the cortical level in elderly listeners with normal peripheral hearing.

We hypothesized that auditory temporal processing could be impaired in normally hearing elderly listeners due to the aging process. In other words, aging auditory systems could have more difficulty processing temporal cues, which might lead to a delay in n1 latency in older adult listeners compared with younger adult listeners in the absence of any peripheral hearing loss. We further hypothesized that the three developed temporal processing paradigms would be sensitive to such impairment.

Methods

Fifteen elderly participants (7 males, 8 females)

aged 64-80 years (mean 70.6, SD=4.6) with no history of neurological, otological or dementia problems were recruited. All had pure-tone air conduction thresholds ≤ 25 dBHL at octave frequencies from 250Hz to 8kHz with type A tympanogram (suggestive of normal middle ear function), and all reported no hearing difficulty. Air conduction and bone conduction pure-tone thresholds were determined using a calibrated clinical audiometer (AC33 Interacoustics two-channel, Denmark) using modified version of Hughson and Westlake procedure [26].

Tympanometry used a calibrated immittance meter (GSI-Tymp star V2, USA, calibrated as per ANSI, 1987). Tympanograms were obtained for 226 Hz probe tone.

To develop the stimuli for this test, two sounds were generated: un-modulated broadband noise (BBN) and amplitude modulated BBN, of 500 ms duration with a rise/fall (ramp) of 20ms. The stimuli were generated using a 16-bit digital-to-analogue converter with a sampling frequency of 44.1 kHz and low pass filtered with a cut-off frequency of 20 kHz. The modulated BBN stimuli were derived by multiplying the BBN by a dc-shifted sine wave. Modulation depth of the AMBBN (FM 16 Hz) stimuli was controlled by varying the amplitude of the modulating sine wave [27,28].

Amplitude modulation detection threshold at low modulation rate (FM 16Hz) was obtained using an adaptive two down one up, forced choice procedure (2I-2AFC) that estimates modulation depth necessary for 70.7% correct detection [29]. The participants' task was to identify the interval containing the modulation. No feedback was given after each trial. The step size and thresholds of modulation were based on the modulation depth in decibels ($20 \log m$, where m refers to depth of modulation). The step size of modulation was initially 4 dB and reduced to 2 dB after two reversals. The mean of the last three reversals in a block of 14 were taken as threshold. The poorest detection threshold that could be measured was 0 dB, which corresponded to an AM of 1 (100% modulation depth); the more negative the value of $20 \log m$, the better the detection threshold.

Stimuli were played in a computer (a Toshiba PC); the participant received the output of the stimuli that were calibrated using Bruel and Kjaer SLM type 2250, microphone number 419 presented at 65 dB SPL sound field.

The consonant-nucleus-consonant (CNC) word test was used to assess open-set speech perception ability. For present purposes, lists 1 and 2 of the 12 possible CNC lists were used, with taped stimuli of 50 monosyllabic words in each list. The percentage of correct phonemes score was used to evaluate the open-set speech perception ability of the older adult participants. Four naturally produced stop consonant-vowel-syllables, /da/-/ta/ and /ba/-/pa/, were recorded by an Australian native English speaking female. Speech stimuli were recorded using an AKG C535 condenser microphone connected to a Mackie sound mixer, with the microphone positioned 150 mm in front and at 45 degrees to the speaker's mouth. The mixer output was connected via an M-Audio Delta 66 USB sound device to a Windows computer running Cool Edit audio recording software and captured at 44.1 kHz 16 bit wave format. All speech stimuli were collected in a single session to maintain consistency of voice quality.

After speech stimuli were selected and recorded, they were modified using Cool Edit 2000 software. All speech stimuli of 200 ms duration were ramped with 20 ms rise and fall time to prevent any audible click arising from the rapid onset or offset of the waveform. The inter-stimulus interval (ISI), calculated from the onset of the preceding stimulus to the onset of the next stimulus was 1207 ms, as it has been shown that a slower stimulation rate results in more robust CAEP waveforms in immature auditory nervous systems [30].

Varying signal-to-noise ratios (SNR) with the speech stimulus /da/ were developed to measure speech-in-noise (SIN) ability. The speech stimulus /da/ was naturally recorded by an Australian female speaker who was chosen because of her accent clarity. The noise levels were changed to produce different SNRs. After a speech stimulus was selected and recorded, the BBN of 600 ms was generated using Praat

software, which changed the SNR using Matlab software with respect to the 65 dB SPL /da/ sound and then combined them to create a /da/ embedded in different noise levels. Noise levels were 45, 60, 65, 70, 75 dB SPL.

These noise levels were chosen to create five SNRs (Quiet (+20dB), +5 dB, 0dB, -5dB, -10dB and -20dB). The ISI, calculated from the onset of the preceding stimulus to the onset of the next stimulus was 1667ms.

Two stimuli were used: an un-modulated BBN of 600ms and amplitude modulated broadband noise of 300ms. The modulated frequency was 16 Hz, and overall duration was 900ms with 20ms rise and fall time. The stimuli were generated using a 16-bit digital-to-analogue converter with a sampling frequency of 44.1 kHz, and low pass filtered with a cut-off frequency of 20 kHz. The depth of the modulation was controlled by varying the amplitude of the modulating sine wave. The ISI, calculated from the onset of the preceding stimulus to the onset of the next stimulus was 1307 ms for the first condition and 1907ms for the second condition.

All stimuli used in these procedures were presented at 65 dB SPL (as measured at the participant's head), which approximates normal conversational level. It was confirmed with each participant that this level was at a loud but comfortable listening level. Presentation was via a loudspeaker speaker placed 1 meter from the participant's seat at 0 azimuth. The participants were asked to focus and keep watching the DVD and to reduce the head movement.

Participants sat on a comfortable chair in a quiet room at Macquarie University Electrophysiology Clinic and watched a DVD of their own choice. The volume was silenced and subtitles were activated to ensure that participants would be engaged with the movie and pay no attention to the stimuli. All participants were instructed to be relaxed, stay awake and pay no attention to the sounds being presented. The noise presentation was simultaneous.

A NeuroScan and 32-channel NuAmps evoked potential system was used for evoked potential

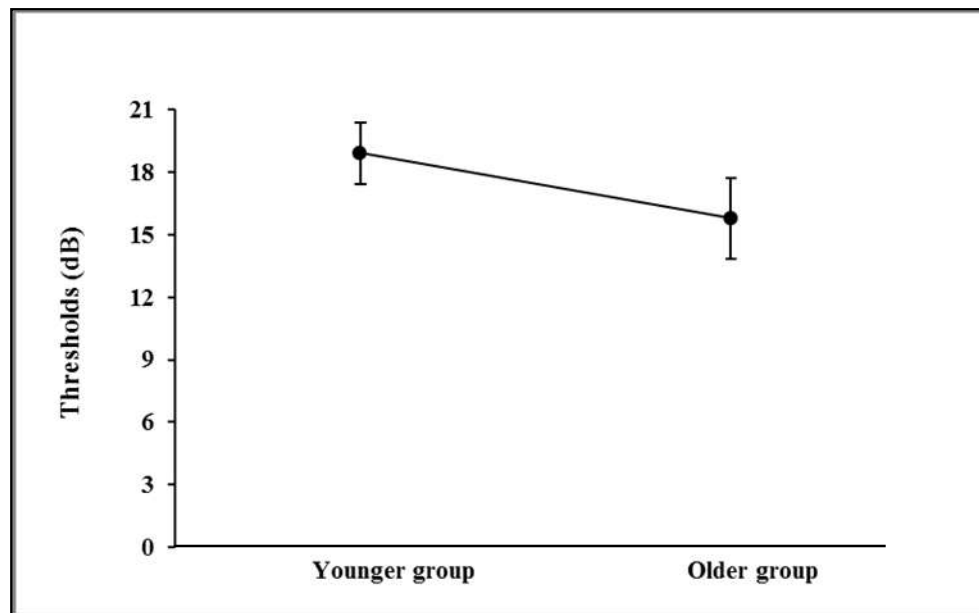


Fig. 1. Mean (standard deviation) of the behavioural temporal modulation transfer function (TMTF) using 16 Hz in younger group versus older group.

recording. All sounds were presented using Neuroscan STIM 2 stimulus presentation system.

Evoked potentials were recorded in continuous mode (gain 500, filter 0.1-100 Hz) and converted using analogue-to-digital sampling rate of 1000 Hz using scan (version 4.3) via gold electrodes placed at C3, C4, Cz with reference electrode A2 on the right mastoid bone and ground on the contralateral ear on the mastoid bone.

Elderly adults participated in two 2-hour recording sessions, including the electrode application and CAEP recording. None of the participants showed signs of fatigue during the testing. All sound levels were calibrated using Bruel and Kjaer SLM type 2250, microphone number 419.

EEG files with a time window of -100 to 500 ms were obtained from the continuous file. Any responses on scalp electrodes exceeding ± 50 μ V were rejected. Prior to averaging, EEG files were baseline corrected using a pre-stimulus period (-100 ms). Averaging was digitally band pass filtered from 1 to 30 Hz (zero shift, 12

dB/octave). For each participant, the individual grand average waveform was computed, visually identified, and subjected to suitable statistical analyses using SPSS 18 to investigate the aims of the current study. The smaller groups of participants necessitated the use of non-parametric analysis.

The Wilcoxon Signed-Rank test was performed to compare the mean n1 latency of the stop CV voiced /da/ vs. voiceless /ta/, and between /ba/ vs. /pa/ in older adult participants. The Friedman test was used to compare the mean n1 latency of all SNR conditions in elderly listeners. Mann-Whitney U test was performed to compare the mean n1 latency for each condition separately between younger and older adults.

Results

Mean speech perception score in quiet was 95% (SD=5.8) and the mean of the behavioural temporal modulation transfer function (TMTF) in dB was -15.78 dB (SD=1.5), as shown in Fig. 1.

Mean n1 latency for /da/ was 120ms

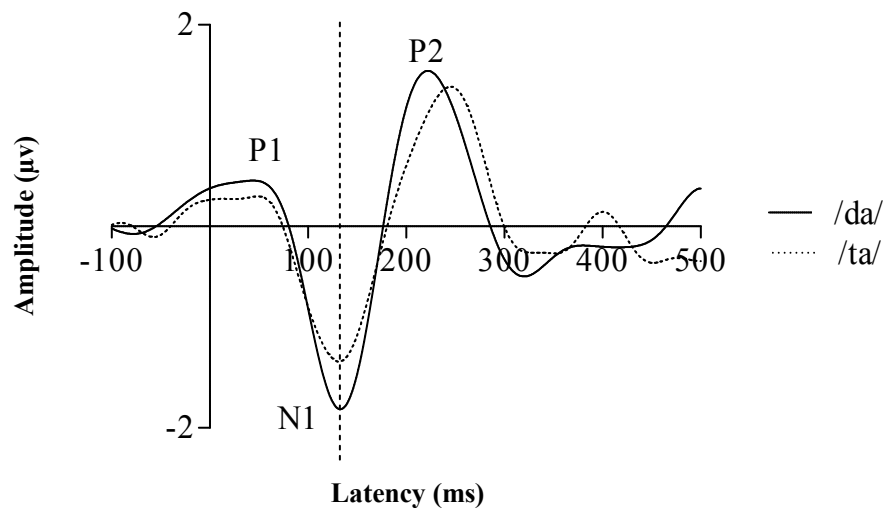


Fig. 2. Grand averaged waveforms for older listeners recorded from electrode Cz in responses to /da/ vs. /ta/ speech stimuli.

(SD=2.83ms), /ta/ was 122ms (SD=3.26ms), /ba/ was 119ms (SD=2.31ms) and /pa/ was 120ms (SD=3.06ms). Results showed no significant mean difference in n1 latency between either /da/ and /ta/ ($Z=-2.409$, $p>0.05$) or /ba/ and /pa/ ($Z = -2.308$, $p>0.05$), as shown in Fig. 2. A Mann-Whitney U test was performed to compare the difference on each speech sound separately between the groups of elderly and young adults. Results showed a statistically significant difference in n1 latency between the two groups for speech sound /da/ ($U=1$, $Z=-3.887$, $p<0.05$), /ta/ ($U=1.5$, $Z=-3.630$, $p<0.05$), /ba/ ($U=1$, $Z=-3.851$, $p<0.05$) and /pa/ ($U=1.5$, $Z=-3.686$, $p<0.05$). Hence, compared with young adult listeners, n1 latencies for all speech tokens were significantly prolonged in elderly listeners, as shown in Fig. 3.

Results indicated a statistically significant difference in n1 latency of the CAEP across the SNR conditions (+20 dB, +5dB, 0dB, -5dB, -10dB SNR) (Chi square, [4] = 18.427, $p<0.05$). Interestingly, with the most difficult listening situation, no observable response was measured at -10dB SNR for all elderly listeners.

Further analysis was conducted using a Mann-

Whitney U test to compare the mean n1 latency for each condition separately between young and old adults. Results showed a statistically significant mean difference in each SNR condition tested +20dB ($U=8$, $Z=-3.317$, $p<0.05$), +5 dB ($U=9$, $Z=-3.230$, $p<0.05$), 0dB ($U=10.5$, $Z=-3.141$, $p<0.05$), -5dB ($U=8.5$, $Z=-3.288$, $p<0.05$). Hence, compared with young adult listeners, n1 latencies for all SNR conditions were significantly prolonged in elderly listeners, as shown in Figures 4A and 4B.

Two conditions were evaluated to measure the temporal processing ability of older versus younger adults with normal hearing: (i) 300 ms AM-BBN and (ii) 300 ms AM-BBN following a 600 ms BBN. Results showed a statistically significant mean difference in each condition tested: 100% AM ($U=2$, $Z=-3.743$, $p<0.05$), 50% AM ($U=4$, $Z=-3.602$, $p<0.05$), 25% AM ($U=1$, $Z=-3.882$, $p<0.05$) Hence, compared with young adult listeners, n1 latencies for all depths were significantly prolonged in elderly listeners, as shown in figures 5A and 6.

For the responses measured to the AM-BBN following a BBN, a Mann-Whitney U test was used to compare the mean n1 latency for each

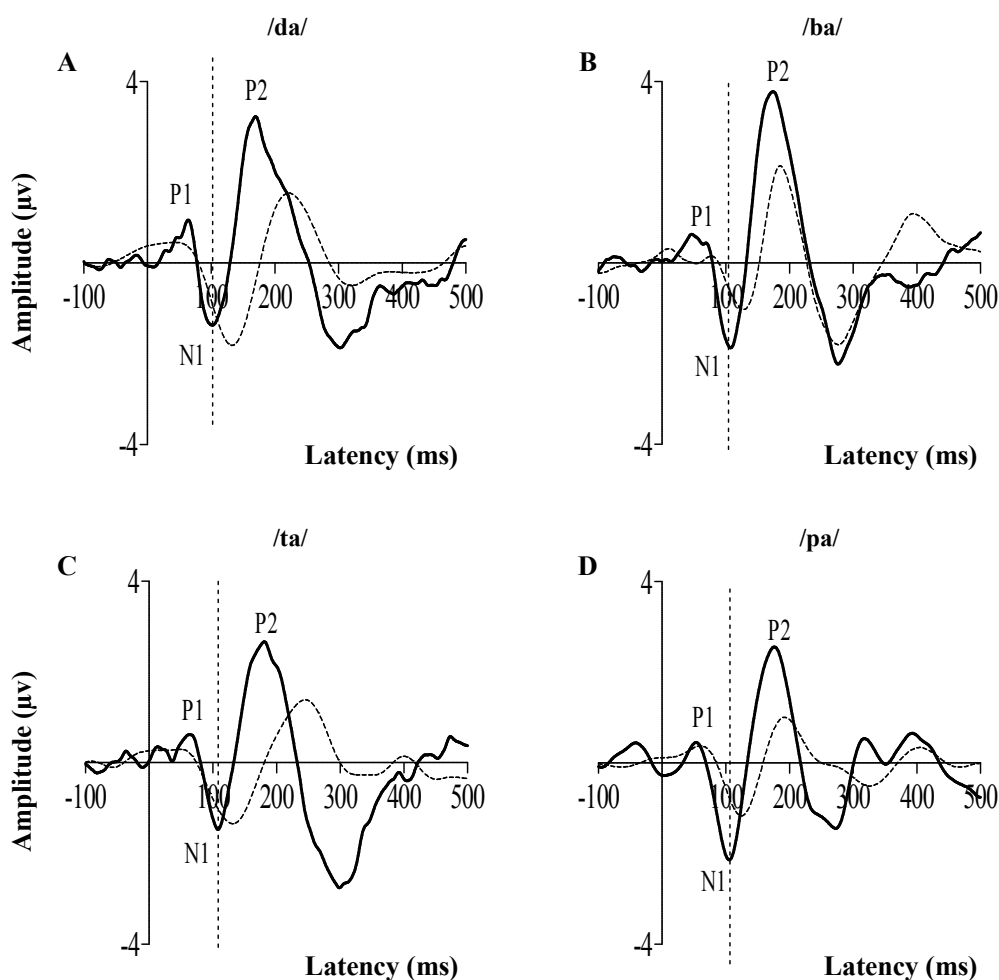


Fig. 3. Grand averaged waveforms for younger (solid line) and older (dashed line) normal-hearing listeners recorded from electrode Cz in response to A) /da/; B) /ba/; C) /ta/; and D) /pa/ speech stimuli.

condition separately between young and old adults. Results showed a statistically significant ($U=15$, $Z=-2.818$, $p<0.05$), 25% AM ($U=6$, $Z=-3.455$, $p<0.05$). Hence, compared with young adult listeners, n1 latencies for all depths were significantly prolonged in elderly listeners, as shown in Fig. 5B.

Discussion

The aim of the current study was to examine whether the three temporal processing paradigms were sensitive to temporal processing disruptions in elderly listeners, using n1 latency of the CAEP. Results demonstrated that n1 latency in elderly listeners compared with

mean difference in each condition tested: 100% AM ($U=14$, $Z=-2.891$, $p<0.05$), 50% AM young adult listeners was prolonged when evoked by all paradigms, demonstrating abnormal timing processing for acoustic stimuli that vary in time cues.

From these results, it is clear that elderly participants demonstrated abnormal timing when perceiving a VOT of short duration, such as /da/, since there was no significant difference in n1 latency between /da/-/ta/ and /ba/-/pa/ stop CV syllables. These results suggest that elderly people have a compromised temporal processing ability to differentiate between two stop CV syllables that are different in VOT,

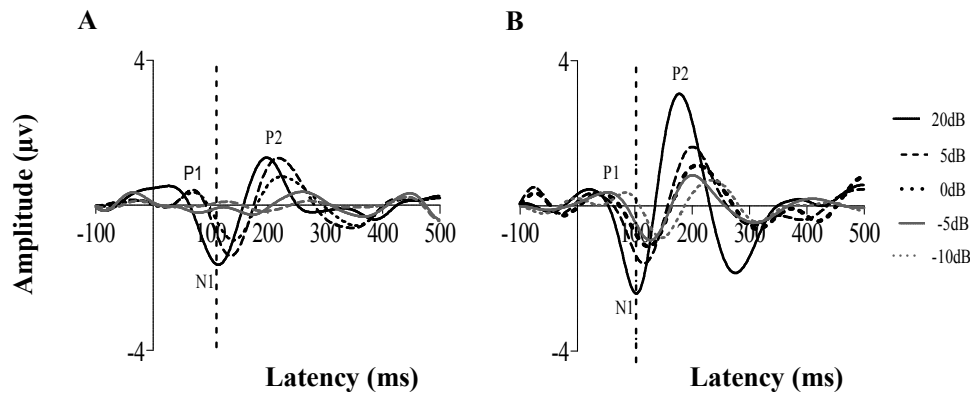


Fig. 4. Grand averaged waveforms for A) older and B) younger normal-hearing listeners recorded from electrode Cz in responses to five SNRs conditions.

when compared with normal-hearing young adults who showed a significant mean difference between the stop CV syllables /da-/ta/ and /ba-/pa/. We suggest, therefore, that elderly people require longer VOTs to differentiate between two speech stimuli with varying timing cues such as VOT. This finding might also explain why some elderly people have more difficulty in discriminating speech sounds, and frequently complain that they can hear but they cannot understand what they hear. Our results are in line with those of Tremblay et al. (24,25), who found that elderly people with and without hearing loss had more difficulty discriminating 10ms VOT contrast, and had prolonged n1 latency in response to /ba/ and pa/. Tremblay et al. suggested that aging auditory systems are less able to time lock to the onset of voicing when there is a gap between the burst onset time and voicing time, compared with younger auditory systems, and this may be due to the ability of younger auditory systems to recover more quickly than older systems, resulting in earlier latencies of n1 for the younger listeners in their study.

Previous research has shown that older listeners have more difficulty than younger listeners in understanding speech, particularly in noise [1]. Part of the reason for this increased difficulty, despite normal pure-tones thresholds, may be related to poor temporal resolution of the speech signal in the presence of noise [31], and the fact

that the aging auditory system becomes slower and more asynchronous in processing time [7]. Individuals must also possess adequate temporal resolution in order to effectively process the amplitude fluctuations of the speech signal in noise, thus an inability to process such temporal fluctuations could underlie older listeners' difficulty in background noise [32]. Such changes in temporal processing associated with an aging auditory system may underlie the coding of the durational and transitional time-varying cues of the input signal, as well as the extraction of the speech signal from noise. Moreover, behavioural studies in both normally hearing young and older listeners demonstrated that, as the SNR decreases, both groups have difficulties understanding speech, although older adults enter the most difficult zone sooner than younger adults [31]. This difference between young and old adults could be explained electrophysiologically from our results, in that both groups showed delay in n1 latencies as noise level increased; however, elderly patients experienced significant delays in n1 latencies at all SNRs, suggesting additional timing delays to perceive the speech signal /da/ in the presence of noise. In addition, with the most difficult listening condition (-10dB SNR), no elderly participants showed an identifiable n1 component compared with young adults, who showed no identifiable n1 latency at -20dB SNR. This difference indicates that

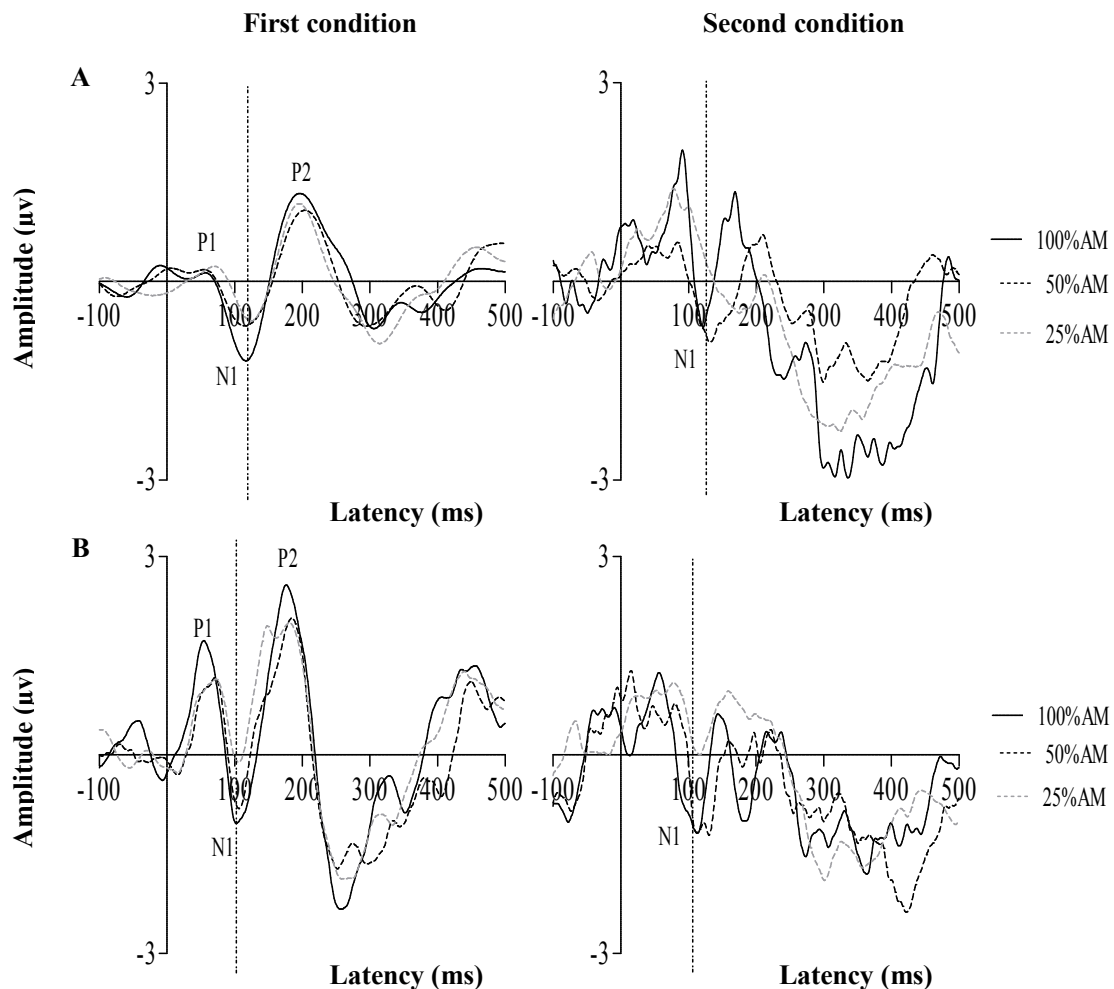


Fig. 5. Grand averaged waveforms for A) older normal-hearing listeners, B) younger normal-hearing listeners recorded from electrode Cz in response to three levels of amplitude modulation for both conditions. AM; amplitude modulation.

elderly listeners might require higher SNR to understand speech in background noise and thus elderly populations have SNR problems. Generally, the elderly listeners were observed to be less sensitive to amplitude modulation at slow amplitude modulation (16 Hz) than younger listeners; that is, older adults with normal hearing demonstrated less sensitivity to detecting the behavioural TMTF than did younger adults with normal hearing. Detecting information from slow amplitude modulation is a crucial aspect of speech perception, as amplitude modulation features in speech are

known to provide important cues for discrimination of specific phonemic features, such as syllable, voicing and consonant identification [33].

Our results show that n1 latencies at all modulation depths were delayed in older adults compared with young adults, suggesting that older auditory systems may require a longer period of time than younger systems to perceive and detect the amplitude modulation within the stimulus. The behavioural TMTF and objective amplitude-modulation detection results might suggest disrupted temporal processing in older

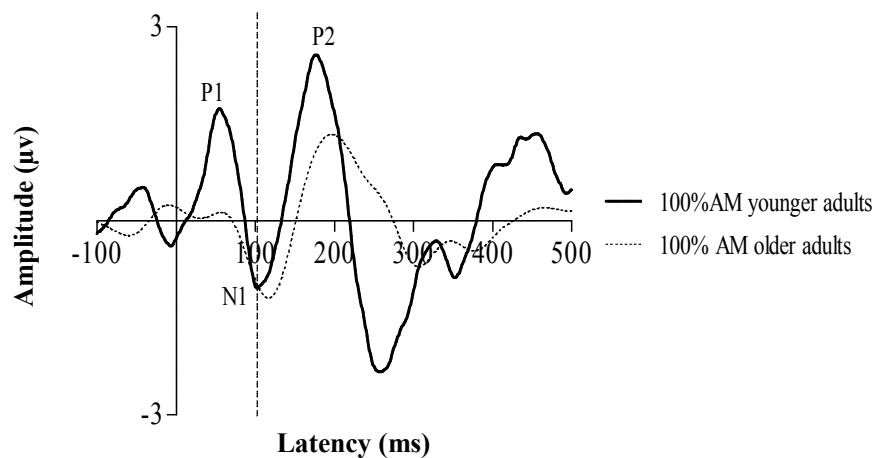


Fig. 6. An example of differences between older and younger normal-hearing listeners recorded from electrode Cz in response to 100% AM; amplitude modulation.

adult listeners, despite a normal peripheral auditory system, given that the n1 latency of the CAEP is more related and sensitive to the temporal cues of the acoustics than other CAEP components [34,35,36], and the TMTF is a measure of temporal processing [37,38]. Consequently, this disruption might account for some of the perceptual difficulties commonly experienced by elderly people. This assumption is based on previous psychoacoustic studies showing that the severe perceptual difficulties experienced by populations with disrupted temporal processing, such as auditory neuropathy spectrum disorder (ANSD), may be due to the impaired ability to follow and detect amplitude changes in speech sounds, which smears temporal representation of the speech signal [37,38].

Models of aging in the rat auditory cortex have shown that temporal processing deterioration occurs with increasing age [39]. Single unit recordings using fast and slow frequency-modulation (FM) sweeps stimuli in 19 rats (young and aged) showed that the majority of cortical cells in young rats responded more strongly to the fast and medium speeds, with relatively fewer cortical cells responding to

slower speeds. On the other hand, the majority of cortical cells recorded from the aged rats responded best to slow speeds while few cortical cells preferred the faster speed. This animal model demonstrates age-related differences in cortical temporal processing speed for FM stimuli, suggesting a reduced ability to process formant transition efficiently, since the frequencies may be changing too rapidly for the auditory system to accurately encode. In addition, the researchers reported that the reduced ability might be related to general slowing of temporal processing in the auditory system [39].

Animal and human studies can provide evidence of how aging reduces the ability to utilize timing cues in acoustic stimuli. Using three different temporal processing paradigms, we have also provided evidence that aging alters the timing processing of time-varying stimuli which is unrelated to the degree of the hearing loss. A disruption in processing timing cues such as these may be an important cause of deteriorated speech perception in the elderly population.

Taken together, the general delay in n1 latencies as a function of the three temporal processing paradigms in our elderly listeners suggests age-

related slowing and delays in synchronous firing among neural populations generating the n1 response. Thus, older auditory systems appear to be less able to time lock to the onset of a given stimulus. Therefore, our results support the hypothesis that aging might affect synchronized neural activity underlying the perception of critical time-varying speech cues and may partially explain some of the difficulties older listeners experience in understanding speech.

Conclusion

Results suggest that the three temporal processing paradigms used in this study are sensitive in evaluating the temporal processing and n1 latency of the CAEP, which could be used as an objective measure of temporal processing ability in individuals who are suspected to have a temporal processing disorder and are not able to provide reliable behavioural responses. In addition, our findings support the hypothesis that temporal processing deteriorates as a result of the aging process, as evidenced by age-related changes in n1 latency, although normal peripheral hearing is present. This study supports earlier research findings that older adults have more difficulty than younger adults in perceiving temporal cues; specifically, n1 latencies were prolonged for older listeners in response to all stimuli. Delayed n1 latencies in the elderly suggest age-related delays in neural synchronous activity. Although it is difficult to measure the temporal processing in elderly individuals behaviourally due to many factors, the objective measures of temporal processing can be promising.

Acknowledgment: The authors acknowledge the financial support of the Hearing Cooperative Research Centre (CRC), established and supported under the CRC Program, an Australian Government Initiative.

REFERENCES

1. Frisina DR, Frisina RD. Speech recognition in noise and presbycusis: relations to possible neural mechanisms. *Hear Res.* 1997;106(1-2):95-104.
2. Strouse A, Ashmead DH, Ohde RN, Grantham DW. Temporal processing in the aging auditory system. *J Acoust Soc Am.* 1998;104(4):2385-99.
3. Schneider BA, Daneman M, Murphy DR. Speech comprehension difficulties in older adults: cognitive slowing or age-related changes in hearing? *Psychol Aging.* 2005;20(2):261-71.
4. Moore BC, Peters RW, Glasberg BR. Detection of temporal gaps in sinusoids by elderly subjects with and without hearing loss. *J Acoust Soc Am.* 1992;92(4 Pt 1):1923-32.
5. Schneider BA, Pichora-Fuller MK, Kowalchuk D, Lamb M. Gap detection and the precedence effect in young and old adults. *J Acoust Soc Am.* 1994;95(2):980-91.
6. Fitzgibbons PJ, Gordon-Salant S. Aging and temporal discrimination in auditory sequences. *J Acoust Soc Am.* 2001;109(6):2955-63.
7. Schneider BA, Pichora-Fuller K. Age-related changes in temporal processing: implications for speech perception. *Semin Hear.* 2001;22(3):227-40.
8. Fitzgibbons PJ, Gordon-Salant S. Auditory temporal processing in elderly listeners. *J Am Acad Audiol.* 1996;7(3):183-9.
9. Schneider BA, Hamstra SJ. Gap detection thresholds as a function of tonal duration for younger and older listeners. *J Acoust Soc Am.* 1999;106(1):371-80.
10. Snell KB, Frisina DR. Relationships among age-related differences in gap detection and word recognition. *J Acoust Soc Am.* 2000;107(3):1615-26.
11. Fitzgibbons PJ, Wightman FL. Gap detection in normal and hearing-impaired listeners. *J Acoust Soc Am.* 1982;72(3):761-5.
12. Lutman ME. Degradations in frequency and temporal resolution with age and their impact on speech identification. *Acta Otolaryngol Suppl.* 1990;476:120-5; discussion 126.
13. Snell KB. Age-related changes in temporal gap detection. *J Acoust Soc Am.* 1997;101(4):2214-20.
14. Pichora-Fuller MK, Schneider BA, Benson NJ, Hamstra SJ, Storzer E. Effects of age on detection of gaps in speech and nonspeech markers varying in duration and spectral symmetry. *J Acoust Soc Am.* 2006;119(2):1143-55.
15. Heinrich A, Schneider B. Age-related changes in within- and between-channel gap detection using sinusoidal stimuli. *J Acoust Soc Am.* 2006;119(4):2316-26.
16. Corso JF. Sensory processes and age effects in normal adults. *J Gerontol.* 1971;26(1):90-105.
17. Pearson JD, Morrell CH, Gordon-Salant S, Brant LJ, Metter EJ, Klein LL, et al. Gender differences in a longitudinal study of age-associated hearing loss. *J Acoust Soc Am.* 1995;97(2):1196-205.
18. Irwin RJ, Hinchcliff LK, Kemp S. Temporal acuity in normal and hearing-impaired listeners. *Audiology.* 1981;20(3):234-43.
19. Bacon SP, Viemeister NF. Temporal modulation transfer functions in normal-hearing and hearing-impaired listeners. *Audiology.* 1985;24(2):117-34.
20. He NJ, Horwitz AR, Dubno JR, Mills JH. Psychometric functions for gap detection in noise measured from young and aged subjects. *J Acoust Soc Am.* 1999;106(2):966-78.
21. Takahashi GA, Bacon SP. Modulation detection, modulation masking, and speech understanding in noise in the elderly. *J Speech Hear Res.* 1992;35(6):1410-21.

22. Wightman F, Allen P, Dolan T, Kistler D, Jamieson D. Temporal resolution in children. *Child Dev.* 1989;60(3):611-24.
23. Green DM. Stimulus selection in adaptive psychophysical procedures. *J Acoust Soc Am.* 1990;87(6):2662-74.
24. Tremblay KL, Piskosz M, Souza P. Aging alters the neural representation of speech cues. *Neuroreport.* 2002;13(15):1865-70.
25. Tremblay KL, Piskosz M, Souza P. Effects of age and age-related hearing loss on the neural representation of speech cues. *Clin Neurophysiol.* 2003;114(7):1332-43.
26. Cahart R, Jerger J. Preferred method for clinical determination of pure tone thresholds. *J Speech Hear Disord.* 1959;24:330-45.
27. Viemeister NF. Temporal modulation transfer functions based upon modulation thresholds. *J Acoust Soc Am.* 1979;66:1364-80.
28. Lorenzi C, Dumont A, Füllgrabe C. Use of temporal envelope cues by children with developmental dyslexia. *J Speech Lang Hear Res.* 2000;43(6):1367-79.
29. Levitt H. Transformed up-down methods in psychoacoustics. *J Acoust Soc Am.* 1970;49(2):Suppl 2:467-77.
30. Gilley PM, Sharma A, Dorman M, Martin K. Developmental changes in refractoriness of the cortical auditory evoked potential. *Clin Neurophysiol.* 2005;116(3):648-57.
31. Festen JM, Plomp R. Effects of fluctuating noise and interfering speech on the speech-reception threshold for impaired and normal hearing. *J Acoust Soc Am.* 1990;88(4):1725-36.
32. Pichora-Fuller MK, Souza PE. Effects of aging on auditory processing of speech. *Int J Audiol.* 2003;42 Suppl 2:S11-6.
33. Van Tasell DJ, Soli SD, Kirby VM, Widin GP. Speech waveform envelope cues for consonant recognition. *J Acoust Soc Am.* 1987;82(4):1152-61.
34. Onishi S, Davis H. Effects of duration and rise time of tone bursts on evoked V potentials. *J Acoust Soc Am.* 1968;44(2):582-91.
35. Michalewski HJ, Starr A, Nguyen TT, Kong YY, Zeng FG. Auditory temporal processing in normal-hearing individuals and patients with auditory neuropathy. *Clin Neurophysiol.* 2005;116(3):669-80.
36. Michalewski HJ, Starr A, Zeng FG, Dimitrijevic A. N100 cortical potentials accompanying disrupted auditory nerve activity in auditory neuropathy (AN): effects of signal intensity and continuous noise. *Clin Neurophysiol.* 2009;120(7):1352-63.
37. Zeng FG, Oba S, Garde S, Sininger Y, Starr A. Temporal and speech processing deficits in auditory neuropathy. *Neuroreport.* 1999;10(16):3429-35.
38. Zeng FG, Kong YY, Michalewski HJ, Starr A. Perceptual consequences of disrupted auditory nerve activity. *J Neurophysiol.* 2005;93(6):3050-63.
39. Mendelson JR, Ricketts C. Age-related temporal processing speed deterioration in auditory cortex. *Hear Res.* 2001;158(1-2):84-94.