PROCEEDINGS of the
23rd International Congress on Acoustics

9 to 13 September 2019 in Aachen, Germany

Selective attention in the brainstem and speech-in-noise comprehension

Marina SAIZ-ALIA[1]; Antonio Elia FORTE [2]; Tobias REICHENBACH[1]

[1] Imperial College London, London, U.K

[2] Harvard University, Cambridge, U.S.A.

ABSTRACT

Understanding speech in noise is a challenging task. Moreover, the ability to understand speech in background noise varies considerably from person to person, even for people that have normal audiograms and hence no measurable hearing loss. Recently we proposed a method for measuring the brainstem's response to natural non-repetitive speech and showed that this response is modulated by selective attention to one of two competing speakers. Here we investigate to what extent this brainstem response varies from subject to subject. We find significant between-subject variation in the amplitude of the brainstem response to continuous speech, in its latency, signal-to-noise-ratio, as well as in its modulation by selective attention. This variability may result from impairments in the auditory periphery, such a cochlear synaptopathy, as well as from damages of the neural pathways in the brainstem and in the central nervous system that are responsible for sound processing, and may potentially lead to deficits with speech-in-noise comprehension.

Keywords: auditory brainstem response, speech processing, selective attention

[1] {marina.saiz-alia15, reichenbach}@imperial.ac.uk

[2] aeforte@seas.harvard.edu

1. INTRODUCTION

People with normal hearing thresholds can nonetheless experience difficulty with understanding speech in noisy backgrounds. Indeed, 5-10 % of the patients seeking audiological treatment complain of speech-in-noise (SIN) understanding besides having normal audiograms (1). A potential source of such supra-threshold hearing deficits is cochlear synaptopathy, the loss of synaptic connection between the hair cells in the inner ear and the auditory-nerve fibers, which presumably leads to a degraded neural encoding of the speech signal compromising speech in noise comprehension (2). Furthermore, central auditory processing disorders and cognitive deficits affecting attention, memory or language can also be responsible for speech-in-noise deficits.

Auditory-brainstem responses to clicks are used clinically to assess the functioning of the auditory periphery. The effects of stimulus and recording parameters, and patient factors such as age and gender, on the amplitude and latency of the click-evoked brainstem response are well documented (4,5). The response can inform on different types of hearing impairments, in particular on sensorineural hearing loss but also on brainstem impairments such as caused by lesions.

To what extent auditory-brainstem responses can inform on other types of speech-in-noise deficits remains, however, debated. Animal studies have shown that cochlear synaptopathy results in a reduction of wave I of the click-evoked brainstem response (6–8), as well as in changes in other brainstem measures such as a latency shift of wave V with level of background noise and the amplitude of envelope-following responses (11–13). To which extent these findings translate to humans is, however, unclear: studies on human volunteers have produced conflicting results regarding the correlation of such brainstem measures with a putative cochlear synaptopathy as well as with speech-in-noise comprehension (11–17).

We recently proposed a method for measuring the brainstem response at the fundamental frequency of continuous speech. Furthermore, we demonstrated that this brainstem response is modulated by selective attention to one of two competing speakers, evidencing a neural mechanism that contributes to listening in noisy backgrounds. However, we also observed significant variability in the brainstem response to speech, as well as in its attentional modulation, among our volunteers (3). Because of the involvement of the brainstem response to speech in speech processing and in attention to speech in noise, deficiencies in the response may indicate speech-in-noise deficits. Here we therefore seek to quantify the variability in different aspects of the speech-evoked auditory-brainstem response.

2. METHODS

2.1 Brainstem response to speech and selective attention

To measure the brainstem response to speech as well as its attentional modulation we followed our recently developed procedure (3). In brief, we employed empirical mode decomposition (EMD) of the speech stimuli to extract a “fundamental waveform” of the speech signal (3,27). The fundamental waveform oscillates, at each time point, at the fundamental frequency of the speech signal, with a corresponding amplitude. We then recorded the brainstem response to continuous speech from scalp electrodes placed at the vertex, Cz, as well as the mastoids. The brainstem response to speech can be quantified through computing the cross-correlation of the fundamental waveform, as well as its Hilbert transform, with the scalp recordings. The resulting complex cross-correlation can be characterized in terms of its amplitude and latency. We measured brainstem responses from 37 volunteers that had no hearing impairment.

We studied the response to a single-talker male speaker, as well as to two competing speakers, one male and one female. All stimuli were presented diotically. The stimulation with two competing voices allowed us to assess the modulation of the brainstem response by selective attention (28). For half of the corresponding stimuli, participants attended the male voice, and vice versa for the remaining parts. We computed the attentional modulation as the relative difference in attended to ignored conditions for the male voice.

3. RESULTS

3.1 Variability in speech-evoked brainstem activity and in selective attention modulation

We observed considerable variability in the brainstem response to speech as well as in its modulation by selective attention among the different participants. The brainstem responses of three exemplary subjects are shown in Figure 1. One subject exhibits a strong brainstem response with a signal-to-noise ratio (SNR) of 18 dB (Figure 1A), the second subject has a medium response (SNR of 14 dB, Figure 1B), and the brainstem response of the third subject is barely measurable (SNR of 4 dB, Figure 1C).

Confirming the results from our previous study we found brainstem responses to speech in quiet at a mean latency of 8.5 ± 0.4 ms, evidencing a subcortical origin of the response (3). Nevertheless, we observed a significant between-subject variation of this response. In particular, the latency (Figure 2A) and amplitude (Figure 2B) of the brainstem response to speech in quiet varied considerably among the participants. The coefficient of variation (CV) was 29% and 46%, respectively.

We further computed the noise floor of the recordings for each participant (gray horizontal areas Figure 1A-C and Figure 2C). A null model was computed by cross-correlating the brainstem response to the fundamental waveform of a different story that had not been heard. The noise floor was determined as the 80th percentile of the complex cross-correlation in the noise model, for latencies from -100 ms to 100 ms. Both the noise floor and the SNR of the responses, computed using the peak amplitude as the signal, exhibited a large variance as well (Figure 2C&D; CV: 12% and 36%, respectively). However, the noise level was not correlated with the peak amplitude of the responses ($p = 0.12$).

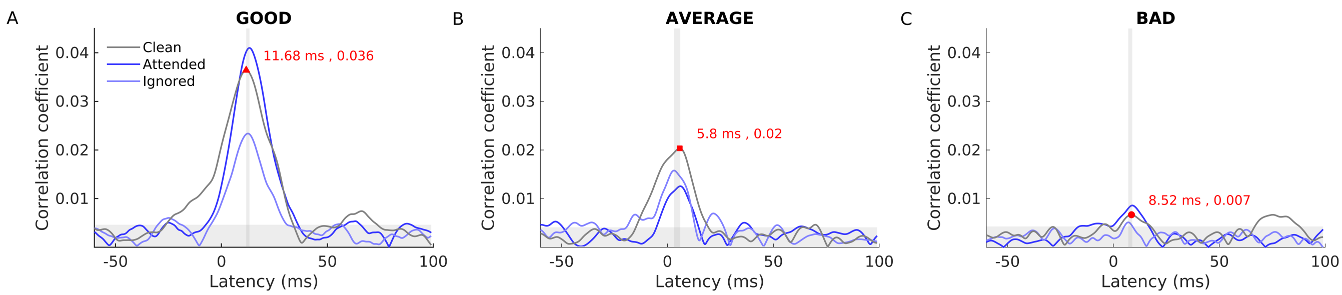

Figure 1 – Brainstem response to speech in quiet (grey) as well as to two competing speakers, namely for the attended voice (dark blue) and for the ignored voice (light blue). We show the brainstem responses of three representative subjects, one with a high SNR (A), one with a medium SNR (B), and one with a low SNR (C). The peak amplitude of the brainstem response to speech in quiet is labeled by a red marker, and the noise floor is marked in grey. The range of the latency of the three responses for each subject is indicated through a vertical grey bar.

Selective attention can modulate the brainstem response to speech. Confirming the results from our previous study, we found that selective attention led to a larger response to the attended than to the ignored speaker (population average). However, we observed large individual differences in the attentional modulation. Indeed, some subjects displayed the opposite modulation, leading to negative attentional differences, which increased the variability in the sample (CV: 250%). The differences in attentional modulation did not result from the SNR of the responses as these two variables were not correlated (p = 0.51).

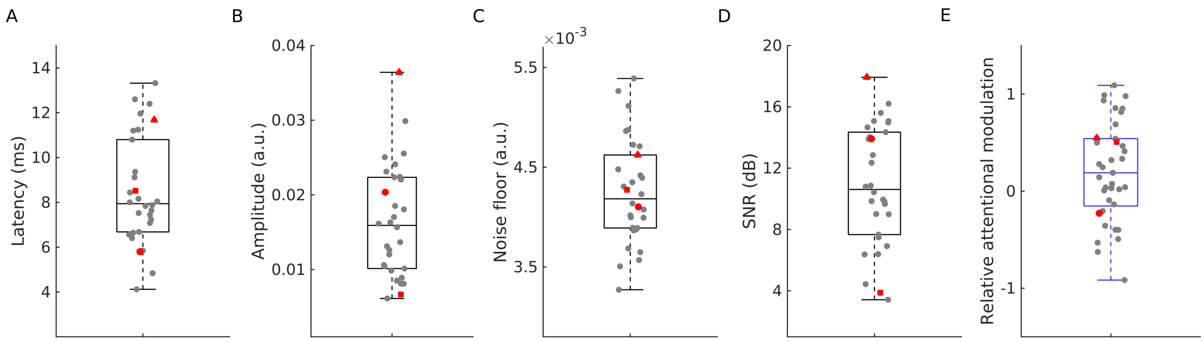

Figure 2 – Between-subject variability in different aspects of the brainstem response : latency (A), amplitude (B), noise-floor (C), SNR (D) and relative attentional modulation (E). Red markers represent the three representative subjects that are shown in Figure 1: high SNR (triangle), medium SNR (square), and low SNR (circle).

4. CONCLUSIONS

We studied the variability in the brainstem response to natural non-repetitive running speech. Consistent with our previous study we found a mean latency of 8.5 ± 0.4 ms, evidencing a subcortical origin of the response. We also found a significant attentional modulation of the response when participants listened to two competing speakers.

Importantly, we found significant between-subject variation of the amplitude, latency and SNR of the response, as well as of the selective attention modulation. The variability was comparable to individual differences in subcortical measures previously reported in cochlear synaptopathy studies (13,29) and in speech-in-noise reports (30).

Because the brainstem response to speech is driven by the neural activity in the auditory-nerve fibers, variation in the response may result from impairments in the auditory periphery such as cochlear synaptopathy. In addition, since the attentional modulation of the brainstem response to speech relies on neural feedback loops from the cerebral cortex, variability in the attentional modulation may have more central origins. Future studies are required in order to assess to what extent the variability in the speech-evoked brainstem activity and its modulation by attention can be explained by individual measures of peripheral function (such as those proposed for cochlear synaptopathy), by more central processing, and whether it may account for differences in speech-in-noise comprehension.

ACKNOWLEDGEMENTS

This research was supported by the Royal British Legion Centre for Blast Injury Studies, from the "la Caixa" Foundation (LCF/BQ/EU15/10350044) as well as by EPSRC grants EP/M026728/1 and EP/R032602/1.

REFERENCES

1. Hind SE, Haines-Bazrafshan R, Benton CL, Brassington W, Towle B, Moore DR. Prevalence of clinical referrals having hearing thresholds within normal limits. International Journal of Audiology. 2011;50(10):708–16.
2. Lopez-Poveda EA. Why do I hear but not understand? Stochastic undersampling as a model of degraded neural encoding of speech. Frontiers in Neuroscience. 2014;8.
3. Forte AE, Etard O, Reichenbach T. The human auditory brainstem response to running speech reveals a subcortical mechanism for selective attention. eLife. 2017;6.
4. Dalebout SD, Robey RR. Comparison of the Intrsubject and Intrasubject Variability of Exogenous and Endogenous Auditory Evoked Potentials. J Am Acad Audiol. 1997;8:343–54.
5. Hood LJ. Clinical applications of the auditory brainstem response. First. Thomson Delmar Learning; 1998. 49-63 p.
6. Hickox AE, Larsen E, Heinz MG, Shinobu L, Whitton JP. Translational issues in cochlear synaptopathy. Hear Res. 2017;349:164–71.
7. Valero MD, Burton JA, Hauser SN, Hackett TA, Ramachandran R, Liberman MC. Noise-induced cochlear synaptopathy in rhesus monkeys (Macaca mulatta). Hear Res. 2017;353:213–23.
8. Kujawa SG, Liberman MC. Adding Insult to Injury : Cochlear Nerve Degeneration after " Temporary " Noise-Induced Hearing Loss. J Neurosci. 2009;29(45):14077–85.
9. Viana LM, Malley JTO, Burgess BJ, Jones DD, Oliveira CACP, Santos F, et al. Cochlear neuropathy in human presbycusis : Confocal analysis of hidden hearing loss in post-mortem tissue. Hear Res. 2015;327:78–88.
10. Wu P, Liberman L, Bennett K, Gruttola VD, Omalley J, Liberman M. Primary Neural Degeneration in the Human Cochlea: Evidence for Hidden Hearing Loss in the Aging Ear. Neuroscience. 2018; 1–13.
11. Bharadwaj HM, Masud S, Mehraei G, Verhulst S, Shinn-Cunningham BG. Individual Differences Reveal Correlates of Hidden Hearing Deficits. J Neurosci. 2015;35(5):2161–72.
12. Mehraei G, Gallardo AP, Shinn-Cunningham BG, Dau T. Auditory brainstem response latency in forward masking, a marker of sensory deficits in listeners with normal hearing thresholds. Hear Res 2017;346:34–44.
13. Mehraei G, Hickox AE, Bharadwaj HM, Goldberg H, Verhulst S, Liberman MC, et al. Auditory brainstem response latency in noise as a marker of cochlear synaptopathy. J Neurosci. 2016;36(13):3755–64.
14. Guest H, Munro KJ, Prendergast G, Plack CJ. Reliability and interrelations of seven proxy measures of cochlear synaptopathy. Hear Res. 2019;375:34–43.
15. Guest H, Munro KJ, Prendergast G, Millman RE, Plack CJ. Impaired speech perception in noise with a normal audiogram: No evidence for cochlear synaptopathy and no relation to lifetime noise exposure. Hear Res. 2018;364:142–51.
16. Prendergast G, Millman RE, Guest H, Munro KJ, Kluk K, Dewey RS, et al. Effects of noise exposure on young adults with normal audiograms II: Behavioral measures. Hear Res. 2017;356:74–86.
17. Prendergast G, Guest H, Munro KJ, Kluk K, Léger A, Hall DA, et al. Effects of noise exposure on young adults with normal audiograms I: Electrophysiology. Hear Res. 2017;344:68–81.
18. Bajo VM, Nodal FR, Moore DR, King AJ. The descending corticocollicular pathway mediates learning-induced auditory plasticity. Nature Neuroscience. 2009;13(2):253–60.
19. Winer JA, Larue DT, Diehl JJ, Hefti BJ. Auditory cortical projections to the cat inferior colliculus. J Comp Neurol. 1998;400:147–74.
20. Song, Judy H., Skore, Erika, Wong, Patrick C. M., Kraus N. Plasticity in the adult human auditory

brainstem following short- term linguistic training Judy. J Cogn Neurosci. 2010;20(10):1892–902.
21. Skoe E, Kraus N. Auditory brainstem reponse to complex sounds : a tutorial. Ear Hear. 2010;31(3):302–24.
22. Musacchia G, Sams M, Skoe E, Kraus N. Musicians have enhanced subcortical auditory and audiovisual processing of speech and music. Proc Natl Acad Sci. 2007;104(40):15894–8.
23. Krishnan A, Xu Y, Gandour J, Cariani P. Encoding of pitch in the human brainstem is sensitive to language experience. Cognitive Brain Research. 2005;25(1):161–8.
24. Galbraith G. Two-channel brain-stem frequency-following responses to pure tone and missing fundamental stimuli. Electroencephalography and Clinical Neurophysiology. 1994;92(4):321–30.
25. Chandrasekaran B, Kraus N. The scalp-recorded brainstem response to speech: Neural origins and plasticity. Psychophysiology. 2010;47(2):236–46.
26. Rønne FM, Harte JM, Dau T. Modelling human auditory evoked brainstem responses to speech syllables. Proc Meet Acoust. 2013;19(1):050120–050120.
27. Huang H, Pan J. Speech pitch determination based on Hilbert-Huang transform. Signal Processing. 2006;86(4):792–803.
28. Ding N, Simon JZ. Emergence of neural encoding of auditory objects while listening to competing speakers. Proceedings of the National Academy of Sciences. 2012;109(29):11854–9.
29. Bharadwaj HM, Verhulst S, Shaheen L, Liberman MC, Shinn-Cunningham BG. Cochlear neuropathy and the coding of supra-threshold sound. Frontiers in Systems Neuroscience. 2014;8.
30. Prell CGL. Effects of noise exposure on auditory brainstem response and speech-in-noise tasks: a review of the literature. International Journal of Audiology. 2018;58(sup1).