Review Article

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Speech in noise: hearing loss, neurocognitive disorders, aging, traumatic brain injury and more

Abstract

In this article, we explore and report the prevalence of speech in noise difficulties across multiple patient populations and reveal and speculate on management of the same. Speech in Noise problems is commonly associated with sensorineural hearing loss. The inability to understand speech in noise is often associated with, and attributed to, sensorineural hearing loss. However, some 12-15% of adults with normal hearing thresholds (i.e., pure tones) have difficulty hearing and struggle to understand speech in noise. Many of these same symptoms are present in people with neurocognitive disorders, advanced age, traumatic brain injury and more. As such, we recommend speech in noise testing on all adults who report these same difficulties. Further, once speech in noise difficulty has been objectively identified and quantified, an appropriate goal would be to improve the patient's speech in noise ability through aural rehabilitation, as well as modern technological advances.

Volume 10 Issue 4 - 2018

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Received: May 31, 2018 | **Published:** July 13, 2018

Introduction

When people complain about not being able to listen or attend to a primary speaker, or when they report difficulty understanding speech in noise (SIN), audiologists should test and diagnose deeper than a simple audiogram. Indeed, simply hearing pure tones at normal loudness levels does not predict or correlate with understanding speech in noise. A comprehensive audiologic evaluation which includes acoustic reflexes, SIN tests, auditory brainstem response, otoacoustic emissions and more is often useful and worthy of exploration, to lead the clinician to a detailed and patient-specific diagnosis, as well as to set appropriate and realistic aural rehabilitative goals.

For "traditional" audiology patients, SIN complaints are common among people with sensorineural hearing loss such as presbycusis and/or noise induced hearing loss, as well as people with auditory processing disorders, auditory neuropathy spectrum disorder, synaptopathy and more. Therefore, it seems apparent a complete audiometric evaluation should include speech-in-noise testing. Likewise, a complete listening and/or communication assessment should include listening difficulties, a history of cognitive challenges or changes, as well as queries regarding traumatic brain injury (TBI), dementia or other neurocognitive disorders (NCDs). Many individuals with acquired cognitive challenges such as dementia, cognitive decline, traumatic brain injury (TBI), Alzheimer's disease (AD) and other Neurocognitive Disorders (NCDs) report SIN difficulties. Tremblay and colleagues[1] reported twelve percent of normal hearing adults experience difficulty attending to the primary speaker in the presence of multi-speaker babble. Hannula et al.[2] reported up to 21% of normal hearing adults have difficulty following conversations which occur in noise. Thus, SIN difficulties represent a vast and common symptom across multiple disorders and etiologies.

Hearing and listening

Many people confuse hearing and listening. Hearing, is simply perceiving sound and is reflected by thresholds on an audiogram; a graph of intensity by frequency. Listening is the ability to make sense of sound by assigning meaning to it. Clearly, one must hear before one can listen. That is, sounds must be audible and detected before the brain can assign meaning. The most common type and degree of hearing loss is bilateral sensorineural high frequency hearing loss (usually age-related hearing loss, often similar to, and often, indistinguishable from hearing loss due to noise exposure). However, patients with these types of hearing loss do not typically complain about sounds not being loud enough. Rather, they complain about the inability to understand speech in noise (SIN), which is (arguably) more of a listening problem than a hearing problem. The essence of the SIN problem is not that sound is absent; it is the inability of the brain to organize and apply meaning to the perceived sounds. Further, SIN problems can be present with or without hearing loss. That is, SIN problems might be thought of as a reflection of the brain's inability to organize and decode sounds.[3]

Listening is where hearing meets brain

In the current audiology literature, the impact of hearing loss on NCDs is under intense investigation. Recent findings indicate untreated hearing loss often has a statistically significant, negative impact on quality of life and hearing loss has been identified as a modifiable risk factor for dementia.[5,7] Indeed, among adults with reported normal audiometric hearing, some 12 to 15% report difficulty listening.[2,7] The potential etiologies of hearing difficulty and/or speech in noise problems in the presence of normal hearing sensitivity is vast an includes; auditory processing disorders, spatial hearing disorders, central presbycusis, obscure auditory dysfunction (OAD), King-Kopetzky syndrome (KKS), auditory disability with normal hearing, idiopathic discriminatory dysfunction, hidden hearing loss (HHL), auditory neuropathy, deficits in auditory temporal processing, age-related factors affecting neural synchrony, synaptopathy, and more.[7]

When people complain about not being able to listen, or they are unable to attend to the primary speaker, or they report difficulty understanding speech in noise (SIN), a deeper analysis is recommended In addition to a complete audiometric evaluation, we recommend dementia screenings in appropriate patients (21, 79) to facilitate appropriate referrals, to better manage expectations and counsel as to realistic outcomes, and in referred patients, to offer an improved opportunity for early diagnosis and intervention, to effectively manage the primary complaint.[8]

Improving the SNR in the presence of normal peripheral hearing

Roup, Post, and Lewis[9] investigated the effectiveness of mild amplification for adults with normal hearing thresholds and subjective hearing difficulties. Their control group consisted of 20 adults with an average age of 22 years, the experimental group consisted of 17 adults with an average age of 31 years. Five of the 17 adults in the experimental group were diagnosed with Traumatic Brain Injury (TBI). Measurements were obtained using Hearing Handicap Inventory for Adults (HHIA), the Auditory Processing Questionnaire (APQ), the SCAN, Gaps in Noise Test, 500 Hz Masking Level Differences, Dichotic Digit Tests and the Speech Perception in Noise (SPIN) test at multiple signals to noise ratios (SNRs). Subjects were fitted with receiver-in-the-canal (RIC) hearing instruments with adaptive directional microphones and noise reduction circuits enabled. Hearing aids were worn about 4 hours daily for four weeks. The authors note significant differences between the two groups upon conclusion of the study. The experimental group (fitted with mild gain hearing aids) demonstrated improvement on the HHIA and APQ, as well as the SPIN test. Roup, Post, and Lewis[9] note mild gain hearing aids (with noise reduction and directional microphones) are a viable option for people with normal hearing and subjective hearing difficulties.[9]

Post, Roup, and Lewis[10] reported an increasing body of evidence demonstrating that adults with normal hearing sensitivity may report substantial difficulty understanding speech in complex listening situations. They report some hearing care professionals have fitted personal mild-gain amplification as an option to help these adults. Their results show that participants with normal hearing who received mild gain amplification demonstrated significant improvements with regard to their hearing handicap, as well as improvements in self-perceived auditory processing difficulties, and improved speech-in-noise performance, when compared to pre-fitting baseline measures. They report that for some adults with normal hearing sensitivity, hearing aid amplification may provide benefit with regard to reducing the difficulty of understanding speech in noise.[10]

Shojaei et al.,[11] report the critical role of SNR for speech perception in the elderly and state the SNR is the most effective physical characteristic of speech sounds for understanding SIN. They note that older adults require a 3-4 dB more advantageous SNR than younger adults to achieve the same understandsame understanding. Smaldino & Crandell[12] reported that children with normal hearing require a 10 dB better SNR than do adults, to achieve the same/similar performance.

Saunders et al.,[13] reported TBI can impact the central auditory nervous system leading to speech understanding problems which are disproportionate to the often reported normal hearing thresholds. As such, FM systems allowed better speech perception in noise for some veterans with blast exposure.[13]

Beck, Doty-Tomasula and Sexton[14] reported that for many people with Auditory Processing Disorders (APDs), improved access to the auditory signal is paramount. They noted the advantages of FM systems includes a realized reduction in distance (between the talker and listener), reduced reverb and reduced background noise, resulting in a more realistic/improved auditory signal and an improved SNR, allowing easier, less stressful and more enjoyable listening.[14]

Jensen reported 29 people with normal hearing (for their age group, average age approximately 66 years) and addressed listening effort (as measured by pupillometry) and speech understanding in noise. She notes there is a point at which people with normal hearing and people with hearing loss give up trying to listen, as the task becomes too difficult.She reported that in challenging acoustic situations, people with hearing loss give up more readily than people with normal hearing. That is, people with normal hearing can participate in more challenging SNRs than people with hearing loss. However, specific modern hearing aid technologies have been shown to increase the SNR with a concomitant decrease in the amount of listening effort required. These technologies allow people with hearing loss to participate in more deleterious SNRs, than would have otherwise been expected (5). Therefore, people with hearing loss who use these technologies, may be able to communicate more effectively in difficult listening situations.

Hearing loss and cognitive decline

Cognitive differences have been noted between older adults with hearing loss and matched groups without hearing loss.[15,16] Like many other ailments, whether or not an individual experiences cognitive decline associated with aging is highly individualized. However, recent literature suggests it is likely that a history of brain injury and a history of hearing loss increase the likelihood of cognitive decline.[17]

Lin and colleagues[18] stated, "Hearing loss is independently associated with accelerated cognitive decline and incident cognitive impairment in community-dwelling older adults" [18 p.1]. Pichora-Fuller & Kramer[19] stated, "we hear with our ears, we listen with our brains, and we exert listening effort because we are motivated to communicate" [19 p.4S]. Edwards[20] reported hearing loss and cognitive function interact via both top-down (i.e., cognitive) and bottom-up (i.e., sensory) processes. He stated the effects of hearing ability on cognitive function has been well-documented. Further, Edwards[20] reported directional microphone technologies and noise reduction algorithms improve reaction time, presumably based on a reduced cognitive load. As such, a reduced cognitive load potentially supports improved comprehension, improved memory access, and improved memory storage of information delivered via audition, as well as increased working memory capacity. and increased working memory capacity. Listening fatigue is also reduced via modern hearing aid technology presumably due to reduced listening effort. Edwards[20] reported the ability of modern hearing aids to capture and deliver spatial cues is important regarding locating sound sources and using spatial separation to improve speech understanding.

Untreated hearing Loss: a medical issue and a modifiable risk factor for NCD

In a new report of more than 7000 people, hearing loss was statistically significantly associated with all-type workplace and non-workplace injuries.[6] Hearing loss has also been associated with increased use of emergency department visits for males and females. Thus, is seems clear, hearing loss is a medical issue, untreated hearing loss has a multitude of medical and social consequences, and indeed, "hearing care is healthcare".[21]

Livingston et al.,[22] details nine potentially reversible (i.e., modifiable) population attributable fractions (PAFs, or risk factors) linked to dementia. That is, if one were to reduce or avoid these PAFs, their risk of dementia would decrease. They reported isolation, or lack of social contact has a PDF of 2.3%. Lack of exercise and lack of

Citation: Beck DL, Larsen DR, Bush EJ. Speech in noise: hearing loss, neurocognitive disorders, aging, traumatic brain injury and more. *J Otolaryngol ENT Res.* 2018;10(4):199–205. DOI: 10.15406/joentr.2018.10.00345

physical activity have a PAF of 2.6%, high blood pressure, type 2 diabetes & obesity combined have a PDF of 4%, depression has a PDF of 4%, smoking has a PDF of 5.5%, less education has a PAF of 7.5% and of note, hearing loss has a PAF of 9.1%. The evidence suggests approximately a third of dementia cases might be modifiable and they suggest anti-hypertensive medications (in appropriate patients), the consumption of a Mediterranean Diet (more fruits, veggies, nuts, beans, fish…) and regular exercise, as well as cognitive and brain training protocols and social activities are neuroprotective.[22]

Weinstein[23] adds that by partially restoring communication ability, modern hearing aid amplification may serve as a buffer to maintain cognitive ability. Regarding social and emotional loneliness and depression, hearing aid amplification may improve the quality and quantity of social interactions, thus enabling participation in cognitively-stimulating activities.[23] Amieva and colleagues[16] reported, "hearing loss is associated with accelerated cognitive decline in older adults" and suggested "hearing aid use attenuates such decline" [16 p.2099].

Neuro cognitive disorders

Untreated hearing loss may eventually be recognized as a substantial pre-cursor to dementia and/or cognitive decline. Although people use "dementia" and "Alzheimer's Disease" (AD) interchangeably in common usage, they are not synonymous. AD is a specific type of dementia, and is the most common of all dementias. Of note, the term 'dementia' does not indicate a specific disease. Rather, it indicates a range of symptoms such as a decline in memory or thinking skills significant enough to negatively impact a person's ability to perform everyday activities. Dementia has recently been re-named by the American Psychiatric Association (APA) in their DSM-5 as Neuro Cognitive Disorder (NCD).[23] Thus, the term "NCD" will be primarily used throughout the remainder of this article.

Modifiable NCDs?

The Mayo Clinic[24] reported certain causes of dementia [NCD] or dementia-like symptoms are reversible, such as those originating from infections and immune disorders, metabolic and endocrine abnormalities (e.g. thyroid problems), nutritional deficiencies (e.g. a vitamin B-12 or vitamin D deficiency), reactions to medications, depression, diabetes, smoking, cardiovascular factors, subdural hematomas or traumatic brain injuries (TBIs) and anoxia or sleep apnea.[24] Irreversible and progressive contributors appear to originate in association with age, genetics, Alzheimer's disease, mild cognitive impairment (MCI), Lewy body dementia, and frontotemporal dementia. Contemporary literature suggests AD is characterized by amyloid beta plaques and tau tangles facilitated by aging, genetics and multiple disease processes.[25]

The Livingston, Sommerlad & Orgeta[22] report stated the most significant contributor to dementia is aging and age related processes. However, in the last decade, researchers have identified multiple modifiable contributors to NCD and/or AD and exploration and documentation of these same factors continues.[22]

Dolgin[22] reported that after more than 200 ineffective pharmaceutical trials, the hope of finding a medication to effectively stop or reverse Alzheimer's has faded. However, the "one intervention at a time approach" has been enthusiastically replaced by a combined synergistic constellation of interventions, addressing a variety of modifiable risk factors and success has been reported.[26]

The Finnish geriatric intervention study to prevent cognitive impairment and disability involved more than 1200 seniors with Mild Cognitive Impairment (MCI, a common precursor to Alzheimer's and other forms of NCD) evaluated the combined impact of diet, physical, mental, and social activity on cognitive ability, as compared to standard medical practice.[27] After two years, this multi-domain lifestyle intervention proved to be significantly more effective in slowing or delaying cognitive decline than standard medical practice. Neuropsychological test scores improved for the majority of participants and performance on complex memory tasks was 40% higher in the intervention group. Executive functioning was 83% better in the intervention group, and processing speed was 150% higher in the intervention group. Of note, the control group (standard medical practice) had a 30% greater risk for cognitive decline. Indeed, the intervention group experienced an overall improvement in vitality, social function and general health, while the control group continued to decline overall.[27,28]

Furthermore, Fotuhi and colleagues[29] reported a 12-week study of 127 seniors diagnosed with MCI using a multi-pronged "brain fitness" approach to address multiple risk factors. Their approach included a Mediterranean diet, physical and mental exercise, richer social interaction, meditation, sleep enhancement, and other stress management strategies. Of the 127 participants, 84% showed significant improvements in three or more areas of cognitive functioning. Of the 17 who had pre-and-post MRIs, more than half experienced volume growth in their hippocampus (i.e., the brain center for emotion and memory) thus supporting the idea that various multi-tiered, health-oriented interventions may be beneficial with respect to cognitive function.[29]

Traumatic brain injury

Traumatic brain injury (TBI) is an often-neglected, public health problem with more than one million injuries occurring in the United States annually.[30,31] TBIs often result in lifelong consequences and outcomes which negatively impact a person's physical abilities, cognitive skills, and emotional well-being.[30,32] TBI survivors' quality of life (QOL) frequently is diminished due to physical, cognitive, and emotional outcomes, as well as their frequent, resultant inability to return to work, school, or other pre-injury activities.[30]

The traditional view of TBI recovery was that once the survivors' outcomes plateaued, they were stable.[33] Recently, researchers have found this is not the case based on large longitudinal data sets. The CDC-initiated TBI Surveillance National Database and the National Institute on Disability and Rehabilitation's Traumatic Brain Injury Model Systems (TBIMS) are national databases.[34] These databases provide extraordinary opportunities for research due to the vast number of individuals represented and the follow-up measures reported.[35] The CDC currently has nearly 30 years of data[34] and the TBIMS[35] contains information on over 12,000 individuals with TBI.[36] Many researchers have conducted long-term follow-up studies and have recently began to publish these results.[37–42] Contrary to the traditional view, this contemporary data suggest that at 10 years post-injury, approximately 30% of survivors with moderate or severe injuries experience a decline in their outcomes. Furthermore, a subset of these survivors experienced degenerative and progressive motor and/or cognitive dysfunction.[43,44]

Researchers have shown associations between a history of TBI and dementia. Gardner and colleagues[35] studied 164,661 patients admitted to hospitals with various types of traumatic injuries, 31.5% had a TBI. Between one and seven years after the traumatic incident, the researchers analyzed records for a diagnosis of dementia. They reported that 8.4% of those with a TBI later developed dementia, compared to 5.9% of the patients who admitted for a traumatic injury that did not include TBI. Hazard ratio calculations indicated ([HR], 1.46; 95% CI, 1.41-1.52; P < .001), those with a history of TBI are at increased risk for dementia. Adjusted analyses, further suggested that for both moderate and severe TBIs, the increased risk of dementia was significant across all ages (55-74 yr.), whereas those with mild TBI had an increased risk after they turned 65 years of age[35] suggesting that TBIs may increase the likelihood of young onset-dementia.[44] Young onset-dementia is defined as dementia diagnosed prior to age 65.[35,45] Corrigan & Hammond[33] reported an incident of moderate or severe TBI is also associated with Parkinsonism.

Individuals who have had multiple mild TBIs (or concussions) have been the focus of contemporary research studies. Chronic Traumatic Encephalopathy (CTE), has garnered increased attention. CTE, defined as a disease with an etiology of repeated head trauma, was the focus of a 2017 New York Times article. The article reported findings from a study in which researchers examined deceased football players' brains. Eighty-seven percent of the players were diagnosed, posthumously, with CTE, including 110 of the 111 NFL players.[46] In 2018, researchers reported that 42 of 100 New England Patriots members of the first three Super Bowl teams have alleged concussions and brought legal action against the National Football League and a helmet manufacturer. These professional football players have reported symptoms of brain injury caused by repetitive head impacts from practices and professional sporting competitions.[46]

While some researchers hesitate to call CTE a type of dementia as there is not consensus on all of its diagnostic features,[44] there is some consensus on its description with notable commonalities to other types of NCDs. That is, CTE is a chronic, progressive, neurodegenerative condition which can affect individuals' cognitive performance, behavior, affect/mood, and sensory/motor skills.[47–49]

Studies have demonstrated those with mild TBI are at a 3-fold greater risk of dementia than un-injured counterparts[51] and males who sustained brain injuries at an early age were more likely to have young onset-dementia.[45] Nordstrom and colleagues[45] found that Swedish men (n=811,622) who served in the Army at an average age of 18, were at an increased risk for young onset-dementia three decades later, if they had incurred a TBI. This risk was not associated with AD, but was strongly associated with other types of NCD after only one mild TBI (HR=1.7; p<0.05) and more so after a severe injury (HR=2.6; p<0.05), after adjusting for premorbid cognitive functioning and alcohol abuse.[45]

Traumatic brain injury and hearing loss

Clinicians, medical professionals, case managers and others should ensure that complete audiometric evaluations (including speech in noise tests) are completed and that audiology-based evaluation and management are addressed and obtained early in the diagnostic and rehabilitation process so therapies addressing concomitant challenges (e.g., psychological, speech, language, educational/vocational, and emotional) are more impactful, effective and allow the individual to experience greater participation in therapeutic processes.[52,53] The United States has experienced a dramatic upsurge in TBI. Some of this may be due to a heightened awareness of brain injury[54] such as the recent media attention to CTE cited previously. Additionally, U.S. military personnel returning home from combat are more likely to have brain injuries (or Blast injuries) as well as hearing loss, tinnitus, or dizziness.[55]

Blast injuries are often associated with other concomitant health problems. Injuries sustained from improvised explosive devices (IEDs) account for 78% of battlefield head and neck injuries[56] and frequently result in brain injury.[51] Blast waves are often destructive to the auditory system as they impact gas and/or fluid-filled structures[57,58] and may cause middle and/or inner ear damage.[59] Xydakis and colleagues[60] estimated 35% of blast injury survivors had a tympanic membrane rupture. Further, research with Marines who sustained combat injuries during operation Iraqi Freedom II, revealed ear injuries are the most common singular combat injury.[61] Despite the frequency with which the ear is injured and a conductive hearing loss occurs, the most frequently experienced hearing loss is sensorineural.[62]

Civilian survivors of brain injuries frequently contend with hearing loss. In fact, hearing loss after pediatric brain injury occurred in 50% of survivors. Sixty-four percent of children with brain injuries had conductive hearing losses, half of which resulted from temporal bone fractures. Sensorineural hearing losses for this population were variable in terms of severity, and with respect to unilateral versus bilateral.[63]

A recent review of 99 veterans exposed to munition blasts, indicated that increasing the SNR via FM systems was useful. All veterans had hearing thresholds consistent with normal or mild hearing loss, all scored appropriately on the Mini Mental State Exam (MMSE), and all reported difficulty understanding speech in noise. The veterans who were issued a wireless FM system reported it was useful in work related meetings, as well as restaurants, lectures, while watching television and more. The authors reported that in some locations, veterans were issued low gain hearing aids with remote (wireless) microphones and the authors stated these were more or less equivalent to FM systems. The authors reported that in the lab, these systems are "highly effective at improving speech understanding in noise…".[67] Finally, the authors concluded that FM systems, or remote microphone systems via Blue Tooth, offer effective intervention for blast exposed veterans with normal or near-normal hearing and should be routinely considered as an intervention approach for this population when possible."

Hoover and colleagues reported that of 13 listeners (average age 47 years) who had experienced mild TBI (mTBI), 84% had SIN tests which revealed SIN deficits. In the control group of 11 listeners (average age 49 years) only 9% demonstrated SIN deficits. SIN The authors note that in addition to a thorough auditory and communications need assessment, the role of the audiologist may include counseling and the provision of technology to facilitate greater participation regarding skills of daily living and participation in rehabilitation.[76] Gallun, Papesh, and Lewis[77] noted an advantage for younger and middle-aged people who experienced TBI, is their willingness and enthusiasm to embrace technological advantages. They note audiologists who have worked with blast exposure patients with normal and near normal hearing have successfully prescribed minimal gain hearing aids for these people, with and without FM systems.[77]

Citation: Beck DL, Larsen DR, Bush EJ. Speech in noise: hearing loss, neurocognitive disorders, aging, traumatic brain injury and more. *J Otolaryngol ENT Res.* 2018;10(4):199‒205. DOI: 10.15406/joentr.2018.10.00345

Treatment options and considerations

Individuals with and without hearing loss who experience hearing difficulty and/or speech in noise difficult, and/or listening disorders, and/or those who have a history of TBI or NCDs, may receive significant benefit from an improved signal-to-noise ratio. Hearing difficulty and speech in noise problems are common ailments, which generally occur with hearing loss, but may be present with normal (or nearly normal) hearing thresholds. All of these symptoms are worthy of further exploration and patient-specific diagnosis and treatment plans are highly recommended. As the signal to noise ratio decreases (i.e., as background noise approaches and exceeds the loudness of the signal of primary interest) a considerable reduction of speech perception occurs.[66] Improved SNRs can be accomplished with modern hearing aids, remote microphones, assistive listening devices (ALDs) and more.[64,65]

Of course, deleterious SNRs are not exclusively a problem of the elderly or those with NCDs, the same problem has had a consistent negative influence in modern school systems. Children in school must exert considerable listening effort as the SNR deteriorates, thus leaving fewer cognitive resources available ffor the learning task at hand.[66]

Indeed, vast technological offerings in modern hearing aids are available through which patients can wear cosmetically pleasing and sophisticated hearing aids with and without wireless remote microphones, to enhance the SNR, while offering excellent sound quality, excellent user satisfaction and improved speech in noise results. Beck & Le Goff[5] suggested that improving the SNR might ultimately be considered among the most pragmatic goals of modern hearing aid fittings. Their recently reported results (with regard to user satisfaction, improved word recognition and speech in noise improvements) are substantially better than results obtained just a few years earlier.

People with sensorineural hearing loss and many users of traditional hearing aids struggle to understand speech in noise. Of note, those with better cognitive skills often achieve improved outcomes, as cognitive skills contribute significantly to listening ability.

Contemporary, sophisticated HAs offer processing strategies which increase the quality of sound,[72] user satisfaction[73] and reduce noise levels[5] while availing an improved SNR which may reduce brain processing load (i.e., listening effort) while listening or attending to speech in noise.[74] These strategies can provide easier and more effective listening in difficult listening situations (where social interaction takes place) such as restaurants, cafes or at a family dinner at home, and are often characterized by annoying, distracting and elevated noise levels.[75–79]

Summary & discussion

Hearing and listening are often confused as synonymous, yet they are unique processes. To hear is to perceive sound. Listening is dependent on hearing, as listening is the ability to make sense of sound. Listening is arguably a derivative of cognitive ability, and cognitive ability results from the vast interactions of many regions of the brain and central nervous system.

Finally, with regard to hearing aid amplification and cognitive health, it appears imperative to provide the brain with maximal auditory information. Technologies are available which substantially reduce background noise, increase the signal-to-noise ratio, avail improved sound quality, and of tremendous importance, some sophisticated hearing aids maintain realistic and naturally occurring spatial cues, so the wearer knows where to focus their attention during speech in noise challenges.[5,72,73] Improving these factors is essential to improving the brain's ability to listen (i.e., derive meaning from sound), particularly in deleterious background noise. Amplification interventions (such as those noted above) may improve the SNR, which can enhance an individuals' quality of life, and may soon prove to serve a neuroprotective role with regard to cognitive decline associated with hearing loss and some neurocognitive disorders.

Acknowledgments

None.

Conflict of interest

The author declares there is no conflict of interest.

References

1. Tremblay KL, Pinto A, Fischer M, et al. Self-reported hearing difficulties among adults with normal audiograms: The beaver dam offspring study. *Ear Hear.* 2015;36(6):e290–e299.
2. Hannula S, Bloigu R, Majamaa K, et al. Self-reported hearing problems among older adults: Prevalence and comparison to measured hearing impairment. *J Am Acad Audiol.* 2011;22(8):550–559.
3. Liberman MC, Epstein MJ, Cleveland SS et al. Toward a differential diagnosis of hidden hearing loss in humans. *PLoS One.* 2016;11(9):e0162726.
4. Beck DL, Flexer C. Listening is where hearing meets brain...in children and adults. *J Hear Sci*. 2011;18(2):30–35.
5. Beck DL, Le Goff N. Contemporary hearing aid amplification: Issues and outcomes in 2018. *J Otolaryngol ENT Res*. 2018;10(1):00303.
6. Mick P, Foley D, Lin F, et al. Hearing difficulty is associated with injuries requiring medical care. *Ear Hear. Forthcoming*. 2018;39 (4):631–644.
7. Tremblay KL, Pinto A, Fischer, ME, et al. Self-reported hearing difficulties among adults with normal audiograms: the beaver dam offspring study. *Ear Hear.* 2015;36(6):e290–e299.
8. Saunders GH, Frederick MT, Arnold ML, et al. A randomized controlled trial to evaluate approaches to auditory rehabilitation for blast-exposed veterans with normal or near-normal hearing who report hearing problems in difficult listening situations. *J Am Acad Audiol.* 2018;29(1):44–62.
9. Roup CM, Post E, Lewis J. Mild-Gain hearing aids as a treatment for adults with self-reported hearing difficulties. *J Am Acad Audiol.* 2018;29(6):477–494.
10. Post E, Roup CM, Lewis J. Mild-gain hearing aids as treatment for adults with subjective hearing difficulties. Department of Speech and Hearing Sciences, The Ohio State University. PP 1027, Audiology NOW! 2016.
11. Shojaei E, Ashayeri H, Jafari Z, et al. Effect of signal to noise ratio on the speech perception ability of older adults. *Med J Islam Repub Iran.* 2016;30:342.
12. Smaldino JJ, Crandell CC. Classroom amplification technology: Theory and practice. *Lang Speech Hear Serv Sch.* 2000;31(4):371–375.
13. Saunders GH, Frederick MT, Chisolm TH, et al. Use of a frequency modulated system for veterans with blast exposure, perceived hearing problems and normal hearing sensitivity. *Sem Hear.* 2014;35(3):227–238.

14. Beck DL, Doty Tomasula M, Sexton J. FM made friendly. *Audiology Online*. 2006.

15. Rosenblum G, Beck D. Over the counter hearing aids: Opinions and perspectives. *Int Hear Soc*. 2018:35–40.

16. Amieva H, Ouvrard C, Giulioli C, et al. Self-reported hearing loss, hearing aids, and cognitive decline in elderly adults: A 25-year study. *J Am Geriatr Soc*. 2015;63(10):2099–2104.

17. Salthouse T A. Initiating the formalization of theories of cognitive aging. *Psychol Aging*. 1988;3(1):3–16.

18. Lin FR, Yaffe K, Xia J, et al. Hearing loss and cognitive decline among older adults. *JAMA Intern Med*. 2013;173(4):293–299.

19. Pichora Fuller MK, Kramer SE. Ericksholm workshop on hearing impairment and cognitive energy. *Ear Hear*. 2016;37(Suppl 1):1S–4S.

20. Edwards B. A model of auditory-cognitive processing and relevance to clinical applicability. *Ear Hear*. 2016;37(Suppl 1):85S–91S.

21. Beck DL, Weinstein BE, Harvey M. Issues in cognitive screenings by audiologists. *Hearing Review*. 2016;23(2):36.

22. Livingston G, Sommerlad A, Orgeta V. Dementia prevention, intervention, and care. *Lancet*. 2017;390(10113):2673–2734.

23. Weinstein BE. Dementia and age-related hearing loss–Part II. *Hear J*. 2017;70(11):26–30.

24. Dementia: Symptoms & causes [internet]. The Mayo Clinic; c1998–2018.

25. Alzheimer's disease [internet]. Chicago (IL): Alzheimer's Association; c2018.

26. Dolgin E. The new offensive on alzheimer's disease: Stop it before it starts. *Newsweek*. 2017.

27. Ngandu T, Lehtisalo J, Solomon A, et al. A 2 year multidomain intervention of diet, exercise, cognitive training, and vascular risk monitoring versus control to prevent cognitive decline in at-risk elderly people (FINGER): A randomised controlled trial. *Lancet*. 2015;385(9984):2255–2263.

28. Kivipelto M, Mangialasche F, Ngandu T. Can lifestyle changes prevent cognitive impairment? *Lancet Neurol*. 2017;16(5):338–339.

29. Fotuhi M, Lubinski B, Trullinger M, et al. A personalized 12-week brain fitness program for improving cognitive function and increasing the volume of hippocampus in elderly with mild cognitive impairment. *J Prev Alzheimers Dis*. 2016;3(3):133–137.

30. Langlois JA, Rutland-Brown W, Wald M. The epidemiology and impact of traumatic brain injury: A brief overview. *J Head Trauma Rehabil*. 2006;21(5):375–378.

31. Thurman DJ, Alverson C, Dunn KA, et al. Traumatic brain injury in the united states: A public health perspective. *J Head Trauma Rehabil*. 1999;14(6):602–615.

32. Raymont V, Salazar AM, Krueger F, et al. "Studying injured minds"–the Vietnam head injury study and 40 years of brain injury research. *Front Neurol*. 2011;2:15.

33. Corrigan JD, Hammond FM. Traumatic brain injury as a chronic health condition. *Arch Phys Med Rehabil*. 2013;94(6):1199–1201.

34. Brooks JC, Strauss DJ, Shavelle RM, et al. Long-term disability and survival in traumatic brain injury: Results from the national institute on disability and rehabilitation research model systems. *Arch Phys Med Rehabil*. 2013;94(11):2203–2209.

35. Gardner RC, Yaffe K. Traumatic brain injury may increase risk of young onset dementia. *Ann Neurol*. 2014;75(3):339–341.

36. Traumatic brain injury model systems national data base [internet]. *National Data and Statistical Center*. 2015.

37. Harrison Felix C, Whiteneck G, DeVivo M, et al. Mortality following rehabilitation in the Traumatic Brain Injury Model Systems of Care. *NeuroRehabilitation*. 2004;19(1):45–54.

38. Harrison Felix C, Whiteneck G, DeVivo M, et al. Causes of death following 1 year post injury among individuals with traumatic brain injury. *J Head Trauma Rehabil*. 2006;21(1):22–33.

39. Harrison Felix C, Whiteneck G, Jha A, et al. Mortality over four decades after traumatic brain injury rehabilitation: A retrospective cohort study. *Arch Phys Med Rehabil*. 2009;90(9):1506–1513.

40. Harrison Felix CL, Hawley LA, Brown AW. *Life expectancy and wellness*. In: ND Zasler, et al. editors. *Brain injury medicine: principles and practice*. Demos Medical Publishing. *New York*. 2013;319–333.

41. Harrison Felix C, Kreider SE, Arango Lasprilla JC, et al. Life expectancy following rehabilitation: A NIDRR traumatic brain injury model systems study. *J Head Trauma Rehabil*. 2012;27(6):E69–E80.

42. Shavelle RM, Strauss D, Whyte J, et al. Long-term causes of death after traumatic brain injury. *Am J Phys Med Rehabil*. 2001;80(7):510–516.

43. Faden AI, Loane DJ. Chronic neurodegeneration after traumatic brain injury: Alzheimer disease, chronic traumatic encephalopathy, or persistent neuroinflammation? *Neurotherapeutics*. 2015;12(1):143–150.

44. McKee AC, Stein TD, Kiernan VE, et al. The neuropathology of chronic traumatic encephalopathy. *Brain Pathol*. 2015;25(3):350–364.

45. Nordstrom P, Michaelsson K, Gustafson Y, et al. Traumatic brain injury and young onset dementia: A nationwide cohort study. *Ann Neurol*. 2014;75(3):374–381.

46. Mez J, Daneshvar DH, Kiernan PT, et al. Clinicopathological evaluation of chronic traumatic encephalopathy in players of american football. *JAMA*. 2017;318(4):360–370.

47. Kanthasamy A, Anantharam V, Jin H. *Chronic traumatic encephalopathy*. In: Ikezu T, Gendelman H, editors. Neuroimmune Pharmacology. *Springer International Publishing*. 2016;599–620.

48. McKee AC, Stein TD, Kiernanr PT, et al. The neuropathology of chronic traumatic encephalopathy. *Brain Pathol*. 2015;25(3):350–364.

49. Gardner RC, Perry DC. *Chronic traumatic encephalopathy*. In: Miller BL, Boeve BF, editors. Behavioral Neurology of Dementia. Cambridge (England): *Cambridge University Press*. 2014;400–415.

50. Lee YK, Hou SW, Lee CC, et al. Increased risk of dementia in patients with mild traumatic brain injury: A nationwide cohort study. *PLoS One*. 2013;8(5):e62422.

51. Langlois JA, Rutland-Brown W, Thomas KE. Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths. *National Center for Injury Prevention and Control*. 2004.

52. Lew HL, Weihing J, Meyers PJ, et al. Dual sensory impairment (DSI) in traumatic brain injury (TBI)–An emerging interdisciplinary challenge. *NeuroRehabilitation*. 2010;26(3):213–222.

53. Vander Werff KR, Rieger B. Brainstem evoked potential indices of subcortical auditory processing after mild traumatic brain injury. *Ear Hear*. 2017;38(4):e200–e214.

54. Marin JR, Weaver MD, Yealy DM, et al. Trends in visits for traumatic brain injury to emergency departments in the united states. *JAMA*. 2014;311(18):1917–1919.

55. Chandler D. Blast-related ear injury in current U.S. military operations: Role of audiology on the interdisciplinary team. *ASHA Leader*. 2006;11(9):8–9.

56. Owens BD, Kragh MD, John F, et al. Combat wounds in Operation Iraqi Freedom and operation Enduring Freedom. *J Trauma Acute Care Surg*. 2008;64(2):295–299.

57. Explosions and blast injuries: A primer for clinicians [Internet]. *Atlanta (GA): Centers for Disease Control and Prevention*. 2003.

58. Mayorga MA. The pathology of primary blast overpressure injury. *Toxicol.* 1997;121(1):17–28.

59. Patow CA, Bartels J, Dodd KT. Tympanic membrane perforation in survivors of a SCUD missile explosion. *Otolaryngol Head Neck Surg*. 1994;110(2):211–221.

60. Xydakis MS, Bebarta VS, Harrison CD, et al. Tympanic-Membrane Perforation as a Marker of Concussive Brain Injury in Iraq. *N Engl J Med*. 2007;375(8):830–831.

61. Gondusky JS, Reiter MP. Protecting military convoys in Iraq: An examination of battle injuries sustained by a mechanized battalion during operation Iraqi Freedom II. *Mil Med.* 2005;170(6):546–549.

62. Lew HL, Jerger JF, Guillory SB, et al. Auditory dysfunction in traumatic brain injury. *J Rehabil Res Dev.* 2007;44(7):921–28.

63. Zimmerman WD, Ganzel TM, Windmill IM, et al. Peripheral hearing loss following head trauma in children. *Laryngoscope.* 1993;103(1 Pt 1):87–91.

64. Nadol JB. Hearing loss. *N Engl J Med.* 1993;329(15):1092–1102.

65. Ferguson MA, Henshaw H. Auditory training can improve working memory, attention, and communication in adverse conditions for adults with hearing loss. *Front Psychol.* 2015;6:556.

66. Howard CS, Munro KJ, Plack CJ. Listening effort at signal-to-noise ratios that are typical of the school classroom. *Intl J Audiol*. 2010;49(12):928–932.

67. Shojaei E, Ashayeri H, Jafari Z, et al. Effect of signal to noise ratio on the speech perception ability of older adults. *MJIRI.* 2016;30:342.

68. Johnson EE, Dillon H. A comparison of gain for adults from generic hearing aid prescriptive methods: Impacts on predicted loudness, frequency bandwidth, and speech intelligibility. J *Am Acad Audiol.* 2011;22(7):441–459.

69. Foo C, Rudner M, Rönnberg J, et al. Recognition of speech in noise with new hearing instrument compression release settings requires explicit cognitive storage and processing capacity. *J Am Acad Audiol.* 2007;18(7):618–631.

70. Gatehouse S, Naylor G, Elberling C. Benefits from hearing aids in relation to the interaction between the user and the environment. *Int J Audiol.* 2003;42(Suppl 1):77–85.

71. Ng EH, Rudner M, Lunner T, et al. Effects of noise and working memory capacity on memory processing of speech for hearing-aid users. *Int J Audiol*. 2013;52(7):433–441.

72. Chasin M. A novel technique to improve amplified sound quality for both music and speech. *The Hearing Review [Internet]*. 2017.

73. Beck DL, Porath M. Consumer responses to the oticon opn hearing aid. *The Hearing Review [Internet]*. 2017.

74. Wendt D, Hietkamp RK, Lunner T. Impact of noise and noise reduction on processing effort: A pupillometry study. *Ear Hear.* 2007;38(6):690–700.

75. Beck DL, Behrens T. The surprising success of digital noise reduction. *Hearing Review.* 2016;23(5):20.

76. Hoover EC, Souza PE, Gallun FJ. Auditory and cognitive factors associated with speech in noise complaints following mild traumatic brain injury. *J Am Acad Audiol.* 2017;28(4):325–339.

77. Gallun FJ, Papesh MA, Lewis MS. Hearing complaints among veterans following traumatic brain injury. *Brain Inj.* 2017;31(9):1183–1187.

78. Jensen JJ. Closing a Gap to Normal Hearing. *Oticon White Paper.* 2018.

79. Beck DL, Weinstein BE, Harvey MA. Dementia Screening-A Role for Audiologists. Hearing Review. July, 2018