Hearing loss is associated with delayed neural responses to continuous speech

Abbreviated title: Hearing loss delays neural responses to speech

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Abstract

We investigated the impact of hearing loss on the neural processing of speech. Using a forward modelling approach, we compared the neural responses to continuous speech of 14 adults with sensorineural hearing loss with those of age-matched normal-hearing peers.

Compared to their normal-hearing peers, hearing-impaired listeners had increased neural tracking and delayed neural responses to continuous speech in quiet. The latency also increased with the degree of hearing loss. As speech understanding decreased, neural tracking decreased in both population; however, a significantly different trend was observed for the latency of the neural responses. For normal-hearing listeners, the latency increased with increasing background noise level. However, for hearing-impaired listeners, this increase was not observed.

Our results support the idea that the neural response latency indicates the efficiency of neural speech processing. Hearing-impaired listeners process speech in silence less efficiently than normal-hearing listeners. Our results suggest that this reduction in neural speech processing efficiency is a gradual effect which occurs as hearing deteriorates. Moreover, the efficiency of neural speech processing in hearing-impaired listeners is already at its lowest level when listening to speech in quiet, while normal-hearing listeners show a further decrease in efficiency when the noise level increases.

From our results, it is apparent that sound amplification does not solve hearing loss. Even when intelligibility is apparently perfect, hearing-impaired listeners process speech less efficiently.

Key words: neural tracking, hearing loss, speech, EEG

Introduction

It is widely known that hearing loss alters the brain (Eggermont, 2017; Peelle and Wingfield, 2016). To study the functional neural changes, several studies focussed on cortical auditory evoked potentials (CAEP) using electroencephalography (EEG). CAEPs reflect the cortical responses evoked by repetitions of simple sounds such as syllables, tone pips, or clicks. These responses represent the detection and/or discrimination of a sound. The CAEP-response is characterized by a first positive peak (P1) around 50 ms, a first negative peak (N1) around 100 ms and a later positive peak (P2) around 180 ms (Burkard et al., 2007). Harkrider et al. (2009) and Campbell and Sharma (2013) reported increased P2-latencies in hearing impaired listeners (HI listeners) compared to normal hearing listeners (NH listeners). Interestingly, Campbell and Sharma (2013) reported that P2-latency was also correlated with the person's speech perception ability in noise. Although changes in latency are often not reported, in most studies HI listeners showed increased amplitudes compared to NH listeners (Tremblay et al., 2003; Harkrider et al., 2006; Bertoli et al., 2011; Alain, 2014; Maamor and Billings, 2017) while Billings et al. (2015) and Koerner and Zhang (2018) did not observe differences between these two populations or others attributed these differences to decreased audibility of the stimulus (Oates et al., 2002; Van Dun et al., 2016; McClannahan et al., 2019). No consensus has been reached on the impact of hearing loss on the P1-N1-P2-complex. The use of continuous speech as the stimulus can be key to characterize the neural differences between these two populations as it requires more in-depth neural processing of the stimulus to understand the speech.

A limited number of studies has been conducted to study the effect of hearing loss on the neural responses to continuous speech. In these studies, the amount of neural tracking, i.e. to what extent speech is tracked by the brain, has been investigated in a two-talker scenario: an attended speaker and an ignored one (Petersen et al., 2017; Mirkovic et al., 2019; Presacco et al., 2019; Decruy et al., 2020; Fuglsang et al., 2020). In all these studies, both NH listeners and HI listeners, showed a higher neural tracking of the attended speech stream than that of the ignored speech stream. Petersen et al. (2017) reported that adults with a higher degree of hearing loss showed a higher neural tracking of the ignored speech and no change in the attended stream, suggesting that they experience more difficulties inhibiting irrelevant information. Although Mirkovic et al. (2019) and Presacco et al. (2019) did not report a neural difference between the two populations, Decruy et al. (2020) and Fuglsang et al. (2020) observed, in contrast to Petersen et al. (2017), an enhanced neural tracking in HI listeners for the attended-speech compared to their normal-hearing peers. This enhancement can indicate a compensation mechanism: HI listeners need to compensate for the degraded auditory input and therefore show enhanced neural tracking.

The difficulties of researching HI listeners are twofold. First, most HI listeners are older, and ageing also has an impact on brain responses (Tremblay et al., 2003; Harkrider et al., 2006; Burkard et al., 2007; Harkrider et al., 2009; Decruy et al., 2019; Presacco et al., 2016). Therefore, it is important to compare HI listeners to age-matched normal-hearing peers. Second, audibility of the stimulus must be taken into account: sound presented at the same intensity can be less audible for HI listeners than for NH listeners.

Previous studies which reported the differences between HI listeners and NH listeners, focused on differences in neural tracking. In the current study, we investigated whether the characteristics of the neural responses to continuous speech (e.g., latency and topography) differed. Here, we showed that there are differences in the neural responses to continuous speech between HI listeners and their age-matched normal-hearing peers. Our results showed delayed neural responses to continuous speech in HI listeners. We hypothesized that HI listeners recruit more brain regions to understand speech, which is reflected in enhanced neural tracking of speech as well as a delay of neural responses.

Materials and Methods

Participants

We used a dataset containing EEG of 14 HI listeners (8♀) with sensorineural hearing loss and 14 aged-matched normal-hearing peers (13♀) (between 21 and 82 years old). The data were collected in a previous study by Decruy et al. (2020) (medical ethics committee of the University Hospital of Leuven approved the experiment (S57102); all participants signed an informed consent form). Inclusion criteria were: (1) having Dutch as a mother tongue, (2) having symmetrical hearing and (3) absence of medical conditions and learning disorders. A cognitive screening, the Montreal Cognitive Assessment (Nasreddine, 2004), was performed for all participants to ensure the absence of cognitive impairment. Hearing thresholds were determined using pure tone audiometry (125 to 8000 Hz). Normal hearing was defined for all participants where the hearing threshold did not exceed 30 dB HL for frequencies 125 to 4000 Hz (average of hearing thresholds within this frequency range in the stimulated ear is denoted as the pure-tone average (PTA)). The hearing thresholds and PTA are shown in Figure 1 (NH listeners: average PTA= 13.27 ± 5.60 dB HL, HI listeners: average PTA $= 44.46 \pm 10.54$ dB HL).

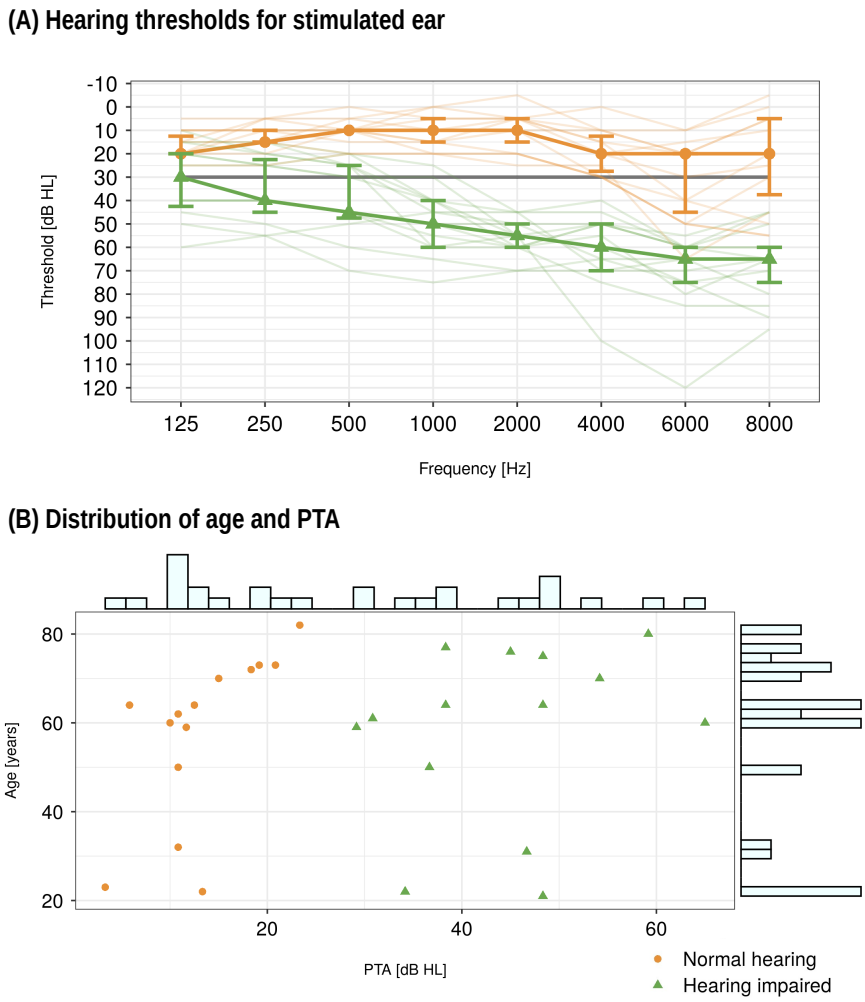

Figure 1: The hearing thresholds for the stimulated ear (panel A) and PTA as a function of age (panel B) for NH listeners (orange) and HI listeners (green).

Experimental Procedures

Behavioural Experiment: Flemish Matrix sentence test

The Matrix sentence test was performed to determine the participant's Speech Reception Threshold (SRT) in speech weighted noise (SWN). These Matrix sentences have a standard grammatical structure, consisting of a name, a verb, a numeral, a colour and an object (Luts et al., 2014). The SRT represents the signal-to-noise ratio (SNR) at which 50% of the presented words are recalled correctly.

EEG Experiment

Data acquisition A BioSemi ActiveTwo system (Amsterdam, Netherlands) was used to measure EEG signals during stimuli presentation. This system uses 64 Ag/AgCl electrodes placed according to the 10-20 system (Oostenveld and Praamstra, 2001). The EEG signals were measured with a sampling frequency of 8192 Hz. All recordings were carried out in a soundproof booth with Faraday cage at ExpORL (Dept. Neurosciences, KU Leuven).

Stimuli presentation The speech stimuli were presented monaurally through ER-3A insert phones (Etymotic Research Inc, IL, USA) using the software platform APEX (Dept. Neurosciences, KU Leuven) (Francart et al., 2008). The stimuli were presented to the right ear unless the participant preferred the left ear (n = 3; 1 NH; 2 HI). All stimuli were set to the same root mean square level and were calibrated.

For all NH listeners, the speech stimuli' intensity was fixed at 55 dB SPL (A-weighted). To ensure audible stimuli for HI listeners, the stimuli were linearly amplified based on the participant's hearing thresholds according to the National Acoustics Laboratory-Revised Profound (NAL) algorithm (Byrne et al., 2001). To ensure a comfortable level, the overall level was adjusted on a subject-specific basis in addition to the linear amplification so that the stimulus was minimally effortful and comfortable to listen to. The individual presentation levels are reported by Decruy et al. (2020).

During the EEG recording, 2 Dutch stories were presented: (1) "Milan", a 12-minute long story narrated by Stijn Vranken (♂) presented in quiet and (2) "De Wilde Zwanen" narrated by Katrien Devos (♀) presented in 5 different levels of background speech-weighted noise (each lasted around 2 minutes). The duration of silences was limited to 200 ms.

The levels of background noise for the second story depended on the participant's speech-in-noise performance. Using an adapted version of the self-assessed Békesy procedure (Decruy et al., 2019), the SRT of the Matrix sentences was adjusted to obtain a SRT of a story (Decruy et al., 2018, 2019). The noise conditions were

calculated on the participant's story adjusted SRT, namely: SRT - 3 dB, SRT, SRT + 3 dB, SRT + 6 dB and a condition without noise, which approximate speech understanding levels of 20%, 50%, 80%, 95% and 100%. A subjective rating of the participant's speech understanding was obtained after each condition (details are described in Decruy et al. (2018, 2019)).

Signal Processing

Processing of the EEG signals

The EEG recording with a sampling frequency of 8192 Hz was downsampled to 256 Hz to decrease processing time. To remove artefacts of eye blinks, we applied multi-channel Wiener filtering to the EEG data to remove artefacts of eye blinks (Somers et al., 2018). Then we referenced the EEG data to the common-average and filtered the data between 0.5 and 25 Hz using a zero-phase Chebyshev filter (Type II with an attenuation of 80 dB at 10% outside the passband). Additional downsampling to 128 Hz was performed.

Extraction of the speech features

In this study, we used 2 speech features: spectrogram and acoustical onsets. Both speech features are continuous features which represent the acoustical properties of the speech stimulus.

To create the spectrogram, the speech stimulus (without amplification) was low-pass filtered below 4000 Hz (zero-phase low-pass FIR filter with a hamming window of 159 samples) because the ER-3A insert phones also low-pass filter at this frequency. A spectrogram representation was obtained using the Gammatone Filterbank Toolkit 1.0 (Heeris, 2014) (centre frequencies between 70 and 4000 Hz with 256 filter channels and an integration window of 0.01 second). This toolkit calculates a spectrogram representation based on a series of gammatone filters inspired by the structure of the human auditory system (Slaney, 1998). The resulting 256 filter outputs were averaged into 8 frequency bands (each containing 32 outputs). Additionally, each frequency band was downsampled to the same sampling frequency as the processed EEG, namely 128 Hz. The NAL filtering introduced a delay of 5.334 ms, which was compensated for. The acoustical onsets were calculated as a half-wave rectification of the spectrogram's derivative.

Prediction accuracies, temporal response function & peak picking method

In this study, we focused on a linear forward modelling approach that predicts the EEG based on a linear combination of speech features of the presented speech. This forward modelling approach results in 2 outcomes: (a) a temporal response function (TRF) and (b) a prediction accuracy. (a) A TRF is a linear approximation of the brain's impulse response. It is a signal over time that describes how the brain responds

to the speech features. (b) TRFs can be used to predict the EEG by convolving it with the speech features. The predicted EEG is then correlated with the actual EEG to obtain a prediction accuracy. Prediction accuracy is considered a measure of neural tracking: the higher the prediction accuracy, the better the brain tracks the stimulus.

(a) To estimate TRFs, we used the Eelbrain toolbox (Brodbeck, 2020). The toolbox estimates TRFs using the boosting algorithm by David et al. (2007) (using a fixed step size of 0.005; stopping criteria based on ℓ_2-norm; kernel basis of 50 ms). We used 4-fold cross-validation (4 equally long folds; 3 folds used for training, 1 for validation) and an integration window between 0 and 700 ms. The estimated TRFs, averaged across folds and frequency bands, were used to determine the peak latencies.

(b) To calculate the prediction accuracy, the TRF is applied to left-out EEG to allow a fair comparison between models with a different number of speech features. We used the boosting algorithm with a testing fold. This implies a 4-fold cross-validation with 2 folds for training, 1 fold for validation and 1 fold for testing, which is left-out during training and validation. Each estimated TRF was used to predict the EEG of the left-out testing fold. The predicted EEG of all left-out segments are correlated, using Pearson correlation, with the actual EEG to obtain a prediction accuracy per EEG-electrode. The prediction accuracies were averaged across EEG-electrodes and denoted as neural tracking. Similarly, as Decruy et al. (2020), we calculated the neural tracking of the second story, presented in different level of background noise, using the TRFs estimated on the story in quiet.

From the TRF, we aimed to identify the amplitude and latency of 3 peaks: P1, N1 and P2. As the EEG data contains 64 different channels, 64 different TRFs were estimated, which made peak picking more complex. Therefore we applied principal component analysis (PCA), a dimensionality reduction method. The PCA-method results in (a) signals in component space and (b) corresponding spatial filters which describe the linear combinations of EEG channels to obtain these components. In our analysis, the first component was used. Adding more components up to 4 did not change the findings of this study. In addition to the time course of the component, we also investigated the corresponding spatial filter. As the sign of this spatial filter is arbitrary, we forced the average of occipital and parietal channels (P9, P7, PO7, O1, Oz, O2, PO8, P8, Iz, P10) to be negative by multiplying the spatial filter with -1 when needed. The PCA-method was applied to the data per story for each participant.

To identify the different peaks, we performed a z-score normalization of the TRF in component space and determined the maximal or minimal amplitude for positive and negative peaks in different time regions (P1: 30 to 110 ms, N1: 70 to 210 ms, P2: 110 to 270 ms), respectively. The overlap of these time regions is not an

issue as we identified either the maximal or minimal amplitude to determine the peak latency of a positive or negative peak, respectively. To only identify prominent peaks, a peak was discarded from the analysis if the amplitude of the normalized TRF was smaller than the threshold of 1.

Statistical analysis

We used the R software package (version 3.6.3) (R Core Team, 2020). We used the Buildmer toolbox, which allows identifying the best linear mixed model (LMM) or linear model (LM) given a series of predictors and all their possible interactions based on the likelihood-ratio test (Voeten, 2020). Depending on the analysis, we used the following predictors: (a) hearing status (NH or HI) or the PTA depending on whether we were interested in the group effect or the effect of the degree of hearing loss, (b) age and (c) peak type (P1, N1, P2). To observe an effect of model choice on prediction accuracy, we also included the predictor (d) model type (Spectrogram, Acoustic onsets, Acoustic onsets + Spectrogram) in the statistical analysis. The analysis over different noise conditions also included the predictor (e) speech understanding. For the analysis with regard to peak latency, all continuous predictors were z-scored to minimize effects due to differences in scale. A matching factor indicated the participants belonging to the same age-matched pair. We included a nested random effect: participant nested inside match, as each match contained a pair of participants, and each participant had multiple dependent observations. The models' assumptions were checked with a visual inspection of the residual plots to assure homoscedasticity and normality. The models' outcomes were reported with the unstandardized regression coefficient (β) with standard error (SE), t-ratio and p-value per fixed effect. If significant interaction effects were found or if we aimed to identify differences between different levels of a factor, additional Holm-adjusted post-hoc tests were performed by looking at the estimates of the estimated marginal means or estimated marginal means of linear trends or pairwise comparisons of these estimates, implemented by the Emmeans toolbox (Lenth, 2020). A significance level of $\alpha = 0.05$ was used.

To compare differences in spatial filters or topographies of the peaks between the 2 groups, we used a related cluster-based permutation test proposed by Maris and Oostenveld (2007) to determine whether the topography differs between NH listeners and HI listeners, using the Eelbrain implementation (Brodbeck, 2020). For these related cluster-based permutation tests the age-matching was preserved. For instance to test whether the topography differed between HI listeners and NH listeners, only the peak topographies of the age-matched participants were considered if both participants showed a prominent peak. A significance level of $\alpha = 0.05$ was used.

Results

Neural differences when listening to speech in quiet

Hearing-impaired listeners show higher neural tracking

We identified the speech feature(s) that resulted in the highest neural tracking: acoustic onsets, spectrogram or a combination of both speech features. As shown in Figure 2 and verified by the statistical analysis, the highest neural tracking was obtained with a combination of both speech features (analysis using LMM: Table 1). Additionally, HI listeners showed higher neural tracking compared to the group of NH listeners (on average 0.012 higher; SE = 0.0054, df = 26, t-ratio = 2.2715, p = 0.0316). Age did not have a significant effect on the neural tracking of speech. A Holm-adjusted pairwise comparison confirmed that the highest neural tracking was obtained with a combination of both speech features which was higher compared to the model using just the acoustic onsets (on average the prediction accuracy of the combined model is 0.003 higher; SE = 0.000651; df = 54, t-ratio = 4.521, p = 0.0001) and higher compared to the model using the spectrogram variable (on average the prediction accuracy of the combined model is 0.005 higher; SE = 0.000651; df = 54; t-ratio = 7.230; p < 0.0001).

Table 1: Linear mixed model: the effect of hearing status and model type on neural tracking. Estimates of the regression coefficients (β), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in match was included as a random effect.
Formula: neural tracking $\sim$ 1 + hearing status + model type + (1 | match/participant)

fixed effect term	β	SE	df	t-Ratio	p-value
Intercept (for NH / Spectrogram)	0.0252	0.0039	26.4961	6.5042	p < 0.001
Hearing status: HL	0.0124	0.0054	26	2.2715	p = 0.0316
Model type = Acoustic onsets	0.0018	7e-04	54	2.7088	p = 0.0090
Model type = Acoustic onsets + spectrogram	0.0047	7e-04	54	7.2301	p < 0.001

Delayed peak latencies for hearing-impaired listeners

In Figure 5.A, all TRFs in component space are shown for both populations and speech features. The TRFs of HI listeners show delayed neural responses to speech compared to those of NH listeners. Additionally, the average TRF for each speech feature show 2 prominent peaks: P1-peak of acoustic onsets ($P1_{AO}$) and N1-peak of acoustic onsets ($N1_{AO}$) for the acoustic onsets, N1-peak of spectrogram ($N1_S$) and P2-peak of spectrogram ($P2_S$) for the spectrogram. The best LMM predicting latency included a main effect of the considered peak, hearing status and age (Table 2). Adults with hearing loss showed later peak latencies (an increase of 23 ms, SE = 6.7665, df = 24.0459, t-ratio = 3.3822, p = 0.0025). The effect of age depended

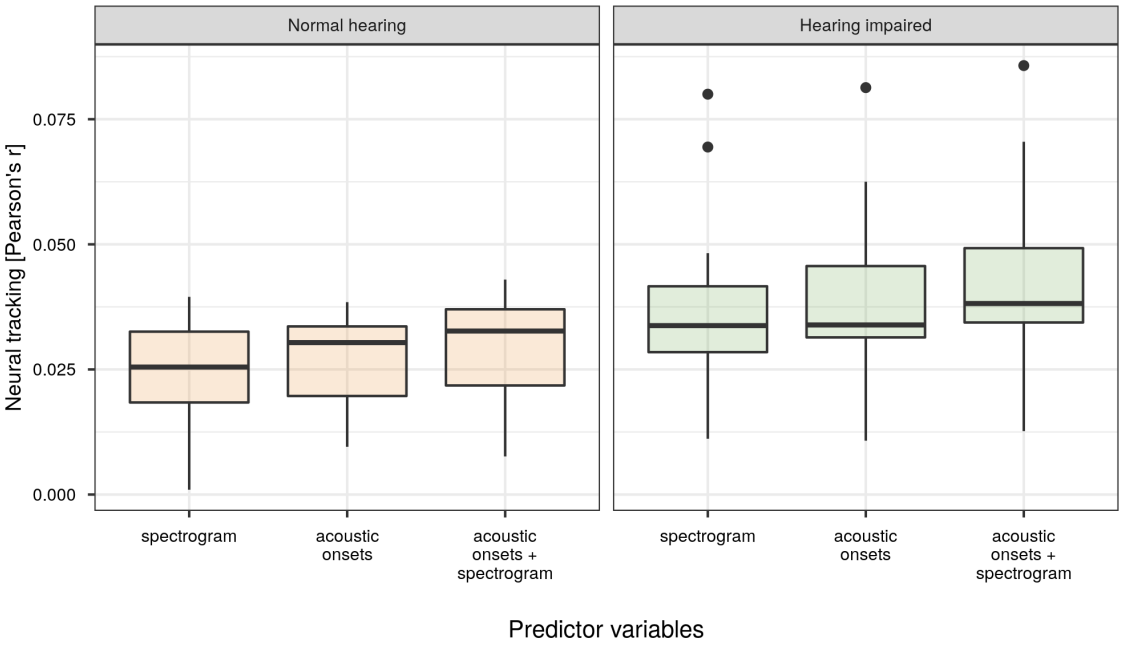

Figure 2: Neural tracking (Pearson's r) as a function of different combinations of speech features ('spectrogram', 'acoustic onsets' and 'acoustic onsets + spectrogram', respectively) for both NH listeners (left; orange) and HI listeners (right; green).

on the considered peak. No significant interaction between age and hearing status was observed. Post-hoc testing showed a significant decrease in latency with increasing age for the $N1_S$-latency (estimate of marginal trend: -0.793, SE = 0.271, df = 68.8, t-ratio = -2.926, p = 0.0186) while no significant trend was observed for the other peak latencies.

We did not observe a significant difference between the spatial filters of HI listeners and NH listeners. HI listeners showed a significantly different topography for $N1_S$ compared to NH listeners (Figure 3). HI listeners showed a more prominent central negativity and a higher occipital positivity which was slightly left-lateralized.

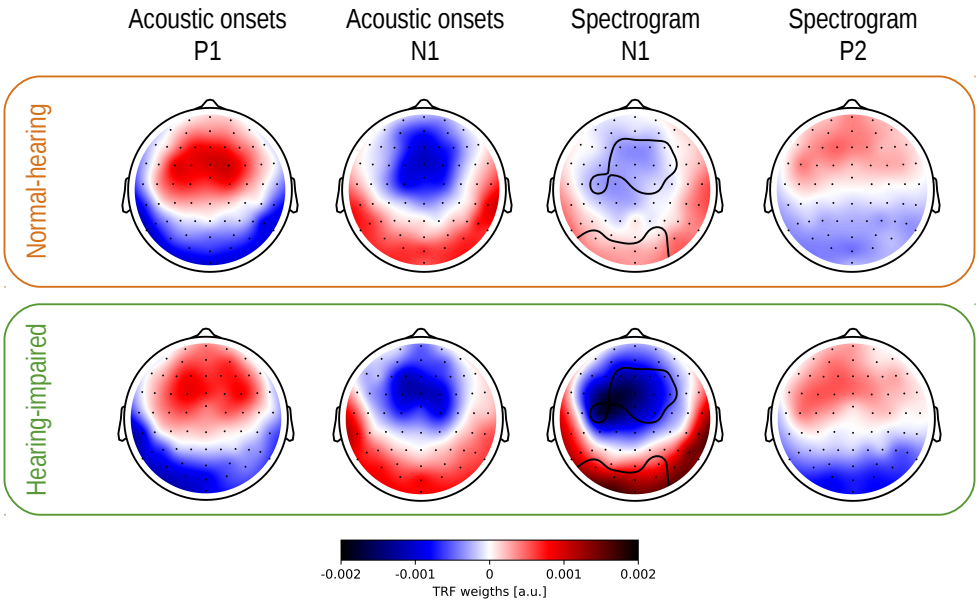

Figure 3: Visualization of the topographies of the peaks in the TRFs in sensor space for both speech features, spectrogram and acoustic onsets, and for NH listeners and HI listeners.

Longer latencies are associated with higher degrees of hearing loss

As significant differences in peak latencies were observed between NH listeners and HI listeners, we hypothesized that a higher degree of hearing loss is associated with increased latency of the peaks. Similarly as above, we identified the LMM which explains the variance in the latency. However, instead of using the factor hearing status, we used the continuous variable describing the degree of hearing loss. We justify this approach because the degree of hearing loss (represented by the PTA) is rather continuously distributed across the participants (Figure 1).

The latency depended on the considered peak, the degree of hearing loss and age of the listener (Table 3; using scaled predictors). The effect of degree of hearing loss on the latency depended on the considered

Table 2: Results of the linear mixed model in order to assess peak type, hearing status and age on the peak latency of $P1_{AO}$, $N1_{AO}$, $N1_S$, $P2_S$. Estimates of the regression coefficients (β), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in match was included as a random effect.
Formula: latency $\sim$ 1 + peak + hearing status + age + peak:age + (1 | match/participant)

fixed effect term	β	SE	df	t-Ratio	p-value
Intercept (for NH / for P1 - acoustic onsets)	65.5364	13.9654	49.1829	4.6928	$p < 0.001$
peak = N1 - acoustic onsets	73.013	17.5116	57.2985	4.1694	$p < 0.001$
peak = N1 - spectrogram	74.7323	17.7336	58.4244	4.2142	$p < 0.001$
peak = P2 - spectrogram	105.822	16.1937	56.478	6.5348	$p < 0.001$
Hearing status: HL	22.8857	6.7665	24.0459	3.3822	$p = 0.0025$
Age	-0.3274	0.2266	52.8304	-1.4449	$p = 0.1544$
peak = N1 - acoustic onsets:age	-0.0341	0.2805	56.788	-0.1214	$p = 0.9038$
peak = N1 - spectrogram:age	-0.4657	0.2781	57.2401	-1.6747	$p = 0.0994$
peak = P2 - spectrogram:age	0.7456	0.2649	56.0189	2.8147	$p = 0.0067$

peak and age of the listener. The Holm-adjusted estimates of the marginal trend showed that the trend of increasing latency with increasing degree of hearing loss is only significant for older adults for the peak latency of $N1_{AO}$ (estimate of trend = 21.0, SE = 6.44, df = 61.0, t-ratio = 3.260, p = 0.0146) and $N1_S$ (estimate of this trend = 20.4, SE = 6.38, df = 60.5, t-ratio = 3.192, p = 0.0157). However, this trend did not significantly differ between the different peaks nor between younger and older adults.

Looking at Figure 1, age is not evenly distributed. Therefore, the age effects in the above-mentioned analysis might be biased towards 3 younger age-matched pairs. We replicated the above-mentioned analysis using only participants above 40 years old. Indeed, in this analysis no interaction was found between degree of hearing loss and age nor degree of hearing loss, age and the considered peak. The latency of the peaks depended on the considered peak, age and the degree of hearing loss and the effect of age depended on the considered peak (Table S.1; using scaled predictors). The peak latency increased with increasing degree of hearing loss independent of the considered peak (estimate = 12.8954, SE = 3.5184, df = 17.8724, t-ratio = 3.6652, p = 0.0018). The Holm-adjusted estimates of the effect of age on the peak latency were not significant for any of the peak latencies.

The results of the above analysis suggest that the effect of age on the peak latencies is not robust. Therefore, this effect was not visualized in Figure 5. With increasing degree of hearing loss, the latency of neural responses increased (estimate = 14.4906, SE = 3.9794, df = 53.1663, t-ratio = 3.6414, $p < 0.001$; Table 3; Figure 5.B).

For each peak latency, we identified whether the variance in latency is explained by age and/or degree of hearing loss when only considering HI listeners. Although this reduced the statistical power, we observed a

significant effect of degree of hearing loss on the $N1_{AO}$-latency: HI listeners with a more severe hearing loss showed an increased latency (analysis using LM and scaled predictors; Table S.2; estimate = 2.0006, SE = 0.6996, t-ratio = 2.860, p = 0.0188).

Table 3: Results of the linear mixed model in order to assess the effects of degree of hearing loss (PTA) and age on the peak latency of $P1_{AO}$, $N1_{AO}$, $N1_S$, $P2_S$. Estimates of the regression coefficients (β), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in the matching factor was included as a random nested effect.
Formula: latency $\sim$ 1 + peak + PTA + age + peak:age + peak:PTA + peak:PTA:age + (1 | match/participant)

fixed effect term	β	SE	df	t-Ratio	p-value
Intercept (for P1 - acoustic onsets)	57.5671	4.1993	57.7904	13.7087	$p < 0.001$
peak = N1 - acoustic onsets	71.513	4.9762	50.0303	14.371	$p < 0.001$
peak = N1 - spectrogram	45.1675	5.0515	50.412	8.9414	$p < 0.001$
peak = P2 - spectrogram	150.0076	4.7256	47.5163	31.7433	$p < 0.001$
Degree of hearing loss (PTA)	14.4906	3.9794	53.1663	3.6414	$p < 0.001$
Age	-8.1202	3.8447	52.9895	-2.1121	p = 0.0394
peak = N1 - acoustic onsets:age	-2.2935	5.2705	53.8308	-0.4352	p = 0.6652
peak = N1 - spectrogram:age	-9.1196	4.9517	51.9372	-1.8417	p = 0.0712
peak = P2 - spectrogram:age	14.7078	4.721	50.1196	3.1154	p = 0.0030
Degree of hearing loss (PTA):age	1.1324	3.7797	52.3099	0.2996	p = 0.7657
peak = N1 - acoustic onsets:PTA	2.0488	5.0542	51.1439	0.4054	p = 0.6869
peak = N1 - spectrogram:PTA	-11.919	5.3951	51.9705	-2.2092	p = 0.0316
peak = P2 - spectrogram:PTA	-2.2075	4.4718	46.5795	-0.4936	p = 0.6239
peak = N1 - acoustic onsets:PTA:age	3.3177	5.2119	55.1995	0.6366	p = 0.5270
peak = N1 - spectrogram:PTA:age	16.6746	5.7976	55.4212	2.8761	p = 0.0057
peak = P2 - spectrogram:PTA:age	-0.5236	4.3161	47.3511	-0.1213	p = 0.9040

Neural differences when speech understanding decreases

Increased neural tracking with increased speech understanding

The effect of increased neural tracking for HI listeners was robust over different levels of background noise (estimate = 0.0154, SE = 0.0046, df = 25.1566, t-ratio = 3.3594, p = 0.0025); Table 4; Figure 4). Additionally, higher neural tracking was observed with increasing age (estimate = 3e-04, SE = 1e-04, df = 24.9812, t-ratio = 2.2452, p = 0.0339); Table 4; Figure 4) and with increasing speech understanding (estimate = 2e-04, SE = 0, df = 115.3178, t-ratio = 5.6547, $p < 0.001$; Table 4; Figure 4). No significant interaction effect was observed between hearing status and speech understanding.

Normal hearing listeners show a prominent increase in latency when speech understanding decreases, while this is less prominent for hearing-impaired listeners

We analysed the effects of speech understanding, age and degree of hearing loss on the peak latencies for the second story presented in different levels of background noise. The latency of the neural responses depends

Table 4: Linear mixed model: the effect of hearing status and speech understanding on neural tracking. Estimates of the regression coefficients (β), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in match was included as a random effect. Formula: neural tracking $\sim$ 1 + hearing status + age + speech understanding + (1 | match/participant)

fixed effect term	β	SE	df	t-Ratio	p-value
Intercept (for NH)	-0.0068	0.0081	29.4306	-0.8397	$p = 0.4078$
Hearing status: HL	0.0154	0.0046	25.1566	3.3594	$p = 0.0025$
Age	3e-04	1e-04	24.9812	2.2452	$p = 0.0339$
Speech understanding	2e-04	0	115.3178	5.6547	$p < 0.001$

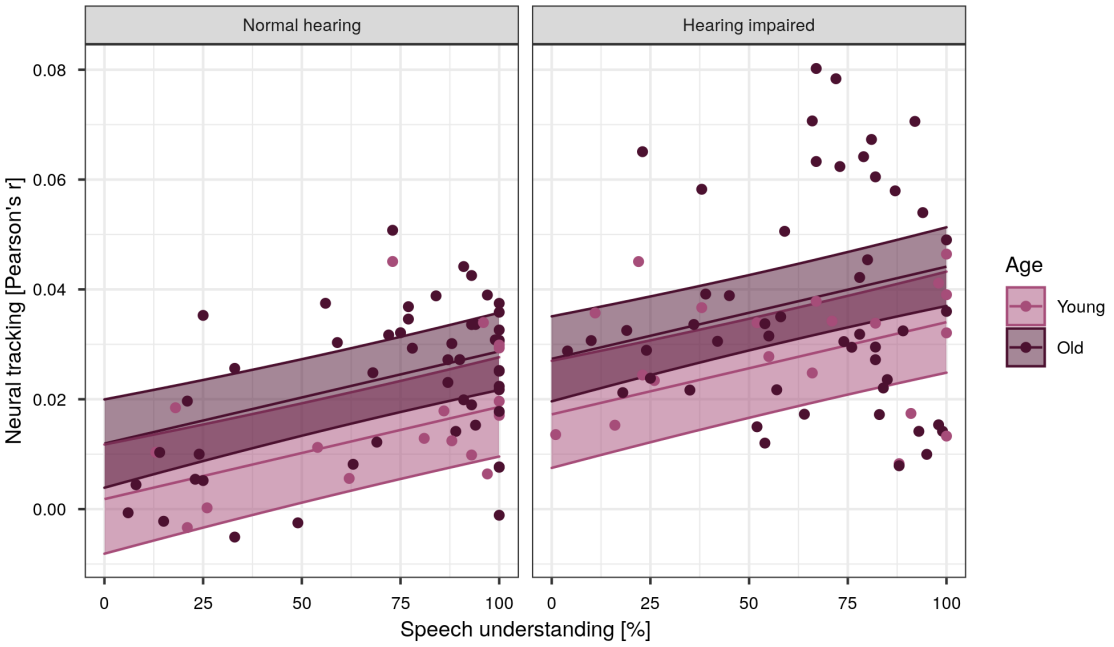

Figure 4: Neural tracking (Pearson's r) as a function of speech understanding. The effect of age was discretized with 2 levels: the average average age of participants younger than 50 years (level young; 31 years; pink) and participants with hearing loss (level old; 68 years; purple) and average average age of participants older than 50 years

on the considered peak, age and degree of hearing loss (analysis using LMM with scaled predictors; Table 5; Figure 5.C). The trends of age and speech understanding on peak latency depended on the considered peak. However, post-hoc tests did not show a significant effect of age on any of the peak latencies. Therefore this effect is not visualized in Figure 5.C.

Interestingly, a significant interaction effect between speech understanding and degree of hearing loss was found (estimate = 4.441, SE = 1.071, df = 396.822, t-ratio = 4.148, p < .001). Post-hoc testing showed that NH listeners showed a significant increase in latency when speech understanding decreased (estimate = -10.5, SE = 1.42, df = 390, t-ratio = -7.401, p < 0.0001) while no significant increase was observed for HI listeners. This trend significantly differed between the NH listeners and HI listeners (estimate = -1.6, SE = 1.68, df = 399, t-ratio = -0.955, p = 0.3402).

Table 5: Results of the linear mixed model in order to assess the effects of degree of hearing loss, speech understanding and age on the peak latency of $P1_{AO}$, $N1_{AO}$, $N1_S$, $P2_S$. Estimates of the regression coefficients (β), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in the matching factor was included as a random nested effect.
Formula: latency ∼ 1 + peak + SI + degree of hearing loss + speech understanding:degree of hearing loss + peak:speech understanding + age + degree of hearing loss:age + peak:age + speech understanding:age + (1 | match/participant)

fixed effect term	β	SE	df	t-Ratio	p-value
Intercept (for P1 - acoustic onsets)	65.523	3.149	69.524	20.808	p < .001
peak = N1 - acoustic onsets	71.012	3.015	384.446	23.557	p < .001
peak = N1 - spectrogram	39.875	3.203	385.734	12.449	p < .001
peak = P2 - spectrogram	144.887	3.097	385.778	46.783	p < .001
Speech understanding	-2.46	2.336	388.475	-1.053	p = 0.293
Degree of hearing loss (PTA)	5.489	2.324	23.74	2.362	p = 0.027
Age	-6.421	2.882	59.27	-2.228	p = 0.03
Speech understanding:Degree of hearing loss (PTA)	4.441	1.071	396.822	4.148	p < .001
peak = N1 - acoustic onsets:speech understanding	-4.487	2.968	381.779	-1.512	p = 0.131
peak = N1 - spectrogram:speech understanding	-1.148	3.163	382.021	-0.363	p = 0.717
peak = P2 - spectrogram:speech understanding	-8.703	3.066	383.334	-2.839	p = 0.005
Degree of hearing loss (PTA):Age	4.797	2.232	24.283	2.149	p = 0.042
peak = N1 - acoustic onsets:age	4.906	2.832	379.405	1.732	p = 0.084
peak = N1 - spectrogram:age	0.94	3.196	384.574	0.294	p = 0.769
peak = P2 - spectrogram:age	13.103	2.897	384.584	4.523	p < .001
Speech understanding:Age	-2.121	1.082	390.971	-1.96	p = 0.051

The effect of the degree of hearing loss on the peak amplitude was not consistent for all peaks.

Discussion

We compared the neural responses to continuous speech of adults with a sensorineural hearing loss with those of age-matched normal-hearing peers. We found that HI listeners show higher neural tracking and increased

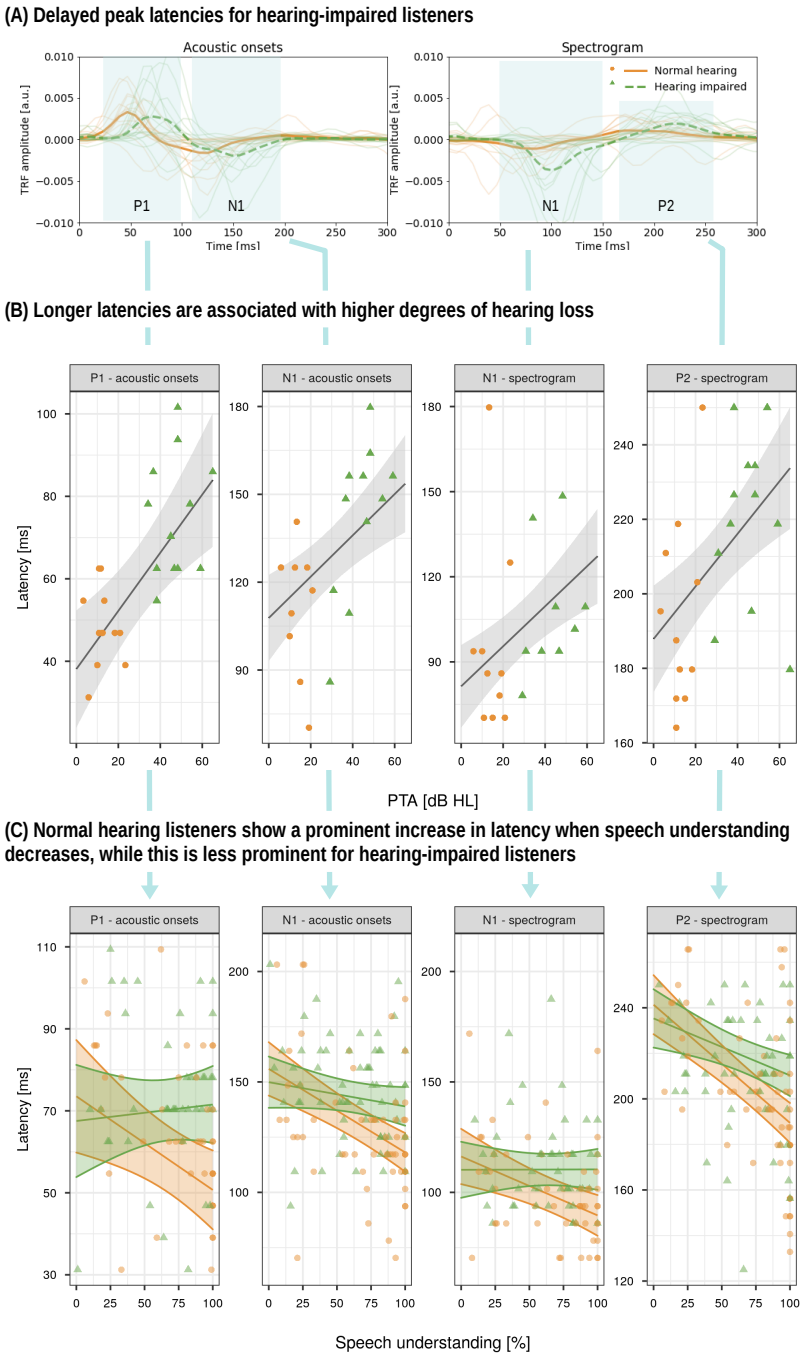

Figure 5: An overview of the neural responses of HI listeners (HI; striped line; green, triangle) and NH listeners (NH; orange, dot). Panel A: TRF in component space when listening to a story in quiet for both speech features evaluated for both HI listeners and NH listeners. The thick line represents the average TRF over participants. The lighter lines represent the subject-specific TRFs. Panel B: The peak latency in function of the degree of hearing loss (PTA) derived from the neural responses when listening to a story in quiet. Panel C: The peak latencies in function of speech understanding derived from the neural responses when listening to a story presented in multiple levels of background noise. The effect of degree of hearing loss was made discrete at 2 levels, the average hearing thresholds of all NH listeners (level NH listeners: 13 dB HL) and subjects with hearing loss (level HI listeners: 44 dB HL) and is represented by the regression lines with confidence intervals (shaded area).

peak latencies in their neural responses. Across noise conditions, NH listeners showed increased latencies as speech understanding decreased. However, for adults with hearing loss, this increase in latency was not observed.

Higher neural tracking of speech in hearing-impaired listeners

By evaluating neural tracking, we concluded that (1) higher neural tracking is observed for a combination of the spectrogram and acoustic onsets compared to the speech features individually and (2) HI listeners show enhanced neural tracking compared to normal-hearing peers.

The combination of speech features results in higher neural tracking, which implies that both speech features encode unique information. Following Hamilton et al. (2018) and Brodbeck et al. (2020), both speech features allow a differentiation between sustained activity, represented by the spectrogram, and transient activity, represented by acoustic onsets.

Using a forward modelling approach (instead of backward), we observed enhanced neural tracking in HI listeners. This agrees with results of Decruy et al. (2020) and Fuglsang et al. (2020) who also reported that HI listeners have higher neural tracking than NH listeners of the attended speaker. Nevertheless, Presacco et al. (2019) did not find a difference in neural tracking between the two populations. However, in their study, the populations were not closely age-matched, while ageing is known to increase neural tracking (Presacco et al., 2016; Decruy et al., 2019).

Like previous literature, we also observed that neural tracking decreases with decreasing speech understanding (Vanthornhout et al., 2018; Lesenfants et al., 2019; Decruy et al., 2020). Even when the speech is presented with background noise, HI listeners showed enhanced neural tracking of speech. This suggests evidence for a compensation mechanism: higher neural tracking indicates more neural activity to compensate for the degraded auditory input (Eggermont, 2017; Fuglsang et al., 2020). Although the enhanced neural tracking of speech in HI listeners, the effect of speech understanding on the neural tracking was similar for both populations.

Hearing-impaired listeners process speech less efficiently

HI listeners showed significantly increased latencies compared to their age-matched normal-hearing peers when they listened to a story presented in quiet (Figure 5.A). Additionally, the delay in neural responses increased with a higher degree of hearing loss (Figure 5.B).

Investigating the CAEP-response, Campbell and Sharma (2013) and Bidelman et al. (2019b) have reported an

increased P2 latency with worse speech perception in noise but not with the degree of hearing loss. However, in both studies, the same intensity was presented to both HI listeners and NH listeners. McClannahan et al. (2019) remarked that differences in the audibility of the stimulus might explain the differences in neural response latency. Indeed, Verschueren et al. (2020) observed that reduced audibility increases the latency of the neural responses to continuous speech. However, at a comfortable loudness (at intensities of 60 dB or higher in a NH population), the latency reaches a plateau. Our stimulus is amplified based on the participants' hearing thresholds and is presented at a subject-specific intensity to assure comfortable listening for HI listeners, therefore, we minimized the effects of differences in audibility of the stimulus.

Even though the sound was amplified, HI listeners showed increased latencies. Therefore, we hypothesize that there are some intrinsic differences in neural speech processing between HI listeners and NH listeners. A possible explanation may be that HI listeners process speech less efficiently, as proposed by Bidelman et al. (2019a). Using functional connectivity analysis, Bidelman et al. (2019a) showed that HI listeners have (a) more extended communication pathways and therefore (b) less efficient information exchange among these brain regions. (a) More extended communication paths may reflect a form of compensation in which additional brain regions are recruited to understand the degraded auditory input. This is supported by increased frontal activation in HI listeners in the neural responses to simple sounds (Campbell and Sharma, 2013; Bidelman et al., 2019b). Similarly, using continuous speech rather than simple repeated sounds, we showed that HI listeners have a significantly different $N1_S$ peak topography which suggests the recruitment of additional and/or different underlying neural sources (Figure 3). (b) When more or different brain regions are involved to process the speech, it causes longer communication pathways in the brain and therefore decreases the neural speech processing efficiency (Bidelman et al., 2019b). Here, we propose the neural response latency as a marker for the efficiency of neural processing of continuous, natural speech: less efficient speech processing is reflected by increased neural response latency as information exchange is hampered due to more involved brain regions and longer communication pathways.

When the intensity of the background noise increases in which speech is presented, NH listeners showed a prominent increase in latency when speech understanding decreases, while this was less prominent for HI listeners. In several studies, it has been shown that NH listeners show an increased neural response latency with increasing task demand due to lower stimulus intensity, increasing background noise or stimulus vocoding. This is the case for neural processing of continuous speech (Mirkovic et al., 2019; Verschueren et al., 2020; Kraus et al., 2020) as well as simple sounds (Billings et al., 2015; Van Dun et al., 2016; Maamor and Billings, 2017; McClannahan et al., 2019). Our results show that this increase in latency is absent for adults with a higher degree of hearing loss (Figure 5.C). This can explain why Mirkovic et al. (2019) did not

find a difference in latency between NH listeners and HI listeners as they presented only two noise conditions. As the noise level increases, the difference in latency between the two populations becomes smaller, which reduces the likelihood of a statistical difference between the two populations.

Bidelman et al. (2019b) did not report an effect of noise on the P2-latency. However, investigating the functional brain connectivity in the same data, Bidelman et al. (2019a) reported that as noise was added to the stimulus, NH listeners showed more long-range neural signalling whereas this was not seen for HI listeners (Bidelman et al., 2019a). The latter finding is supported by our data: in NH listeners the neural response latency increases as the speech understanding decreases due to increasing level of background noise while this was less prominent for HI listeners. This suggests that NH listeners process speech in noise less efficiently: more processing time is required to attend the speech stream and ignoring the noise. However, for HI listeners, this is not the case: when background noise increases, processing efficiency does not decrease. If longer latencies are a marker for less efficient neural processing and thus the number of recruited brain regions, our results in noise suggest that HI listeners recruit already recruit a maximum number of brain regions in the speech network to understand speech in quiet as their neural response latency does not increase with increasing amount of background noise.

Finally, we would like to highlight the difference in the trend of neural tracking and neural response latency. As speech understanding decreases, neural tracking decreases for both NH and HI listeners while the neural response latency remains constant (HI) or increases (NH). This difference in trend suggests that both measures represent different underlying neural processes for speech comprehension.

Conclusion

In this study, we compared the neural responses to continuous speech of adults with a sensorineural hearing loss with those of age-matched normal-hearing peers. HI listeners showed increased peak latencies of their neural responses. Interestingly, the latency increases as the degree of hearing loss increases. Across noise conditions, latency generally increases as the listening conditions become more difficult. However, for HI listeners, this increase in latency is not observed. We here suggest latency as a marker for the efficiency of neural processing to understand continuous, natural speech.

References

Alain, C. (2014). Effects of age-related hearing loss and background noise on neuromagnetic activity from auditory cortex. *Frontiers in systems neuroscience*, 8:8.

Bertoli, S., Probst, R., and Bodmer, D. (2011). Late auditory evoked potentials in elderly long-term hearing-aid users with unilateral or bilateral fittings. *Hearing research*, 280(1-2):58–69.

Bidelman, G. M., Mahmud, M. S., Yeasin, M., Shen, D., Arnott, S. R., and Alain, C. (2019a). Age-related hearing loss increases full-brain connectivity while reversing directed signaling within the dorsal–ventral pathway for speech. *Brain Structure and Function*, 224(8):2661–2676.

Bidelman, G. M., Price, C. N., Shen, D., Arnott, S. R., and Alain, C. (2019b). Afferent-efferent connectivity between auditory brainstem and cortex accounts for poorer speech-in-noise comprehension in older adults. *Hearing research*, 382:107795.

Billings, C. J., Penman, T. M., McMillan, G. P., and Ellis, E. (2015). Electrophysiology and perception of speech in noise in older listeners: effects of hearing impairment & age. *Ear and hearing*, 36(6):710.

Brodbeck, C. (2020). Eelbrain 0.32. http://doi.org/10.5281/zenodo.3923991.

Brodbeck, C., Jiao, A., Hong, L. E., and Simon, J. Z. (2020). Neural speech restoration at the cocktail party: Auditory cortex recovers masked speech of both attended and ignored speakers. *bioRxiv*, page 866749.

Burkard, R. F., Eggermont, J. J., and Don, M. (2007). *Auditory evoked potentials: basic principles and clinical application*. Lippincott Williams & Wilkins.

Byrne, D., Dillon, H., Ching, T., Katsch, R., and Keidser, G. (2001). Nal-nl1 procedure for fitting nonlinear hearing aids: characteristics and comparisons with other procedures. *Journal of the American academy of audiology*, 12(1).

Campbell, J. and Sharma, A. (2013). Compensatory changes in cortical resource allocation in adults with hearing loss. *Frontiers in systems neuroscience*, 7:71.

David, S. V., Mesgarani, N., and Shamma, S. A. (2007). Estimating sparse spectro-temporal receptive fields with natural stimuli. *Network: Computation in neural systems*, 18(3):191–212.

Decruy, L., Das, N., Verschueren, E., and Francart, T. (2018). The self-assessed Békesy procedure: validation of a method to measure intelligibility of connected discourse. *Trends in hearing*, 22:2331216518802702.

Decruy, L., Vanthornhout, J., and Francart, T. (2019). Evidence for enhanced neural tracking of the speech envelope underlying age-related speech-in-noise difficulties. *Journal of neurophysiology*, 122(2):601–615.

Decruy, L., Vanthornhout, J., and Francart, T. (2020). Hearing impairment is associated with enhanced neural tracking of the speech envelope. *Hearing Research*, page 107961.

Eggermont, J. J. (2017). Acquired hearing loss and brain plasticity. *Hearing Research*, 343:176–190.

Francart, T., van Wieringen, A., and Wouters, J. (2008). Apex 3: a multi-purpose test platform for auditory psychophysical experiments. *Journal of neuroscience methods*, 172(2):283–293.

Fuglsang, S. A., Märcher-Rørsted, J., Dau, T., and Hjortkjær, J. (2020). Effects of sensorineural hearing loss on cortical synchronization to competing speech during selective attention. *Journal of Neuroscience*, 40(12):2562–2572.

Hamilton, L. S., Edwards, E., and Chang, E. F. (2018). A spatial map of onset and sustained responses to speech in the human superior temporal gyrus. *Current Biology*, 28(12):1860–1871.

Harkrider, A. W., Plyler, P. N., and Hedrick, M. S. (2006). Effects of hearing loss and spectral shaping on identification and neural response patterns of stop-consonant stimuli. *The Journal of the Acoustical Society of America*, 120(2):915–925.

Harkrider, A. W., Plyler, P. N., and Hedrick, M. S. (2009). Effects of hearing loss and spectral shaping on identification and neural response patterns of stop-consonant stimuli in young adults. *Ear and hearing*, 30(1):31–42.

Heeris, J. (2014). Gammatone filterbank toolkit 1.0. https://github.com/detly/gammatone.

Koerner, T. K. and Zhang, Y. (2018). Differential effects of hearing impairment and age on electrophysiological and behavioral measures of speech in noise. *Hearing research*, 370:130–142.

Kraus, F., Tune, S., Ruhe, A., Obleser, J., and Woestmann, M. (2020). Unilateral acoustic degradation delays attentional separation of competing speech. *bioRxiv*.

Lenth, R. (2020). *emmeans: Estimated Marginal Means, aka Least-Squares Means*. R package version 1.4.8.

Lesenfants, D., Vanthornhout, J., Verschueren, E., Decruy, L., and Francart, T. (2019). Predicting individual speech intelligibility from the cortical tracking of acoustic-and phonetic-level speech representations. *Hearing research*, 380:1–9.

Luts, H., Jansen, S., Dreschler, W., and Wouters, J. (2014). Development and normative data for the Flemish/Dutch matrix test.

Maamor, N. and Billings, C. J. (2017). Cortical signal-in-noise coding varies by noise type, signal-to-noise ratio, age, and hearing status. *Neuroscience letters*, 636:258–264.

Maris, E. and Oostenveld, R. (2007). Nonparametric statistical testing of eeg-and meg-data. *Journal of neuroscience methods*, 164(1):177–190.

McClannahan, K. S., Backer, K. C., and Tremblay, K. L. (2019). Auditory evoked responses in older adults with normal hearing, untreated, and treated age-related hearing loss. *Ear and hearing*, 40(5):1106–1116.

Mirkovic, B., Debener, S., Schmidt, J., Jaeger, M., and Neher, T. (2019). Effects of directional sound processing and listener's motivation on eeg responses to continuous noisy speech: Do normal-hearing and aided hearing-impaired listeners differ? *Hearing Research*, 377:260–270.

Nasreddine, Z. (2004). *Montreal cognitive assessment (MoCA)*. École des sciences de la réadaptation, Sciences de la santé, Université d'Ottawa.

Oates, P. A., Kurtzberg, D., and Stapells, D. R. (2002). Effects of sensorineural hearing loss on cortical event-related potential and behavioral measures of speech-sound processing. *Ear and hearing*, 23(5):399–415.

Oostenveld, R. and Praamstra, P. (2001). The five percent electrode system for high-resolution eeg and erp measurements. *Clinical neurophysiology*, 112(4):713–719.

Peelle, J. E. and Wingfield, A. (2016). The neural consequences of age-related hearing loss. *Trends in neurosciences*, 39(7):486–497.

Petersen, E. B., Wöstmann, M., Obleser, J., and Lunner, T. (2017). Neural tracking of attended versus ignored speech is differentially affected by hearing loss. *Journal of neurophysiology*, 117(1):18–27.

Presacco, A., Simon, J. Z., and Anderson, S. (2016). Evidence of degraded representation of speech in noise, in the aging midbrain and cortex. *Journal of neurophysiology*, 116(5):2346–2355.

Presacco, A., Simon, J. Z., and Anderson, S. (2019). Speech-in-noise representation in the aging midbrain and cortex: Effects of hearing loss. *PloS one*, 14(3):e0213899.

R Core Team (2020). *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing, Vienna, Austria.

Slaney, M. (1998). Auditory toolbox. *Interval Research Corporation, Tech. Rep*, 10(1998).

Somers, B., Francart, T., and Bertrand, A. (2018). A generic eeg artifact removal algorithm based on the multi-channel wiener filter. *Journal of neural engineering*, 15(3):036007.

Tremblay, K. L., Piskosz, M., and Souza, P. (2003). Effects of age and age-related hearing loss on the neural representation of speech cues. *Clinical Neurophysiology*, 114(7):1332–1343.

Van Dun, B., Kania, A., and Dillon, H. (2016). Cortical auditory evoked potentials in (un) aided normal-hearing and hearing-impaired adults. In *Seminars in hearing*, volume 37, page 9. Thieme Medical Publishers.

Vanthornhout, J., Decruy, L., Wouters, J., Simon, J. Z., and Francart, T. (2018). Speech intelligibility predicted from neural entrainment of the speech envelope. *Journal of the Association for Research in Otolaryngology*, 19(2):181–191.

Verschueren, E., Vanthornhout, J., and Francart, T. (2020). The effect of stimulus intensity on neural envelope tracking. *bioRxiv*.

Voeten, C. C. (2020). *buildmer: Stepwise Elimination and Term Reordering for Mixed-Effects Regression*. R package version 1.6.

Supplementary Material

Supplementary Statistical Material

Table S.1: Results of the linear mixed model in order to assess the effects of degree of hearing loss (PTA) and age on the peak latency of $P1_{AO}$, $N1_{AO}$, $N1_S$, $P2_S$. Estimates of the regression coefficients (β), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in the matching factor was included as a random nested effect.
Formula: latency $\sim$ 1 + peak + PTA + age + peak:age + peak:PTA + peak:PTA:age + (1 | match/participant)

fixed effect term	β	SE	df	t-Ratio	p-value
Intercept (for P1 - acoustic onsets)	59.7545	5.7913	45.0463	10.318	$p < 0.001$
peak = N1 - acoustic onsets	63.1639	7.1839	46.8945	8.7925	$p < 0.001$
peak = N1 - spectrogram	32.1969	8.2213	48.341	3.9163	$p < 0.001$
peak = P2 - spectrogram	141.4255	6.1896	42.5149	22.8488	$p < 0.001$
Degree of hearing loss (PTA)	12.8954	3.5184	17.8724	3.6652	$p = 0.0018$
Age	-11.3602	9.4021	41.6263	-1.2083	$p = 0.2338$
peak = N1 - acoustic onsets:age	14.8838	11.4562	45.5116	1.2992	$p = 0.2004$
peak = N1 - spectrogram:age	18.3418	12.275	46.5449	1.4942	$p = 0.1419$
peak = P2 - spectrogram:age	32.7614	9.9271	41.8241	3.3002	$p = 0.0020$

Table S.2: Results of the linear model in order to assess the effect of degree of hearing loss on the peak latency of $N1_{AO}$ when only HI listeners are takening into account. Estimates of the regression coefficients (β), standard errors (SE), t-Ratios and p-values are reported per fixed effect term.
Formula: $N1_{AO}$-latency $\sim$ 1 + PTA
R^2_{adj} *= 0.4125, F = 6.618 on 1 and 7 df, p = 0.03688*

fixed effect term	β	SE	t-Ratio	p-value
Intercept	60.1035	31.0682	1.9346	0.0943
Degree of hearing loss (PTA)	1.8454	0.7174	2.5725	0.0369