JSLHR

Review Article

Cortical and Sensory Causes of Individual Differences in Selective Attention Ability Among Listeners With Normal Hearing Thresholds

Barbara Shinn-Cunningham[a]

Purpose: This review provides clinicians with an overview of recent findings relevant to understanding why listeners with normal hearing thresholds (NHTs) sometimes suffer from communication difficulties in noisy settings.
Method: The results from neuroscience and psychoacoustics are reviewed.
Results: In noisy settings, listeners focus their attention by engaging cortical brain networks to suppress unimportant sounds; they then can analyze and understand an important sound, such as speech, amidst competing sounds. [illegible]

The challenge of understanding spee
where there are multiple sound sources is l
cocktail party problem, a term originally co
(1953). Understanding how listeners with n
ing solve the cocktail party problem has re
of study for over 50 years (e.g., see Bee & M
Bodden, 1993; Hafter et al., 2013; Wood &
Yost, 1997), in no small part because of its

Kujawa, & Liberman, 2013; Kujawa & Liberman, 2009; Lin, Furman, Kujawa, & Liberman, 2011; Rosen, Cohen, & Vanniasegaram, 2010), a catchall diagnosis that says nothing about the underlying causes of the communication difficulties, making it difficult to develop effective treatments.

to provide appropriate counseling and care management and, ultimately, targeted interventions.

At first, it may seem surprising that listeners can have difficulty understanding speech in cocktail party settings but do not report difficulty in other listening situations. However, solving the cocktail party problem places much greater cognitive and sensory demands on the listener than does listening in quiet. As discussed in the section on We Rely on Selective Attention to Communicate in Noisy Social Settings, audibility often is not the factor that limits understanding for listeners with NHTs. Instead, central processing resources can limit what we can consciously perceive. We manage this limitation by focusing attention on whatever acoustic source we will process, which relies on engaging cortical networks to filter inputs in ways that are unnecessary in quiet. Some listeners may have problems controlling cortical control networks and, therefore, have difficulty focusing selectively on whatever sound they want to hear, an idea developed further in the section Individuals Differ in Their Ability to Control Selective Attention. More attending to a different object on the screen—even though the input reaching the animal's retina is identical (e.g., see Buschman & Miller, 2007). In auditory neuroscience, single enhanced activation (Hill & Miller, 2010; Larson & Lee, 2014; Lee et al., 2013; Michalka et al., 2015, 2016). Thus, top-down attention to nonspatial auditory features differentially engages areas associated with auditory-specific processing

Importantly, the effect on cochlear function can be negligible; cochlear tuning and behavior detection thresholds can be normal in exposed animals (Kujawa & Liberman, 2009).

Most hearing screenings reveal losses associated with damage to inner and outer hair cells. Yet, with cochlear synaptopathy, measures of cochlear function are normal, making the deficit “invisible” to typical hearing screenings (explaining the use of the colloquial term hidden hearing loss to describe these problems; see Schaette & McAlpine, 2011).

Although detection thresholds may be normal in animals with cochlear synaptopathy, the loss of independent ANFs degrades temporal processing, which particularly degrades the coding of temporal modulation in suprathreshold sound. These effects can be seen, for instance, in the fidelity of phase locking in brainstem responses to amplitude modulation and the effects of additive noise and forward masking on subcortical neural responses (e.g., see Chambers et al., 2016; Furman et al., 2013; Hickox & Liberman, 2014).

Although it is difficult to prove directly that cochlear synaptopathy causes hearing problems in humans with normal cochlear mechanical function, a growing number of studies suggest that it does. Listeners with NHTs differ in their ability to use fine temporal cues (see Grose & Mamo, 2010; Mehraei, Gallardo, Shinn-Cunningham, & Dau, 2017; Mehraei et al., 2016; Strelcyk & Dau, 2009). This variability correlates with difficulties in using spatial selective attention to focus on and understand speech in a noisy background (Bharadwaj, Masud, Mehraei, Verhulst, & Shinn-Cunningham, 2015; Paul, Bruce, & Roberts, 2017; Ruggles & Shinn-Cunningham, 2011), underscoring the clinical relevance of these differences.

Listeners with NHTs show large intersubject variability in the magnitude of auditory brainstem response (ABR) Wave I (Schaette & McAlpine, 2011; Stamper & Johnson, 2015), supporting the view that some listeners with normal audiograms may suffer from cochlear synaptopathy, albeit to varying degrees. As in animal studies, while ABR Wave I amplitude varies significantly across individuals, the magnitude of ABR Wave V does not (Schaette & McAlpine, 2011; Stamper & Johnson, 2015). One study has shown that perceptual differences correlate with these differences in human ABRs: In young adults with no known hearing deficits, Wave I magnitude correlates with ITD sensitivity, whereas Wave V magnitude is unrelated to Wave I magnitude or perceptual ability (Mehraei et al., 2016). Indeed, cochlear synaptopathy reduces the strength of auditory nerve responses; the auditory system then seems to respond by increasing some internal gain to amplify the weak response that remains (e.g., see Chambers et al., 2016). Based on these findings, one proposed method for identifying cochlear synaptopathy in humans computes the ratio of the summation potential (the response of the hair cells in the cochlea) to the action potential (the auditory nerve response; Liberman, Epstein, Cleveland, Wang, & Maison, 2016); however, neither this metric nor any other has yet been proven to be diagnostic of cochlear synaptopathy in humans (see comments in the section on Future Impact in the Clinic).

In one study in my own laboratory, young adult subjects were recruited with no special criteria except that they had NHTs and no known auditory deficits (Bharadwaj et al., 2015). Individual differences among this cohort were nonetheless large. Perceptual abilities (including the one 15° to the left of center and one 15° to the right of

A [illegible]

The research reported in this publication was supported by the National Institute on Deafness and Other Communication Disorders of the National Institutes of Health under Award Numbers R13DC003383 and R01DC013825. The content is

Frontiers in Human Neuroscience, 10,

selective auditory attention. Journal of the Association for Research in Otolaryngology, 13(1), 119–129. https://doi.org/10.1007/s10162-011-0299-7

M , C. A., , J., K , . G., L , M. C., & M , . N. (2011). Age-related primary cochlear neuronal degeneration in human temporal bones. Journal of the Association for Research in Otolaryngology, 12(6), 711–717. https://doi.org/10.1007/s10162-011-0283-2

M D , J. H., , D., & O , A. J. (2011). Recovering sound sources from embedded repetition. Proceedings of the National Academy of Science, 108(3), 1188–1193. https://doi.org/10.1073/pnas.1004765108

M , G., G , A. P., -C , B. G., & D , . (2017). Auditory brainstem response latency in forward masking, a marker of sensory deficits in listeners with normal hearing thresholds. Hearing Research, 346, 34–44. https://doi.org/10.1016/j.heares.2017.01.016

M , G., H , A. E., B , H. M., G , H., , ., L , M. C., & -C , B. G. (2016). Auditory brainstem response latency in noise as a marker of cochlear synaptopathy. Journal of Neuroscience, 36(13), 3755–3764. https://doi.org/10.1523/Jneurosci.4460-15.2016

M , N., & C , E. F. (2012). Selective cortical representation of attended speaker in multi-talker speech perception. Nature, 485(7397), 233–236. https://doi.org/10.1038/nature11020

M , ., K , L., , M. L., -C , B. G., & , D. C. (2015). Short-term memory for space and time flexibly recruit complementary sensory-biased frontal lobe attention networks. Neuron, 87(4), 882–892. https://doi.org/10.1016/j.neuron.2015.07.028

M , ., , M. L., K , L., -C , B. G., & , D. C. (2016). Auditory spatial coding flexibly recruits anterior, but not posterior, visuotopic parietal cortex. Cerebral Cortex, 26(3), 1302–1308. https://doi.org/10.1093/cercor/bhv303

M , C., , B., C , . P., & , J. P. (2005). Perceptual organization of tone sequences in the auditory cortex of awake macaques. Neuron, 48(1), 139–148. https://doi.org/10.1016/j.neuron.2005.08.039

M , M. M., P , ., - , P., , J., - , . A., & H , . A. (1998). Effects of spatial selective attention on the steady-state visual evoked potential in the 20–28 Hz range. Brain Research. Cognitive Brain Research, 6(4), 249–261.

N , K., & G - , . (2011). Speech perception in quiet and noise using the hearing in noise test and the Japanese hearing in noise test by Japanese listeners. Ear and Hearing, 32(1), 121–131. https://doi.org/10.1097/AUD.0b013e3181eccdb2

N , ., N , G., B , N., & A , M. A. (2012). Self-assessed hearing abilities in middle- and older-age adults: A stratified sampling approach. International Journal of Audiology, 51(3), 174–180. https://doi.org/10.3109/14992027.2011.621899

O , D., , ., C , K., M , ., & , D. (2015). Structural and functional connectivity of visual and auditory attentional networks: Insights from the Human Connectome Project. Journal of Vision, 15(12), 223. https://doi.org/10.1167/15.12.223

O’ , J. A., P , A. J., M , N., , ., F , J. J., -C , B. G., . . . L , E. C. (2014). Attentional selection in a cocktail party environment can be decoded from single-trial EEG. Cerebral Cortex, 25(7), 1697–1706. https://doi.org/10.1093/cercor/bht355

O’ , J. A., , . A., & L , E. C. (2015). Evidence for neural computations of temporal coherence in an auditory scene and their enhancement during active listening. Journal of Neuroscience, 35(18), 7256–7263. https://doi.org/10.1523/JNEUROSCI.4973-14.2015

O , A. J., & D , . (2001). Modulation detection interference: Effects of concurrent and sequential streaming. The Journal of the Acoustical Society of America, 110(1), 402–408.

P , B. N., D , ., M , N., F , A., , . A., C , N. E., . . . C , E. F. (2012). Reconstructing speech from human auditory cortex. PLoS Biology, 10(1), [illegible], [illegible]. A., M[illegible], J. H., & B[illegible], F. A. (1996). Age-related loss of activity of auditory-nerve fibers. Journal of Neurophysiology, 76(4), 2799–2803.

[illegible], A., M D[illegible], J. H., & [illegible]-C[illegible], B.