Dementia and the Hearing Healthcare Provider

The HHCP is uniquely suited to screen their patient's dementia risk as it relates to their information and auditory processing abilities, audiometric findings, and ability to hear and listen in complex listening environments.

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Introduction

Hearing healthcare providers (HHCPs) must hold the welfare of each patient paramount by incorporating evidence-based clinical judgement.[1,2] With more than 50 years of data[3] indicating decreased cognitive function associated with the presence of hearing loss, the HHCP has a responsibility to understand, incorporate, and provide services to assess the adult patient's risk of cognitive decline, including promoting the treatment of hearing loss which often positively impacts cognitive health[4,5] and may reduce the risk of dementia.[6]

The notion that hearing loss impacts cognitive function was first documented when deficits in memory recall were observed at higher rates in persons with hearing loss.[3] Nearly twenty years later, the increased prevalence of dementia was noticed in persons with hearing loss.[7,8] For a thorough review of the literature on hearing and cognition, see Beck and Clark,[9] Powell et al.[10] and Beck.[11]

The shift to integrate cognitive health into hearing healthcare was propelled by Lin and colleagues who demonstrated the correlation of increased risk of cognitive decline and dementia with hearing loss, i.e., the risk of cognitive decline and dementia increased an additional 130% per 10 dB drop in hearing (see **Figure 1**).[12-14] Additionally, the emerging relationship of speech-in-noise handicap with incident rates of dementia[15] have compelled many HHCPs to begin screening for cognitive decline (see the accompanying article by Weinstein, 2023, this edition of Hearing Review).

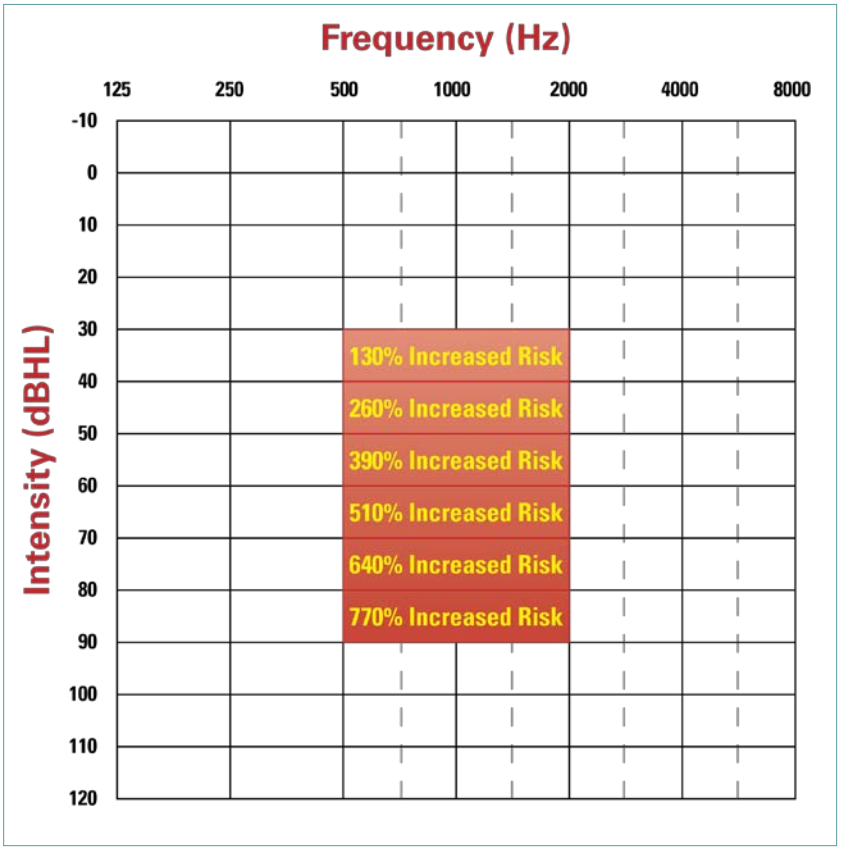

Figure 1. Dementia risk as it relates to hearing loss levels. Lin et al., (2011) documented the risk of developing all-cause dementia that increased an additional 130% per 10dB of hearing loss (as defined by pure tone average).

With 40% of all dementias considered potentially modifiable and 8.2% of all global dementia rates attributed to hearing loss,[16,17] the HHCP is uniquely positioned to potentially reduce the patient's risk of cognitive decline, or slow the progression of decline, thereby reducing conversion rates from Mild Cognitive Impairment (MCI) to Major Neurocognitive Disorder, e.g. dementia.

Understanding neurocognitive disorders as well as the age/genetic risks, the patient profile of the person with dementia (PWD), and the 12 modifiable lifestyle/health risk factors[17] may all prove beneficial for the HHCP to increase their professional services and to actively refer appropriate patients for diagnosis and management.

Mild and Major Neurocognitive Disorders

The diagnostic classification for a person with diminished cogni-

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tive function has been revised to either mild neurocognitive disorder (MiNCD) or major neurocognitive disorders (MaNCD) formerly known as Mild Cognitive Impairment (MCI) and dementia, respectively.[18] These newer terms clarify the presentation of the disorders by not including psychological illness and they reduce the stigma associated with the word dementia (i.e., dementia is derived from the Latin word meaning 'insane'). Each of these disorders goes beyond the typical deficits of aging, which often include slower recall, limited issues with concentration, and maintenance of the normal capacity to learn. Mild Neurocognitive Disorder (MiNCD) describes an individual whose cognitive deficits do not interfere with capacity for independence in everyday living and typically entail a mild, or modest decline from a previous level of performance. In contrast, Major Neurocognitive Disorder (MaNCD) does interfere with the ability to perform daily independent tasks, also known as activities of daily living (ADLs). The person with MiNCD is at risk for developing MaNCD, with approximately 20% of patients converting to MaNCD on an annual basis.[19]

Cognitive deficits associated with neurocognitive disorders typically affect at least 1 (in MiNCD) or 2 (in MaNCD) cognitive domains, including complex attention, executive function, learning and memory, language, perceptual - motor/visual, and social cognition.

- Complex Attention: Generally involves sustained attention, divided attention, selective attention, and information processing speed.
- Executive Function: Generally involves planning, decision-making, working memory, responding to feedback, error correction, overriding habits, and mental flexibility.
- Learning and Memory: Generally involves immediate short-term memory, working memory, recent memory (free recall, cued recall, and recognition memory), and long-term memory.
- Language: Generally involves expressive language (naming, fluency, grammar, and syntax) and receptive language.
- Perceptual (Motor/Visual): Generally involves the coordination of visual and motor functions for everyday tasks such as picking up the telephone, handwriting, and using a fork/spoon/knife.
- Social Cognition: Generally involves the ability to interact with others.

Although hearing abilities are not explicitly mentioned in any of the cognitive domains, the ability to hear (perceive sound) and listen (i.e., comprehend sound) are directly associated with complex attention (i.e., the ability to follow a conversation with visual or auditory distractions in the background),[20] executive functions (i.e., the ability to perceive what others are saying and appropriately respond),[21] learning and memory (i.e., the ability to instantly associate ongoing speech with auditory memory to determine meaning and intent),[22] language (i.e., receptive language impairments resulting from hearing loss and other etiologies)[23] and social cognition (i.e., the known correlations of hearing loss and social isolation in older adults).[24]

The Person With Dementia

In addition to the previously noted inability to competently perform ADLs, the signs and symptoms of the PWD (MaNCD) vary and may include memory loss, poor judgment, difficulty communicating with others, wandering, repeatedly asking questions, anomia, loss of interest, and difficulty completing everyday tasks.[25] These symptoms present gradually and represent a change from past behaviors. The HHCP provider must be acutely aware of these presentations and explore these behavioral changes during routine and thorough case history and clinical evaluations. It is important that these behavioral changes be reported to the primary care physician for diagnosis and management and to rule out pseudo-dementia or delirium, or pharmaceutical-induced causes, which may manifest as more typical MiNCD or MaNCD.

Clinical manifestation of the PWD, pseudo-dementia, and delirium can be similar; however, their etiology and treatment are considerably different. Pseudo-dementia, or potentially reversible dementia, is often the result of adverse drug interactions, emotional disorders (e.g., schizophrenia), sensory loss (e.g., visual or hearing impairment), infection (e.g., urinary tract infection), nutritional deficits (e.g., vitamin B12 deficiency), metabolic changes (e.g., thyroid disease) or intracranial pathology (e.g., head trauma or tumor).[26] Delirium, on the other hand, is a rapid (i.e., days to weeks) onset change in mental state and awareness resulting from acute brain dysfunction. A person with MaNCD can exhibit episodes of delirium, and these observations must be addressed immediately if suspected.[27]

The Prevalence of MaNCDs

MaNCD's are a collection of heterogenous progressive neurodegenerative diseases that directly impact cognition and share a common feature of cerebral atrophy. The four most common types of dementia, include Alzheimer's disease (AD), Vascular Dementia (VaD), Lewy Body Dementia (LBD), and Frontotemporal Lobar Degeneration (FTLD).

AD, the most common form of dementia, accounts for nearly 2/3 of all diagnosed MaNCD cases. There continues to be a lack of consensus regarding specific diagnostic criteria for AD, with most clinicians using the terms 'probable' and 'possible' AD.[28] Typical AD diagnostic criteria include ruling out the presence of any clinical or other evidence of co-existing cerebrovascular disorders, medication-induced dementia (or delirium), any other condition affecting cognition, or any other known form of dementia.[29] Indeed, all post-mortem cases of Alzheimer's disease present with aggregate beta-amyloid plaques in the brain; however, these plaques are also found in individuals without diagnosed cognitive disorders. With the recent introduction of aducanumab as an approved treatment for mild AD, there is the potential for reducing the presence of amyloid plaques and potentially slowing the progression of the disease in selected candidates who are as of yet not clearly identified. Unfortunately, this targeted therapy does present with significant side effects.[30]

VaD accounts for nearly 20% of all cases of dementia. Vascular damage is often characterized by cerebral infarct, white matter lesions, myelin loss, and amyloid angiopathy, resulting in neuronal loss and synaptic degeneration.[31] In addition to deficits in at least two cognitive domains, VaD diagnosis requires neuroimaging evidence of cerebrovascular disease.[32,33] Further, type-2 diabetes is a significant risk factor for all types of dementia. Rates of VaD more than double in individuals with type-2 diabetes as compared to other forms of dementias. Although no targeted therapeutics exist for VaD, addressing cardiovascular health (e.g., lowering blood pressure, reducing cholesterol and anticoagulants, and controlling blood sugar) may slow the rate and potentially prevent further decline.[3]

LBD, like AD, is associated with abnormal protein deposits in

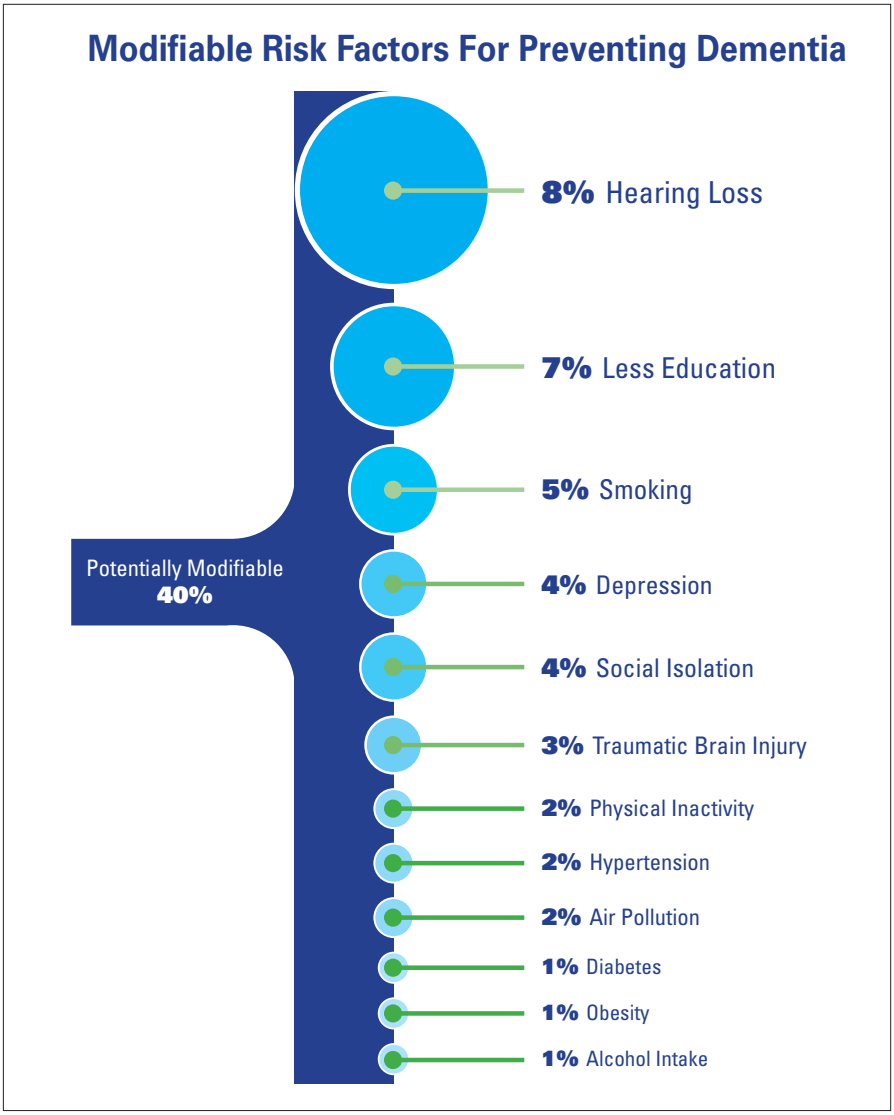

Figure 2. Illustration of the 12 potentially modifiable risk factors for reducing the risk of cognitive decline and dementia,[17] addressing hearing loss is the most significant modifiable factor.

the brain that can only be definitively diagnosed post-mortem. LBD is often associated with Parkinson's disease (PD) and represents approximately 15% of all dementias. Diagnosis is often identified as concurrent cognitive impairment with supporting neuroimaging of the basal ganglia and REM sleep behavior disorder.[35] LBD is the result of genetic mutations to the SCNA gene, identified as producing alpha-synuclein protein clusters (proteins normally found in the pre-synaptic terminal of neurons). These abnormal clusters of the alpha-synuclein protein are present primarily in the basal ganglia region in patients with Parkinson's disease (PD) and are even more widespread in the brains of people with LBD + PD. The comorbidity of dementia in patients with PD can exceed 80% with a long enough survival time (i.e., > 20 years), implying dementia may be inevitable in PD.[35-37] There is no targeted therapy for LBD other than those medications typically prescribed to patients with cognitive impairment (i.e., cholinesterase inhibitors). Unfortunately, treatment targeting PD may only aggregate the LBD by increasing delusions, confusion, and hallucinations.

Frontotemporal Dementia, also known as FTLD or Picks Disease, is considered the earliest-onset neurodegenerative dementia[38] with an average age of diagnosis of 53 years.[39] This MaNCD accounts for approximately 5% of all dementia cases.[40] As the name implies, primary lesion sites in the brain include both the frontal and temporal lobes, causing significant personality and behavioral changes, motor neuron disease, and accompanying non-fluent aphasias.[39] There is no specific treatment for FTLD. Interestingly, the most common medications often prescribed for AD to temporarily relieve cognitive symptoms may exacerbate the symptoms of FTLD. Most often, antidepressants, antipsychotics, and speech-language therapy are indicated to manage the symptoms.

Pharmaceutical Treatments for MaNCD

With approximately 60% of all dementias considered the result of genetic predisposition (i.e., the presence of APOE genes and first-degree family history) and the other 40% considered modifiable and potentially preventable,[17] there must be a significant emphasis placed on both treatment and prevention. Currently, there are no widely available pharmacological interventions to directly target the cause of MaNCD, regardless of type. Most prescribed medications for MaNCD intend to moderate the symptoms of cognitive impairment, including memory and learning deficits, behavioral changes, and depression.

Acetylcholinesterase inhibitors (e.g., Aricept, Razadyne, and Exelon). Acetylcholine (AcH) is a primary neurotransmitter of the central nervous system (CNS) and essential for processing memory and learning. Levels of AcH are typically reduced in the CNS of patients with MaNCD. Therefore, acetylcholinesterase inhibitors, which increase the amount of free AcH in the synapse, are often prescribed to patients with mild to moderate AD. These medications, administered orally or by patch, may improve or stabilize the symptoms of dementia for up to 1 year.[41]

Glutamate Antagonist (e.g., Namenda). Glutamate, another primary neurotransmitter of the CNS, is involved in learning and memory and is critical for neuronal survival. Unfortunately, excessive amounts of glutamate promotes cell death and may be an underlying cause of neurodegeneration in MaNCD. Glutamate antagonist may be prescribed in moderate to severe dementia to limit the damage caused by the excessive release of glutamate.

Antipsychotics (e.g., Risperdal, Abilify, Zyprexa, etc.). This class of drugs may offer modest effects in treating psychosis, aggression, agitation, and erratic behaviors more common in late-stage dementia.[42,43]

Antidepressants (e.g., Celexa, Zoloft, Prozac, Paxil, etc.) Depression may be dif-

ficult to diagnose in patients with MaNCD and may be a prodrome to the disorder. The two disorders may also share a common etiologic pathway, particularly white matter disease.[44] When depression is present in MaNCD it can further reduce the quality of life, increase disability, and reduce the lifespan.[44,45] Many patients with dementia are prescribed antidepressants, although evidence of efficacy (as defined by depression rating scales) is moderate at best.

Aducanumab (e.g., Aduhelm). The FDA recently (July 2021) approved aducanumab to treat the underlying pathophysiology of AD and is recommended to treat MCI and mild AD.[46] This human monoclonal antibody specifically targets the buildup of amyloid plaques in the brain. Initial studies found slowing of clinical decline as measured by the Clinical Dementia Rating and Mini-Mental State Examination scores.[47] However, additional studies did not provide evidence of improvements in cognitive function. Ultimately, aducanumab was fast-track approved by the FDA based on its ability to reduce the presence of amyloid plaques without any corroborating evidence of less cognitive or functional decline.[48] The required FDA confirmation study is anticipated to be complete in 2026. The side effects of aducanumab include a 40% incidence of brain swelling and bleeding. Aducanumab has been rejected by the European Medicines Agency.

Preventing MaNCD

Approximately 55-60 million people globally are affected by dementia, with rates anticipated to nearly triple by 2050.[49] This increase is due to a substantial rise in life expectancy and an aging population globally. This increase is anticipated to discriminately impact low- and middle-income populations the most, possibly increasing their risk of dementia by a factor of 4-5x in the next 25 years. As reviewed, no (widely accepted) treatments are available to directly target the underlying pathophysiology of MaNCD. With approximately 4 in 10 cases of dementia considered preventable, extraordinary focus must be placed on prevention and slowing disease progression.

Primary prevention of MaNCD focuses on delaying the disease onset by modifying lifestyle and behavioral risk factors.[17,50] Estimates that a delay in the onset of dementia by 1% could reduce global rates of dementia by more than 10% in 2050[51] place all healthcare providers, including the HHCP, on the frontlines of prevention.

Prevention through lifestyle modifications may specifically target cognition in normal aging individuals, as well as improve cognition in persons with MiNCD. Currently, it is estimated that 15-20% of people with MiNCD transition to MaNCD each year.[19] Unfortunately, with no intervention or modification of lifestyle, and given a long enough survival time, conversion from MiNCD to MaNCD may be inevitable.[52]

Each of the twelve modifiable lifestyle risk factors listed includes their population attributable factor (PAF) value **(**see **Figure 2)**.

1. Hearing Loss (8%) – encourage the early treatment of hearing loss for those affected and reduce exposure to excessive noise[53-58]

2. Education (7%) – provide all people with the opportunity for lifelong learning, especially primary and secondary education[59-62]

3. Smoking (5%) – avoid smoking[63-66]

4. Depression (4%) – reducing depression may curb dementia neuropathology[67-69]

5. Isolation (4%) – engage in more frequent social contact, especially during late middle age[70-72]

6. Traumatic Brain Injury (3%) – prevent head injury[73-76]

7. Physical Activity (2%) – sustain midlife and possibly later life physical activity[77-78]

8. Reduce Hypertension (2%) – maintain systolic blood pressure of 130mmHG or less[79-82]

9. Air Pollution (2%) – reduce exposure to air pollution and second-hand smoke[83-85]

10. Diabetes (1%) – reduce rates of acquired diabetes (i.e., Type-2 Diabetes)[86,87]

11. Obesity (1%) – reduce obesity (as defined by BMI)[88,89]

12. Alcohol Intake (1%) – Limit alcohol consumption to less than 21 units per week (the equivalent of 2 bottles of wine per week)[90-93]

Although not specifically assigned a PAF value by the Lancet Commission, sleep and diet were included as significant contributing factors to dementia risk. Consistently sleeping more than 6 hours per night is considered important for reducing the risk of decline and dementia.[94-96] In addition, The World Health Organization guidelines recommend a Mediterranean diet (i.e., plant-based foods, whole grains, nuts, seeds, and moderate amounts of lean poultry and fish) to reduce the risk of cognitive decline or dementia, as it might help and does not harm.[97]

Discussion: Role of the HHCP in MaNCD

Rates of MaNCD exponentially increase every 5 years between the age of 65-90 years old,[98] with the pre-clinical stage of disease beginning up to 20 years before symptoms manifest.[99] The challenge facing the HHCP is that presbycusis often starts in the 4th to 5th decade of life,[100] but the average age of the first-time hearing aid user is 74 years old.[101] These pre-clinical and pre-symptomatic years for MaNCD and presbycusis are likely the most crucial years to successfully impact the trajectory of cognitive function and conversion from typical aging to MiNCD to MaNCD.

The HHCP is uniquely suited to screen their patient's dementia risk as it relates to their information and auditory processing abilities,[11] audiometric findings,[13] and ability to hear and listen in complex listening environments.[15] In recent years, many HHCPs have adopted formal cognitive screening measures,[102] including dementia risk questionnaires, paper-based cognitive screenings, and sophisticated computerized cognitive screeners which do not depend on sound systems to deliver test questions, thereby eliminating tester bias, as well as eliminating tester and /or patient-based hearing or listening problems (as confounding sources of variability) and are automatically scored and a detailed report for the physician is provided.

HHCPs must continue to advocate for the early detection and treatment of hearing loss as it directly impacts social, emotional, physical, and cognitive health. Although a cumulative effect has not yet been demonstrated, given that treating hearing loss increases social engagement,[103] increases physical activity[104-107] (i.e., potentially lowering rates of obesity, diabetes, and hypertension) and reduces depression,[108] the impact of treating hearing loss on reducing an individuals' dementia risk may be profoundly underestimated.

References can be found in the online version of this article at: hearingreview.com