Journal of Otology 21 (2026) 160 – 166

Journal of Otology

journal homepage: www.sciopen.com/journal/1672-2930

Auditory Biomarkers in Neurodegenerative Disorders: A Literature Review

Sreevidhya Palakkot[1] ✉ and Arun Banik[2]

[1] *Department of Audiology and Speech Language Pathology, School of Rehabilitation and Behavioural Sciences, Vinayaka Mission's Research Foundation (DU), Aarupadai Veedu Medical College and Hospital, Puducherry, India*

[2] *School of Rehabilitation and Behavioural Sciences, Vinayaka Mission's Research Foundation (DU), Aarupadai Veedu Medical College and Hospital, Puducherry, India*

ABSTRACT

The prevalence of neurodegenerative diseases is escalating globally. However, the conventional diagnostic framework fails to identify the pathology until substantial neuronal damage occurs. The evidence from the recent literature indicates that auditory dysfunction commonly precedes motor and cognitive symptoms across multiple neurodegenerative diseases. The pathophysiology involves both the peripheral and cortical auditory structures, which produce distinctive patterns reflects systemic neurodegeneration. Hence, this suggests auditory assessment as a valuable tool for early identification of neural degeneration. This review synthesises the contemporary literature on auditory dysfunctions and underlying pathophysiology in Alzheimer's disease, Parkinson's disease, Frontotemporal dementia, Amyotrophic lateral sclerosis, etc. The analysis included the temporal trajectories of auditory impairment, subjective-objective measurements, and evaluated the importance of early identification and longitudinal tracking. Objective measures of central auditory processing, including Auditory Brainstem Responses, P300, Mismatch Negativity, and speech-in-noise testing, provide objective, non-invasive diagnostic tools and its sensitivity comparable to established biomarkers. Integration of standardised auditory assessment batteries into clinical protocols could enable the early identification of pathology, differential diagnosis and the development of novel therapeutic strategies for both auditory and cognitive deficits. Current review suggests that future longitudinal studies with neurodegenerative conditions should focus on the clinical utility of auditory, cognitive, and neuroimaging biomarkers and facilitate clinical translation of these findings.

KEYWORDS

auditory dysfunction, central auditory processing disorder, electrophysiology, biomarkers, neurodegenerative disorder, early diagnosis.

1 Introduction

Neurodegenerative disorders are long-term progressive, chronic, heterogeneous conditions (Toader et al., 2024), which include Alzheimer's disease (AD), Parkinson's disease (PD), Frontotemporal dementia (FTD), etc. These are characterised by loss of neurons, protein aggregation, and selective neuronal vulnerability. This adversely affects not only those who are diagnosed but also caregivers and society. The number of affected individuals was around 55 to 60 million and is projected to be >150 million by 2050 (Zhou et al., 2025). This may underscore the critical need for early detection and patient-specific treatments (Agarwal et al., 2025). The diagnostic paradigms largely rely on clinical detection after significant neuronal loss, which limits therapeutic efficacy. Interventions are effective only if initiated during prodromal or early symptomatic stages (Benatar et al., 2022). The major challenge is lack of reliable and cost-effective biomarkers for screening neurodegeneration in its early stage (Pal et al., 2025).

Sun et al. (2025b) suggest that age-related decline in hearing may accelerate AD pathogenesis. This serves as a prodromal sign that appears years before cognitive impairment (Sun et al., 2025b). Hearing loss not only impacts auditory processing but also accelerates cognitive decline in neurodegenerative pathology (Sardone et al., 2019). This establishes a bidirectional relationship between sensory and cognitive deterioration. Auditory impairment is more central rather than peripheral and is crucial for identifying neurodegenerative diseases. The cholinergic hypothesis that links a connection between auditory and cognitive processing. This drifted perspectives of auditory deficit in dementia from peripheral age-related hearing loss to central auditory processing dysfunction (Hampel et al., 2018).

Electrophysiological methods such as Auditory Brainstem Response (ABR) testing have expanded research to sensory-cognitive interactions in neurodegeneration (Anderson et al., 2012). Central auditory dysfunction is now regarded as an early warning sign of neurodegeneration, with diagnostic methods and the temporal relationships between auditory and cognitive functions under focused study. This review consolidates interdisciplinary evidence for auditory impairment as an early neurodegeneration biomarker, highlighting clinical prospects for improved diagnostic

Peer review under responsibility of PLA General Hospital Department of Otolaryngology Head and Neck Surgery.

* Corresponding author Sreevidhya Palakkot, svpalakkotaslp@gmail.com

https://doi.org/10.26599/JOTO.2026.9540067

Received 13 March 2026; Revised 8 June 2026; Accepted 10 June 2026

and therapeutic frameworks.

2 Auditory Dysfunction and Alzheimer's Disease

2.1 Epidemiology and Clinical Presentation.

Auditory deficit in early stage of dementia has been identified as one of the most treatable risk factors (Thomson et al., 2017). The severity of hearing loss, AD risk and cognitive decline acceleration are mutually connected (Thomson et al., 2017). AD patients often complaint that difficulty in understanding speech, especially in noisy environments. This highlights the persistence of central deficit rather than purely peripheral impairment, despite the near-normal pure-tone thresholds (Durrani et al., 2025). Sardone et al. (2019) reported the supportive evidence from caregivers that patients can "hear" sounds but fail to "understand" speech. Recent literature elucidates speech-in-noise deficits in early stage of AD and its correlation with quality of life, communication ability, and cognitive function (Sardone et al., 2019; Durrani et al., 2025). AD patients often exhibit both peripheral hearing loss and auditory processing deficits (Jafari et al., 2020; Hamed et al., 2025). Hearing impairment frequently precedes dementia diagnosis by years, beyond what is expected from ageing alone (Hong et al., 2024).

2.2 Temporal Linkage to Cognitive Symptoms.

Benatar et al. (2022) found that deficits in auditory functions often precede cognitive symptoms by three to five years in AD (Figure 1). Hence, this offers a critical window for early diagnosis and intervention (Benatar et al., 2022). Studies with prodromal stages of AD, i.e., mild cognitive impairment (MCI) and subjective cognitive decline, found that progressive auditory processing deficits occur beyond normal ageing (Kim et al., 2022). Temporal processing is not only fundamental to auditory functions but also closely linked with working memory and executive functions (Nagaraj, 2024). Deficits in processing rapid auditory cues adversely affect cognitive functions. This may serve as an early indicator of neural degeneration.

2.3 Electrophysiological Markers.

Auditory evoked potentials (AEPs) provide objective measures of central auditory processing. A differentiation between AD patients and controls has been reported with prolonged P300 latency and reduced amplitude (Bayat et al., 2026). P300 often shows the largest effect size among AEPs (Bayat et al., 2026). Whereas auditory P50 gating and mismatch negativity (MMN) often reflect central auditory deficits in AD. These objective, low-cost, non-invasive electrophysiological methods serve as both diagnostic and screening tools (Bayat et al., 2026).

2.4 Central Auditory Processing and Speech Perception Deficits.

Speech perception deficits in noise arise from damage to temperoparietal cortical circuits that mediate auditory-language integration. These deficits correlate with reduced grey matter volume and cognitive impairment (Durrani et al., 2025). Speech-in-noise testing is considered an affordable, effective screening tool across neurodegenerative diseases like AD (Durrani et al., 2025) (Figure 2). Patients with MCI show marked reductions in central auditory processing (CAP), including on frequency/duration pattern tests, Gaps-in-Noise (GIN) tests, dichotic digits tests, and speech-in-noise assessments (Kim et al., 2022). The gap detection threshold (GDT) significantly increases in cognitive impairment (e.g., 8.25±6.14 ms vs. 5.98±3.44 ms in controls, p=0.034) and correlates negatively with cognitive scores (Ma et al., 2025). Electrophysiological and connectivity analyses were associated with high GDT, altered auditory networks and reduced ERP amplitudes. This identifies GDT as a sensitive early biomarker of cognitive decline (Ma et al., 2025).

3 Parkinson's Disease and Auditory Dysfunction

3.1 Prevalence and Clinical Features.

Recent studies reported that around 68% of patients with PD had motor symptoms (tremor, rigidity, bradykinesia) coexisting

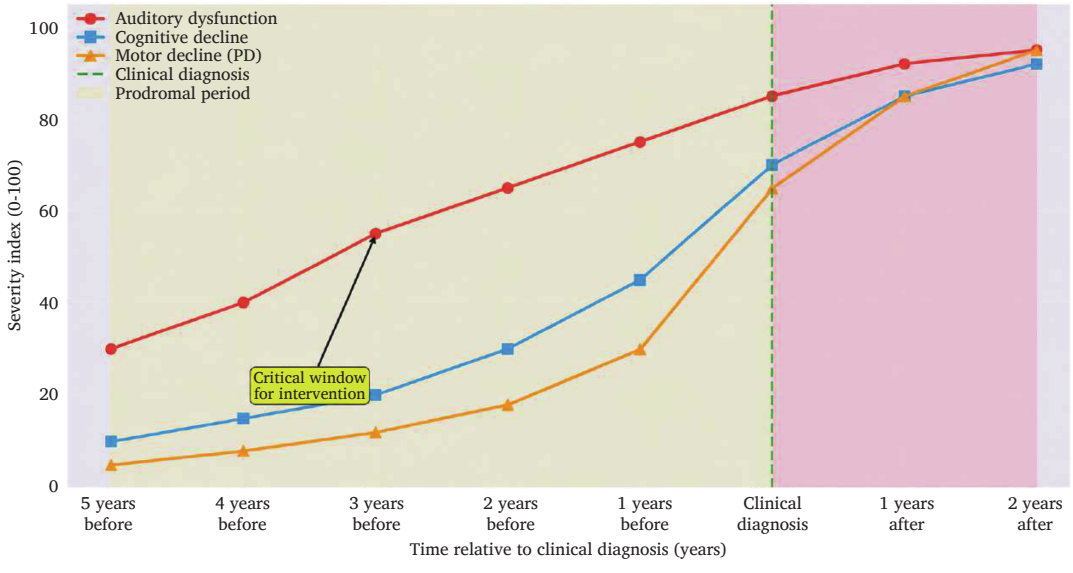

Figure 1 Temporal precedence of auditory dysfunction in neurodegenerative disease progression (auditory changes precede clinical diagnosis by 3-5 years)

(Note: The figure shown is a schematic representation synthesised from the reviewed literature and is intended for illustrative purposes only.)

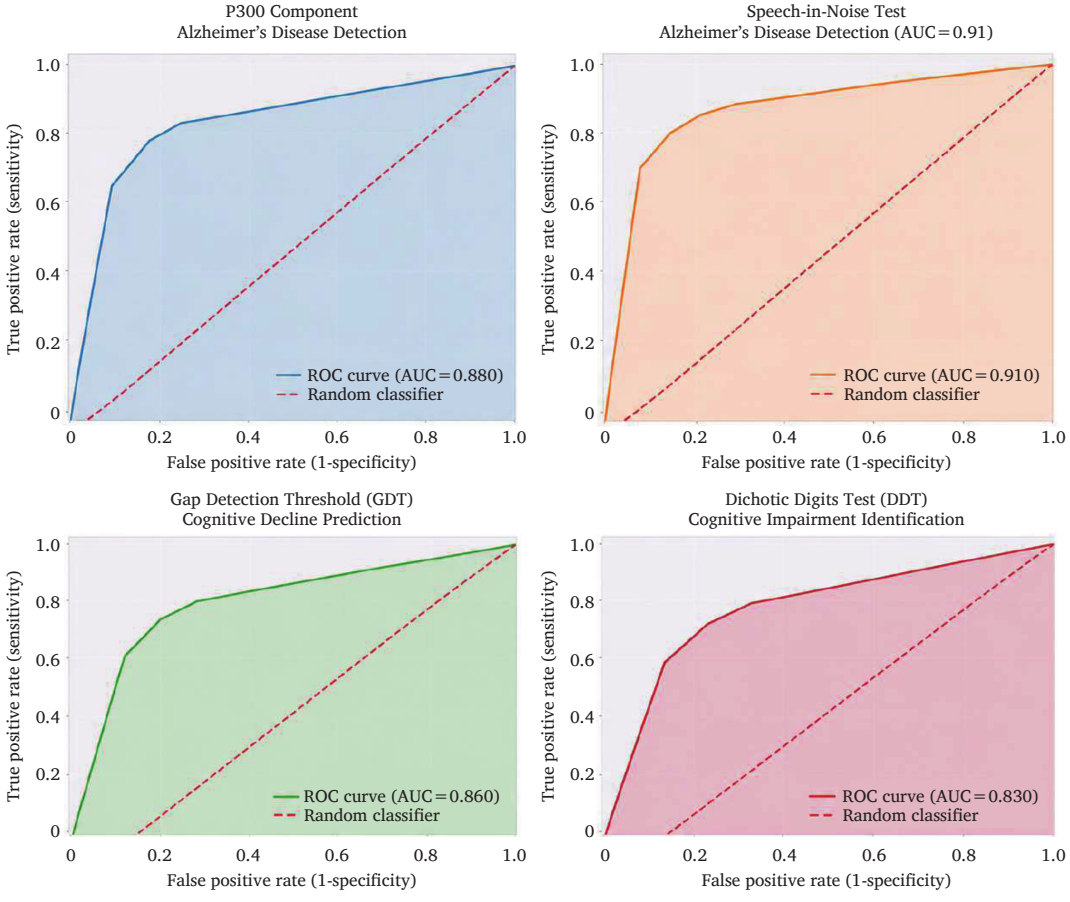

Figure 2 Diagnostic accuracy of auditory biomarkers in neurodegenerative diseases (ROC curve)

(Note: The figure shown is a schematic representation synthesised from the reviewed literature and is intended for illustrative purposes only)

with auditory dysfunction (Jafari et al., 2020; Hamed et al., 2025). Auditory deficits in these patients range from peripheral to central. They exhibit ABR abnormalities and habituation deficits in the prodromal stages of PD (Jafari et al., 2020). Hamed et al. (2025) reported poor scores in the dichotic digits test (DDT) and duration pattern test (DPT) that reveal significant central processing dysfunction. In addition, speech-in-noise performance correlates with cognitive deficits as well as elevated speech reception thresholds at high frequencies. However, auditory deficits in PD are often underrecognized, but it carries a significant impact on disease burden (Jafari et al., 2020).

3.2 *Asymmetric Auditory Deficits and Motor Lateralisation.*

Lasheen et al. (2025) reported that asymmetry of the auditory deficits parallels and lateralized motor symptoms in PD. Higher pure-tone thresholds and delayed ABR wave latencies tend to be more pronounced on the side of the body with more severe motor symptoms (Lasheen et al., 2025). Similarly, distortion product otoacoustic emission amplitudes are diminished ipsilateral to the motor-affected side. This lateralisation provides a potential non-motor diagnostic marker that helps with the differential diagnosis of PD. This reflects the negative impact of asymmetric dopaminergic degeneration on auditory and motor functions (Shalash et al., 2017; Lasheen et al., 2025).

3.3 *Brainstem Auditory Pathway Dysfunction.*

Previous literature studies were reported that degeneration of brainstem nuclei, including auditory pathways were integral to both motor and non-motor symptoms. Auditory Brainstem Responses (ABRs) and Vestibular Evoked Myogenic Potentials (VEMPs) showed abnormal waveforms and delayed latencies, especially prolonged wave V in PD (Hassan and Shalash, 2017; Shalash et al., 2017). VEMP abnormalities were indicate vestibular and brainstem dysfunction. These findings suggested that delayed neural conduction is due to dopamine-related dysfunction along the brainstem and cortico-subcortical auditory regions. These abnormalities in electrophysiological findings correlate with motor rigidity and bradykinesia, as well as with nonmotor symptoms like sleep disturbances, autonomic complaints, cognition, and urinary issues (Hassan and Shalash, 2017; Shalash et al., 2017). Hence, ABR and VEMP can be considered as multifunctional biomarkers in PD.

3.4 *Dopaminergic Mechanisms.*

Auditory processing systems involve neurotransmitters, such as dopamine and glutamate, within basal ganglia circuitry. Dopaminergic therapies may modulate auditory responses through shared neural substrates between auditory and motor circuits (Jafari et al., 2020). The encoding of the fundamental frequency was intensified in Frequency Following Responses (FFR). This indicates auditory processing dysfunction related to the basal ganglia (Mollaei et al., 2022). Event-related potentials such as P300 correlate with cognitive status and disease duration in PD. Thus, it serves as a marker for cognitive decline (Ferrazoli et al., 2022). The heterogeneous nature of auditory dysfunction in PD likely requires broader therapeutic approaches. However, dopaminergic

drugs can partially reduce auditory processing deficits (Jafari et al., 2020).

3.5 Auditory Impact in Early-Onset PD.

Shetty et al. (2019) reported that Parkinson's disease patients below 55 years had high rates (approximately 65%) of asymptomatic pure-tone hearing loss, which is distinct from age-related loss. This implies that auditory dysfunction may be intrinsic to PD and independent of ageing. Brainstem evoked responses were more or less similar to those of controls in their earlier stage (Shetty et al., 2019). This suggests the pathology is primarily peripheral in nature, although central involvement cannot be excluded. Systematic audiological screening in PD could improve detection of these deficits and quality of life by enabling earlier intervention (Shetty et al., 2019).

4 Auditory Dysfunction in Other Neurodegenerative Disorders

4.1 Amyotrophic Lateral Sclerosis (ALS) and Motor Neuron Disease.

Previous studies reported auditory processing deficits in ALS, as well as the disease mechanism in progressive motor neuron loss (Iyer et al., 2017; Sardone et al., 2019). Mismatch negativity (MMN) serves as an electrophysiological marker of auditory discrimination. Iyer et al. (2017) reported MMN was altered in ALS, which correlates with cognitive deficits in specific sub-domains. It indicates auditory–cognitive network involvement distinct from pure motor effects (Iyer et al., 2017). Even though peripheral hearing is relatively preserved, central auditory processing deficits are reported in ALS. This reflects disruptions in the common network and synaptic dysfunction (Sardone et al., 2019). Such auditory biomarkers could provide valuable information for patient stratification and monitoring in clinical trials (Iyer et al., 2017).

4.2 Frontotemporal Dementia (FTD) and Language-Dominant Presentations.

FTD is a language-variant form that demonstrates prominent auditory-linguistic dysfunction extending beyond semantic and syntactic impairments. Häggström et al. (2020) reported that left-ear deficits in dichotic listening tests as well as <50% correct responses in overall scores. This significantly elevates dementia risk among FTD patients. FTD reveals functional connectivity alterations that affect integration between auditory, language, and executive networks. This mainly contributes to complex communication impairments (Jalilianhasanpour et al., 2019).

4.3 Lewy Body Dementia (LBD) and α-Synucleinopathies.

Patients with LBD show similar auditory dysfunction patterns as in the case of AD and PD, which reflects its mixed α-synuclein and amyloid pathology (Galvin, 2024). Patients with dementia with Lewy bodies often have early visual hallucinations and fluctuating cognition alongside auditory processing deficits that exceed what peripheral hearing loss would predict (Galvin, 2024). The auditory phenotypes of Parkinson's disease dementia and dementia with Lewy bodies overlap brainstem auditory pathway dysfunction, asymmetric deficits, and correlations between auditory and cognitive measures (Milán-Tomás et al., 2021; Galvin, 2024). The dopamine imaging shows abnormalities in results that correlate with both motor and auditory symptoms in LBD. This is supporting evidence for shared pathophysiological mechanisms across α-synucleinopathies (Galvin, 2024).

5 Mechanisms Linking Auditory and Cognitive Decline

5.1 Protein Aggregation and Auditory Pathology.

Recent literature highlights that misfolded protein aggregation is a hallmark of neurodegeneration and affects the auditory system at multiple levels (Calabresi et al., 2023). α-synuclein Lewy bodies accumulate in the superior olivary complex, inferior colliculus, and auditory cortex in PD. This may disrupt auditory function via dopaminergic and non-dopaminergic brainstem nuclei (Calabresi et al., 2023). The pathology in peripheral and central auditory structures, including multiple molecular pathways for hearing, implicates auditory as well as cognitive deterioration (Hong et al., 2024).

5.2 Neuroinflammation and Immune Dysregulation.

Padhi et al. (2022) reported that neuroinflammatory processes and immune dysregulation are key pathways that link neurodegeneration to auditory impairment. Peripheral inflammation exacerbates protein aggregation, mitochondrial dysfunction, and synaptic damage across the auditory system. This pathological feedback loop promotes progressive auditory and cognitive decline through interwind inflammatory and prion-like cascades (Padhi et al., 2022).

5.3 Synaptic Dysfunction and Neurotransmitter Deficits.

Synaptic pathology in neurodegenerative progression with auditory dysfunction is pivotal in nature. Cerebrospinal fluid analyses reveal altered synaptic proteins (SNAP-25, 14-3-3ζ/δ, β-synuclein, neurogranin) in AD patients. These results were strongly correlated with tau and amyloid PET imaging and cortical atrophy (Nilsson et al., 2024). These synaptic biomarkers predict cognitive decline and reflect the vulnerability of auditory synapses. Cholinergic deficits impair central auditory processing by damaging basal forebrain cholinergic projections in AD (Hampel et al., 2018). Dopamine and glutamate deficits in Parkinson's disease disrupt auditory processing via basal ganglia circuits. Although dopaminergic treatment can partially remediate some deficits (Jafari et al., 2020; Mollaei et al., 2022).

5.4 Vascular and Metabolic Dysfunction.

Vascular insufficiency, oxidative stress, and metabolic dysregulation collectively result in auditory dysfunction (Sun et al., 2025b) Peripheral cochlear and central auditory neurons are sensitive to cerebrovascular pathology and mitochondrial impairment, which lead to reduced ATP generation and increased oxidative damage (Huang et al., 2023). Dynamic contrast-enhanced MRI shows early blood–brain barrier permeability changes in auditory and cognitive regions during mild cognitive impairment. This precedes visible neurodegeneration (Chen et al., 2024; Sun et al., 2025a). These vascular–metabolic insults accelerate cochlear degeneration and central auditory pathway dysfunction (Sun et al., 2025b).

5.5 Network Connectivity and Compensatory Mechanisms.

Neurodegeneration disrupts large-scale brain networks integral to auditory processing, which include the default mode, salience, central executive, and auditory-specific networks. Functional connectivity within and between these networks decreases with age

and neurodegeneration. This may increase the severity of hearing deficit and cognitive impairment (Huang et al., 2023; Tong et al., 2025). Compensatory recruitment of frontal executive regions may help to maintain auditory task performance by reducing sensory processing. However, this compensation eventually becomes insufficient and leads to accelerated decline (Yang et al., 2024). This network-level helps to elucidate the interrelation of auditory and cognitive decline and supports multi-domain therapeutic strategies.

6 Electrophysiological Assessment Methods

6.1 *Auditory Brainstem Response (ABR).*

ABR provides an objective assessment of the auditory pathway from the auditory nerve through brainstem nuclei to the inferior colliculus, approximately 10 ms post-stimulus (Anderson et al., 2012). It records vertex-positive waveforms and is linked to the auditory nerve, cochlear nucleus, superior olivary complex, lateral lemniscus and inferior colliculus (Anderson et al., 2012). The more important waveforms in diagnosis and differentiation are I, III, and V. Typical ABR findings in PD include prolonged wave V latencies, increased interpeak intervals, and reduced amplitudes (Lasheen et al., 2025). The asymmetric responses could be a valuable non-motor marker in PD, helpful for differential diagnosis.

6.2 *Speech-Evoked ABRs.*

Fu et al. (2019) use complex syllabic stimuli like /da/ in speech-evoked ABRs that incorporate spectrotemporal features essential for speech perception. These responses capture sustained frequency-following activity and transient onset/offset brainstem potentials that offer ecological validity beyond clicks. Speech is more complex stimulus with higher cognitive load than clicks. Hence, this demands good cognitive skills to complete the task in speech-evoked ABR. Speech-evoked ABRs have shown greater sensitivity than conventional audiometry or click-ABR for detecting early central auditory dysfunction (Fu et al., 2019; Mollaei et al., 2022). This suggests their utility for identifying prodromal neurodegenerative changes.

6.3 *Event-Related Potentials (ERPs).*

Auditory event-related potentials assess higher-order auditory cognition, including attention, memory, and stimulus discrimination. P300 is the positive deflection around 250-400 ms following an "oddball" stimulus. It reflects cognitive resource allocation and can distinguish AD patients from controls with high accuracy (Bayat et al., 2026). Mismatch Negativity (MMN) is a pre-attentive response and present approximately around 100 to 250 ms following a deviant stimulus. It measures auditory discrimination and sensory memory without requiring active attention. AD, PD, and FTD patients show reduced MMN amplitudes and increased latencies that correlate with disease severity (Näätänen et al., 2011). Bayat et al. (2026) highlighted the clinical utility of auditory evoked potentials. They suggested AEP can be included in the auditory test battery for neurodegenerative cases.

6.4 *Gap Detection and Temporal Processing.*

Temporal auditory processing is crucial for cognitive well-being. The gap detection threshold (GDT) is elevated in cognitive impairment and negatively correlates with cognitive test scores (Ma et al., 2025). Gaps-in-Noise (GIN) tests measure gap perception abilities and have shown strong diagnostic value (Kim et al., 2022). EEG analyses confirm altered cortical networks and reduced ERP amplitudes associated with elevated GDT (Ma et al., 2025). This establishes gap detection as a non-invasive biomarker for early cognitive decline. Geriatric patients who have cognitive impairment beyond an age-related decline exhibited temporal processing deficits with increased right ear advantage (Thamizhmani et al., 2025)

7 Specific Auditory Processing Assessment Methods

7.1 *Dichotic Listening Tests.*

Dichotic listening tasks assess the ability to integrate competing auditory inputs that are delivered simultaneously to both ears. In addition, it assesses executive control, selective attention, and corpus callosum-mediated interhemispheric transfer. The Dichotic Digits Test (DDT) reveals significant auditory-cognitive impairment in PD and dementia. The test findings correlated with cognitive deficits and auditory impairment (Jalaei et al., 2019; Hamed et al., 2025). Lower DDT scores predict increased dementia risk (odds ratio ~2.5), which reflects sensitivity to declines in auditory processing speed (Hamed et al., 2025). Dichotic tests show poorer left-ear scores in FTD (Häggström et al., 2020). Whereas the Staggered Spondaic Word (SSW) test detects early binaural integration deficits in mild cognitive decline (Jalilianhasanpour et al., 2019).

Most previous literature reported a predominant right-ear advantage in AD on dichotic tasks (Bouma and Gootjes, 2011; Tuwaig et al., 2017; Utoomprurkporn et al., 2020). Neuroimaging studies link dichotic sentence identification scores with cortical thinning in temporal and frontal gyri (Golden et al., 2015), and Synthetic Sentence Identification with ipsilateral competing message (SSI-ICM) scores correlate with inferior parietal lobule, Heschl's gyrus, parahippocampal gyrus, and entorhinal cortex thickness (Brun and Englund, 1981). These areas are crucial in prodromal AD (Jack and Holtzman, 2013).

8 Conclusion

Auditory impairment is an early diagnostic marker in multiple neurodegenerative diseases. These auditory changes often precede clinical diagnosis by years and strongly correlate with cognitive decline. Their temporal precedence aids early detection, differential diagnosis, risk stratification, and treatment monitoring (Bayat et al., 2026). Electrophysiological assessments and behavioural tests demonstrate diagnostic sensitivity and specificity comparable to established biomarkers, while offering non-invasive, accessible, and cost-effective advantages (Durrani et al., 2025; Bayat et al., 2026). Hence, auditory assessment can facilitate early diagnosis and intervention to improve the quality of life in patients with neurodegenerative disorders.

9 Future Directions

Neural degeneration correlates with early and multifaceted auditory dysfunction. Hence, future research should focus on temporal changes with auditory, cognitive, neuroimaging, and biological measures. This may help to explore the underlying relationships and temporal dynamics (Durrani et al., 2025). Standardised, culturally-equitable auditory assessment batteries will promote cross-study meta-analyses and clinical relevance (Durrani

et al., 2025). Intervention studies should address auditory deficits, neuromodulation, neurotransmitter augmentation, and auditory training. This may improve cognitive and functional outcomes (Zhou et al., 2025). Integrating auditory biomarker frameworks and clinical guidelines will enable earlier diagnosis, more accurate risk stratification, and better outcomes for individuals at risk or suffering from neurodegenerative diseases.

Funding

No external sources of funding for this scientific research.

Data Availability Statement

Not applicable.

Ethical Approval

Not applicable.

Informed Consent Statement

Not applicable.

Conflicts of Interest

The authors have declared that no conflict of interest exists.

Acknowledgements

The authors acknowledge the researchers from all over the world whose work served as the basis for this review.

Author Contributions

S.P - concept, review of literature, working with the review article, writing the manuscript, editing the manuscript, analysis and review of publications on the topic of the article; A.B – Review of the manuscript, supervision and coordination of work, review of publications on the topic of the article.

References

Agarwal, U., Paliwal, S., Yadav, V., et al., 2025. Pathological insights into neurodegenerative and neurodevelopmental disorders: Perspectives for the development of novel treatment approaches. CNS Neurol. Disord. Drug Targets https://doi.org/10.2174/0118715273402657250905055635.

Anderson, S., Parbery-Clark, A., White-Schwoch, T., et al., 2012. Ageing affects neural precision of speech encoding. *J. Neurosci.* 32(41), 14156–14164.

Bayat, A., Mirmomeni, G., Aiken, S., et al., 2026. Meta-analyses of auditory evoked potentials as Alzheimer biomarkers. *Ear Hear.* 47(1), 95–106.

Benatar, M., Wuu, J., McHutchison, C., et al., 2022. Preventing amyotrophic lateral sclerosis: Insights from pre-symptomatic neurodegenerative diseases. *Brain* 145(1), 27–44.

Bouma, A., Gootjes, L., 2011. Effects of attention on dichotic listening in elderly and patients with dementia of the Alzheimer type. *Brain Cognit.* 76(2), 286–293.

Brun, A., Englund, E., 1981. Regional pattern of degeneration in Alzheimer's disease: Neuronal loss and histopathological grading. *Histopathology* 5(5), 549–564.

Calabresi, P., Mechelli, A., Natale, G., et al., 2023. Alpha-synuclein in Parkinson's disease and other synucleinopathies: From overt neurodegeneration back to early synaptic dysfunction. *Cell Death Dis.* 14(3), 176.

Chen, T.L., Dai, Y., Hu, C.H., et al., 2024. Cellular and molecular mechanisms of the blood-brain barrier dysfunction in neurodegenerative diseases. *Fluids Barriers CNS* 21(1), 60.

Durrani, F.G., Marshall, C.R., Waters, S., et al., 2025. (DIADEM) digital assessment of auditory perception in dementia: Study protocol. *Alzheimers Dementia* 21(S2), e098358.

Ferrazoli, N., Donadon, C., Rezende, A., et al., 2022. The application of P300-long-latency auditory-evoked potential in Parkinson disease. *Int. Arch. Otorhinolaryngol.* 26(1), e158–e166.

Fu, Q.Y., Wang, T., Liang, Y., et al., 2019. Auditory deficits in patients with mild and moderate obstructive sleep apnea syndrome: A speech syllable evoked auditory brainstem response study. *Clin. Exp. Otorhinolaryngol.* 12(1), 58–65.

Galvin, J.E., 2024. Lewy body dementia. *Continuum* 30(6), 1673–1698.

Golden, H.L., Agustus, J.L., Goll, J.C., et al., 2015. Functional neuroanatomy of auditory scene analysis in Alzheimer's disease. *NeuroImage: Clin.* 7, 699–708.

Häggström, J., Hederstierna, C., Rosenhall, U., et al., 2020. Prognostic value of a test of central auditory function in conversion from mild cognitive impairment to dementia. *Audiol. Neurotol.* 25(5), 276–282.

Hamed, S.A., Abd-ElHamed, M.A., Oseily, A.M., et al., 2025. Assessment of auditory perceptual functions in patients with Parkinson's disease. *Am. J. Neurodegener. Dis.* 14(3), 82–99.

Hampel, H., Mesulam, M.M., Cuello, A.C., et al., 2018. The cholinergic system in the pathophysiology and treatment of Alzheimer's disease. *Brain* 141(7), 1917–1933.

Hassan, D.M., Shalash, A., 2017. Auditory brainstem evoked responses and vestibular evoked myogenic potentials: Potential biomarkers in Parkinson's disease. *Egypt. J. Otolaryngol.* 33(2), 508–517.

Hong, S.J., Baek, S.H., Lai, M.K.P., et al., 2024. Ageing-associated sensory decline and Alzheimer's disease. *Mol. Neurodegener.* 19(1), 93.

Huang, H.M., Chen, G.S., Liu, Z.Y., et al., 2023. Age-related hearing loss accelerates the decline in fast speech comprehension and the decompensation of cortical network connections. *Neural Regen. Res.* 18(9), 1968–1975.

Iyer, P.M., Mohr, K., Broderick, M., et al., 2017. Mismatch negativity as an indicator of cognitive sub-domain dysfunction in amyotrophic lateral sclerosis. *Front. Neurol.* 8, 395.

Jack, C.R. Jr., Holtzman, D.M., 2013. Biomarker modeling of Alzheimer's disease. *Neuron* 80(6), 1347–1358.

Jafari, Z., Kolb, B.E., Mohajerani, M.H., 2020. Auditory dysfunction in Parkinson's disease. *Mov. Disord.* 35(4), 537–550.

Jalaei, B., Valadbeigi, A., Panahi, R., et al., 2019. Central auditory processing tests as diagnostic tools for the early identification of elderly individuals with mild cognitive impairment. *J. Audiol. Otol.* 23(2), 83–88.

Jalilianhasanpour, R., Beheshtian, E., Sherbaf, G., et al., 2019. Functional connectivity in neurodegenerative disorders: Alzheimer's disease and frontotemporal dementia. *Top. Magn. Reson. Imaging* 28(6), 317–324.

Kim, G.Y., Kim, H., Kim, H.J., et al., 2022. Central auditory processing disorder in patients with amnestic mild cognitive impairment. *Behav. Neurol.* 2022, 9001662.

Lasheen, R.M., Elsheikh, M.N., Tomoum, M.O., 2025. Asymmetrical auditory dysfunction as a potential nonmotor lateralizing sign in Parkinson's disease: A case-control study. *Otol. Neurotol.* 46(9), e370–e376.

Ma, X.R., Li, J.Y., Wang, Y., et al., 2025. Can auditory processing dysfunction indicate early cognitive decline? Alzheimers Dement. 17(4), e70189.

Milán-Tomás, Á., Fernández-Matarrubia, M., Rodríguez-Oroz, M.C., 2021. Lewy body dementias: A coin with two sides? Behav. *Sci.* 11(7), 94.

Mollaei, F., Shiller, D.M., Baum, S.R., et al., 2022. Early auditory responses to speech sounds in Parkinson's disease: Preliminary data. *Sci. Rep.* 12, 1019.

Näätänen, R., Kujala, T., Kreegipuu, K., et al., 2011. The mismatch negativity: An index of cognitive decline in neuropsychiatric and neurological diseases and in

ageing. *Brain* 134(12), 3435–3453.

Nagaraj, N.K., 2024. Hearing loss and cognitive decline in the aging population: Emerging perspectives in audiology. *Audiol. Res.* 14(3), 479–492.

Nilsson, J., Pichet Binette, A., Palmqvist, S., et al., 2024. Cerebrospinal fluid biomarker panel for synaptic dysfunction in a broad spectrum of neurodegenerative diseases. *Brain* 147(7), 2414–2427.

Padhi, P., Worth, C., Zenitsky, G., et al., 2022. Mechanistic insights into gut microbiome dysbiosis-mediated neuroimmune dysregulation and protein misfolding and clearance in the pathogenesis of chronic neurodegenerative disorders. *Front. Neurosci.* 16, 836605.

Pal, S., Kaushik, A., Goel, F., et al., 2025. Revolutionizing early diagnosis: The role of artificial intelligence in neurodegenerative disorders. *J. Adv. Sci. Res.* 16(3), 31–37.

Sardone, R., Battista, P., Panza, F., et al., 2019. The age-related central auditory processing disorder: Silent impairment of the cognitive ear. *Front. Neurosci.* 13, 619.

Shalash, A.S., Hassan, D.M., Elrassas, H.H., et al., 2017. Auditory- and vestibular-evoked potentials correlate with motor and non-motor features of Parkinson's disease. *Front. Neurol.* 8, 55.

Shetty, K., Krishnan, S., Thulaseedharan, J.V., et al., 2019. Asymptomatic hearing impairment frequently occurs in early-onset Parkinson's disease. *J. Mov. Disord.* 12(2), 84–90.

Sun, L., Xu, M.R., Zhou, C.Y., et al., 2025a. Diagnostic dynamic contrast-enhanced magnetic resonance imaging blood-brain barrier assessment combined with plasma biomarkers for mild cognitive impairment. *World J. Psychiatry* 15(6), 103661.

Sun, S.C., Zhao, Q., He, H.J., et al., 2025b. Pathophysiological insights and therapeutic developments in age-related hearing loss: A narrative review. *Front. Aging Neurosci.* 17, 1657603.

Thamizhmani, L., Ganapathy, K., Palaniswamy, H.P., et al., 2025. Auditory processing and its cognitive correlates in older adults with mild cognitive impairment. *BMC Geriatr.* 25(1), 373.

Thomson, R.S., Auduong, P., Miller, A.T., et al., 2017. Hearing loss as a risk factor for dementia: A systematic review. *Laryngoscope Investig. Otolaryngol.* 2(2), 69–79.
Toader, C., Tataru, C. P., Munteanu, O., et al, 2024. "Decoding neurodegeneration: A Review of molecular mechanisms and therapeutic advances in Alzheimer's, Parkinson's, and ALS", International Journal of Molecular Sciences, 25(23), 12613.

Tong, Z.P., Xing, C.H., Xu, X.M., et al., 2025. Impaired network organization in mild age-related hearing loss. *MedComm* 6(1), e70002.

Toader, C., Tataru, C. P., Munteanu, O., et al, 2024. Decoding neurodegeneration: A Review of molecular mechanisms and therapeutic advances in Alzheimer's, Parkinson's, and ALS, International Journal of Molecular Sciences, 25(23), 12613.

Tuwaig, M., Savard, M., Jutras, B., et al., 2017. Deficit in central auditory processing as a biomarker of pre-clinical Alzheimer's disease. *J. Alzheimers Dis.* 60(4), 1589–1600.

Utoomprurkporn, N., Hardy, C.J.D., Stott, J., et al., 2020. "The Dichotic Digit Test" as an index indicator for hearing problem in dementia: Systematic review and meta-analysis. *J. Am. Acad. Audiol.* 31(9), 646–655.

Yang, L., Wang, S.J., Chen, Y.N., et al., 2024. Effects of age on the auditory cortex during speech perception in noise: Evidence from functional near-infrared spectroscopy. *Ear Hear.* 45(3), 742–752.

Zhou, Y., Han, H.L., Zhang, X.L., et al., 2025. Dual improvement of cognitive function and auditory ability in elderly patients with hearing impairment by transcranial direct current stimulation-assisted auditory rehabilitation training. *Front. Aging Neurosci.* 17, 1591496.