

Too Tired to Listen? Assessing Fatigue in Children with Hearing Loss

Ben Hornsby

Abstract:

Fatigue is a common, but important, patient complaint in a variety of chronic health conditions. Subjective reports from the literature have suggested for many years that fatigue was an important, but overlooked, consequence of hearing loss. Consider this anecdotal report from a person with hearing loss: "I crashed. This letdown wasn't the usual worn-out feeling after a long day. It was pure exhaustion, the deepest kind of fatigue. I took a nap hoping it would refresh me, but when I woke up three hours later I was still so tired I gave up on the day.... The only cause of my fatigue I could identify was the stress of struggling to understand what those around [me] were saying..." (Copithorne, 2006).

Despite numerous patient reports of fatigue, there is a paucity of research on fatigue in hearing loss, particularly in children. In this presentation the constructs of subjective and objective fatigue and their impact on adults and children with hearing loss will be introduced. Methods for assessing fatigue will be discussed and preliminary data from an ongoing study examining fatigue in children with hearing loss will be presented.

Neural synchrony and auditory processing

Nina Kraus

Consistency in how the brain responds to sound represents a mechanism that contributes to accurate auditory processing and language skills. Synchrony of neural firing in the auditory pathway is critical to hearing. Sufferers of an extreme example of dyssynchrony—auditory neuropathy—are often labeled “deaf” even though their audiometric thresholds are normal. So, neural synchrony occurs along a continuum and less extreme cases of dyssynchrony can result in an array of auditory processing disorders, often involving hearing in noise. How can effective sound-to-meaning relationships be formed if responses to sound are unstable? An “ideal” brain will respond in a consistent manner each time an identical sound—such as a speech syllable—is heard. The extent to which this ideal synchrony is achieved can be gauged with the auditory brainstem response to a complex sound (cABR).

There is a systematic relationship between neural response consistency and reading skills (Hornickel et al., *J Neuroscience*, 2013). Additionally, there is an intriguing corollary to the idea of synchrony and auditory processing in auditory-motor coupling. It has been discovered that the better you are at keeping a beat—for example accurately tapping along to a metronome or tapping out a beat in music—the better you are at reading. Using the same cABR response consistency measure, we found that the link between reading and auditory-motor synchronization in children is consistent biological processing of sound (Tierney & Kraus, *J. Neuroscience*, 2013). Thus, neural synchrony can serve as a focus in assessment and rehabilitation of individuals with auditory processing disorders across the lifespan. Interventions such as music training, computer-based auditory training or assistive listening devices in the classroom can bolster auditory processing skills by driving consistent neural synchrony in the auditory system.

Knowles Center Symposium
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Title: Unilateral Hearing Loss in Children
Speaker: Judith E. C. Lieu, MD MSPH

Abstract

Although bilateral hearing loss in children has long been recognized to have the potential to adversely affect speech-language acquisition, literacy, and educational attainment, unilateral hearing loss (UHL) in children has been often ignored as a possible obstacle in the development of communication skills. Several important studies from the 1980s and 1990s raised the specter that UHL in children was not as benign as common wisdom suggested. Research from the past 5 years has documented that in elementary school-aged children, UHL was associated with significantly poorer oral language skills and vocabulary IQ scores. Children with UHL were at higher risk of receiving individualized educational plans in school and speech therapy. Quality of life in children may also be negatively affected by UHL, similar to children with bilateral hearing loss. Although a growing number of audiologists, deaf educators, and otolaryngologists acknowledge the problems that many children with UHL experience, the larger medical and educational communities still tend to discount UHL as a problem for children. This presentation will review the speech-language, educational and behavioral consequences of unilateral hearing loss in children. Possible reasons for these findings will be explored from the context of research into the differences in brain interconnections with UHL.

The multidimensional effects of hearing loss on word learning in children
Andrea Pittman

There is evidence that the listening and learning strategies of children with hearing loss compete with one another such that they are not able to detect and learn new information as well as their age-matched or hearing-matched peers. This competition may be responsible, in part, for the 2-year difference in vocabulary development between children with mild-to-moderate hearing losses and children with normal hearing. Our work focuses on understanding the listening and learning strategies of these children in an effort to help them make the most of every opportunity to learn new information. Results from several recent studies will be discussed with particular attention on factors that affect their ability to recognize familiar words and to detect and learn new words.

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New therapies for otitis media

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Otitis media (OM) is the most frequent cause of hearing loss in children. It is also the most common etiology of hearing loss for all ages worldwide, due to untreated disease in developing countries where it causes an estimated 50,000 deaths/year. Current treatments for OM include systemic antibiotics, tympanostomy tubes and adenoidectomy, each of which have drawbacks and side effects. There is therefore a need for novel therapies to treat this condition.

To develop new treatments, we have explored the molecular substrates of middle ear defense against infection and pathogenic host responses that contribute to sequelae of the disease. Using whole-genome gene arrays we identified genes and gene networks that play a critical role in different forms of OM. This in turn has led to studies in gene deletion models, which clarify the role of critical genes, and to potential therapeutic interventions. We have identified important mediators of mucosal hyperplasia and leukocytic infiltration, including chemokines and novel sentinel molecules. When supplemented by exogenous application, these mediators enhance recovery and reduce pathogenic sequelae of OM in animal models.

Finally, we have used molecular methods to identify a novel means of delivering therapeutic agents to the middle ear. Sequential biopanning of bacteriophage peptide display libraries was employed to identify a family of peptides with the capacity to transit the intact membrane, and deliver cargo into the tympanic cavity. Further developed, this will provide a simple and noninvasive method for local delivery of pharmaceuticals or gene therapy for the treatment of OM.

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Neural Synchrony: A Developmental Perspective
Jason Sanchez

Abstract 2

Auditory brainstem neurons fire synchronized action potentials by “locking” to a specific phase of incoming sound waves. This neural synchrony depends on specialized time coding properties found in brainstem neurons and is important for normal auditory temporal processing. Animal research has shown that potassium channels contribute to time coding abilities in the mature cochlear nucleus, a first order brainstem structure. However, the extent to which they regulate time coding in the developing cochlear nucleus is lacking. My talk will address this by giving a developmental perspective on time coding properties in the avian cochlear nucleus. Our research shows that action potential speed, reliability and phase locking improves with maturation due in part to a significant upregulation in potassium channel conductances. When potassium channels are blocked, action potentials become significantly slower and less reliable, resulting in poorer phase locking. Deficits in a neuron’s ability to phase lock and fire synchronized action potentials to sound are thought to underlie aspects of auditory (dys) synchrony and central auditory processing disorders. Our results suggest that the development of potassium channels contributes to specialized neural features critical for auditory time coding, phase locking and neural synchrony in the cochlear nucleus.

Mild hearing loss disrupts the maturation of auditory cortex function

Dan Sanes, New York University

Abstract

The emphasis of deafness research has been to understand the consequences of permanent hearing loss. However, auditory processing deficits are also associated with transient hearing loss during childhood, and may persist for years, long after normal audibility is restored. One explanation for the persistence of auditory processing deficits is that transient hearing loss causes irreversible changes to cellular properties within the central nervous system that may lead to degraded stimulus encoding. I will present evidence supporting each of these assertions. Following a transient period of mild hearing loss during development, we find long-lasting impairments of synaptic and membrane properties within the gerbil auditory cortex. These neural deficits are found only when the transient hearing loss occurs during a well-defined developmental critical period. Furthermore, an identical manipulation leads to a perceptual deficit on an auditory task, amplitude modulation detection. The behavioral deficit is closely correlated with a diminished cortical representation of amplitude modulated stimuli, recorded while animals perform the auditory task. Together, these findings suggest that temporary periods of mild-to-moderate hearing loss, when induced during a critical period of development, cause persistent deficits to central auditory function that may impair auditory perception despite the return of normal peripheral function.