Development and Validation of a Prognostic and Assessment Checklist for Sudden Sensorineural Hearing Loss (SSNHL)

Shruti Darshana Mishra

Registration No: P01II24S023031

A Dissertation Submitted in Part Fulfilment of the

Degree of Master of Science

Audiology

University of Mysore

ALL INDIA INSTITUTE OF SPEECH AND HEARING,

MANASAGANGOTHRI, MYSORE – 570006

JUNE, 2026

CERTIFICATE

This is to certify that this dissertation entitled **"Development and Validation of a Prognostic and Assessment Checklist for Sudden Sensorineural Hearing Loss (SSNHL)"** is a bonafide work submitted as part of fulfilment of the degree of Master of Science **(Audiology)** of the student with Registration Number **P01II24S023031.** This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any other Diploma or Degree.

M. Pushpavathi

Mysuru
9th June, 2026

Dr. M. Pushpavathi
Director
All India Institute of Speech and Hearing
Manasagangothri, Mysuru-570 006

CERTIFICATE

This is to certify that the dissertation entitled **"Development and Validation of a Prognostic and Assessment Checklist for Sudden Sensorineural Hearing Loss (SSNHL)"** has been prepared under my supervision and guidance. It is also certified that this dissertation has not been submitted earlier to any other university for the award of any diploma or degree.

Mysuru
June, 2026

Dr. Mamatha N M
Guide
Assistant Professor
Department of Audiology
All India Institute of Speech and Hearing
Manasagangothri
Mysore – 570006

Dr. G. Rajeshwari
Co-guide
Professor & HOD
Department of ENT & Otorhinolaryngology
All India Institute of Speech and Hearing
Manasagangothri
Mysore – 570006

DECLARATION

This is to certify that the dissertation entitled **"Development and Validation of a Prognostic and Assessment Checklist for Sudden Sensorineural Hearing Loss (SSNHL)"** is a result of my own study done under the guidance of Dr. Mamatha N M, Assistant Professor Department of Audiology and Dr. G. Rajeshwari, Professor & HOD of ENT Department of Otorhinolaryngology, All India Institute of Speech and Hearing, Mysore-06. This dissertation has not been submitted earlier to any other university for the award of any diploma or degree.

Mysuru **Registration No: P01II22S023031**

June, 2026

ACKNOWLEDGEMENT

"The journey of a thousand miles begins with a single step, but it is the people who walk beside us who make the journey meaningful."

The completion of this dissertation marks an important milestone in my academic journey filled with learning, challenges and growth. It would not have been possible without the guidance, encouragement, and support of many individuals, to whom I extend my sincere gratitude.

First and foremost, I extend my heartfelt thanks to **Dr M. Pushpavathi**, Director, AIISH, Mysore, for providing an environment that fosters learning, research, and professional growth.

My deepest gratitude goes to my guide, **Dr N. M. Mamatha**, whose mentorship has been invaluable throughout this journey. Her expertise, thoughtful guidance, constructive feedback, patience, and constant encouragement helped shape this dissertation at every stage. Beyond research, her calm approach, compassionate outlook, and ability to approach challenges with patience and perspective have been a source of inspiration, teaching lessons that extend far beyond research.

I am equally grateful to my co-guide, **Dr Rajeshwari G.**, for her invaluable support and insights throughout this study. Her broader clinical perspective greatly strengthened the quality and relevance of this research. Her suggestions, encouragement, and ability to bridge the gap between audiological and medical viewpoints helped me appreciate the interdisciplinary nature of patient care and research.

I also extend my heartfelt appreciation to the esteemed panel of experts, **Dr Animesh Barman, Dr Ajith Kumar U, Dr Sharath Kumar, Dr Arunraj K, and Dr T. K. Prakash**, for their valuable feedback, insightful suggestions, and professional expertise. Their guidance played a significant role in refining the study and strengthening its overall structure and scientific rigour.

I would also like to express my deepest gratitude to my guru, **Dr Swarup Bikash Mishra**, whose mentorship extends far beyond academics. His guidance, belief in my abilities, and passion for the field of Audiology & Vestibular sciences have been a constant source of inspiration. In many ways, he laid the foundation of my journey in practical Audiology. The confidence to pursue my dreams and eventually study at my dream institute was nurtured by his encouragement and support. For that, I will always remain grateful.

To my beloved parents, **Mom and Dad**, words can hardly capture my gratitude. Thank you for believing in me even during the times when I doubted myself. Your sacrifices, unconditional love, endless encouragement, and quiet strength have been the pillars supporting every achievement in my life. This accomplishment belongs to you as much as it does to me.

To my dear Sakhi Shalei group, **Sonal, Swasti, Varsha, and Shivani**, thank you for turning a demanding postgraduate journey into a collection of cherished memories. From the countless conversations, shared laughter, mutual understanding, last-minute panic sessions, late-night rants, and for always reminding me that I was not alone in this journey. The dissertation may have been written individually, but surviving it was definitely a group project.

A very special thank you to my best friend, **Samresh**, for being a constant source of support throughout this entire journey. Thank you for understanding me at my level, for

listening without judgment, for celebrating the highs, helping me through the lows, and for always showing up when I needed a friend. Your encouragement and belief in me have meant more than words can express.

A special note of gratitude goes to **Ms Bhuvana** ma'am from the Department of ENT for her unwavering support throughout the data collection process and beyond. Her willingness to help and constant guidance made the research journey much smoother. I am also thankful to my junior, **Ambika**, for her valuable assistance during data collection, which greatly contributed to the successful completion of this study.

Finally, I would like to thank all the participants who contributed to this study and everyone who, directly or indirectly, supported me throughout this journey.

As this chapter comes to a close, I carry forward not only the knowledge I have gained but also the friendships, memories, lessons, shared struggles, and stories that made these years so special. This dissertation may be bound between covers, but the experiences, growth, and people behind it will stay with me forever. While the dissertation is now complete, the learning and perhaps the occasional academic chaos will undoubtedly continue.

Thank you all for being a part of it !!

Shruti Darshana Mishra

31.05.2026

AIISH

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

LIST OF FREQUENTLY USED ABBREVIATIONS

Abbreviation	Full Form
AAO-HNS	American Academy of Otolaryngology–Head and Neck Surgery
ABR	Auditory Brainstem Response
AICA	Anterior Inferior Cerebellar Artery
AIISH	All India Institute of Speech and Hearing
BPPV	Benign Paroxysmal Positional Vertigo
CI	Confidence Interval
CMV	Cytomegalovirus
COVID-19	Coronavirus Disease 2019
CT	Computed Tomography
dB HL	Decibels Hearing Level
DHI	Dizziness Handicap Inventory
DPOAE	Distortion Product Otoacoustic Emission
ENT	Ear, Nose, and Throat
HADS	Hospital Anxiety and Depression Scale
HBOT	Hyperbaric Oxygen Therapy
HHIA	Hearing Handicap Inventory for Adults
HIV	Human Immunodeficiency Virus
HSV	Herpes Simplex Virus
IL-6	Interleukin-6

IRB	Institutional Review Board
ISSNHL	Idiopathic Sudden Sensorineural Hearing Loss
IT	Intratympanic
MRI	Magnetic Resonance Imaging
NF-κB	Nuclear Factor Kappa B
NHS	National Health Service
OAE	Otoacoustic Emission
OR	Odds Ratio
PCOS	Polycystic Ovary Syndrome
PTA	Pure-Tone Average
r_s	Spearman Rank Correlation Coefficient
SARS-CoV-2	Severe Acute Respiratory Syndrome Coronavirus 2
SD	Standard Deviation
SNHL	Sensorineural Hearing Loss
SPSS	Statistical Package for the Social Sciences
SSD	Single-Sided Deafness
SSNHL	Sudden Sensorineural Hearing Loss
TEOAE	Transient Evoked Otoacoustic Emission
THI	Tinnitus Handicap Inventory
TIA	Transient Ischemic Attack
TNF-α	Tumor Necrosis Factor-Alpha
TSH	Thyroid-Stimulating Hormone

USNHL	Unilateral Sensorineural Hearing Loss
VEMP	Vestibular Evoked Myogenic Potential
VZV	Varicella-Zoster Virus
WRS	Word Recognition Score

ABSTRACT

Introduction: Sudden sensorineural hearing loss (SSNHL) is a well-recognised otological emergency, defined as a rapid decline in hearing sensitivity of ≥30 dB across three or more consecutive audiometric frequencies within 72 hours. Despite its clinical urgency, there is no standardised, condition-specific instrument that systematically captures the full range of prognostic indicators, associated clinical features, and etiological variables relevant to patient assessment and outcome counselling. The present study sought to address this gap by developing and preliminarily validating a structured prognostic and assessment checklist for SSNHL, and by examining its association with post-treatment audiometric outcomes following steroid therapy.

Aim: To develop and preliminarily validate a condition-specific Assessment and Prognostic Indicator Checklist for Sudden Sensorineural Hearing Loss (SSNHL) and to examine its association with post-treatment audiometric outcomes.

Methods: A cross-sectional design for checklist development and validation was employed. The checklist was developed through an extensive review of existing literature and clinical resources. It was organised into two sections: Section A, comprising seven established prognostic indicators with an ordinal scoring system, and Section B, documenting associated clinical symptoms, systemic risk factors, and possible etiological variables. Content validation was carried out by a panel of six experienced audiologists and otolaryngologists with a minimum of 10 years of clinical and academic experience, whose structured feedback guided iterative refinement of the instrument. The validated checklist was subsequently administered to 24 adults (18 males, 6 females; mean age 46.25 years; range 20–70 years) diagnosed with SSNHL at the All India Institute of Speech and Hearing (AIISH), Mysuru. Statistical analyses were performed using IBM SPSS Statistics (Version 26), with Spearman's rank correlation

used to examine associations between Section A prognostic scores and post-treatment audiometric outcomes.

Results: Spearman's rank correlation analysis revealed a statistically significant moderate positive correlation between the total Section A prognostic score and post-treatment pure-tone average (r_s = 0.490, p = .018), indicating that participants with less favourable prognostic profiles at presentation demonstrated poorer audiometric outcomes following steroid therapy. Among individual prognostic variables, treatment initiation time (r_s = 0.525, p = .008) and audiogram configuration (r_s = 0.513, p = .010) showed the strongest associations with the cumulative prognostic score. Based on the criterion of a ≥15 dB post-treatment PTA gain, 13 participants (54.2%) demonstrated audiometric improvement. Supplementary classification using Siegel's criteria revealed complete recovery in 2 participants (8.3%), partial recovery in 4 (16.7%), slight improvement in 7 (29.2%), and no improvement in 11 (45.8%). Descriptive analysis of Section B variables identified diabetes mellitus and lifestyle-related risk factors as the most prevalent comorbidities (37.5% each), with tinnitus (66.7%), vertigo (54.2%), and aural fullness (50.0%) as the most frequently reported associated symptoms. Notably, all participants with hypercholesterolemia were in the non-improvement group, consistent with the vascular hypothesis for SSNHL.

Conclusions: The developed checklist demonstrated face and content validity as confirmed by expert review, and the preliminary correlation analysis provided initial evidence for its prognostic utility. The checklist offers a structured, evidence-informed approach for the comprehensive clinical evaluation of SSNHL, with potential to support prognostic profiling, patient counselling, and interdisciplinary communication across clinical settings. Larger prospective studies are recommended to establish the predictive validity and inter-rater reliability of the instrument.

Keywords: sudden sensorineural hearing loss, prognostic checklist, audiometric outcome, corticosteroid therapy, clinical assessment, content validation, Siegel's criteria.

Chapter I

INTRODUCTION

Sudden sensorineural hearing loss represents an abrupt disruption of auditory function, leading to partial or complete loss of hearing. The medical term is preferred to be used when the hearing level is reduced by more than 30 dB in three or more consecutive audiometric frequencies within a period of 72 hours (Wilson et al., 1980). Sudden loss of hearing expresses a form of sensorineural injury that appears abruptly, normally in minutes, hours, or within the span of a few days (Byl, 1984; Mattox & Simmons, 1977).

Incidence of sudden sensorineural hearing loss (SSNHL) varies greatly worldwide, with reported figures varying from approximately 5 to over 400 per 100,000 people annually (Yang et al., 2024). Higher incidence rates of SSNHL were reported from Japan (60.9 per 100,000) compared to the USA (approximately 27 per 100,000) and Korea (17.76 per 100,000) (Alexander & Harris, 2013; S. H. Kim et al., 2017; Nakashima et al., 2014). In the USA alone there are an estimated 60,000-66,000 new cases per year, though the actual number may be higher, given that mild cases of SSNHL and those with spontaneous resolution are often not reported (Chandrasekhar et al., 2019). Epidemiologically, the incidence of SSNHL worldwide is slowly increasing. From 2000 to 2013, the incidence of SSNHL in Taiwan increased from 5.15 to 13.97 per 100,000 (Kuo et al., 2019).

Middle-aged and elderly patients are more commonly affected by SSNHL than young people. Incidence has been found to increase with age, and is highest during the 5 th to 6 th decades of life (Byl, 1984; S. H. Kim et al., 2017). There appears to be a slight female preponderance in some series and a slight male preponderance in others, so this may vary between populations (Byl, 1984; S. H. Kim et al., 2017).

Hearing loss disorders have been studied in a wide range of countries though relatively little has been written regarding the extent of single-sided deafness in India. Gupta et al. (2021), in a prospective observational study of 130 adult patients with unilateral sensorineural hearing loss (USNHL), estimated that the incidence of adult-onset deafness to be approximately 7.6%, and unilateral hearing loss to affect between 7.9%-13.3% of the population. The peak incidence was between the ages of 36 and 45 (36.9%), with a male predominance. There was a delay in presentation, as 30% presented between 1-5 years of symptoms and 43% presented with profound hearing loss. At a community level, Konadath et al. (2017) assessed the prevalence of single-sided deafness (SSD) within an island group by screening an at-risk population, identified through a high-risk register methodology. In total 13 out of 106 subjects (approximately 0.05%) screened (22,558 people screened) had unilateral hearing loss. The risk factors were not assessed in this population-based study.

Sudden sensorineural hearing loss (SSNHL) can be further divided into primary (idiopathic) and secondary forms. Primary SSNHL is typically associated with viral illness, vascular issues and immune-mediated disorders (Schreiber et al., 2010), while secondary forms may arise from a known condition, such as a tumour or cerebrovascular event or from external sources, such as radiation. Causes such as acoustic trauma, head injury, Ménière's disease, perilymph fistula and ototoxicity are usually described separately because, despite sudden hearing loss as an initial presentation, their clinical course often differs from other causes of sudden sensorineural hearing loss (Chau et al., 2010).

Sudden sensorineural hearing loss (SSNHL) is predominantly a unilateral condition, with over 90% of patients presenting with a single-sided deficit (Byl, 1984). Although there is limited specific data regarding SSNHL in India, information on general hearing loss implies that single-sided presentations are common. Bilateral SSNHL is rarer, though it should alert to a more systemic cause, either immune-mediated or vascular in origin (Alexander & Harris,

2013; Chau et al., 2010). Simultaneous bilateral SSNHL is an extremely rare presentation. Patients typically realise they have suffered hearing loss either upon waking or during routine activity, hearing the change in one ear (Byl, 1984). The hearing loss may be realised to have been instantaneous or developed over a period of time (Chandrasekhar et al., 2019).

SSNHL is classified as an otological emergency due to the rapid nature of the insult and the potential for a permanent and severe disability if treatment is delayed. Idiopathic sudden sensorineural hearing loss (ISSNHL), the most common form, affects primarily middle-aged patients, and there is no consistent preference for gender or side (Byl,1984). In approximately 10% of cases, a definite underlying cause can be identified following detailed evaluation; however, in most patients, no specific etiology is found, and the condition is therefore classified as idiopathic (Chau et al., 2010).

Several mechanisms have been proposed to explain the pathogenesis of ISSNHL, including viral infections, vascular compromise, and autoimmune processes. Autoimmune-related conditions such as systemic lupus erythematosus, Behçet's disease, and Cogan's syndrome have been implicated in some cases (Schreiber et al., 2010). Despite ongoing research, the exact etiology, optimal treatment strategies, and overall management of sudden sensorineural hearing loss remain areas of ongoing debate and uncertainty (Chandrasekhar et al., 2019; Rauch, 2008).

In many cases of sudden sensorineural hearing loss (SSNHL), no specific underlying cause can be identified, and such cases are classified as idiopathic (Chau et al., 2010). However, several systemic and vascular factors have been proposed to contribute to its pathogenesis, including cardiovascular disease, hypertension, and smoking (Chandrasekhar et al., 2010). The prognosis of SSNHL is reported to be highly variable. Poorer outcomes have been associated with increasing age, greater severity of initial hearing loss, and the presence of vertigo at the

initial presentation of hearing loss. Additionally, certain audiometric configurations have been suggested to be associated with prognosis, although their predictive value remains less consistent compared to other clinical factors (Alexander & Harris, 2013).

The abrupt onset of sudden sensorineural hearing loss can significantly disrupt daily functioning and overall well-being. Patients often experience immediate communication difficulties, and commonly associated symptoms such as tinnitus and vertigo may further increase the functional burden. These symptoms can have a substantial impact on daily activities and quality of life (Chandrasekhar et al., 2019; Chau et al., 2010).

The prognosis is highly variable, with some patients experiencing partial or complete recovery, while others continue to have persistent hearing deficits. Recovery outcomes are influenced by several factors, including age, severity of initial hearing loss, presence of vertigo, and timing of intervention. Early initiation of treatment has been associated with improved hearing outcomes (Chandrasekhar et al., 2019; Kuhn et al., 2011).

In clinical practice, assessment typically involves pure-tone audiometry, speech audiometry, and tympanometry, with additional investigations such as imaging performed when indicated to exclude secondary causes (Chandrasekhar et al., 2019; Stachler et al., 2012).

While these tools are essential for confirming the diagnosis and characterising the degree of hearing loss, they provide limited insight into prognostic indicators such as symptom onset, associated features, and comorbid conditions. This highlights the need for a more comprehensive clinical evaluation that goes beyond standard audiological measures to better understand patient outcomes and recovery potential.

Need for the study

Sudden sensorineural hearing loss (SSNHL) is an otological emergency that needs to be recognised quickly so as to afford the patient the optimal chance of regaining some of their hearing. Studies indicate that approximately a third to two-thirds of patients regain hearing spontaneously to some extent, while others recover only partially, if at all (Byl, 1984; Chau et al., 2010). Despite advances in diagnostics and management, the clinical team may still face difficulties with systematic evaluation of potential causes, prognostic factors and clinically derived characteristics of the condition.

Various pathogenic mechanisms may underlie SSNHL, including viral, vascular and autoimmune pathways, while the specific therapy may be guided by the presumed etiology, with corticosteroids being the primary treatment modality, supplemented in the right setting by additional treatment regimens (Chandrasekhar et al., 2019; Kuhn et al., 2011). Since there is often no single test that can accurately identify the cause of sudden hearing loss, diagnosis largely depends on careful clinical assessment. While audiological tests help determine the degree of hearing loss, they may not provide enough detail on their own. For this reason, it is important to document relevant symptoms, associated conditions, and potential triggering factors in a clear and structured manner to support early recognition of sudden sensorineural hearing loss (SSNHL) and ensure prompt treatment.

A simple checklist or questionnaire could assist clinical evaluation in this way, serving as a clinical tool that systematically documents relevant patient symptoms, signs and medical history that are important to elicit, and reducing the chance that important information is missed and also improving the consistency of examination at different centers and improving overall clinical assessment and facilitating appropriate documentation of characteristics related to the patient's symptoms and the patient themselves (Chandrasekhar et al., 2019; Chau et al., 2010).

Not only does the treatment need to be timely, but patients require a good explanation with realistic prognostics regarding their chance of recovery, variability in prognosis and importance of rapid treatment initiation, and the likely outcomes may depend on various factors such as the patient's age and severity of hearing loss, presence of vestibular dysfunction, etc. (Kuhn et al., 2011). It is important to give patients the relevant information regarding their specific prognosis and condition so that their anxiety is lessened and compliance with treatment is encouraged, along with seeking early medical help.

While there exist numerous standard questionnaires to elicit the extent of hearing related disability and patient's quality of life, e.g. Hearing Handicap Inventory for Adults (HHIA) and the Tinnitus Handicap Inventory (THI), they have been designed for psychosocial or vestibular assessment (Ventry & Weinstein, 1982; Newman et al., 1996) rather than documenting the important clinical features and prognostic indicators of SSNHL, in a similar way as is seen for The Dizziness Handicap Inventory which is a common measure for vestibular assessment (Jacobson & Newman, 1990) and general psychological instruments such as The Hospital Anxiety and Depression scale (HADS) for patient anxiety and depression in the context of hearing related problems (Zigmond & Snaith, 1983). Therefore, a clear requirement for a condition-specific questionnaire is to help doctors make an early, comprehensive assessment for SSNHL.

Thus, there is a need for a condition-specific, clinically directed screening and assessment checklist for Sudden Sensorineural Hearing Loss to facilitate more effective evaluation, cross-disciplinary referral and patient education, which is so critical for appropriate clinical decision making.

Aim of the study

This study aims to develop and validate a practical prognostic and assessment checklist for sudden sensorineural hearing loss (SSNHL) to help clinicians more comprehensively assess relevant clinical indicators in affected patients.

Objectives of the study

- To develop a condition-specific screening and assessment checklist for sudden sensorineural hearing loss (SSNHL), enabling to systematically capture key prognostic indicators, associated symptoms, and systemic risk factors often missed in routine practice.
- To evaluate the association between the Section A prognostic score and post-treatment audiometric outcome (post-treatment PTA) following steroid therapy in patients with SSNHL.
- To descriptively assess outcome trends across different prognostic score ranges and individual Section A prognostic variables in patients with SSNHL.
- To describe the distribution of clinical risk factors (Section B1) and possible etiological factors (Section B2) among patients with SSNHL using descriptive statistics.

Chapter II

REVIEW OF LITERATURE

Several modifications have been made to the definition of SSNHL over the course of its history since it was first conceptualised. The specific condition, sudden hearing loss without any apparent etiology has been identified as a distinct clinical entity since the mid-twentieth century, as it was first described by De Kleyn et al in 1944 (Mathews et al., 2020). In subsequent decades, criteria have been formulated to define the condition and have shown inconsistent results as early and mid-range studies show more lax audiometric standards than current treatment trials which reveal inconsistency in definitive diagnosis criteria (Eisenman & Arts, 2000).

In an attempt to standardise diagnosis and so to make studies comparable, an agreed definition for Sudden Sensorineural Hearing Loss was established, and is defined as an unexplained sensorineural hearing loss (SNHL) affecting at least three consecutive frequencies and totalling 30 dB or more that develops within 72 hours (Wilson et al., 1980). Most recent treatment trials continue to follow this definition as outlined by the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) guideline, which explicitly uses this definition and notes that hearing loss is typically quantified relative to the thresholds of the unaffected ear when premorbid audiometry is unavailable (Chandrasekhar et al, 2019). The literature contains multiple review articles that use the ≥30 dB at three frequencies over 72 hours criterion as the most frequently applied diagnostic standard, which demonstrates its essential role in maintaining uniformity during diagnostic assessments and research. (Kuhn et al., 2011; Mathews et al., 2020).

The literature shows agreement among researchers yet displays multiple differences. The first study defined SSNHL through a≥20 dB loss, whereas the second study included cases that showed two frequencies at the same time (He et al., 2024). The second clinical guideline explains SSNHL through a simultaneous loss of 10-20 dB at two or three frequencies, which occurs within 12 hours according to various clinical descriptions that show different patterns (Chen & Fraser-Kirk, 2024). The different study methods that researchers used for their investigations lead to distinct epidemiological measurement outcomes, which affect the way people estimate their recovery progress. The use of less strict criteria in research studies results in higher reported incidence rates and improved recovery results because diagnostic differences between studies cause different findings to emerge in published research.

The clinical progression and prognosis of Sudden Sensorineural Hearing Loss (SSNHL) have received major insights from essential research studies. The first complete study about the natural progression by Mattox & Simmons (1977) found that most patients achieved natural recovery without any medical treatment, which resulted in a recovery percentage between 32% and 65%, depending on their illness intensity and accompanying symptoms. Their study results showed positive outcomes for patients who had mild loss but no vertigo. Later another large study over eight years with 85 patients was presented and the results illustrated a prognostic profile for sudden sensorineural hearing loss (Byl, 1977). The severity of initial hearing loss and treatment delay were one of the greatest indicators of success while other signs for a poor outcome included a severe hearing loss, as well as delayed treatment (Byl, 1977).

The modern research about diagnosing pathophysiology and treatment methods for idiopathic SSNHL was summarised in a recent comprehensive study. The study proved that clinicians use the diagnostic standard of hearing loss greater than 30 dB, which affects three continuous pure tone frequencies within 72 hours, as the official hearing impairment definition

(Rauch, 2008). The review also acknowledged multifactorial etiological hypotheses and variability in recovery patterns that previous studies had documented.

2.1 Epidemiology

Sudden Sensorineural Hearing Loss (SSNHL) represents a rare yet critical hearing emergency, for which different communities and research methods have established its occurrence rate at different levels. Population-based studies generally report the annual incidence of SSNHL to be approximately 5 to 27 cases per 100,000 people per year(Alexander & Harris, 2013; Chandrasekhar et al., 2019), with about 66,000 new cases per year in the United States (Chandrasekhar et al., 2019).The range underestimates the actual incidence because some patients with less severe symptoms will get better without treatment, and they do not seek medical help (Chandrasekhar et al., 2019; Rauch, 2008).The incidence increased with increasing age, ranging from 11 per 100,000 for patients younger than 18 years to 77 per 100,000 for patients 65 years and older (Alexander & Harris, 2013).

Current systematic reviews and clinical overviews repeat this age incidence pattern, which shows different rates between regions and healthcare-seeking patterns (Chandrasekhar et al., 2019). Large population-based studies confirm that different regions show different rates of occurrence. The Taiwanese nationwide retrospective analysis of hospitalised SSNHL patients showed that annual incidence increased steadily from 2000 to 2010, starting at 5.15 per 100,000 people and reaching 13.97 per 100,000, while middle-aged adults (45–64 years) showed the highest occurrence rate (Kuo et al., 2019). Similarly, population health insurance data from South Korea reported an annual incidence of 17.76 cases per 100,000, with increasing occurrence in older age groups (Kim et al., 2017).

In terms of age distribution, SSNHL has been observed across all age groups, but most frequently affects middle-aged and older adults. Review articles note that the peak incidence

tends to occur in the fifth and sixth decades of life, although younger and older age groups can also be affected (Chandrasekhar et al., 2019; Kim et al., 2017; Rauch, 2008). Gender distribution in SSNHL appears to be relatively balanced, with most epidemiological studies reporting either a near equal distribution or a slight male predominance (Kuhn et al., 2011). However, some cohorts have demonstrated a slight female preponderance, with reported male-to-female ratios of approximately 1:1.35 (Kim et al., 2017). Other large population-based analyses have observed an overall slight male predominance of 1.07:1, which becomes more pronounced in individuals aged 65 years and older, 1.30:1 (Alexander & Harris, 2013). These findings suggest that sex distribution may vary across populations and age groups. It is predominantly unilateral in nature, with fewer than 2% of cases bilateral (Kuhn et al., 2011).

Despite the lack of comprehensive national epidemiological data for Indian patients with SSNHL, some Indian clinical trials do provide context. An observational study by Gupta et al. (2021) involving 130 adults with unilateral sensorineural hearing loss (USNHL) reported that about 36% of cases had sudden hearing loss, most commonly observed among individuals aged 36–45 years, with a higher prevalence among males. Although the study had elements of sudden and gradual USSNHL, it provides some demographic clues for SSNHL presentation in the Indian population.

Another study by Konadath et al. (2017) looked into the incidence of communication disorders in an island group in India. The investigators used a high-risk register methodology during the survey to identify individuals affected by communication disorders. This implied that of the population size of 22,558, a total of 106 individuals were found with hearing loss, of which 13 suffered from unilateral hearing loss. The prevalence of single-sided deafness in all age groups was reported as 0.05%. Factors related to SSD, however, were not highlighted in the study. In addition, prospective Indian data related to post-COVID SSNHL have suggested that incidence in infected populations can appear elevated compared with pre-

pandemic baseline rates, although these findings require confirmation with larger representative samples (Kandakure et al., 2023).

Taken together, these epidemiological findings indicate that while global SSNHL incidence is relatively low compared with other auditory disorders, the condition affects tens of thousands of individuals annually, predominantly in middle to older age groups. Variability in reporting and region-specific healthcare systems, as well as factors such as spontaneous recovery and under-reporting, contribute to differences in reported incidence rates across studies and populations.

2.2 Clinical Importance of SSNHL

Sudden sensorineural hearing loss (SSNHL) is considered an otologic emergency and warrants rapid assessment and treatment (Chandrasekhar et al., 2019). Of all patients with SSNHL 32% to 65% recover spontaneously, and oral/intratympanic steroid therapy has been reported to help those seen within the first days of symptom onset (Saba et al., 2023). It has been established in guidelines that all patients require hearing assessment before steroid treatment initiation since the outcome of early treatment is better than that of later treatment (Chandrasekhar et al., 2019; Kuhn et al., 2011). Sudden hearing loss presentations are generally accompanied by other symptoms such as aural fullness, vertigo, and tinnitus, which are important factors influencing treatment outcomes and patient management (Rauch, 2008). It is clinically significant due to the potential for permanent hearing loss. Patients either recover completely or suffer persistent hearing loss leading to difficulty with communication, social implications, and a reduction in quality of life (Rauch, 2008). The potential for permanent auditory deficit underscores the clinical significance of SSNHL. Some patients achieve spontaneous recovery while other patients face ongoing hearing challenges, which lead to communication problems, psychosocial issues and decreased life satisfaction (Rauch, 2008).

The American Academy of Otolaryngology–Head and Neck Surgery guideline further reinforces the need for urgent assessment to exclude retrocochlear pathology and initiate evidence-based treatment strategies (Chandrasekhar et al., 2019).

2.3 Idiopathic Nature of SSNHL

The standard definition of sudden sensorineural hearing loss (SSNHL) requires that people develop their hearing impairment, which affects three or more tones, to a level of at least 30 dB within three days (Chandrasekhar et al., 2019). When no specific cause can be identified after appropriate clinical evaluation, the condition is classified as idiopathic sudden sensorineural hearing loss (ISSNHL). Traditional epidemiological studies have reported annual incidence rates that range from 5 to 20 cases per 100,000 population (Kuhn et al., 2011; Rauch, 2008). Recent population-based studies that use national health databases show that the actual incidence rate is higher than earlier estimates, and it grows with increasing age (Yoon et al., 2023).

Diagnostic techniques have improved since their inception, but they still fail to identify most SSNHL cases because 85 to 90 per cent of patients remain without a diagnosed cause(Kuhn et al., 2011; Nosrati-Zarenoe et al., 2007; Rauch, 2008). Contemporary research supports the finding which shows that approximately 85 to 90 per cent of patients with sudden sensorineural hearing loss present with idiopathic cases (Kuhn et al., 2011; Nosrati-Zarenoe et al., 2007; Rauch, 2008). Researchers have suggested various causes for the disease, including viral infections, vascular compromise, autoimmune inner ear disease, and membrane rupture, yet the actual pathophysiological mechanism behind ISSNHL remains unknown to scientists(Rauch, 2008; Tsuzuki & Wasano, 2024). The absence of specific laboratory markers or imaging findings that conclusively identify the underlying cause further contributes to the classification of most cases as idiopathic (Chandrasekhar et al., 2019). The identification of

SSNHL as an idiopathic condition results in major consequences for clinical practice. The unknown origin of the condition leads doctors to use empirical treatment methods, which depend on corticosteroids as the primary treatment, because doctors believe that the condition involves either inflammatory or immune-mediated processes (Rauch, 2008). The different proposed causes of the disease explain why patients with the disease show different symptoms and recovery patterns according to the study results (Kuhn et al., 2011). Hence, SSNHL represents a different condition that requires additional research to discover its fundamental causes.

2.4 Etiology of Sudden Sensorineural Hearing Loss

Despite advances in diagnostic and imaging modalities, the aetiology of sudden sensorineural hearing loss (SSNHL) remains somewhat undetermined. After completion of clinical, audiological and radiological assessments, a significant proportion of cases were designated as idiopathic. Repeatedly, between 85-90% of cases were stated as having an unknown etiology (Kuhn et al., 2011; Rauch, 2008); however, reports have varied from as low as ~7% up to ~45% concerning the number of SSNHL cases where an etiology was identified, depending on methodology and criteria (Kuhn et al., 2011).

The differential diagnosis for SSNHL is extensive, with over a hundred potential causes (Mattox & Simmons, 1977; Fetterman et al., 1996; Chau et al., 2010). In identifiable causes of SSNHL, all causes fall into either infectious, autoimmune, traumatic, vascular, neoplastic, metabolic or neurologic categories.

In a meta-analysis of 23 studies on identifiable causes, Chau et al. (2012) found that infectious causes were most common (13%), followed by otologic (5%), traumatic (4%), vascular or hematologic (3%), neoplastic (2%), and other (2%). Malingering, conversion

disorder and ototoxic drugs were not discussed, but must also be included in identifiable causes of SSNHL (Kuhn et al., 2011).

Contemporary reviews support this overwhelming proportion of idiopathic cases, suggesting that the exact mechanisms of the disease remain to be fully elucidated (Lee & Chung, 2024). This diverse range of causes means that treatment management of SSNHL is predominantly empirical rather than based on aetiology; hence, a focus has been placed on what are the most probable pathophysiological mechanisms of SSNHL presentations. Among the potential pathophysiological mechanisms that have received the most attention have been virus-induced lesions, compromised blood supply and autoimmune attack of the inner ear (Kuhn et al., 2011; Rauch, 2008; Tripathi & Deshmukh, 2022a). Each of these hypotheses has supporting clinical and experimental evidence, but none has been confirmed as the definite explanation for SSNHL.

Viral theory of SSNHL

The viral hypothesis remains one of the most widely discussed and historically supported mechanisms in the pathogenesis of sudden sensorineural hearing loss (SSNHL). Early clinical observations noted that a proportion of patients report antecedent upper respiratory tract infections before the onset of hearing loss, suggesting a possible viral trigger (Rauch, 2008). Additionally, the typically unilateral presentation and abrupt onset resemble patterns seen in viral labyrinthitis, further strengthening this hypothesis (Kuhn et al., 2011).

Several viruses have been implicated, including herpes simplex virus (HSV), varicella-zoster virus (VZV), cytomegalovirus (CMV), HIV, hepatitis virus, measles virus, rubella virus, mumps virus, Lassa virus, and enterovirus (Chen et al., 2019; Kuhn et al., 2011).

Viral infection has been implicated in the pathogenesis of sudden sensorineural hearing loss (SSNHL) through four principal mechanisms: direct viral invasion, reactivation of latent virus, immune-mediated injury, and stress-response–mediated damage. Direct invasion involves viral entry into the cochlear nerve or inner ear structures via hematogenous spread during viremia or through cerebrospinal fluid pathways (Irwin, 2006), leading to structural injury such as hair cell loss, spiral ganglion degeneration, and stria vascularis atrophy, as demonstrated in infections caused by mumps, herpes viruses, and Lassa virus(Chen et al., 2019). Measles and rubella have been temporally or serologically associated with isolated cases of sudden deafness. Despite widespread immunisation, which has led to a marked decline in these infections, the incidence of idiopathic SSNHL has not decreased correspondingly. This suggests that these viruses are unlikely to account for the majority of ISSNHL cases (Merchant et al., 2008).

Reactivation of latent viruses, particularly members of the Herpesviridae family, including herpes simplex virus and varicella zoster virus, has been strongly implicated, given their lifelong persistence in neural ganglia and high adult seroprevalence; clinical entities such as Ramsay Hunt syndrome support this mechanism, although epidemiological evidence remains inconsistent (Chen et al., 2019). Unfortunately, there are no good serologic tests to diagnose reactivation. Once an individual is seropositive for a latent herpes virus, an increase in titer does not diagnose reactivation (Merchant et al., 2008).

The immune-mediated hypothesis proposes that systemic viral infections trigger cross-reactive antibodies or autoimmune responses against inner ear antigens, resulting in cochlear inflammation, vasculitis-like changes, and secondary hair cell damage even after viral clearance (Chen et al., 2019; Hashimoto et al., 2005). Finally, the stress-response hypothesis highlights the role of cytokines such as TNF-α and IL-6, oxidative stress, and NF-κB pathway activation in cochlear microcirculatory compromise and apoptotic signalling, with evidence

suggesting that modulation of these inflammatory mediators correlates with hearing recovery (Chen et al., 2019). Collectively, current literature suggests that SSNHL is likely multifactorial, involving complex interactions between viral factors and host immune and inflammatory responses rather than a single uniform pathogenic pathway.

A recent systematic review by Tripathi & Deshmukh (2022) further consolidate the evolving understanding of viral-associated SSNHL. A comprehensive review highlighted the contribution of virus-triggered inflammatory cascades, emphasising that dysregulated cytokine signalling and sustained immune activation may perpetuate cochlear injury beyond the acute infectious phase. Another study by Yamada et al. (2022) underscored the association between viral infections and endothelial dysfunction, increased oxidative stress markers, and impairment of cochlear microcirculation, thereby strengthening the proposed link between viral and vascular mechanisms. Together with other contemporary evidence, these findings support a multifactorial model in which immune–inflammatory and microvascular alterations play a central role in viral-associated SSNHL.

The research review showed that viruses activate inflammatory pathways, which lead to cochlear damage through their uncontrolled cytokine production and ongoing immune system activity (Tripathi & Deshmukh, 2022). The study demonstrated that viral infections lead to endothelial dysfunction and elevated oxidative stress levels while also damaging cochlear microcirculation, which establishes a connection between viral and vascular mechanisms (Yamada et al., 2022). The current research findings establish a multifactorial model that explains how immune and microvascular changes contribute to SSNHL, which is linked to viral infections. The clinical studies have shown that 20 to 40 per cent of patients with SSNHL report experiencing an upper respiratory tract infection, cold or fever before their hearing loss begins (Byl, 1977; Mattox & Simmons, 1977; Rauch, 2008).The temporal relationship provides evidence that a virus might be responsible for the disease yet the common

occurrence of respiratory infections and the lack of consistent virological evidence make it difficult to prove a direct causal link.

The medical community now considers SARS-CoV-2 as a possible viral cause of SSNHL according to current research about COVID-19. It explains different ways that viruses can reach the inner ear through their neurotropic abilities and through the immune response which produces inner ear inflammation and through thrombotic microangiopathy which disrupts the cochlear blood supply. The existing research mainly consists of case studies and limited observational research while there is a lack of extensive epidemiological studies that show a direct causal connection (Yamada et al., 2022).

Despite the biological plausibility of viral involvement, routine viral serological testing is not recommended in standard clinical evaluation due to limited specificity and minimal impact on management decisions (Kuhn et al., 2011; Rauch, 2008). Thus, while the viral theory remains one of the most compelling explanatory models, definitive proof of a single viral etiology in most SSNHL cases remains lacking.

Vascular Theory

The vascular hypothesis proposes that sudden sensorineural hearing loss (SSNHL) results from acute compromise of cochlear blood flow. Idiopathic sudden sensorineural hearing loss (ISSNHL) has been proposed by some authors to have a vascular etiology, based on the cochlea's unique blood supply from small terminal arteries with no collateral circulation, rendering it highly vulnerable to ischemic insults (Kuhn et al., 2011). Vascular impairments are considered to be the most likely cause of ISSNHL. This hypothesis is consistent with the results of large cohort studies that show a positive association between ISSNHL and stroke (J. Y. Kim et al., 2018; S. Y. Kim et al., 2018). The clinical presentation of unilateral sudden SNHL has been compared to transient ischemic attacks, and proposed mechanisms include

vascular occlusion, embolism, vasospasm, haemorrhage, and altered blood viscosity(Tsuzuki & Wasano, 2024). However, several clinical and radiologic observations challenge a primary vascular cause in most cases, as hearing loss following definite vascular occlusion is typically permanent and associated with cochlear fibrosis, whereas most ISSNHL cases show partial or complete recovery and lack radiologic evidence of fibrosis (Kuhn et al., 2011). A distinct entity termed "vestibulo-cochlear artery syndrome" has been described, characterized by high-frequency hearing loss with vertigo due to vascular occlusion. Intralabyrinthine haemorrhage is another recognized but uncommon vascular cause, identifiable on T1-weighted and 3D-FLAIR MRI, particularly in patients with hematologic disorders or anticoagulant use (Tsuzuki & Wasano, 2024).

The relationship between ISSNHL and atherosclerotic risk factors remains inconsistent. Several systematic reviews and meta-analyses report associations with smoking, alcohol consumption, hypertriglyceridemia, elevated total cholesterol, diabetes mellitus, and hypertension (Kuhn et al., 2011; Tsuzuki & Wasano, 2024), whereas others find no significant association with hypertension, diabetes, LDL-C, HDL-C, triglycerides, or BMI, but a medical history of myocardial infarction was significantly associated with developing ISSNHL (Tsuzuki & Wasano, 2024). Among these factors, elevated total cholesterol appears to show the most consistent association (Tsuzuki & Wasano, 2024).

Additionally, a large systematic review and meta-analysis by Saba et al. (2023) reported that 35–40% of ISSNHL patients have hypercholesterolemia. The study also demonstrated a significant association between ISSNHL and elevated total cholesterol, further reinforcing the role of dyslipidemia in the vascular hypothesis. These findings strengthen the evidence suggesting that vascular compromise related to atherosclerotic changes may contribute to a subset of ISSNHL cases.

Further support for a vascular contribution to ISSNHL is provided by a review on endothelial dysfunction by Quaranta et al. (2016), which proposes endothelial impairment as a central pathophysiological mechanism linking cardiovascular risk factors and cochlear injury. Endothelial dysfunction results in reduced nitric oxide bioavailability and impaired vasodilation and increased oxidative stress which creates a state that promotes blood clotting and leads to potential damage of cochlear microcirculation. Patients with ISSNHL show signs of systemic vascular involvement because they display both flow-mediated dilation problems and high levels of endothelial activation markers.

The findings demonstrate that ISSNHL develops in some patients as a result of systemic microvascular dysfunction. Histopathologic investigations have provided mixed support for the vascular theory. A temporal bone examination showed that idiopathic sudden deafness cases resulted in deteriorative changes to both the organ of Corti and the spiral ganglion cells. The histopathologic pattern presented greater similarity to viral labyrinthitis than to classic arterial infarction, since the major vascular occlusion pattern showed only intermittent instances of widespread hemorrhagic necrosis (Merchant et al., 2008).

Current research asserts that SSNHL development occurs through microcirculatory damage and endothelial dysfunction, and inflammatory cytokine production and prothrombotic states. The mechanisms indicate that vascular problems develop through hidden processes involving microvascular damage and inflammatory responses rather than through visible large-vessel blockage, which supports the existence of multiple causes for cochlear ischemia (Yamada et al., 2022). SSNHL patients show strong anatomical evidence for vascular occlusion, yet most patients cannot receive decisive diagnostic proof of their condition. Vascular imaging tests do not show major blockage because they fail to detect specific obstructions and thrombosis. Laboratory tests produce inconsistent results. Thus, while

vascular compromise remains a compelling and widely accepted theoretical mechanism, it has not been definitively established as the sole or predominant cause of idiopathic SSNHL.

Autoimmune theory

The year 1958 saw Lehnhardt present his theory about sudden hearing loss, which develops rapidly through the process of bodily immune defense systems attacking inner ear structures (Almeida et al., 2017). Later, the autoimmune hypothesis about sudden sensorineural hearing loss (SSNHL) as a separate medical condition was established, which causes most patients to experience hearing loss that develops rapidly but later shows improvement through immunosuppressive treatment (McCabe, 1979).

The subsequent medical review by Broughton et al. (2004) demonstrated that patients with autoimmune inner ear disease experience progressive hearing loss through their bilateral condition, which leads that 79% of cases showing hearing loss in both ears. The progression exhibits irregular patterns that result in alternating periods of advancement and stagnation. The majority of people experience their first symptoms at age 50; however, cases have been documented between ages 22 and 80, and there exists no evident gender preference. Tinnitus affects approximately 83 per cent of patients, while vestibular symptoms occur in about 79 per cent of patients, and nearly half of patients show symptoms that resemble Ménière's disease (Broughton et al., 2004) .

The researchers examined whether immune system components participate in the process of developing sudden hearing loss or hearing loss that affects only one ear. The case series documented both unilateral and bilateral sudden and progressive SNHL cases showed that approximately two thirds of cases demonstrated positive nonspecific immune markers. The rates remained constant whether the hearing loss affected one ear or both ears. The study found that 59 percent of patients with sudden onset one-sided SSNHL showed positive results for

immunologic testing (Atturo et al., 2017). Therefore, an immune cause cannot be ruled out just because the hearing loss is sudden or only affects one ear.

Researchers have further explored immunologic markers associated with inner ear disease. Moscicki and colleagues found circulating antibodies against a 68-kDa inner ear antigen, suggesting the immune system targets cochlear proteins (Moscicki et al., 1994). A decade-long clinical review determined that immune-mediated inner ear disease accounts for only a minority of cases of sudden or progressive hearing loss, but these patients often respond well to corticosteroids, which is an important clue (Broughton et al., 2004). Overall, there is evidence that immune mechanisms contribute to some cases of SSNHL, even though they represent only a small proportion.

Systemic autoimmune diseases are associated with sensorineural hearing loss, including sudden hearing loss, based on research evidence from both population studies and their combined data. A study by Jeong et al. (2019) found that 1.09% of patients with autoimmune diseases developed sudden sensorineural hearing loss during the research study, while 0.73% of matched controls developed the condition. The elevated risk appeared in patients who had rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, antiphospholipid syndrome, Sjögren syndrome or Behçet disease. Another systematic review and meta-analysis by Li et al. (2023) showed that 21.3% of systemic lupus erythematosus patients experience sensorineural hearing loss, while 16.1% of rheumatoid arthritis patients experience the same condition, yet vitiligo, which is another autoimmune disease, exhibits higher rates of hearing loss. The meta-analysis evaluated sensorineural hearing loss in general, although the data demonstrated that autoimmune diseases lead to significant cochlear damage, which results in sudden hearing loss for some patients.

Immune-mediated sensorineural hearing loss occurs more frequently in women, especially those between 30 and 50 years old. It is probably less than 1% of all hearing loss cases, but determining the exact prevalence is difficult since there is no single definitive test for diagnosis. (Almeida et al., 2017). When immune mechanisms cause sudden SNHL, these cases are particularly rare and challenging to identify because their symptoms overlap significantly with idiopathic cases. (Almeida et al., 2017). In general, the evidence suggests that immune factors can cause hearing loss in a subset of SSNHL patients, although this remains an uncommon cause.

Other causes

Temporal bone fractures represent one of the most established medical causes, which research has shown to result in sensorineural hearing loss among 14 to 23% of fracture cases and complete sensorineural loss among 14% of cases (Antoniades et al., 2022; Oulghoul et al., 2021). In polytrauma cohorts, approximately 16% of patients with temporal bone fractures developed acute sensorineural hearing loss. (Padmakumar et al., 2019). Labyrinthine concussion functions as a cause for sudden cochlear dysfunction even when the patient shows no visible fracture (Chiaramonte et al., 2013).

Barotrauma exists as an established medical condition that affects both divers and people who experience sudden changes in atmospheric pressure, such as those in aviation (Selvanayagam & Mathialagan, 2021). The pressure changes that occur during flights to high altitudes can cause temporary ear problems, yet these changes do not cause any lasting hearing impairment. In contrast, divers are exposed to much greater and more rapid pressure variations, even at shallow depths, which increases the risk of structural injury to the middle and inner ear and may lead to permanent hearing loss (Selvanayagam & Mathialagan, 2021), while a perilymphatic fistula serves as a recognisable but rare explanation for SSNHL cases (Kuhn et

al., 2011). Barotrauma can be caused by blast trauma. The most common form of hearing impairment that blast trauma victims reported was hearing loss. The eardrum gets perforated, and the ossicular system experiences disruption, and people develop tinnitus from hearing blast-like sounds, together with gunshots and bombs and loud firework explosions (Selvanayagam & Mathialagan, 2021).

In etiologic reviews, toxic causes are included within the minority of non-idiopathic cases, with identifiable etiologies overall accounting for roughly 10% of SSNHL, and toxic exposures forming a small subset of these (Kuhn et al., 2011). Acute threshold shifts have been documented following high-dose or rapid intravenous administration of aminoglycosides and loop diuretics, particularly in patients with renal impairment (Rybak & Ramkumar, 2007). Although ototoxic hearing loss is more often progressive, sudden deterioration during exposure has been reported in clinical series (M. Rybak et al., 2009). Occupational toxic exposures are also relevant. Organic solvents such as toluene, styrene, and xylene have been associated with sensorineural hearing loss independent of noise exposure (Fuente & McPherso, 2012; Selvanayagam & Mathialagan, 2021). A systematic review reported significantly increased odds of hearing loss among workers exposed to solvents, particularly when combined with noise (Fuente & McPherso, 2012). While most occupational toxic hearing loss is chronic, acute high-level exposure has been described as capable of producing rapid cochlear injury.

Vestibular schwannoma stands as a crucial diagnosis to rule out when evaluating patients with sudden unexplained hearing loss that affects only one ear. A study conducted on past medical records assessed MRI results from patients who exhibited SSNHL and discovered that vestibular schwannoma appeared in about 4.5% of the examined patients who received imaging tests (Jeong et al., 2016). Intralabyrinthine schwannoma causes hearing disability for almost all of its affected individuals because 99% of those with this condition experience hearing loss. The presentation of this condition shows different patterns because 61% of

patients experience hearing loss that develops gradually, while 7% have fluctuating hearing loss and 39% experience sudden hearing loss, considering 38% of tumors which do not affect the cochlea or internal auditory canal still lead to hearing loss, which demonstrates that direct cochlear invasion does not need to occur for people to experience auditory dysfunction (Jeong et al., 2016).

All possible neurological vascular causes require thorough examination in patients who experience sudden sensorineural hearing loss (SSNHL) with additional focal symptoms that include facial numbness and weakness, and limb deficits (Chandrasekhar et al., 2019). Research demonstrates that acute infarction of the anterior inferior cerebellar artery (AICA) territory results in sudden hearing loss, commonly accompanied by vertigo and other brainstem symptoms. A clinical report that sudden hearing loss and audiovestibular dysfunction constituted the primary symptoms of AICA infarction (Lee et al., 2002). Migraines have also been linked to an increased risk of SSNHL. A nationwide population-based cohort study demonstrated that individuals with migraine had a significantly higher likelihood of developing SSNHL, with an adjusted hazard ratio of approximately 1.8 compared with non-migraine controls (Hwang et al., 2018). The connection between migraine and hearing loss demonstrates that both conditions share common neurovascular pathways, which lead to an increased risk of sudden hearing impairment (Hwang et al., 2018).

Researchers have studied various metabolic and endocrine disorders to determine their impact on sudden sensorineural hearing loss (SSNHL). A diabetes mellitus study conducted across multiple regions showed that patients with the condition had a higher SSNHL risk, which the researchers calculated at an adjusted hazard ratio of 1.59. Regarding prognosis, a propensity score-matched analysis showed that diabetes did not affect hearing recovery after steroid treatment because all confounding variables had been eliminated from the analysis. The two factors that affected strongly were initial hearing level and time to treatment (Seo et al.,

2020). Another clinical research study published showed that 28% of SSNHL patients tested positive for diabetes, whereas diabetic patients demonstrated lower complete recovery rates from corticosteroid treatment than non-diabetic patients (Sajid et al., 2021).

The evidence from population studies shows that thyroid disorders lead to an increased risk of sudden sensorineural hearing loss (SSNHL). A study by Tsai et al. (2020) found that both hypothyroidism and hyperthyroidism lead to higher SSNHL risk in a nationwide Taiwanese population because they both provide increased risk for this condition (adjusted odds ratio 1.54 for hypothyroidism and 1.41 for hyperthyroidism). Furthermore, analysis of Korean National Health Insurance data demonstrated that, after adjusting for potential confounders, patients with SSNHL had significantly higher prevalence rates of goitre and hypothyroidism compared to matched control subjects (Kim et al., 2020).

Regarding autoimmunity, a study discovered that 24.8% of SSNHL patients tested positive for thyroid autoantibodies, which resulted in greater high-frequency hearing loss among antibody-positive individuals, while their recovery process remained unchanged (Sun et al., 2022). In a prospective study, patients with moderately severe-to-profound SSNHL demonstrated significantly lower TT3, TT4, FT3, and TSH levels compared to healthy controls, and lower FT3 and TSH levels were associated with poorer hearing recovery (Zheng et al., 2021). Similarly, another study reported that 24.4% of 676 SSNHL patients had abnormal thyroid function, and reduced T3 and FT3 levels were significantly associated with poorer hearing outcomes, particularly among those who initiated treatment more than 14 days after onset (Zhu et al., 2020).

Patients with SSNHL have been reported to exhibit significantly lower serum 25-hydroxyvitamin D levels compared with controls, and reduced vitamin D levels were associated with greater initial hearing loss severity; however, these studies did not identify

vitamin D as an independent predictor of recovery (Zhao et al., 2025; Zheng et al., 2023). In contrast, Ghazavi et al. (2020) published in the American Journal of Otolaryngology demonstrated a significant association between serum vitamin D level and treatment response ($P = 0.004$), with 100% recovery in patients with sufficient vitamin D levels, compared to 87.5% recovery and 12.5% non-response among vitamin D–deficient patients, and comparatively poorer outcomes in those with insufficient levels. Vitamin D deficiency may influence auditory function through its role in calcium homeostasis, affecting cochlear fluid balance, neural transmission, bone remodelling of the otic capsule, and susceptibility to ischemic or degenerative changes.

A retrospective study was conducted by Kurioka et al. (2020) to determine whether patients with idiopathic sudden sensorineural hearing loss would benefit from extended vitamin B12 treatment combined with adenosine triphosphate. After six months, about one-third of patients showed meaningful recovery, with roughly one in five achieving complete recovery, while more than a quarter did not improve. The extended duration of vitamin B12 treatment did not produce any noticeable effects on total recovery. The research findings demonstrated that patients who underwent shorter treatment periods experienced greater hearing loss between 16 and 24 weeks.

Recent research shows a potential connection between hormonal levels and sudden-onset sensorineural hearing loss. A retrospective cohort study by Wang et al. (2024) resulted in the discovery that perimenopausal women with SSNHL showed reduced serum estradiol and progesterone levels when compared to healthy controls. The researchers demonstrated that reduced hormone levels resulted in increased hearing loss severity, which established a connection between endogenous sex hormone reduction and SSNHL. Another study demonstrated that postmenopausal women who used hormone therapy did not experience an

increased risk of developing SSNHL according to their nationwide cohort research (P. J. Chen et al., 2019).

Research has also explored whether stress-related hormones relate to idiopathic sudden sensorineural hearing loss. A study by Chae et al. (2021) reported that patients with increased dehydroepiandrosterone sulfate levels had worse hearing, while those with higher adrenocorticotropic hormone levels experienced declining hearing abilities, and individuals with elevated depressive stress scores achieved poorer hearing results at their final assessment. The authors proposed that stress hormones affect the disease state because depressive stress serves as a treatment response predictor, but their research only allows them to establish relationships between variables.

2.5 Clinical characteristics of SSNHL

Sudden sensorineural hearing loss is clinically characterised by a rapid onset of sensorineural hearing loss of at least 30 dB across three consecutive frequencies occurring within 72 hours, and patients commonly present with sudden unilateral hearing loss that may be accompanied by symptoms such as tinnitus, vertigo, or aural fullness (Chandrasekhar et al., 2019). The clinical presentation can vary between individuals, and these associated symptoms are often considered during the initial clinical evaluation. The prognosis of sudden sensorineural hearing loss is variable, with recovery ranging from complete improvement to persistent hearing impairment, and several clinical factors, such as the severity of hearing loss at presentation and the presence of vertigo, have been reported to influence hearing outcomes (Lin et al., 2022).

Tinnitus

Tinnitus is one of the most common accompanying symptoms of sudden sensorineural hearing loss. Studies have reported a high prevalence of tinnitus in patients with SSNHL; for example, a clinical study found that tinnitus occurred in approximately 93.3% of affected patients (Nogueira-Neto et al., 2016). Similarly, a recent clinical study reported that tinnitus was present in 79.7% of patients with sudden deafness, indicating its high prevalence in this condition (Zou et al., 2023). In addition, a study described tinnitus as a frequent clinical feature in patients presenting with sudden hearing loss undergoing imaging evaluation (Brennan et al., 2025). Studies focusing on tinnitus characteristics in SSNHL, further describe tinnitus as commonly appearing at the onset of sudden hearing loss and often accompanying the acute episode (Cvorovic et al., 2021; Mokhatrish et al., 2023).

Vertigo:

Approximately 30–60% of SSNHL patients also complain about symptom of vertigo, which may appear at the onset of hearing loss or be delayed for hours or even days (Wang et al., 2020). Pogson et al. (2016) examined audio-vestibular characteristics of patients with sudden hearing loss and vertigo and reported that patients presenting with vertigo frequently demonstrated abnormalities on vestibular function testing, suggesting concurrent vestibular dysfunction in some cases. Furthermore, a systematic review and meta-analysis found that the presence of vertigo in patients with sudden sensorineural hearing loss was associated with poorer hearing recovery outcomes compared with patients who did not experience vertigo (Yu & Li, 2018).

Aural fullness

Aural fullness is one of the commonly reported associated symptoms in patients with Sudden Sensorineural Hearing Loss, although it is less frequently discussed compared to tinnitus and vertigo. In an editorial review of patient presentations, it is stated that patients with SSNHL are often accompanied by other symptoms such as tinnitus and dizziness, and descriptions are common to include an ear fullness sensation at the onset of hearing loss (Kuhn et al., 2011). Similarly, the clinical features of ear fullness have been investigated by Park et al. (2012) and showed that ear fullness is a symptom that is predominantly associated with inner-ear pathology and specifically with those that include idiopathic sudden sensorineural hearing loss and Ménière's disease. It was noted that the patients presenting with ear fullness showed an increase in the number of accompanying signs of hearing loss and tinnitus, thus auras of middle-ear pathology are often overshadowed by symptoms of cochlear/inner-ear disorder (Park et al., 2012). In more recent clinical investigations of SSNHL, aural fullness is still documented among accompanying signs that patients are often questioned about in the clinic. (Y. Wang et al., 2020)

Fluctuating hearing loss

The finding of a fluctuant course of hearing loss during the course of an SSNHL has been noted by numerous clinicians within the literature on the illness and prognosis. Contemporary reviews have stated that full hearing restoration may not always occur with SSNHL, and healing of the loss is not always steady. Prognostic reviews on SSNHL indicate that following SSNHL, not all patients regain complete and stable hearing levels, and hearing can fluctuate and show improvements based upon the mechanism and associated characteristics. Prognostic research has also stated that the hearing loss must be monitored with serial audiometry and varied in all individuals. Many studies have described poor and some have indicated moderate and full recovery. Narrative reviews of SSNHL have often mentioned

fluctuating hearing, stated that recovery is not always complete and may fluctuate over time with degree of loss dependant on various factors (Tripathi & Deshmukh, 2022).

Together, these studies document that fluctuating or variable hearing recovery is recognized in the literature as part of the clinical course of SSNHL, particularly during the early phase and follow-up period.

2.6 Audiological evaluation in SSNHL

Pure tone audiometry: audiogram shape as a prognostic marker

SSNHL audiograms are typically classified into several configurations: ascending (low-frequency loss), descending (high-frequency loss), flat, U-shaped/concave, and profound/total. The consistent finding across multiple retrospective studies is that audiogram shape at presentation predicts recovery. Ascending (low-frequency) and U-shaped patterns show the best recovery rates, while descending (high-frequency), flat, and especially profound patterns carry a worse prognosis (Waissbluth et al., 2022; Watanabe & Suzuki, 2018).

Low frequencies consistently recover better than high frequencies. Study by Jun et al. (2012) in 99 patients with SSNHL, recovery frequency was analysed and found that low-frequency hearing loss responded better to treatment across all audiogram patterns except the descending type. Similarly, Psillas et al. (2019) compared low-tone vs. high-tone SSHL over ~3 years of follow-up. Complete recovery was 77.7% in the low-tone group versus only 15% in the high-tone group, a dramatic difference. However, low-tone SSHL had higher recurrence rates (22% vs. 10%), while high-tone SSHL was more often accompanied by persistent tinnitus (65% vs. 11%).

A large retrospective study on 156 patients found that upsloping and U-shaped patterns had significantly better hearing outcomes, while flat and downsloping curves were associated

with poorer recovery and also with adverse metabolic and inflammatory markers (lower HDL, elevated neutrophil-to-lymphocyte ratio) (Cadoni et al., 2025).

An interesting recent proposal by Tseng et al. (2025) offered a simplified three-type classification based solely on the 4 kHz threshold position, whether 4 kHz is the best, intermediate, or worst threshold. Overall recovery rates in each category were found to be 49%, 36% and 20%, respectively and were more favourable than the previous 7-type classification. Apart from shape, level also has a profound effect, with a high degree of initial loss (>80dB) or presenting with a profound hearing loss, shown to consistently have poorer outcomes in SSNHL. (Penido et al., 2009).

In a large study examining 848 patients, it was discovered that trough-shaped (mid frequency) loss is significantly more common in acoustic neuroma patients compared to idiopathic SSNHL and therefore mid-frequency dip presentation of SSNHL should warrant an investigation for retrocochlear pathology (Hosokawa et al., 2018).

Speech audiometry: word recognition as an independent prognostic factor

Speech discrimination testing yields important clinically relevant information that pure tone thresholds do not provide alone. Word recognition score recovery has been looked into as an independent predictor of the outcome following SSNHL alongside recovery in pure tone levels and shows significant variation, with those suffering from accompanied vertigo achieving significantly worse initial speech PTAs and lower levels of speech PTA recovery (Waissbluth et al., 2022). Results from a study investigating 180 patients showed that the initial word recognition score (WRS) alone can predict the outcome in SSNHL, particularly with a poor presenting score (< 52%). In multivariate analysis, WRS remained a significant predictor even after accounting for audiogram configuration, age, and treatment timing (Kwak et al., 2022).

Perhaps most striking is the finding that speech intelligibility deficits in sudden sensorineural hearing loss (SSNHL) are not always fully explained by pure-tone thresholds. **I**n a study of 166 patients, reported that many patients with SSNHL demonstrated poorer speech recognition than would be predicted from their audiometric thresholds (Okada et al., 2021). Similarly, a study analysing approximately 96,000 ears, found that the discrepancy between measured and predicted word-recognition scores was among the largest in patients with SSNHL (along with Ménière's disease and vestibular schwannoma) (Grant et al., 2022). These findings suggest that functional hearing outcomes in SSNHL may not be fully reflected by pure-tone recovery alone.

Jan et al. (2016) identified a novel cohort of patients who failed treatment by pure tone criteria but still showed improved word recognition over time, suggesting that PTA and WRS can dissociate and that tracking both is important for counselling. Conversely, it was reported that steroid-treated patients showed significant improvement in word recognition scores, particularly in severe-to-profound cases (C. Y. Chen et al., 2003).

Study by Xu et al. (2024) distinguished "neural-type" SSNHL (presenting WRS <60%, disproportionately worse than expected from PTA) from "sensory-type" loss. While overall hearing recovery rates were similar between groups, the neural-type group showed substantially greater WRS improvement with treatment, with the neural group showing a mean change of 46.9% ± 29.8 compared to 3.2% ± 25.8 in the non-neural group.

Hence, across these studies, pure tone and speech audiometry capture partly independent dimensions of cochlear and neural damage in SSNHL. Audiogram configuration is a reliable prognostic indicator, with low-frequency and U-shaped losses doing best. Speech discrimination adds prognostic value beyond PTA, particularly in identifying neural involvement, and residual speech deficits often persist even after threshold recovery.

ABR findings: wave I latency as the key prognostic marker

The most consistent ABR finding in SSNHL is prolonged wave latencies in the affected ear. H. C. Lin et al. (2017) found that waves I, III, and V latencies were all significantly prolonged in affected versus unaffected ears, with reduced wave I amplitude. Critically, wave I latency showed a significant association with hearing outcome; it was progressively more prolonged from the complete recovery group to the slight recovery group. Lim et al. (2019) confirmed this finding that hearing improvement correlated specifically with the interaural latency delay of wave I. Patients with wave I interaural delay under 0.2 ms had significantly better outcomes than those over 0.2 ms or with absent wave I. Additionally, in another study, it was reported that wave I latency was an independent predictor of good response on logistic regression, with a large odds ratio (Bang et al., 2019).

The prognostic significance of ABR extends beyond wave latencies to wave presence. A study found that patients with normal ABR had a 65% recovery/improvement rate versus 43% for those with abnormal ABR. Among steroid-treated patients, this gap widened to 83% vs. 56% (Busaba & Rauch, 1995). Furthermore, a study in 114 patients reported that the detection of wave V early in recovery and within the first month was a significant, favourable prognostic factor (Xenellis et al., 2006).

Multivariate analysis by C. Te Wang et al. (2009) found that ABR waveform presence was a significant independent outcome predictor. In the severe SSHL subgroup, the presence of ABR waveforms carried an adjusted odds ratio of 14.7 for better hearing outcomes. They also found VEMP presence was independently prognostic and proposed a combined ABR/VEMP predictive table.

ABR also plays a role in distinguishing cochlear from retrocochlear pathology. A study found retrocochlear involvement in 19 of 61 sudden deafness cases using electrocochleography and ABR, including 4 acoustic neuromas confirmed by CT (Portmann et al., 1985).

OAE findings: outer hair cell function and prognostic value

A key early finding showed that despite hearing thresholds exceeding 35 dB (where OAEs normally disappear in chronic SNHL), about half of SSNHL ears still had detectable evoked OAEs at onset. The majority of these OAE-detectable ears showed good recovery. This suggests that in acute SSNHL, the presence of OAEs despite elevated thresholds indicates preserved OHC function and a mechanism beyond pure OHC damage possibly strial dysfunction, neural involvement, or endolymphatic disturbance (Sakashita et al., 1991). Another prospective study provided the most specific prognostic data; patients with detectable TEOAEs at day 7 had 62% average hearing improvement versus only 11% for those without; for DPOAEs, the figures were 71% vs. 10%. DPOAE detectability at day 7 had 83% sensitivity and 100% specificity for predicting significant hearing improvement (Shupak et al., 2014).

The prognostic value of otoacoustic emissions was evaluated by Gaafar et al. (2022) in 30 patients with unilateral idiopathic sudden sensorineural hearing loss who were followed at baseline, one week, one month, and three months after treatment. The study reported a statistically significant improvement in pure-tone thresholds across 250–8000 Hz at one and three months, particularly at 500–4000 Hz. Patients with detectable TEOAEs and DPOAEs at baseline showed significantly greater hearing improvement and better speech scores. In contrast, those with absent otoacoustic emissions did not demonstrate statistically significant improvement during follow-up. In addition, the number of patients with measurable otoacoustic emissions increased progressively at one and three months, indicating recovery of cochlear function during the follow-up period.

Another study added nuance by stratifying by severity. In mild-to-moderate hearing loss (≤55 dB), initial DPOAE levels were not different from contralateral normal ears, suggesting OHC function was relatively spared and that the hearing loss mechanism in these cases is not primarily OHC damage. In moderately severe-to-profound cases (>55 dB), DPOAEs were clearly reduced, and DPOAE recovery correlated with hearing improvement (H. Park et al., 2010).

A systematic review concluded that the evidence overwhelmingly supports including OAEs in the SSNHL monitoring protocol, though they noted that no standard protocol yet exists (Babich & Dunckley, 2020).

Other major prognostic indicators:

The timing of treatment initiation is one of the most consistently reported prognostic factors in sudden sensorineural hearing loss (SSNHL). A landmark 8-year prospective study by Byl (1984), which included 225 patients, found that the time of presentation with the first audiogram and the start of treatment were the primary predictors of a favourable outcome. In this study, early presentations showed significantly higher rates of recovery.

A modern approach in prognostication was seen in research completed by Mandavia et al. (2024), who created a predictive model in 498 patients throughout 76 NHS trusts within the UK. It was discovered that initiating steroid treatment within 7 days from onset had the greatest statistical power as an independent prognosticator of a complete recovery, with an OR of 5.23 for steroid therapy versus non-treatment. Similarly, Edizer et al. (2015) identified delayed treatment with a duration >10 days from the onset of hearing loss as a negative predictor for SSNHL outcome; some hearing restoration was observed in 59% of cases.

Vertigo at presentation in SSNHL is widely accepted to be a negative prognostic indicator. A meta-analysis study comprising 10 studies that investigated 4,814 patients found the hearing recovery rate for patients who presented with vertigo to be 42.13% compared to 60.29% for those presenting without. (Yu & Li, 2018). This resulted in a pooled odds ratio of 2.22 for poorer recovery in the vertigo group. Interestingly, this negative association was not observed in patients who received intratympanic corticosteroids. Further, a study of 125 patients demonstrated that the type of vestibular involvement also plays an important role in determining prognosis. Recovery was poorest in patients with concurrent benign paroxysmal positional vertigo (BPPV), particularly in the high-frequency range, followed by those with unilateral vestibular hypofunction (H. M. Park et al., 2001). This observation was further supported by Ben-David et al. (2002), who reported a significant association between vertigo and persistent hearing loss, particularly at 8 kHz.

Advanced age has also been identified as an unfavorable prognostic factor in several studies, although its independent contribution appears to vary. Mandavia et al. (2024)confirmed that younger patient age was an independent predictor of complete recovery in their multivariate model. Similarly, Edizer et al. (2015)reported significantly lower complete recovery rates in patients older than 60 years. However, Atay et al. (2016) found that treatment timing and audiogram configuration were more significant predictors of recovery than age alone.

Systemic comorbidities, particularly diabetes mellitus and hypertension, have also been implicated in the pathogenesis and prognosis of SSNHL. A large meta-analysis, which included 27 studies involving 77,566 patients, reported significantly higher odds of diabetes and hypertension among patients with SSNHL compared with matched controls (Saba et al., 2023). Similarly, the negative influence of diabetes and hypercholesterolemia on prognosis was also reported (C.-F. Lin et al., 2016). However, Seo et al. (2020) reported that diabetes itself did not

independently influence hearing recovery after matching for age, sex, and initial hearing level, as diabetic patients tended to present with more severe hearing loss at baseline. Similar findings were reported that identified diabetes and vertigo as negative prognostic factors but found no significant effect of age or gender (Attia Askar et al., 2023).

Fluctuating hearing in the context of SSNHL appears to carry important prognostic implications and may reflect a distinct underlying pathophysiological mechanism. Domínguez et al. (2021), using gadolinium-enhanced MRI in 170 patients, found that the incidence of endolymphatic hydrops was minimal in idiopathic SSNHL but significantly higher in patients with fluctuating sensorineural hearing loss. This suggests that fluctuating hearing following sudden deafness may indicate a hydropic mechanism and a potential risk of progression to Ménière's disease. In addition, Pecorari et al. (2019), in a long-term follow-up study of 50 patients over a mean period of 5.26 years, reported that although most patients demonstrated stable hearing, 26% experienced worsening over time, with alcohol consumption and vasculopathies identified as possible contributing factors.

2.7 Management of SSNHL

Systemic corticosteroids

Systemic corticosteroids remain the most widely used first-line treatment for SSNHL; however, the evidence supporting their efficacy appears to be weaker than often assumed. Conlin & Parnes (2007) conducted a meta-analysis of all randomised controlled trials published between 1966 and 2006 and found no clear evidence of the benefit of systemic steroids over placebo. The analysis pooled data from two randomised controlled trials comparing steroids with placebo and reported a non-significant improvement (OR 2.47; 95% CI 0.89–6.84; $p = 0.08$). In addition, no significant difference in outcomes was observed between systemic steroids and other active treatments. This analysis was later updated by Crane et al. (2015),

who similarly concluded that although systemic steroids are widely adopted in clinical practice, the supporting evidence remains limited because of the small number and heterogeneity of available randomised trials. More recently, a multicenter randomised controlled trial compared high-dose intravenous prednisolone and high-dose oral dexamethasone with standard-dose oral prednisone and found that high-dose systemic glucocorticoid therapy was not superior to the lower-dose regimen in terms of improvement in hearing threshold at 30 days and was associated with a higher risk of adverse effects (Plontke et al., 2024).

Intratympanic steroids

Intratympanic (IT) steroid injection has gained increasing acceptance as both a primary alternative treatment and a salvage option for patients who fail systemic therapy. Spear & Schwartz (2011) reviewed 176 articles and reported that intratympanic steroid therapy used as a primary treatment appeared comparable to high-dose oral prednisone. When used as salvage therapy, it was associated with additional hearing improvement, with a limited meta-analysis showing a mean improvement difference of 13.3 dB (95% CI 7.7–18.9; $p < 0.0001$), although the clinical significance of this difference remained uncertain.

Similarly, Garavello et al. (2012) conducted a meta-analysis of 11 randomized controlled trials including 472 patients treated with intratympanic steroids and 453 control patients. Their analysis showed that intratympanic steroid therapy provided significant benefit when used as a salvage treatment, with a pooled odds ratio for recovery of 2.9 (95% CI 1.9–4.5), whereas no significant benefit was observed when it was used as primary treatment (OR 0.9; 95% CI 0.7–1.6).

Combination therapy

The combination of systemic and intratympanic corticosteroids has been investigated as a strategy to improve outcomes beyond either modality alone. A systematic review and meta-analysis of seven randomised controlled trials, including 710 patients, found no evidence that combined treatment resulted in better hearing outcomes compared with either systemic or intratympanic therapy alone (Mirian & Ovesen, 2020). In contrast, another meta-analysis of 20 randomised controlled trials reported that combination therapy significantly improved recovery rates compared with systemic therapy alone and that moderate- and high-dose combination therapy appeared to accelerate hearing improvement (J. Li & Ding, 2020). Supporting these findings, Gundogan et al. (2013) reported in a prospective study of 73 patients that combination therapy using intratympanic methylprednisolone together with oral steroids resulted in significantly greater hearing improvement and better speech discrimination scores than oral steroid therapy alone, particularly in patients with severe hearing loss.

Other adjunct therapies

Hyperbaric oxygen therapy (HBOT) is one of the most extensively studied adjunctive treatments for SSNHL. Olex-Zarychta (2020) provided a comprehensive review of both the molecular mechanisms and clinical effectiveness of HBOT and highlighted its role in improving oxygen delivery to the cochlea, particularly when used in combination with corticosteroid therapy. A controlled study conducted by Fattori et al. (2001) involving 50 patients who were randomised to receive either HBOT or IV vasodilators suggested that a significantly greater hearing recovery was achieved in those who received HBOT, regardless of age and sex, with more patients showing a good or significant improvement.

More recently, Psillas (2023) reviewed the available evidence on HBOT in SSNHL and discussed its role as both a primary and salvage treatment while summarising current data on

its therapeutic effectiveness. About antiviral therapy, Conlin & Parnes (2007) found no significant difference between patients treated with antiviral therapy in combination with steroids and those receiving placebo plus steroids.

Spontaneous recovery

The relatively high rate of spontaneous recovery in SSNHL complicates the interpretation of treatment efficacy and remains an important consideration when evaluating therapeutic outcomes. In a landmark prospective study, Mattox & Simmons (1977) reported that approximately 65% of patients recovered to functional hearing levels spontaneously, independent of medical treatment, with most recovery occurring within the first 14 days. Low-frequency hearing loss was associated with better recovery than high-frequency loss, and none of the treatments commonly used at that time appeared to influence outcome.

Chaushu et al. (2023) conducted a recent systematic review and meta-analysis that pooled data from placebo and untreated control arms across multiple trials to establish a more accurate baseline for spontaneous recovery in ISSNHL. These findings showed that a significant proportion of patients achieved spontaneous recovery and therefore highlight the necessity of comparisons being drawn against the natural course of the condition rather than assumed efficacy of treatment. Similarly, Ying et al. (2024) provided updated spontaneous recovery estimates highlighting a sizable percentage of spontaneous recoverees and showing that age and level of initial hearing loss have a statistically significant association with spontaneous hearing restoration.

Bayomuy et al. (2018) found spontaneous full recovery rates to vary between 32% and 65%, depending on the nature of the studies and the criteria used to define recovery. This data is important for physicians to consider when deciding whether to commence treatment for SSNHL. The findings of this review clearly demonstrated the complex and multifactorial

nature of SSNHL, specifically related to treatment and outcomes. Early intervention and treatment time are identified as one of the most important predictive factors of hearing restoration, as patients treated within the first week showed greater chances of recovery than those who presented later. Besides the time aspect, the age of the patient, initial hearing loss severity and the presence of systemic diseases such as hypertension and diabetes mellitus are all documented as key indicators of SSNHL outcome, though individual results differ between studies.

Several papers also researched the benefits of treatment options. Systemic steroid therapy is the treatment option of choice, but there remains controversy in the literature about its overall effectiveness. Intratympanic steroid treatment is being recognised for its uses, especially in cases where SSNHL requires salvage treatment, combination therapy, and treatments like hyperbaric oxygen therapy have shown varied success. Furthermore, the high rate of spontaneous recovery reported in several studies interprets treatment outcomes as more challenging.

Overall, the existing literature highlights variability in both prognosis and treatment response, indicating the need for further well-designed studies and supporting the relevance of the present study.

Chapter III

METHODS

This research intends to address the identified gap by developing a condition-specific, clinically oriented screening and assessment checklist for sudden sensorineural hearing loss (SSNHL). A cross-sectional tool development and validation design was employed to achieve this aim. The study was carried out using the following methods:

3.1 Informed consent and ethical consideration

The present study employed a questionnaire-based, cross-sectional, observational design with retrospective record review. Participants included individuals with a confirmed diagnosis of sudden sensorineural hearing loss (SSNHL), including both unilateral and bilateral cases, who had initially received their diagnosis either at the ENT department of the All India Institute of Speech and Hearing (AIISH) or at other healthcare facilities and later presented to the audiological clinical services at AIISH for further evaluation and management.

As part of routine clinical practice at the institute, all registered patients provided written informed consent during initial registration and follow-up visits, which includes permission for the use of their clinical data for research purposes. Following approval from the Institutional Review Board (IRB), participants were contacted through telephone conversations and direct interviews, and data were collected using a structured questionnaire.

To access the required clinical data, prior permission was obtained from the Head of the Department of Clinical Services. A formal requisition letter, submitted through the dissertation guide, was provided to the aforementioned authority, outlining the objectives of the study and specifying the data requirements while assuring confidentiality. Following

approval, the investigator accessed the relevant records and systematically extracted the required information. Details pertaining to demographic characteristics, occupation, associated complaints, diagnosis, treatment history, and audiological findings were obtained from the Medical Records Section of the Department of Clinical Services, AIISH, Mysore.

All procedures carried out in the study adhered to established bio-behavioural research standards (AIISH, 2023) and were conducted in accordance with the ethical guidelines of the institutional review board of AIISH. Participants who met the inclusion criteria were adequately informed about the study before participation, and confidentiality of all collected information was strictly maintained throughout the study.

3.2 Participants

A total of 24 participants were recruited using non-random purposive sampling, including 18 males and 6 females. The participants ranged in age from 20 to 70 years, with most participants falling within the 41–50 year age range.

The study population comprised individuals with a confirmed diagnosis of SSNHL, either at the institute's ENT Department or at external healthcare facilities, who subsequently presented to the Audiological Clinical Services for evaluation, management, and follow-up care. The sample included both newly diagnosed cases and individuals previously diagnosed elsewhere who sought continued audiological management and rehabilitative services at the institute.

3.2.1 Inclusion Criteria

Participants who fulfilled the following criteria were selected for the study

- Adults aged 18 years and above.

- Individuals with a clinical diagnosis of sudden sensorineural hearing loss (SSNHL) (Chandrasekhar et al., 2019), based on medical and audiological evaluation.
- Ability to provide informed consent and participate in the study procedures.

3.2.2 Exclusion Criteria

- Individuals with conductive hearing loss.
- Patients with chronic otologic conditions unrelated to SSNHL that could independently affect hearing thresholds (e.g., chronic otitis media, cholesteatoma).
- Individuals with known psychiatric illness or cognitive impairment that could affect reliable communication or participation during the interview process.
- Individuals unwilling or unable to provide informed consent.

3.3 Procedure

The present study adopts a cross-sectional design for the development and validation of a checklist and is carried out in a series of structured phases. Phase I involves the preparation and development of the checklist based on relevant clinical parameters. Phase II focuses on validating the developed tool. In Phase III, the validated checklist is administered to the target population to collect data. Finally, Phase IV involved both quantitative and qualitative analysis of the collected responses to derive meaningful insights.

3.3.1 Phase I- Preparation and Development of the Checklist

In the initial phase of the study, the checklist was developed through a comprehensive review of literature on the prevalence, etiology, pathogenesis, and prognosis of sudden sensorineural hearing loss (SSNHL). Relevant information was gathered from standard

databases such as Google Scholar, PubMed, and Scopus, along with established textbooks in otolaryngology and hearing sciences. Clinical inputs from experienced otologists were also considered to ensure that the items reflected real-world clinical presentation and relevance. Based on these sources, a structured, closed-ended checklist was developed in English using simple and patient-friendly language to facilitate ease of understanding and administration.

The checklist was organised into two main sections to allow a systematic clinical assessment. Section A focused on core prognostic indicators, capturing factors known to influence recovery outcomes in SSNHL. These included the time interval between onset and medical consultation, age, pattern of onset (sudden or gradual), progression of hearing loss, laterality, presence of vestibular symptoms such as vertigo, and severity of hearing loss based on audiological findings or patient-reported perception at onset.

Section B focused on clinical and etiological factors that could influence the presentation and prognosis of SSNHL. This section included associated auditory symptoms such as tinnitus and aural fullness; family history; recent infections; autoimmune features; toxic exposures; vascular and metabolic risk factors; neurological symptoms; and any atypical clinical history. It also covered possible identifiable triggers, including trauma-related events such as loud noise exposure, ear surgery, or head injury, as well as pressure-related events like sudden changes in temperature or barometric pressure, which may contribute to hearing loss through different underlying mechanisms.

Hence, the pre-validated questionnaire consisted of a total of 39 items distributed across both sections.

3.3.1.1 Formulating a rating scale for the questionnaire

All items in the checklist were structured as closed-ended responses to ensure consistency in data collection. The scoring system was designed based on the relative prognostic significance of selected clinical variables in SSNHL.

Section A included established prognostic indicators such as age category, time to treatment initiation, progression of hearing loss, laterality, presence of vertigo and baseline degree of hearing loss. Responses within each variable were arranged from relatively favourable to poorer prognostic categories and assigned ordinal scores accordingly, with higher scores indicating a relatively poorer prognostic profile. For example, age categories of <40 years, 40–60 years, and >60 years were assigned scores of 1, 2, and 3, respectively. Binary variables, such as progression of hearing loss, were scored as 0 or 1 based on the presence or absence of the clinical feature. The cumulative Section A score was used for exploratory prognostic assessment, and its association with post-treatment audiometric outcome was evaluated. Section B originally consisted of a single descriptive section that included associated clinical features, systemic risk factors, and possible etiological factors related to SSNHL. These variables were recorded descriptively and were not incorporated into the cumulative prognostic score due to their exploratory and heterogeneous nature.

3.3.1.2 Other components of the questionnaire

In addition to the main sections, the checklist also included other components to facilitate standardized administration and interpretation. These comprised basic demographic and identification details (such as name, age, gender, case number, & contact information), which were recorded for documentation purposes and not included in scoring. Clear instructions were included at the beginning of the questionnaire to assist the clinician in administering the checklist, selecting appropriate responses, and interpreting the scoring format in a standardized manner.An example item was included to illustrate the scoring procedure.

These components ensured uniform administration of the checklist and consistency in data recording across participants.

3.3.2 Phase II - Content Validation

The developed checklist was reviewed for content validity by a panel of six experts comprising audiologists and otolaryngologists with a minimum of 10 years of clinical and academic experience. The experts were asked to evaluate each item in terms of relevance, clarity, accuracy, grammatical correctness, ambiguity, repetition, and overall suitability for inclusion in the checklist. Responses for each parameter were obtained using a qualitative format with "Yes" or "No" options. In addition, space was provided for experts to give comments, suggestions, and recommendations for modification wherever necessary.

The feedback received from the experts was carefully considered to refine the checklist. Based on their inputs, modifications were made to improve the wording, structure, and overall clarity of the items. This process helped enhance the clinical relevance and ease of understanding of the checklist, ensuring that it could be used effectively in a clinical setting. The content validation format is provided in Appendix I.

3.3.2.1 Preparation of the Final Questionnaire

The questionnaire was revised based on the feedback and suggestions of a panel of six experts to improve its clarity, comprehensiveness, and contextual relevance. The final questionnaire consisted of two major sections. Section A included core prognostic indicator items assessing factors such as time to treatment initiation, age category, progression of hearing loss, laterality, presence of vertigo, baseline severity of hearing loss, and audiogram configuration, and was used for exploratory prognostic assessment through cumulative scoring.

Section B was intended for descriptive clinical profiling and was divided into two subsections. Subsection B1 included 29 items related to associated clinical and systemic risk-modifying factors, while Subsection B2 included 4 items documenting possible etiological or triggering factors such as noise exposure, trauma, and pressure-related events. The finalized English version of the questionnaire is provided in Appendix II.

3.3.3 Phase III- Field Testing & Validation

The refined checklist was administered to individuals with confirmed SSNHL by the investigator. Before administering the questionnaire, informed consent was obtained from all participants. Data collection was carried out through structured telephonic or direct interviews, during which all items in the checklist were systematically asked, and responses were recorded. To ensure accuracy and consistency, the information obtained from participants was cross-verified with available clinical records and case history details wherever applicable. The informed consent forms in English and Kannada are provided in Appendix III and Appendix IV, respectively.

During the interview, participants were asked to report relevant symptoms and clinical history as per the checklist items. These included aspects such as the onset and progression of hearing loss, associated symptoms such as tinnitus and vertigo, and potential risk or etiological factors. For example, participants were asked questions such as whether they experienced vertigo or imbalance at the time of hearing loss, and responses were recorded in the predefined format (e.g., Yes/No) with corresponding scores.

All responses were documented according to the checklist's scoring framework. Scores from Section A were computed to obtain the prognostic classification for each participant, while responses from Section B were recorded descriptively to document associated clinical features, systemic risk factors, and possible etiological factors related to SSNHL.

The collected data were compared with post-treatment audiological outcomes following steroid therapy. Outcome assessment was primarily based on post-treatment pure-tone audiometry (PTA) findings obtained during follow-up evaluation. Hearing recovery was assessed by comparing pre-treatment and post-treatment PTA values, and a hearing gain of ≥15 dB was considered indicative of audiometric improvement, in accordance with commonly used recovery criteria in studies on sudden sensorineural hearing loss (SSNHL).

3.3.4 Phase IV- Quantitative and Qualitative analysis of the observed data

The data obtained from the administered checklist were compiled and organized for analysis. Descriptive statistics were used to summarize the demographic characteristics of the participants and to present the distribution of responses across the checklist items. Section A consisted of ordinal prognostic scores assigned to established prognostic indicators identified from the literature review. Higher total scores reflected a relatively poorer prognostic profile, whereas lower scores indicated a comparatively favorable prognosis. The cumulative Section A score represented the overall prognostic tendency of each participant. The total possible Section A score ranged from 8 to 15.

The association between the total Section A prognostic score and post-treatment PTA was analyzed using Spearman rank correlation analysis. This non-parametric test was selected due to the small sample size and the non-normal distribution of the data. The analysis was performed to determine whether increasing prognostic scores were associated with poorer post-treatment hearing outcomes.

Descriptive statistics were used to summarize demographic characteristics, clinical findings, and the distribution of responses across the checklist items. In addition, tables and graphical representations were used to descriptively observe hearing outcome trends across different prognostic score ranges and individual Section A variables.

Section B included clinical risk factors and possible etiological factors. As these variables were exploratory in nature and were not included in the primary prognostic scoring system, they were analyzed descriptively using frequencies, percentages, and graphical representation. This section aimed to identify commonly observed clinical characteristics and possible etiological trends within the study sample rather than establish direct causal or prognostic relationships.

Overall, the combined quantitative and qualitative approach helped to assess the usefulness of individual checklist items and their contribution to prognosis, thereby supporting the clinical applicability of the developed tool.

Chapter IV

RESULTS

The present study aimed to develop and preliminarily validate a prognostic and assessment checklist for patients with sudden sensorineural hearing loss SSNHL, and to see how Section A prognosis relates to the post-treatment audiometric outcome after steroid therapy. The checklist also had Section B variables for recording related clinical risk factors, plus possible causes or etiological factors, as they were observed among patients with SSNHL. A total of 24 patients diagnosed with SSNHL were enrolled. All statistical analyses were conducted with IBM SPSS Statistics (Version 26), as expected.

First, descriptive statistical analyses were done for demographic, clinical, audiological, and checklist-related variables. Section A of the checklist consisted of seven established prognostic variables: the treatment initiation time, age category, progression of hearing loss, laterality, presence of vertigo, baseline degree of hearing loss, and the audiogram configuration. The scores from these individual variables were added up to get a composite prognostic score for every participant.

Then, Spearman's rank correlation analysis was used to examine the relationship between the composite Section A prognostic score and the post-treatment audiometric outcome. In addition to the primary analyses, descriptive comparisons were performed to identify outcome trends across different prognostic score ranges and individual prognostic variables. Hearing improvement was primarily defined as a post-treatment gain of at least 15 dB in the pure-tone average (PTA). To support clearer clinical interpretation and improve consistency in outcome classification, participants were also categorized according to Lawrence G. Siegel's criteria.

Section B variables were examined using descriptive statistics and frequency distributions. These variables included associated symptoms, vascular and metabolic risk factors, autoimmune markers, previous auditory history, and other possible etiological contributors. Since the study was exploratory in nature and involved a relatively small sample, subgroup findings were interpreted descriptively, and no inferential statistical tests were performed.

4.1 Development of the Checklist

4.1.1 Content Validation

An initial version of the Assessment and Prognostic Indicator Checklist for Sudden Sensorineural Hearing Loss (SSNHL) was developed through an extensive review of the literature and relevant clinical resources. Information was gathered from the AIISH repository, standard textbooks in otolaryngology and hearing sciences, and research articles sourced from Google Scholar, PubMed, and Scopus-indexed journals. In addition, valuable insights were obtained through discussions with experienced audiologists and otolaryngologists.

Based on the evidence reviewed and the clinical relevance of the identified factors, a preliminary checklist comprising 39 items across Sections A and B was formulated. The checklist was designed to provide a structured, comprehensive framework for evaluating and identifying prognostic indicators for SSNHL.

The checklist itself was set into two major sections. The checklist was organised into two major sections. Section A comprised 7 questions focusing on the core prognostic indicators associated with recovery outcomes in SSNHL. These indicators were identified based on evidence from the reviewed literature, their frequency of reporting in previous studies, and their clinical relevance as determined through expert consultation with audiologists and

otolaryngologists. Section B consisted of 33 questions addressing the clinical and etiological factors associated with SSNHL.

Content validation was carried out by a group of six experienced audiologists and otolaryngologists. Each had at least 10 years of combined clinical and teaching experience in evaluating and managing hearing problems. The experts evaluated each item for its relevance, grammatical correctness, and overall accuracy and usefulness. Relevance and grammatical correctness were rated using "Yes" or "No" responses, while accuracy was assessed using the categories "Useful," "Optional," and "Not useful." In addition, experts were asked to indicate whether any item was repetitive, complex, or ambiguous/confusing by marking these parameters only when such issues were identified in a question, as outlined in Table 4.1. Additionally, space was also provided for open-ended comments and recommendations for modification.

Table 4.1

Content Validation Format

Sl. No.	Questions	Relevant		Repetitive	Complex	Ambiguous/ Confusing	Grammatically correct		Accuracy		
		Yes	No				Yes	No	Yes	Optional	No
1											

Note. The format used by six expert reviewers to evaluate each checklist item across the specified parameters.

Based on the expert feedback, the necessary modifications were made to improve the wording, structure and kind of overall clarity of the checklist. In Section A, one extra item was

included, and the response options for the item about treatment delay were revised into more clinically relevant time groupings. In Section B, a new subsection on associated features was introduced, covering tinnitus, aural fullness, and family history of hearing loss. The items under the atypical clinical history domain were rephrased for greater clarity. The exact additions and modifications made to Sections A and B after expert review are shown in Tables 4.2A and 4.2B, respectively.

Table 4.2A

Modifications and Additions Made to Section A Following Expert Review

Sl. No.	Type of Revision	Details
1	Modified	Response options for the item on duration between onset of hearing loss and initial medical consultation were revised into four clinically relevant time categories: "<3 days," "3–7 days," "7–14 days," and ">14 days." (Klein et al., 2023; Kuhn et al., 2011).
2	Added	"What was the audiogram configuration observed in the affected ear during the initial audiological evaluation?"

Table 4.2B

Modifications and Additions Made to Section B Following Expert Review

Sl. No.	Type of Revision	Details
1	Restructured	A new subsection titled "Associated Features" was introduced under Subsection B1, under which the following existing questions were reorganised:

	Modified	"At the time the hearing loss began, did you experience ringing in the same ear?"
	Modified	"Did you experience a feeling of fullness or pressure in the affected ear?"
	Modified	"Is there a history of congenital or unexplained early-onset hearing loss in your immediate family (parents or siblings)?"
2	Restructured	Section B was further divided into Subsection B1 ("Risk-Modifying Factors") and Subsection B2 ("Etiological Factors").

After incorporating all the suggested changes, the final checklist consisted of 40 questions, along with sections for demographic and identification details and instructions for administration. The demographic section included details such as name, age, gender, case number, and contact information, which were recorded for documentation purposes and were not included in scoring. Instructions and an example item were provided at the beginning of the checklist to facilitate standardised administration and response recording. Section A included 7 questions related to core prognostic indicators, while Section B included 33 questions addressing clinical and etiological factors. Section B was further divided into Subsection B1, “Risk Modifying Factors,” with 29 questions, and Subsection B2, “Etiological Factors,” with 4 questions. No scoring system was included, as the checklist was intended to capture the varied and multifactorial etiologies associated with SSNHL.

4.2 Demographic and Clinical Characteristics of the Study Population

In total, 24 participants with a confirmed diagnosis of SSNHL were enrolled in the present study. Among them, 18 were male (75.0%), and 6 were female (25.0%). The participants' ages ranged from 20 to 70 years, with a mean age of 46.25 years (SD not reported).

Using the age categorization from Section A, 6 participants fell in Category 1 (< 40 years), 13 in Category 2 (40–60 years) , and 5 in Category 3 (> 60 years).

Regarding the time of treatment after symptom onset, 2 participants (8.3%) sought their first medical consultation within 3 days, 7 participants (29.2%) presented between 3 and 7 days, 6 participants (25.0%) presented between 7 and 14 days, and 9 participants (37.5%) sought consultation after 14 days of onset. he stratification of treatment delay into four-time intervals (<3 days, 3–7 days, 7–14 days, and >14 days) was done, mostly, to make it possible to look a bit more closely at that early therapeutic window in SSNHL which is known to drop in recovery potential quite steeply over short time spans. A lot of studies, they often pick wider cut-offs like a 7-day or 14-day threshold, but the way this was grouped here, was meant to catch those subtler time-related changes during the acute phase of the disease. That includes, especially, the first week, since recovery is usually at its most dynamic, or at least that's the idea. In the end, this kind of setup allowed a more granular view of how the outcomes shift across clinically relevant stages of delay, without over-smoothing the timing.

Progressive worsening of hearing loss was reported by 4 participants (16.7%), while 20 participants (83.3%) did not report progressive deterioration. In most participants, unilateral involvement was seen, like in 21 people (n = 21, 87.5%), and only 3 participants (12.5%) showed bilateral involvement. Vertigo was mentioned by 13 participants (54.2%), while 11 participants (45.8%) said they did not have any vestibular-type symptoms or anything similar.

According to baseline audiometric severity, 1 participant (4.2%) fell into the mild to moderate hearing loss group. Then 4 participants (16.7%) had moderately severe hearing loss, and the remaining 19 participants (79.2%) were in the severe to profound range. For audiogram shape, 4 participants (16.7%) demonstrated an ascending or "U" shaped configuration, which was considered Type 1. Meanwhile, 14 participants (58.3%) had descending or flat patterns,

labelled Type 2, and 6 participants (25.0%) had profound or total loss setups, labelled Type 3. For classification purposes, descending/ sloping and flat patterns were all pooled under Type 2, and the profound, profound with no response, plus total loss configurations were pooled into Type 3. The full spread of Section A prognostic variables and the demographic characteristics is shown in Tables 4.3 and 4.4, respectively.

Table 4.3

Distribution of Section A Prognostic Variables (N = 24)

Variable	**Category**	**Frequency (n)**	**Percentage (%)**
Treatment Initiation Time	<3 days (Category 1)	2	8.3
	3–7 days (Category 2)	7	29.2
	7–14 days (Category 3)	6	25.0
	>14 days (Category 4)	9	37.5
Age Category	<40 years (Category 1)	6	25.0
	40–60 years (Category 2)	13	54.2
	>60 years (Category 3)	5	20.8
Progression of Hearing Loss	Absent	20	83.3
	Present	4	16.7
Laterality	Unilateral	21	87.5

	Bilateral	3	12.5
Vertigo	Absent	11	45.8
	Present	13	54.2
Baseline Degree of Hearing Loss	Mild to Moderate (Category 1)	1	4.2
	Moderately Severe (Category 2)	4	16.7
	Severe to Profound (Category 3)	19	79.2
Audiogram Configuration	Ascending/U-shaped (Type 1)	4	16.7
	Descending/Flat (Type 2)	14	58.3
	Profound/Total Loss (Type 3)	6	25.0

Note. Tx Time = treatment initiation time; categories reflect duration between symptom onset and initial medical consultation. Baseline Degree Category 1 = mild to moderate; Category 2 = moderately severe; Category 3 = severe to profound. Audiogram Configuration Type 1 = ascending/U-shaped; Type 2 = descending/flat; Type 3 = profound/total loss.

Table 4.4

Demographic Characteristics of the Study Population (N = 24)

Variable	**Category**	**Frequency (n)**	**Percentage (%)**
Gender	Male	18	75.0

	Female	6	25.0
Age (years)	Mean age: 46.25 years	—	—
	Range: 20–70 years	—	—

4.3 Distribution of Clinical Risk Factors and Associated Symptoms (Section B)

Section B of the checklist then assessed associated clinical symptoms, potential risk-modifying factors, and possible etiological clues for SSNHL. The variables were summarised descriptively and then organised into clinically meaningful domains, so that recognisable patterns across the study group can be analysed. Among the linked symptoms that were documented, tinnitus came up the most often, reported by 16 participants (66.7%). Aural fullness was reported by 12 participants (50.0%). With respect to vascular and metabolic risk factors, diabetes mellitus and lifestyle-related risk factors were each found in 9 participants (37.5%). Hypertension was present in 6 participants (25.0%), while hypercholesterolemia and a history of smoking were each observed in 5 participants (20.8%).

Other potential contributing factors, including a recent viral illness, a family history of hearing impairment, autoimmune features, migraine, thyroid dysfunction, hormonal influences, loud noise exposure, and a history of ear trauma, were reported only in smaller subsets of participants. The full distribution of the Section B variables is shown in Table 4.5. Figure 4.1, meanwhile, gives a kind of graphical snapshot of the main Section B variables seen across the study population.

Table 4.5

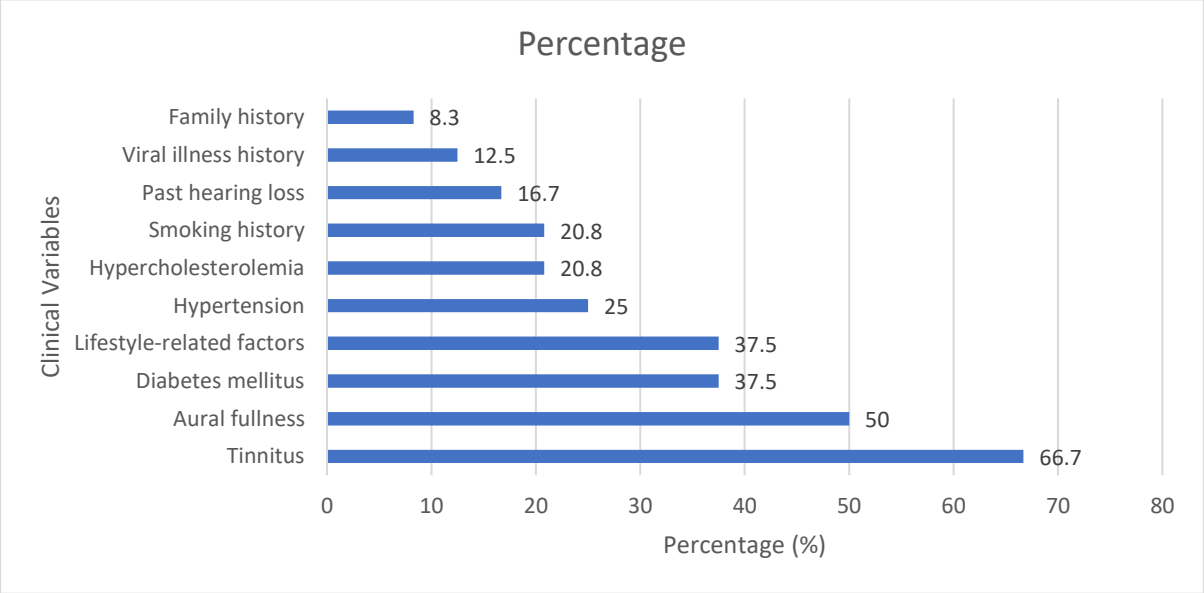
Distribution of Section B Clinical Risk Factors and Associated Symptoms (N = 24)

Clinical Factor	Frequency (n)	Percentage (%)
Associated Symptoms		
Tinnitus	16	66.7
Aural fullness	12	50.0
Family history of hearing loss	2	8.3
Past hearing loss history	4	16.7
Past imbalance history	1	4.2
Infectious / Etiological Factors		
Viral illness history	3	12.5
Blood transfusion history	2	8.3
Trauma / Exposure Related Factors		
Loud sound exposure	1	4.2
Ear trauma	1	4.2
Vascular / Metabolic Risk Factors		
Hypertension	6	25.0
Diabetes mellitus	9	37.5
Hypercholesterolemia	5	20.8
Smoking history	5	20.8
Lifestyle-related risk factors	9	37.5
Autoimmune / Neurological / Hormonal Factors		
Autoimmune history	1	4.2
Migraine history	1	4.2

Stroke history	1	4.2
Thyroid disorder	1	4.2
Hormonal factors	1	4.2

Figure 4.1

Distribution of Major Clinical Variables in Section B



4.4 Prognostic Scoring System

The prognostic scoring system was built from cumulative scores coming from Section A of the checklist. It consisted of seven established prognostic indicators of SSNHL: the time to start treatment, age category, progression of hearing loss, laterality, the presence of vertigo, baseline degree of hearing loss, and audiogram configuration. Scores were assigned to each response category based on the variable's relative prognostic significance, with higher total scores indicating a less favourable prognostic profile. The total possible Section A score ranged from 8 to 15, with a maximum achievable score of 21. The cumulative Section A score for each

participant was subsequently used for prognostic analysis and evaluated in relation to post-treatment audiometric outcomes.

Section B comprised associated clinical symptoms, systemic risk factors, and possible etiological indicators related to SSNHL. Given the exploratory and clinically heterogeneous nature of these variables, they were analyzed descriptively and were not incorporated into the cumulative prognostic scoring system.

4.5 Correlation Between Prognostic Score and Treatment Outcomes

Spearman's rank correlation analysis was performed to examine the relationship between the cumulative Section A prognostic score and post-treatment audiometric outcome. Spearman's correlation was preferred over Pearson's correlation because the prognostic scores represented ordinal/ranked data and the assumptions of normal distribution required for Pearson's correlation were not met.

A statistically significant moderate positive correlation was observed between the total prognostic score and post-treatment PTA ($r_s = 0.490, p = 0.018$), suggesting that participants with higher prognostic scores generally demonstrated poorer hearing outcomes following treatment. In other words, an increase in the prognostic score was associated with an increase in post-treatment PTA values, reflecting reduced hearing recovery.

Among the individual prognostic variables contributing to the total score, treatment initiation time showed a significant positive correlation with the cumulative prognostic score ($r_s = 0.525, p = 0.008$), indicating that delayed initiation of treatment was associated with a comparatively poorer prognosis. Similarly, audiogram configuration demonstrated a significant positive association with the total prognostic score ($r_s = 0.513, p = 0.010$), suggesting that certain audiometric patterns were linked to less favourable post-treatment hearing outcomes.

The remaining variables age category, progression of hearing loss, bilateral involvement, presence of vertigo, and baseline degree of hearing loss—demonstrated weaker, non-significant associations with the cumulative prognostic score. Detailed correlation coefficients and significance values in Table 4.6.

Additionally, post-treatment PTA showed a significant negative correlation with the hearing improvement difference score ($r_s = -0.568,\ p = .005$), indicating that poorer post-treatment hearing thresholds were associated with smaller magnitudes of hearing improvement following treatment.

Table 4.6

Spearman's Rank Correlations Between Individual Section A Prognostic Variables and Total Prognostic Score

Prognostic Variable	**Correlation Coefficient (r_s)**	**p-value**	**Interpretation**
Treatment Initiation Time	**0.525**	.008	Significant*
Age Category	0.342	.102	Not Significant
Progression of Hearing Loss	0.279	.186	Not Significant
Bilateral Involvement	0.333	.111	Not Significant
Presence of Vertigo	0.357	.087	Not Significant
Baseline Degree of Hearing Loss	0.381	.066	Not Significant
Audiogram Configuration	**0.513**	.010	Significant*

Note. * $p < .05$.

4.6 Outcome Distribution According to Hearing Improvement

Participants were split into improved vs not-improved groups using the preset threshold of above or below 15 dB gain in post-treatment PTA, respectively. Of the 24 participants, 13 (54.2%) showed audiometric improvement that met this criterion, while 11 (45.8%) did not. A descriptive look at the Section A prognostic variables across the two outcome groups suggested a few noticeable patterns. People who improved were more often found in the earlier treatment-initiation categories, whereas delayed treatment initiation was more commonly associated with the no-improvement category. Similarly, the absence of vertigo was more frequently noted among participants who showed improvement, while vertigo was relatively more common in those without improvement.

With respect to baseline audiometric severity, participants with severe to profound hearing loss constituted a larger proportion of the improved group. However, this finding should be interpreted cautiously, as individuals with poorer baseline thresholds have a greater numerical range for measurable PTA improvement without necessarily achieving complete functional recovery.

With respect to audiogram configuration, Type 1 patterns (ascending/U-shaped) were more commonly observed among participants who experienced hearing improvement. In contrast, Type 2 patterns (descending/flat) appeared more frequently in participants who did not demonstrate meaningful recovery. The distribution of Section A prognostic variables according to hearing improvement outcomes is summarised in Table 4.7.

Table 4.7

Distribution of Section A Prognostic Variables According to Hearing Improvement Outcome

Prognostic Variable	Category	Improved n (%) (n=13)	Not Improved n (%) (n=11)
Treatment Initiation Time	< 3 days	1 (7.7%)	1 (9.1%)
	3–7 days	5 (38.5%)	2 (18.2%)
	7–14 days	4 (30.8%)	2 (18.2%)
	> 14 days	3 (23.1%)	6 (54.5%)
Age Category	Younger age category	11 (84.6%)	8 (72.7%)
	Older age category	2 (15.4%)	3 (27.3%)
Progression of Hearing Loss	Sudden onset	11 (84.6%)	9 (81.8%)
	Progressive onset	2 (15.4%)	2 (18.2%)
Laterality	Unilateral	11 (84.6%)	10 (90.9%)
	Bilateral	2 (15.4%)	1 (9.1%)
Vertigo	Present	5 (38.5%)	8 (72.7%)
	Absent	8 (61.5%)	3 (27.3%)
Baseline Degree of Hearing Loss	Mild–Moderate	1 (7.7%)	0 (0%)
	Moderately Severe	0 (0%)	4 (36.4%)
	Severe–Profound	12 (92.3%)	7 (63.6%)
Audiogram Configuration	Type 1 (Ascending/U-shaped)	4 (30.8%)	1 (9.1%)
	Type 2 (Descending/Flat)	4 (30.8%)	6 (54.5%)

	Type 3 (Profound/Total loss)	5 (38.5%)	4 (36.4%)

Note. Participants were categorized as "improved" based on a ≥15 dB gain in post-treatment pure tone average (PTA) following steroid therapy. Percentages were calculated within each prognostic category.

4.7 Supplementary Descriptive Classification According to Siegel's Criteria

In addition to the primary outcome measure based on a ≥15 dB improvement in pure-tone average (PTA), hearing recovery was also assessed using Siegel's criteria, a commonly used clinical classification system for outcomes in Sudden Sensorineural Hearing Loss. Under this system, complete recovery is defined as a final hearing threshold better than 25 dB HL. Partial recovery refers to an improvement greater than 15 dB with a final hearing level between 25 and 45 dB HL, while slight improvement indicates a gain greater than 15 dB with residual hearing poorer than 45 dB HL. No improvement is classified as a gain of less than 15 dB or the absence of meaningful recovery.

Based on these criteria, complete recovery was seen in 2 participants (8.3%), whereas 4 participants (16.7%) showed partial recovery. Slight improvement was noted in 7 participants (29.2%). The remaining 11 participants (45.8%) did not demonstrate significant improvement after treatment.

Overall, the findings suggest that although several participants experienced some level of audiometric improvement, full restoration of hearing was relatively infrequent in this study population. Many participants continued to have residual hearing loss despite measurable gains in PTA thresholds. The distribution of recovery categories according to Siegel's criteria is presented in Table 4.8 and illustrated in Figure 4.2.

Table 4.8

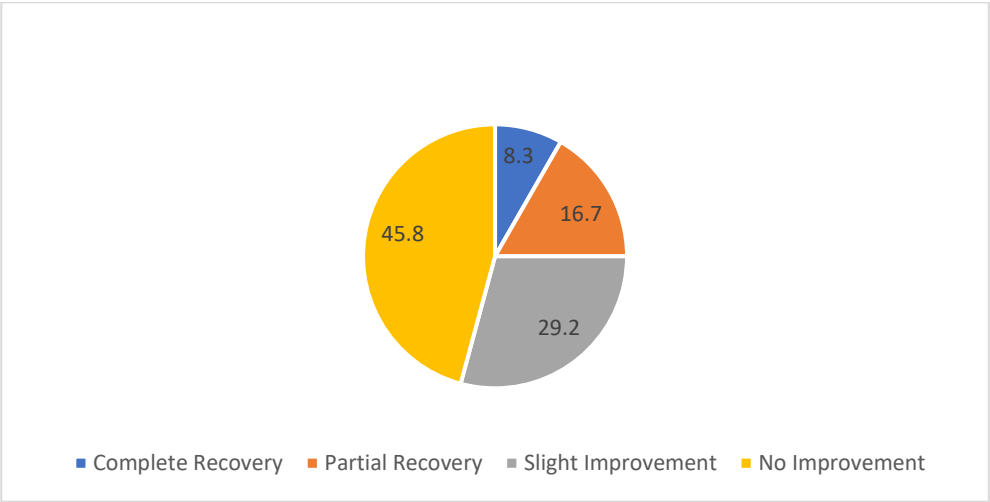
Distribution of Hearing Recovery According to Siegel's Criteria (N = 24)

Siegel's Category	Definition	n (N = 24)	Percentage (%)
Complete Recovery	Final hearing level < 25 dB HL	2	8.3
Partial Recovery	> 15 dB gain; final level between 25–45 dB HL	4	16.7
Slight Improvement	> 15 dB gain; final level > 45 dB HL	7	29.2
No Improvement	< 15 dB gain or no measurable recovery	11	45.8

Note. Siegel's criteria are widely used for classifying hearing recovery outcomes in sudden sensorineural hearing loss. dB HL = decibels hearing level.

Figure 4.2.

Distribution of hearing recovery categories (%) according to Siegel's criteria.



Chapter V

DISCUSSION

Sudden sensorineural hearing loss (SSNHL) is one of the most challenging conditions in clinical audiology and otolaryngology. It often appears without warning, progresses unpredictably, and carries an uncertain prognosis. For both clinicians and patients, the experience can feel unsettling, marked by urgency and significant diagnostic uncertainty. Even with decades of accumulated research, no standardised prognostic tool exists that brings together the clinical, audiological, and etiological dimensions of this condition in a way that can meaningfully guide day-to-day management decisions. This study attempted to address that gap by developing and carrying out a preliminary validation of a condition-specific prognostic and assessment checklist for SSNHL. The core aims were to examine how Section A prognostic scores related to audiometric outcomes following steroid treatment, and to build a descriptive clinical picture using the risk factor and etiological data captured in Section B.

A total of 24 participants with confirmed SSNHL were enrolled and assessed using the developed checklist. Overall, 54.2% (13 out of 24) showed audiometric improvement, defined as a post-treatment pure-tone average (PTA) gain of 15 dB or more.

5.1 Checklist Development and Content Validation

The checklist developed in the present study was guided by existing literature on SSNHL prognostic indicators and refined through expert review. Previous studies on clinical tool and questionnaire development have emphasised the importance of literature-based item generation combined with expert evaluation to ensure clinical relevance, clarity, and usability (Hinderink et al., 2000; Beaton et al., 2000). Following a similar approach, the present checklist

was structured to include both prognostic indicators and associated clinical risk factors relevant to SSNHL assessment.

One of the notable strengths of the checklist was the inclusion of interdisciplinary expert input during the validation process. Contributions from both audiologists and otolaryngologists helped ensure that the checklist reflected the multidimensional nature of SSNHL evaluation in routine clinical practice (Chandrasekhar et al., 2019). The structured format of the tool may also support more systematic documentation of prognostic indicators, particularly in busy clinical settings where important case-history details may otherwise be overlooked.

However, certain limitations should be acknowledged. Additional psychometric properties such as reliability, construct validity, and clinical sensitivity were not evaluated. Furthermore, the checklist was developed and examined within a relatively small clinical sample, which may limit broader generalizability. Future studies involving larger and more diverse populations are, therefore, necessary before wider clinical implementation can be considered.

5.2 Demographic and Clinical Profile of Participants

Participants in this study ranged from 20 to 70 years of age, with a mean of 46.2 years. The largest cluster of participants, 13 of the 24, fell between 40 and 60 years, accounting for 54.2% of the sample. Six participants (25.0%) were younger than 40, and five (20.8%) were older than 60. This age distribution aligns well with what has been reported epidemiologically. SSNHL has consistently been found to be most common in middle-aged and older adults, with the fifth and sixth decades carrying the highest incidence (Byl, 1984; Kim et al., 2017; Kuo et al., 2019). The reasons for this are thought to involve the gradual deterioration of cochlear

vasculature over time, declining regenerative capacity within the inner ear, and the accumulating weight of systemic conditions like hypertension and diabetes mellitus, all of which become more prevalent with age (Chandrasekhar et al., 2019; Tsuzuki & Wasano, 2024).

In terms of gender distribution, the present study included 18 male and 6 female participants, yielding a male-to-female ratio of 3:1. A similar trend toward male predominance was reported by Gupta et al. (2021) in an Indian cohort with unilateral sensorineural hearing loss. However, the literature on gender distribution in SSNHL remains mixed. While some studies have observed a slight female predominance, others have reported an almost equal representation of males and females (Alexander & Harris, 2013; Kim et al., 2017). Given the relatively small sample size of the present study, the observed gender pattern should be interpreted with caution rather than viewed as reflecting a definitive epidemiological trend.

Nearly 21 participants (87.5%) presented with unilateral SSNHL, while the remaining 3 (12.5%) had bilateral involvement. This is broadly consistent with the well-established observation that more than 90% of SSNHL cases affect only one ear (Byl, 1984; Kuhn et al., 2011). When both ears are affected, the clinical picture tends to be more complex, with systemic conditions such as autoimmune disease or vascular pathology more likely to be implicated (Chau et al., 2010; Alexander & Harris, 2013). Although the present study did not investigate etiological mechanisms in detail, it is noteworthy that vascular risk factors such as hypertension and diabetes were present in some of the bilateral cases. However, given the very small number of bilateral participants, these observations should be interpreted cautiously. In this study, two of the three participants with bilateral involvement did improve audiometrically following treatment, though the subgroup is too small to support any generalizable conclusion.

Initial audiometric severity was notable in this cohort, with 19 of 24 participants (79.2%) presenting with severe to profound hearing loss. The predictive weight of initial

severity in SSNHL has been well-established since Byl's (1984) landmark eight-year prospective study, which found that profound loss at presentation was associated with markedly lower rates of recovery.

5.3 Section A: Prognostic Score and Audiometric Outcomes

The primary objective of the present study was to examine whether the cumulative Section A prognostic score was associated with post-treatment audiometric outcomes. Spearman's rank correlation analysis demonstrated a statistically significant moderate positive correlation between the total prognostic score and post-treatment PTA ($r_s = 0.490$, $p = 0.018$). This indicates that participants with higher prognostic scores, representing comparatively less favourable clinical profiles at presentation, generally exhibited poorer audiometric outcomes following steroid therapy. Although these findings should be interpreted cautiously in view of the relatively small sample size, the observed association suggests that the checklist may hold potential as a supportive tool for prognostic assessment in SSNHL.

There was considerable variability among participants who had similar total scores, which reflects something real about SSNHL: it is a heterogeneous condition, and the same clinical profile can produce quite different outcomes in different patients. However, the observation that a composite score incorporating multiple clinical variables demonstrated a statistically significant relationship with outcome is in line with the broader shift in SSNHL research toward multivariable approaches to prognostic assessment (Mandavia et al., 2024; Byl, 1984).

5.3.1 Influence of Treatment Delay on Prognosis

Of all the prognostic variables in Section A, time of treatment showed the strongest individual correlation with the cumulative score ($r_s = 0.525$, $p = 0.008$). Looking at the data

more closely, only two participants had sought medical attention within three days of symptom onset, and only one of those two improved. In the three-to-seven-day window, five of seven participants (71.4%) showed improvement. Among those who came in between seven and fourteen days, four of six (66.7%) improved. But among the nine participants who delayed beyond two weeks, only three (33.3%) demonstrated audiometric gain.

This pattern is consistent with a large and fairly consistent body of evidence on treatment timing in SSNHL. Byl (1984) demonstrated progressively worsening recovery rates as treatment delay increased. More recently, Mandavia et al. (2024), drawing on a multicenter dataset of 498 patients, identified steroid initiation within seven days as the single strongest predictor of complete hearing recovery. Edizer et al. (2015) similarly found that outcomes deteriorated when therapy was delayed beyond ten days. Klein et al. (2023), through a case series and meta-analysis, further reported significantly better recovery outcomes when treatment was initiated within the first 14 days of symptom onset, reinforcing the importance of early intervention in SSNHL. Similarly, Chen et al. (2023), in a retrospective study involving 666 patients, observed that patients treated within the first two weeks showed significantly greater hearing improvement than those treated after 14 days.

The biological rationale here is fairly well understood: inflammatory processes, endolymphatic pressure changes, and cochlear ischemia can cause progressive and potentially irreversible damage to hair cells and supporting structures if left unaddressed during the acute window (Rauch, 2008; Yamada et al., 2022). The longer the cochlea is left without intervention, the narrower the window for recovery becomes.

The finding that nine out of 24 participants (37.5%) in the present study presented more than 14 days after symptom onset is clinically important, particularly given the well-established influence of treatment timing on recovery outcomes in SSNHL. Delayed presentation in such

cases may substantially reduce the likelihood of meaningful hearing improvement. Similar patterns of delayed presentation have also been reported in Indian clinical cohorts by Gupta et al. (2021). Although the present study did not specifically investigate the reasons underlying delayed healthcare-seeking, factors such as limited awareness regarding the urgency of sudden hearing loss, delays in specialist referral, and misinterpretation of symptoms as minor ear-related problems may contribute to late presentation in clinical practice. These findings highlight the importance of improving awareness regarding SSNHL as a medical emergency and strengthening timely referral pathways for early intervention.

5.3.2 Influence of Age on Prognosis

Age was included in Section A as an ordinal variable, with scores assigned across three brackets: under 40 years (score 1), 40–60 years (score 2), and over 60 years (score 3). In this study, participants younger than 40 improved at a rate of 50% (3 out of 6), those in the middle bracket at 61.5% (8 out of 13), and those over 60 at 40% (2 out of 5). The pattern is not a clean linear decline, and the subgroup sizes are too small to read too much into the numbers. But directionally, younger and middle-aged participants did appear to fare somewhat better.

The literature has fairly consistently linked younger age with better prognosis in SSNHL, though the magnitude and independence of this effect vary across studies. Mandavia et al. (2024) identified younger age (below 40-48 years) as an independent predictor of complete recovery in their multivariate model. Edizer et al. (2015) similarly reported lower complete recovery rates in patients over 60. The proposed mechanisms include cochlear degeneration with aging, reduced reserve in cochlear blood flow, greater susceptibility to ischemic injury, and diminished hair cell regenerative potential (Alexander & Harris, 2013; Tsuzuki & Wasano, 2024). At the same time, Atay et al. (2016) found that audiogram shape and treatment timing outweighed age as independent prognostic influences, suggesting that

older patients are not necessarily poor candidates for recovery, particularly when other factors are working in their favour. That nuance is consistent with what was seen in this study, where some older participants did improve meaningfully after timely treatment.

5.3.3 Progression of Hearing Loss

Progressive worsening of hearing loss after the sudden onset was observed in four participants in the present study. Of these, two participants (15.4%) belonged to the improved group, while two (18.2%) were in the non-improved group, suggesting no clear difference between outcome groups within this sample. Consistent with this observation, progression of hearing loss did not demonstrate a statistically significant association with audiometric outcome. However, this may also reflect the influence of multiple interacting prognostic variables, including factors such as initial hearing severity, associated vertigo, and time to treatment initiation, all of which may contribute to recovery outcomes in SSNHL.

Nevertheless, inclusion of this variable in the checklist remains clinically relevant, as it encourages attention to the temporal evolution and trajectory of symptoms rather than focusing solely on severity at presentation. Progressive or fluctuating hearing loss has been discussed in the literature as a potentially less favourable clinical feature and may reflect ongoing cochlear pathology, including hydropic or inflammatory mechanisms (Domínguez et al., 2021). In addition to its prognostic relevance, this pattern may also have diagnostic implications. Previous studies have described progressive auditory symptoms in the context of early Ménière's disease and endolymphatic hydrops, where hearing changes may precede the classical presentation of episodic vertigo, tinnitus, and aural fullness (Domínguez et al., 2021; Pecorari et al., 2019). Although the small number of participants limits broader interpretation, documenting progressive symptom patterns may still help identify patients who require closer follow-up and further otological evaluation.

5.3.4 Severity of Initial Hearing Loss

Baseline hearing severity demonstrated a weak positive correlation with cumulative prognostic score ($r_s = 0.381, p = 0.066$). A large proportion of participants in the present study (79.2%) presented with severe-to-profound hearing loss at baseline, resulting in relatively limited variability across severity categories. This restricted distribution may partly explain the absence of a statistically significant association between baseline severity and audiometric outcome.

Although 54.2% of participants demonstrated audiometric improvement based on the study criterion of a ≥15 dB gain in PTA, these findings should be interpreted cautiously in relation to final functional hearing status. According to Siegel's recovery criteria, only two participants (8.3%) achieved complete recovery, while the majority of participants who demonstrated improvement continued to have hearing thresholds greater than 45 dB HL and therefore fell within the "slight improvement" category. This suggests that measurable audiometric gain did not necessarily correspond to substantial functional recovery in all cases. The predominance of improvement within the severe-to-profound subgroup may also partly reflect a ceiling/floor effect associated with the use of absolute PTA change as an outcome measure, wherein participants with markedly elevated baseline thresholds may demonstrate measurable numerical improvement despite remaining within a functionally severe range of hearing impairment.

Nevertheless, the relationship between baseline severity and prognosis in SSNHL remains one of the most consistently reported findings in the literature. Byl (1984) demonstrated that individuals presenting with profound hearing loss were significantly less likely to achieve complete or substantial recovery compared to those with milder degrees of impairment, a finding later supported by the meta-analytic review of Yu and Li (2018). From

a pathophysiological perspective, more severe initial thresholds may reflect greater cochlear injury involving hair cells, spiral ganglion neurons, and supporting structures, thereby limiting the potential for meaningful recovery (Rauch, 2008; Kuhn et al., 2011). At the same time, the variability in recovery patterns observed even among participants with severe initial hearing loss highlights the multifactorial nature of SSNHL prognosis, where outcomes are likely influenced by the interaction of several clinical variables rather than baseline severity alone.

5.3.5 Audiogram Configuration and Prognostic Trends

Audiogram configuration showed one of the strongest individual associations with cumulative prognostic score ($r_s = 0.513$, $p = 0.010$). Descending and flat patterns were the most common in this cohort, with ascending and U-shaped (trough) configurations appearing in a smaller number of participants. Consistent with what the literature would predict, participants presenting with ascending audiogram patterns reflecting predominantly low-frequency hearing loss showed more favourable recovery trends.

This finding is well-supported by the existing evidence. Mattox and Simmons (1977) were among the first to identify ascending configurations as associated with substantially better recovery. More recent work has reinforced this: Psillas et al. (2019) reported complete recovery in 77.7% of patients with low-frequency SSNHL compared to just 15% of those with high-frequency loss. Flat and down-sloping patterns have been linked to more adverse inflammatory and metabolic profiles in addition to worse audiometric outcomes, possibly indicating more extensive cochlear pathology (Cadoni et al., 2025). The proposed explanation is that ascending patterns may reflect pathology confined mainly to the apical cochlea, perhaps endolymphatic hydrops or transient strial dysfunction, which may be more amenable to reversal than the hair cell destruction typically associated with high-frequency descending loss (Waissbluth et al., 2022; Watanabe & Suzuki, 2018).

One participant in the present study demonstrated a trough-shaped (mid-frequency) audiogram configuration. Although no retrocochlear pathology was identified in this case, previous literature has noted the potential clinical significance of this audiometric pattern. Hosokawa et al. (2018) reported that trough-shaped audiograms were more frequently observed in patients with acoustic neuroma compared to those with idiopathic SSNHL, highlighting the importance of careful otological evaluation when such configurations are encountered. In this context, inclusion of audiogram configuration within the checklist may have clinical utility not only for prognostic profiling but also for identifying patterns that could warrant further diagnostic investigation in selected cases.

5.3.6 Bilateral Involvement and Prognostic Implications

Three participants in the present study demonstrated bilateral SSNHL. Of these, two participants (15.4%) belonged to the improved group, while one (9.1%) belonged to the non-improved group. Interestingly, both participants who demonstrated improvement presented with flat audiogram configurations and did not report associated vertigo, whereas the participant who did not improve demonstrated a high-frequency sloping audiogram configuration along with vestibular involvement. Although the very small subgroup size limits interpretation, these observations suggest that recovery in bilateral SSNHL may be influenced by the interaction of multiple clinical variables rather than bilateral involvement alone.

Bilateral SSNHL is generally considered less common and is often associated with a more complex clinical profile compared to unilateral presentations. Previous literature has reported stronger associations between bilateral involvement and identifiable systemic etiologies, including autoimmune inner ear disease, haematological disorders, and vascular pathology (Alexander & Harris, 2013; Chau et al., 2010). Accordingly, assigning a higher

prognostic score to bilateral involvement within the checklist remains clinically justified, as it may prompt more detailed etiological evaluation and closer clinical monitoring in such cases.

5.4 Section B: Associated Clinical Symptoms (Section B1)

Section B was designed to document associated symptoms, systemic comorbidities, and relevant risk factors in a structured descriptive format. Given the sample size and the exploratory intent of this section, no inferential statistical testing was applied. Instead, findings are discussed descriptively in relation to observed improvement trends, interpreted alongside the relevant literature. Therefore, observed associations within specific subgroups should be interpreted as potential patterns rather than independent causal relationships.

5.4.1 Tinnitus

Tinnitus was the most frequently reported associated symptom in this cohort, present in 16 of 24 participants (66.7%). Of those, 10 (62.5%) still showed audiometric improvement, while 6 (37.5%) did not. The high prevalence of tinnitus here is consistent with what has been reported in larger clinical series. Nogueira-Neto et al. (2016) documented tinnitus in approximately 93.3% of SSNHL patients, and Zou et al. (2023) reported it in 79.7% of patients with sudden deafness. Tinnitus in SSNHL is generally understood as a reflection of the same cochlear injury mechanisms underlying the hearing loss, i.e., hair cell damage, auditory nerve deafferentation, and altered central auditory processing (Cvorovic et al., 2021; Mokhatrish et al., 2023).

Importantly, tinnitus in this sample was not systematically associated with non-improvement; rather, the majority of participants with tinnitus still achieved audiometric gain. However, tinnitus carries its own clinical burden that is separate from audiometric outcome. It

can persist long after hearing thresholds have stabilised and significantly affect quality of life (Nogueira-Neto et al., 2016).

In the present study, patients with tinnitus showed a relatively higher proportion of audiometric improvement. This observation is consistent with some previous studies and pooled evidence suggesting a mild association between tinnitus presence and better hearing recovery in SSNHL. Meta-analytic data have reported slightly higher recovery rates in patients with tinnitus compared to those without, although the effect size is modest and findings are heterogeneous across studies (Wei et al., 2025). Earlier cohort-level observations have also suggested a tendency toward improved outcomes in certain tinnitus-positive subgroups (Hikita-Watanabe et al., 2010; Ben-David et al., 2001). However, this association is not consistently observed, and larger multivariate analyses have shown that tinnitus is not an independent predictor of recovery when adjusted for established prognostic factors such as initial hearing severity, time to treatment, and presence of vertigo (Harada & Kato, 2013; Nogueira-Neto et al., 2016). Documenting it in the checklist is therefore important not just for clinical profiling but for identifying patients who may need ongoing management.

5.4.2 Aural Fullness

Aural fullness was reported by half of the cohort (12/24, 50.0%), with an equal split between those who improved (n = 6) and those who did not (n = 6), suggesting no clear prognostic value in this sample. It is a commonly reported symptom in SSNHL and is thought to result from inner ear pressure dysregulation rather than middle ear disease (Park et al., 2012; Wang et al., 2020).

Previous studies have also shown limited prognostic relevance. Han et al. (2023) found no significant association between aural fullness and hearing outcome ($p = 0.261$), and Zhou et al. (2021) reported that resolution of fullness did not correlate with audiometric recovery.

However, one small study in recurrent SSNHL suggested a possible association between aural fullness and better recovery in selected cases (Jeon et al., 2022). Overall, aural fullness appears to be a frequent accompanying symptom in SSNHL, but it shows limited and inconsistent value as a predictor of hearing recovery.

5.4.3 Vertigo and Vestibular Symptoms

Vertigo was present in 13 of 24 participants (54.2%). Within this subgroup, only 5 of 13 patients (38.5%) demonstrated audiometric improvement, whereas a higher proportion of patients without vertigo improved (8 of 11; 72.7%). This pattern suggests a comparatively poorer hearing outcome in participants presenting with vertigo in this cohort. Yu and Li (2018), in a systematic review and meta-analysis of 4,814 patients, found hearing recovery rates of 42.13% in those with vertigo versus 60.29% in those without, with a pooled odds ratio of 2.22 for poorer recovery among the vertigo group. Park et al. (2001) likewise observed that audiometric recovery was affected by how extensively the vestibular system was involved when patients first presented with sudden hearing loss.

The most plausible explanation is that vertigo reflects more extensive labyrinthine involvement, indicating pathology that extends beyond the cochlea into the vestibular system. This suggests a broader extent of inner ear injury, which is likely associated with reduced potential for auditory recovery (Yamada et al., 2022). This finding supports the inclusion of vertigo as a prognostic indicator in Section A and highlights the clinical importance of early identification of vestibular symptoms, both for informing patient counselling regarding prognosis and for guiding timely consideration of vestibular rehabilitation when indicated.

5.5 Vascular, Metabolic, and Systemic Risk Factors

5.5.1 Diabetes Mellitus

Diabetes mellitus was the most prevalent systemic comorbidity in this sample, present in 9 of 24 participants (37.5%). The diabetic subgroup had a mean age of 53.1 years, with ages ranging from 40 to 68 years. Of these nine participants, four (44.4%) improved, and five (55.6%) did not, which is a slightly less favourable distribution compared to the 15 participants without diabetes, of whom nine (60%) showed improvement. The directional trend is consistent with the literature, even if the group sizes are too small to draw firm conclusions.

Sajid et al. (2021) reported that 28% of SSNHL patients in their clinical series had diabetes, and noted lower complete recovery rates from corticosteroid treatment in the diabetic subgroup. A large meta-analysis by Saba et al. (2023), drawing on 27 studies and over 77,000 patients, identified significantly elevated odds of diabetes among SSNHL patients compared to matched controls. The proposed mechanisms are multiple and interrelated: endothelial dysfunction, microvascular disease affecting the stria vascularis, reduced cochlear blood flow, and impaired glucose metabolism in hair cells have all been implicated (Tsuzuki & Wasano, 2024; Quaranta et al., 2016). It is worth noting, however, that a propensity score-matched analysis by Seo et al. (2020) found that after accounting for baseline hearing severity and treatment timing, diabetes did not independently predict recovery outcomes, suggesting that some of its apparent prognostic impact may operate through more severe initial hearing loss rather than through a direct effect on treatment response.

The prevalence of diabetes in this cohort (37.5%) is notable and reflects an important comorbid profile in this population. Given India's high burden of type 2 diabetes, this comorbidity pattern is not unexpected among patients presenting to tertiary care centres with SSNHL. It highlights the importance of routine metabolic screening as part of SSNHL assessment, particularly in settings with a high background prevalence of diabetes. It also underscores the need for a multidisciplinary approach to care, involving audiology,

otolaryngology, and endocrinology, to ensure comprehensive management of both the auditory condition and its associated systemic risk factors.

5.5.2 Hypertension

Hypertension was present in six participants (25.0%) and was relatively older, with a mean age of 56.5 years (range: 42–70 years). The outcome pattern here was more balanced than with diabetes: four of the six improved, and two did not. This suggests that hypertension alone was not a particularly strong barrier to recovery in this sample, though its co-occurrence with other vascular risk factors in several participants complicates independent interpretation.

Hypertension has been proposed as an SSNHL risk factor through its effects on cochlear microcirculation, including endothelial dysfunction, increased arterial stiffness, and impaired autoregulation of inner ear blood flow, which have all been described as potential pathways (Quaranta et al., 2016; Saba et al., 2023). The meta-analysis by Saba et al. (2023) confirmed elevated hypertension prevalence in SSNHL patients relative to controls. That said, establishing hypertension as an independent prognostic factor for recovery, rather than a risk factor for the occurrence of SSNHL, has proven challenging across studies. Some evidence suggests that, similar to diabetes, its apparent prognostic effect may be mediated by baseline disease severity rather than a direct influence on treatment response (Tsuzuki & Wasano, 2024). The findings of the present study are cautiously consistent with this interpretation.

5.5.3 Hypercholesterolemia

Five participants (20.8%) in this study had hypercholesterolemia, and none of the five showed audiometric improvement of 15 dB or more. All five fell in the non-improvement group. The sample is small, and caution is warranted in interpreting this, the uniformity of this pattern is clinically noteworthy.

Elevated total cholesterol has been identified as one of the more consistent vascular risk factors associated with SSNHL across systematic reviews (Tsuzuki & Wasano, 2024). Saba et al. (2023) specifically reported that 35–40% of idiopathic SSNHL patients had hypercholesterolemia, with a significant association between elevated total cholesterol and SSNHL occurrence. The mechanistic pathway is thought to involve atherosclerotic compromise of cochlear microcirculation, reduced flexibility of the stria vascularis, and impaired oxygen delivery to inner ear structures through endothelial dysfunction (Quaranta et al., 2016; Tsuzuki & Wasano, 2024). Lin et al. (2016) specifically identified that hypercholesterolemia, alongside diabetes, has been reported in some studies as a potential negative prognostic factor for hearing recovery following SSNHL, and this trend is broadly in keeping with the pattern observed in the present study. One plausible explanation is that dyslipidemia may contribute to microvascular compromise and sustained cochlear ischemia, which could limit the degree of recovery following treatment. However, such mechanisms remain speculative, and causation cannot be inferred from the present descriptive data. Overall, these findings highlight the relevance of documenting lipid-related risk factors in SSNHL, while also recognising that their independent prognostic role requires further clarification in larger controlled studies.

5.5.4 Smoking and Lifestyle Factors

Five participants (20.8%) had a documented history of smoking: two improved, and three did not. Lifestyle-related risk factors, as captured in the questionnaire (including regular alcohol consumption and recent physiological stressors such as significant stress, dehydration, or lack of sleep preceding symptom onset), were present in nine participants (37.5%). Among these, four showed audiometric improvement, and five did not. This reflects a modestly higher proportion of non-improvement within this subgroup, although no definitive inference can be made given the descriptive nature of the data. The slightly higher proportion of non-improvers

in the lifestyle risk factor group is consistent with the idea that vascular risk factor accumulation gradually erodes cochlear perfusion reserve, leaving less room for recovery when an acute insult occurs (Tsuzuki & Wasano, 2024).

Smoking is another factor related to SSNHL, as it is thought to influence blood viscosity, endothelial function, and cochlear microcirculation (Saba et al., 2023; Kuhn et al., 2011). Nonetheless, its prognostic impact on hearing recovery following treatment, in contrast to its impact on causation, has yet to be clearly defined. The results in this paper parallel the vascular hypothesis of SSNHL, stating that incremental vascular burden could compromise cochlear reserve and the potential for recovery following an event such as an acute ischemic or inflammatory lesion (Quaranta et al., 2016).

5.5.5 History of Prior Audiological Complaint

Preceding histories of tinnitus before the diagnosis of SSNHL was recorded in four patients (16.7%). Audiometric improvement was noted in all four patients however, the subgroup size was extremely small. Tinnitus occurring with SSNHL can be attributed to cochlear pathology manifested by mechanisms such as hair cell damage, neural deafferentation and alterations in central auditory processing (Cvorovic et al., 2021; Mokhatrish et al., 2023). In these instances, preceding or pre-existing tinnitus may be representative of early or subclinical cochlear pathology prior to the manifestation of significant hearing loss.

Some of the clinical descriptions of SSNHL also describe discrepancies in the course of symptoms including the presence of auditory symptoms prior and concomitant to incidents of sudden hearing loss (Rauch,2008; Byl, 1977). However, tinnitus has not been identified as an independent predictor of outcome, and the available evidence shows mixed and weak relationships between prognostic outcome and this symptom (Nogueira-Neto et al., 2016: Hikita-Watanabe et al., 2010). Therefore, although the present finding may have some

implication in that it suggests a positive trend toward a better recovery in this subset of individuals, it must be interpreted with caution given the small numbers.

5.5.6 Family History and Viral History

A family history of hearing loss was reported by two participants (8.3%), both of whom were in the non-improvement group. Family history has been looked at in connection with idiopathic SSNHL, and a few studies hint at a genetic predisposition. Binnetoğlu et al. (2015) described, in a group of 125 patients, a noticeably higher rate of people who had relatives with similar issues, 33.6% for ISSNHL cases, which seems to link to disease susceptibility. Likewise, Gäckler et al. (2010) reported that 21.4% out of 186 SSNHL patients mentioned a positive family history, and even if the differences in outcome were not statistically significant, there was still a slight inclination toward weaker recovery and a younger age when symptoms started, in that specific subgroup.

A history of recent viral illness before the onset of hearing loss was documented in three participants (12.5%), and all three of those participants also did not show audiometric improvement. The pattern is limited by sample size, but the concentration of these features in the non-improvement group is worth noting. Viral etiology is among the most discussed pathophysiological hypotheses in SSNHL research. The proposed mechanisms include direct viral invasion of cochlear structures, reactivation of latent herpesviruses, immune-mediated inner ear injury, and cytokine-driven cochlear inflammation (Chen et al., 2019; Tripathi & Deshmukh, 2022). The observation that all three participants with antecedent viral illness in this study were non-improvers may reflect the hypothesis that immune-mediated or virus-related cochlear damage tends to be more extensive and less reversible, causing more severe destruction of hair cells and spiral ganglion neurons than some other mechanisms (Yamada et al., 2022; Merchant et al., 2008). Notably, no participant in the present study reported COVID-

19-associated hearing loss, which may reflect the timing of data collection or the healthcare-seeking profile of the cohort.

5.6 Section B2: Possible Etiological and Triggering Factors

Section B2 was designed to capture possible etiological triggers — noise exposure, ear trauma, pressure changes, and other identifiable precipitants. Across this cohort, such triggers were relatively infrequent, which reflects the predominantly idiopathic nature of SSNHL in most clinical populations. One participant reported significant loud sound exposure before the hearing loss, and another reported a history of ear-related trauma; both fell in the non-improvement group. This pattern is conceptually consistent with what the literature describes for noise- and trauma-related cochlear injury, where mechanical disruption of hair cell stereocilia or the tectorial membrane may produce more direct and less reversible damage than vascular or inflammatory mechanisms (Selvanayagam & Mathialagan, 2021; Padmakumar et al., 2019).

No participants reported barotrauma, head trauma, or toxic exposures as possible precipitants. The absence of these triggers in this sample does not diminish their clinical importance rather they carry specific diagnostic and management implications. Noise-induced threshold shifts may warrant different clinical decision-making compared to idiopathic SSNHL, and trauma-related cases may require imaging to exclude perilymph fistula or temporal bone fracture (Antoniades et al., 2022; Kuhn et al., 2011). Including these items in Section B2 ensures that the checklist functions as a comprehensive screening instrument across the full spectrum of SSNHL etiologies.

One participant had a documented autoimmune history, and this individual demonstrated audiometric improvement following steroid treatment. This single observation is consistent with the broader clinical understanding that autoimmune-mediated hearing loss may

respond favourably to corticosteroids in the acute phase, given the overlap between the mechanism of steroid treatment and immune suppression (Broughton et al., 2004; McCabe, 1979). It also supports the rationale for including autoimmune history in the checklist, not only as a clinically meaningful variable but as a prompt to consider longer-duration immunosuppressive therapy or rheumatological co-management in appropriate cases.

5.7 Overall Audiometric Outcomes and Siegel's Criteria

Across the cohort, 13 participants (54.2%) achieved audiometric improvement of 15 dB or more in post-treatment PTA, while 11 (45.8%) did not reach this threshold. The mean PTA before treatment was 86.4 dB HL, and after treatment it was 62.4 dB HL, a mean group-level gain of approximately 23.0 dB. While these findings are clinically meaningful, they should be viewed in light of the heterogeneity of presentations within the sample.

The 54.2% improvement rate is within the range of 32–65% cited across natural history and intervention studies (Mattox & Simmons, 1977; Bayoumy et al., 2018). When outcomes were classified using Siegel's criteria, two participants (8.3%) achieved complete recovery, four (16.7%) showed partial recovery, and seven (29.2%) demonstrated slight improvement, while 11 (45.8%) showed no meaningful change.

The relatively high proportion of participants with slight or no improvement, compared to some published treatment studies, may be explained by several overlapping factors. First, at presentation, the mean PTA was 86.4 dB HL; therefore, on admission, subjects were already severely impaired, and recovery was thus naturally restricted. Secondly, 37.5% presented more than 14 days from onset, and the negative correlation between later presentation and prognosis in SSNHL is known (Byl, 1984; Mandavia et al., 2024). Thirdly, many subjects also had vascular and metabolic co-morbidities, potentially limiting cochlear recovery. Collectively,

these findings highlight the multifactorial nature of recovery in SSNHL and reinforce the importance of early and structured clinical assessment.

5.8 Clinical Utility and Implications of the Checklist

The checklist developed in this study occupies a distinct, real-world role among clinical instruments that are used in audiology and otolaryngology. There are already tools like the Hearing Handicap Inventory for Adults (HHIA), the Tinnitus Handicap Inventory (THI), and the Dizziness Handicap Inventory (DHI), but they were not designed specifically to support prognostic assessment in SSNHL (Ventry & Weinstein, 1982; Newman et al., 1996; Jacobson & Newman, 1990). In contrast, the present checklist was developed specifically to facilitate a structured evaluation of clinical, audiological, and etiological factors that may influence prognosis and management of individuals with SSNHL.

Overall, the checklist is meant to support clinical decisions right at the first presentation of the hearing loss. Section A gives a short outline for estimating the overall prognostic pattern, which may help clinicians counsel the patients about what recovery might look like and why fast, timely intervention matters. Section B documents associated symptoms, comorbidities, and possible etiological triggers that might be missed during routine visits. Put together, the two parts make the checklist useful for both prognostic and wider clinical assessment needs.

The checklist was developed to be practical and adaptable to several settings in which SSNHL is first diagnosed, including ENT outpatient clinics, audiology centres, and emergency departments. The framework may also facilitate multi-disciplinary communication, aiming to consistently provide and effectively communicate key clinical information among professionals involved in patient care. However, the checklist should be considered a preliminary clinical tool requiring further validation. Some further prospective studies in large,

diverse patient groups will be required to assess the predictive validity, inter-rater reliability and universal applicability.

Chapter VI

SUMMARY AND CONCLUSIONS

Sudden sensorineural hearing loss (SSNHL) is classified as an otological emergency characterised by a rapid fall of ≥30 dB in hearing sensitivity across three or more consecutive audiometric frequencies, and this should happen within 72 hours (Wilson et al., 1980; Chandrasekhar et al., 2019). The condition affects tens of thousands of individuals annually, with reported global incidence rates ranging from approximately 5 to over 400 per 100,000 population (Yang et al., 2024). Recovery outcomes are highly variable, with spontaneous or treatment-mediated improvement reported in approximately 32–65% of cases (Mattox & Simmons, 1977; Bayoumy et al., 2018). Even with the clinical importance and, well, the large amount of prognostic work, there is still no standardised, SSNHL-specific instrument in everyday clinical practice that really and truly captures the wide set of clinical, audiological, and etiological factors needed for both assessment and prognosis.

This present study aimed for the following objectives: (a) to design a condition-focused screening and assessment checklist for SSNHL, one that gathers the main prognostic signs, co-occurring symptoms, and systemic risk contributors in a structured way; (b) to test the link between the Section A prognostic score and the audiometric results after steroid therapy ; (c) to look at outcome patterns, descriptively, across different prognostic score intervals, plus across individual prognostic variables; and (d) to outline how clinical risk factors, and possible etiological factors, are distributed among patients with SSNHL, using descriptive statistics.

The study used a cross-sectional approach of tool development and validation. It was carried out at the All India Institute of Speech and Hearing (AIISH), Mysuru, with approval from the Institutional Review Board. The checklist was developed in four structured phases:

(I) literature-based development, (II) expert content validation, (III) field administration, and (IV) quantitative and qualitative data analysis. First, a preliminary checklist with 39 items was reviewed by a panel of six audiologists and otolaryngologists (each with at least 10 years of clinical as well as academic practice). The experts' comments led to one additional item being included in Section A, and Section B was restructured, which finally gave a 40-item checklist. It was then arranged into Section A (7 prognostic indicator items) and Section B (33 items split into Subsections B1 and B2).

Spearman's rank correlation analysis showed a statistically significant, moderate positive association between the total Section A prognostic score and the post-treatment pure-tone average ($r_s = 0.490,\ p = .018$). This implies that participants who came in with less favourable prognostic characteristics had higher composite scores and often ended up with worse audiometric results after steroid treatment. Looking at the individual prognostic components, the time when treatment was first initiated emerged as the strongest correlate with the total prognostic score ($r_s = 0.525,\ p = .008$), followed by audiogram configuration ($r_s = 0.513,\ p = .010$). Age, bilateral involvement, baseline hearing severity, presence of vertigo, and the progression of hearing loss showed weaker, non-significant associations with the cumulative score. Also, the post-treatment PTA by itself had a significant negative correlation with the hearing improvement difference score ($r_s = -0.568,\ p = .005$), which fits with smaller improvements being tied to higher PTA values after therapy.

Using the main outcome rule of at least a ≥15 dB improvement in post-treatment PTA, 13 participants (54.2%) showed audiometric improvement. When we applied an additional stratification approach based on Siegel's criteria, complete recovery occurred in 2 participants (8.3%), partial recovery in 4 (16.7%), slight improvement in 7 (29.2%), and no improvement in 11 (45.8%). Within the descriptive analysis of Section B variables, tinnitus emerged as the most frequently reported associated symptom, present in 66.7% of participants, followed by

vertigo (54.2%) and aural fullness (50.0%). For systemic comorbidities, diabetes mellitus, along with lifestyle-related risk factors, showed up as the most common, each one appearing in 37.5% of participants. All five participants with documented hypercholesterolemia fell within the non-improvement group, a pattern that is broadly consistent with existing literature highlighting the possible role of vascular and metabolic factors in poorer SSNHL outcomes (Lin et al., 2016; Saba et al., 2023). Similarly, histories of recent viral illness and family history of hearing loss, although observed only in small subgroups, were also confined to participants who did not demonstrate audiometric improvement

6.1 Conclusions

This study successfully generated and has produced a preliminary validation for a condition-specific Checklist for Assessment and Prognostic Indicator for Sudden Sensorineural Hearing Loss (SSNHL) using a systematic literature-based framework supported by structured expert validation.

This study offers initial evidence on the prognostic relevance of this checklist. In general, higher cumulative scores derived from the prognostic items were associated with inferior post-treatment audiometric outcomes after steroid therapy, suggesting that the checklist may be a useful clinical aid in identifying individuals at higher risk of having an questionable recovery. Delay in treatment and unfavorable audiometric configuration, among other prognostic indicators, seemed to be more strongly correlated with a worse outcome.

This study also revealed the common association between other otological symptoms like tinnitus, vertigo, and aural fullness and metabolic or vascular risk factors such as diabetes mellitus and hypercholesterolemia. This suggests a potentially multi-factorial basis for SSNHL and stresses the importance of constructing a thorough clinical profile of a patient with SSNHL.

Overall, the developed checklist provides a structured and evidence-informed approach for the systematic evaluation of SSNHL by integrating prognostic indicators, associated symptoms, and potential etiological contributors within a single clinical tool. Its use may assist in improving the organization of clinical information, support patient counselling, facilitate interdisciplinary communication, and contribute to greater consistency in the clinical assessment of SSNHL across different practice settings. However, the findings of this study need to be interpreted within the context of the limitations that exist: a small number of subjects included and a cross-sectional design of the study. Larger, prospective studies are required to provide better information on the predictive validity, reliability and wider generalizability of this checklist across different populations and clinical settings.

6.2 Clinical Implications

The findings of this study highlight the potential value of incorporating patient-reported clinical information into the routine assessment of individuals presenting with sudden sensorineural hearing loss. Even though audiometric testing still stays the mainstay for diagnosis and for tracking the outcome, the questionnaire here gives a practical way to spot clinical features that might connect with hearing recovery.

From a clinician's perspective, having a sense of these prognostic pointers at the first visit may support better, clearer conversations with patients and their families. Patients frequently seek answers regarding their likelihood of recovery, and the questionnaire may serve as an additional tool to support individualised counselling, helping clinicians provide realistic expectations while acknowledging the inherent uncertainty of recovery in SSNHL.

The questionnaire, in addition, really underlines the value of hearing the patient's experience beyond just the audiogram. Things like how the symptoms started, whether there are accompanying complaints, and elements of the medical history may add meaningful clues

about prognosis. This can support stronger clinician to patient dialogue and more shared decision-making during a time that's commonly tied to uncertainty. Also, since the questionnaire uses information that can be collected quickly in normal clinic visits, it could be used in lower-resource environments where advanced prognostic tools might be hard to obtain.

By helping clinicians recognise patterns tied to more or less favourable outcomes, the questionnaire can aid in prioritising follow-up, monitoring, and patient education. In the end, this study highlights the importance of pairing objective audiological results with patient-reported clinical details so it can support a more customised and patient-centred pathway for managing sudden sensorineural hearing loss.

6.3 Limitations of the Study

Though the current study provides a useful preliminary basis for the clinical utility of the developed checklist, certain methodological and contextual issues need to be addressed when considering its findings. Firstly, due to the sample size being restricted to only 24 patients recruited from a single centre, the statistical power is inadequate to provide meaningful insights and the generalizability of the study to different SSNHL populations. Since a purposive sampling technique has been employed, which is more suited for a tool development study, selection bias can occur. The referral population probably has a skew towards severely affected and late-presenting patients and therefore may not accurately represent the general SSNHL population encountered in community settings or primary care centers.

An in-built review of past medical records was conducted to extract audiometric and clinical data, which introduced the possibility of incomplete data, variability in clinical charting, and recall bias related to symptom reporting. In addition, standardized pre-treatment audiometric assessments obtained at uniform time intervals were not available for all participants, making comparisons of baseline hearing severity potentially susceptible to

inconsistency. Post-treatment audiometric evaluations were also not conducted at standardized follow-up intervals across participants. As hearing recovery in SSNHL may continue for up to three months following treatment initiation, outcome assessment at variable time points may not have fully represented the extent of recovery in all cases.

As the study employed a cross-sectional design, longitudinal assessment of hearing outcomes was not possible. Consequently, changes in hearing trajectory, fluctuating recovery patterns, or delayed deterioration following initial improvement could not be evaluated, as participants were assessed only at a single post-treatment time point.

For the Section B variables relating to risk factors and associated conditions, the relatively small sample size limited the analyses to descriptive statistics alone. Therefore, inferential statistical testing and correlation analyses could not be performed, and these findings should be interpreted as exploratory and hypothesis-generating in nature. Furthermore, objective vestibular function tests, serological investigations, and imaging studies were not consistently available for all participants. As a result, etiologically relevant factors contributing to outcome variation between subgroups and the underlying pathophysiological mechanisms could not be comprehensively evaluated.

All participants completed the checklist in English, and no formal linguistic adaptation or validation was conducted for linguistically diverse populations. In a multilingual context such as India, the absence of translated and culturally adapted versions may limit the broader applicability and accessibility of the tool.Finally, test–retest reliability and inter-rater reliability of the checklist were not assessed. It is an important factor for any tool recommended for multi-site clinical use and requires thorough assessment before it can be presented as a validated clinical tool.

6.3 Future Directions

Large-scale prospective validation studies in multiple centers across heterogeneous patient populations to establish the predictive validity, sensitivity and specificity of the checklist as a prognostic tool are essential. Standardized assessment of test-retest reliability and inter-rater agreement of the checklist is needed. Validated translations and culturally adapted versions in regional Indian languages are required for the practical implementation of this tool in our region. Longitudinal assessment of hearing over a uniform period of three months is crucial for a more accurate assessment of the recovery and potential of hearing rehabilitation with treatment. A modified checklist with relevant biomarkers (vestibular tests, metabolic indicators, inflammatory markers) for better etiological and prognostic classification would also be useful. Pilot studies to ascertain its utility and feasibility of implementation in primary care settings and emergency ENT practices could also be undertaken.

REFERENCES

Alexander, T. H., & Harris, J. P. (2013). Incidence of sudden sensorineural hearing loss. *Otology and Neurotology*, *34*(9), 1586–1589. https://doi.org/10.1097/MAO.0000000000000222

Almeida, B., Rossini, A., De Oliveira Penido, N., Lei Munhoz, M. S., Amaro Bogaz, E., & Curi, R. S. (2017). Sudden Sensorioneural Hearing Loss and Autoimmune Systemic Diseases. *Int Arch Otorhinolaryngol*, *21*, 213–223. https://doi.org/10.1055/s-0036-1586162

Antoniades, E., Psillas, G., Polyzoidis, K., & Patsalas, I. (2022). Patient-Assessed Outcomes following Temporal Bone Fractures. *Diagnostics 2022, Vol. 12, Page 547*, *12*(2), 547. https://doi.org/10.3390/diagnostics12020547

Attia Askar, A., Rashad Ghonim, M., & Kamel Shabana, Y. (2023). Discriminant Analysis of the Prognostic Factors for Hearing Outcomes in Patients with Idiopathic Sudden Sensorineural Hearing Loss. *The Journal of International Advanced Otology*, *19*(3), 162. https://doi.org/10.5152/iao.2023.22893

Atturo, F., Colangeli, R., Bandiera, G., Barbara, M., & Monini, S. (2017). Can unilateral, progressive or sudden hearing loss be immune-mediated in origin? *ACTA OTO-LARYNGOLOGICA*, *137*(8), 1–6. https://doi.org/10.1080/00016489.2017.1286035

Babich, K., & Dunckley, K. T. (2020). Idiopathic Sudden Sensorineural Hearing Loss: Should Otoacoustic Emissions Be Added to the Monitoring Protocol? A Systematic Review. *Applied Sciences 2020, Vol. 10, 10*(1). https://doi.org/10.3390/app10010326

Bang, J., Lee, H., Choi, H., Lee, D., Kim, Y., & Kim, D. K. (2019). Analysis of the relationship between changes in the auditory brainstem response and prognosis in patients with sudden hearing loss. *The Journal of Laryngology & Otology*, *133*(12), 1103–1106. https://doi.org/10.1017/S0022215119002500

Ben-David, J., Luntz, M., Podoshin, L., Sabo, E., & Fradis, M. (2002). Vertigo as a Prognostic Sign in Sudden Sensorineural Hearing Loss. *International Tinnitus Journal*, *8*(2), 127–128.

Binnetoğlu, A., Yumuşakhuylu, A. C., Demir, B., Bağlam, T., Derinsu, U., & Sarı, M. (2019). Association between family history and idiopathic sudden sensorineural hearing loss. *Journal of International Advanced Otology, 11*(1), 30–32. https://doi.org/10.5152/iao.2015.607

Brennan, M. J., Patel, S. J., Shin, D., Nayak, N. A., Chang, Y. S., Huang, S. Y., Kozin, E. D., & Reinshagen, K. L. (2025). Retrospective Review of MR Findings in Sudden Sensorineural Hearing Loss and Associated Clinical Features. *American Journal of Neuroradiology*, *46*(11), 2355–2362. https://doi.org/10.3174/ajnr.A8853

Broughton, S. S., Meyerhoff, W. E., & Cohen, S. B. (2004). Immune-mediated inner ear disease: 10-Year experience. *Seminars in Arthritis and Rheumatism*, *34*(2), 544–548. https://doi.org/10.1016/j.semarthrit.2004.07.001

BUSABA, N. Y., & RAUCH, S. D. (1995). Significance of Auditory Brain Stem Response and Gadolinium-Enhanced Magnetic Resonance Imaging for Idiopathic Sudden Sensorineural Hearing Loss. *Otolaryngology–Head and Neck Surgery*, *113*(3), 271–275. https://doi.org/10.1016/S0194-5998(95)70117-6

Byl, F. M. (1977). Seventy-six cases of presumed sudden hearing loss occurring in 1973: prognosis and incidence. *The Laryngoscope*, *87*(5 Pt 1), 817–825. https://doi.org/10.1002/lary.5540870515

Byl, F. M. (1984). Sudden hearing loss: Eight years' experience and suggested prognostic table. *The Laryngoscope*, *94*(5), 647–661. https://doi.org/10.1288/00005537-198405000-00014

Cadoni, G., Rizzuti, A., Sollazzo, M., Picciotti, P. M., & Galli, J. (2025). Audiogram Shape: Does It Have a Significant Prognostic Role in Idiopathic Sudden Sensorineural Hearing Loss Outcome? *Journal of Personalized Medicine 2025, Vol. 15, 15*(8). https://doi.org/10.3390/jpm15080379

Chae, H., Lee, H. S., Lee, J. H., Kim, M., Park, S. Y., & Seo, Y. J. (2021). Usefulness of stress-related hormones as predictors and prognostic factors for idiopathic sudden sensorineural hearing loss. *Acta Oto-Laryngologica*, *141*(7), 695–701. https://doi.org/10.1080/00016489.2021.1936165

Chandrasekhar, S. S., Tsai Do, B. S., Schwartz, S. R., Bontempo, L. J., Faucett, E. A., Finestone, S. A., Hollingsworth, D. B., Kelley, D. M., Kmucha, S. T., Moonis, G., Poling, G. L., Roberts, J. K., Stachler, R. J., Zeitler, D. M., Corrigan, M. D., Nnacheta, L. C., & Satterfield, L. (2019). Clinical Practice Guideline: Sudden Hearing Loss (Update). *Otolaryngology - Head and Neck Surgery (United States)*, *161*(1_suppl), S1–S45. https://doi.org/10.1177/0194599819859885

Chandrasekhar, S. S., Tsai Do, B. S., Schwartz, S. R., Bontempo, L. J., Faucett, E. A., Finestone, S. A., Hollingsworth, D. B., Kelley, D. M., Kmucha, S. T., Moonis, G., Poling, G. L., Roberts, J. K., Stachler, R. J., Zeitler, D. M., Corrigan, M. D., Nnacheta, L. C., Satterfield, L., & Monjur, T. M. (2019). Clinical Practice Guideline: Sudden Hearing Loss (Update) Executive Summary. *Otolaryngology - Head and Neck Surgery (United States)*, *161*(2), 195–210. https://doi.org/10.1177/0194599819859883

Chau, J. K., Lin, J. R. J., Atashband, S., Irvine, R. A., & Westerberg, B. D. (2010). Systematic review of the evidence for the etiology of adult sudden sensorineural hearing loss. *The Laryngoscope*, *120*(5), 1011–1021. https://doi.org/10.1002/LARY.20873

Chen, C. Y., Halpin, C., & Rauch, S. D. (2003). Oral steroid treatment of sudden sensorineural hearing loss: a ten year retrospective analysis. *Otology & Neurotology :*

Official Publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology, *24*(5), 728–733. https://doi.org/10.1097/00129492-200309000-00006

Chen, J., & Fraser-Kirk, K. (2024). Management of sudden sensorineural hearing loss: A time-critical diagnosis. *Australian Journal of General Practice*, *53*(11 Suppl), S14–S18. https://doi.org/10.31128/AJGP-01-24-7088

Chen, P. J., Chung, C. H., Chien, W. C., & Chen, H. C. (2019). Hormone therapy is not associated with the risk of sudden sensorineural hearing loss in postmenopausal women: a 10-year nationwide population-based study. *Menopause (New York, N.Y.)*, *26*(8), 892–898. https://doi.org/10.1097/GME.0000000000001323

Chen, X., Fu, Y. Y., & Zhang, T. Y. (2019). Role of viral infection in sudden hearing loss. *The Journal of International Medical Research*, *47*(7), 2865. https://doi.org/10.1177/0300060519847860

Chiaramonte, R., Bonfiglio, M., D'Amore, A., Viglianesi, A., Cavallaro, T., & Chiaramonte, I. (2013). Traumatic labyrinthine concussion in a patient with sensorineural hearing loss. *The Neuroradiology Journal*, *26*(1), 52–55. https://doi.org/10.1177/197140091302600109

Conlin, A. E., & Parnes, L. S. (2007). Treatment of Sudden Sensorineural Hearing Loss: II. A Meta-analysis. *Archives of Otolaryngology–Head & Neck Surgery*, *133*(6), 582–586. https://doi.org/10.1001/archotol.133.6.582

Crane, R. A., Camilon, M., Nguyen, S., & Meyer, T. A. (2015). Steroids for treatment of sudden sensorineural hearing loss: A meta-Analysis of randomized controlled trials. *Laryngoscope*, *125*(1), 209–217. https://doi.org/10.1002/lary.24834

Cvorovic, L., Arsovic, N., Radivojevic, N., Soldatovic, I., & Hegemann, S. (2021). Acute onset of tinnitus in patients with sudden deafness. *Noise and Health*, *23*(110), 81–86. https://doi.org/10.4103/nah.NAH_42_20

Edizer, D. T. una, Çelebi, Ö., Hamit, B., Baki, A., & Yiğit, Ö. (2015). Recovery of Idiopathic Sudden Sensorineural Hearing Loss. *The Journal of International Advanced Otology*, *11*(2), 122–126. https://doi.org/10.5152/iao.2015.1227

El-sayed El-sayed Gaafar, A., Ibrahem Ismail, E., & Zaghloul, H. S. (2022). Otoacoustic emissions value in patients with idiopathic sudden sensorineural hearing loss. *Journal of Otology*, *17*(4), 183. https://doi.org/10.1016/j.joto.2022.06.001

Fuente, A., & McPherso, B. (2012). Occupational Chemical-Induced Hearing Loss. *Hearing Loss*. https://doi.org/10.5772/33852

Gäckler, A., Eickelmann, A. K., Brors, D., Dazert, S., Epplen, J. T., & Kunstmann, E. (2010). Positive family history of idiopathic sudden sensorineural hearing loss. *European Archives of Oto-Rhino-Laryngology, 267*, 1843–1848.

Grant, K. J., Parthasarathy, A., Vasilkov, V., Caswell-Midwinter, B., Freitas, M. E., de Gruttola, V., Polley, D. B., Liberman, M. C., & Maison, S. F. (2022). Predicting neural deficits in sensorineural hearing loss from word recognition scores. *Scientific Reports 2022 12:1*, *12*(1), 8929-. https://doi.org/10.1038/s41598-022-13023-5

Gundogan, O., Pinar, E., Imre, A., Ozturkcan, S., Cokmez, O., & Yigiter, A. C. (2013). Therapeutic efficacy of the combination of intratympanic methylprednisolone and oral steroid for idiopathic sudden deafness. *Otolaryngology - Head and Neck Surgery (United States)*, *149*(5), 753–758. https://doi.org/10.1177/0194599813500754

Gupta, K., Varshney, S., Tyagi, A., Kumar, A., & Sood, R. (2021). Epidemiological study of unilateral sensorineural hearing loss in adults. *Indian Journal of Otology*, *27*(2), 96–100. https://doi.org/10.4103/indianjotol.indianjotol_27_21

Hashimoto, S., Billings, P., Harris, J. P., Firestein, G. S., & Keithley, E. M. (2005). Innate immunity contributes to cochlear adaptive immune responses. *Audiology & Neuro-Otology*, *10*(1), 35–43. https://doi.org/10.1159/000082306

He, J., Jin, L., Yao, J., Mahmoudi, A., Pan, Z., Fu, J., Yuan, Q., & Liu, W. (2024). Clinical characteristics of patients diagnosed with bilateral sudden sensorineural hearing loss. *Frontiers in Neurology* , *15*, 1378017. https://doi.org/10.3389/fneur.2024.1378017

Hwang, J. H., Tsai, S. J., Liu, T. C., Chen, Y. C., & Lai, J. T. (2018). Association of Tinnitus and Other Cochlear Disorders With a History of Migraines. *JAMA Otolaryngology--Head & Neck Surgery*, *144*(8), 712–717. https://doi.org/10.1001/jamaoto.2018.0939

Irwin, J. (2006). *Basic Anatomy and Physiology of the Ear.In:Newton Valerie E ,Vallely Pamela E.,editors.Infection and hearing impairment;Whurr Publishers Limited (a subsidiary of John Wiley & Sons Ltd) The Atrium, Southern Gate, Chichester, West Sussex PO19 8SQ, England*. 1–17. https://books.google.com/books/about/Infection_and_Hearing_Impairment.html?id=8n9T8-_v7REC

Jan, T. A., Kozin, E. D., Kanumuri, V. V., Sethi, R. K., & Jung, D. H. (2016). Improvement in word recognition following treatment failure for sudden sensorineural hearing loss. *World Journal of Otorhinolaryngology - Head and Neck Surgery*, *2*(3), 168–174. https://doi.org/10.1016/j.wjorl.2016.06.002

Jeong, J., Lim, H., Lee, K., Hong, C. E., & Choi, H. S. (2019). High Risk of Sudden Sensorineural Hearing Loss in Several Autoimmune Diseases according to a Population-Based National Sample Cohort Study. *Audiology & Neuro-Otology*, *24*(5), 224–230. https://doi.org/10.1159/000502677

Jeong, K. H., Choi, J. W., Shin, J. E., & Kim, C. H. (2016). Abnormal Magnetic Resonance Imaging Findings in Patients With Sudden Sensorineural Hearing Loss: Vestibular Schwannoma as the Most Common Cause of MRI Abnormality. *Medicine*, *95*(17), e3557–e3557. https://doi.org/10.1097/MD.0000000000003557

Jun, H. J., Chang, J., Im, G. J., Kwon, S. Y., Jung, H., & Choi, J. (2012). Analysis of frequency loss as a prognostic factor in idiopathic sensorineural hearing loss. *Acta Oto-Laryngologica*, *132*(6), 590–596. https://doi.org/10.3109/00016489.2011.652306

Kandakure, V. T., Kunjumon, R., Dube, Y., More, M. S., & Garje, S. (2023). A Prospective Study on Post Covid Sudden Onset Sensory Neural Hearing Loss and Its Recovery. *Indian Journal of Otolaryngology and Head and Neck Surgery : Official Publication of the Association of Otolaryngologists of India*, *75*(Suppl 1), 451–457. https://doi.org/10.1007/s12070-023-03529-3

Kim, J. Y., Hong, J. Y., & Kim, D. K. (2018). Association of Sudden Sensorineural Hearing Loss With Risk of Cardiocerebrovascular Disease: A Study Using Data From the Korea National Health Insurance Service. *JAMA Otolaryngology–Head & Neck Surgery*, *144*(2), 129–135. https://doi.org/10.1001/jamaoto.2017.2569

Kim, S. H., Kim, S. J., Im, H., Kim, T. H., Song, J. J., & Chae, S. W. (2017). A Trend in Sudden Sensorineural Hearing Loss: Data from a Population-Based Study. *Audiology & Neuro-Otology*, *22*(6), 311–316. https://doi.org/10.1159/000485313

Kim, S. Y., Lim, J. S., Sim, S., & Choi, H. G. (2018). Sudden sensorineural hearing loss predicts ischemic stroke: A longitudinal follow-up study. *Otology and Neurotology*, *39*(8), 964–969. https://doi.org/10.1097/MAO.0000000000001902

Kim, S. Y., Song, Y. S., Wee, J. H., Min, C., Yoo, D. M., & Choi, H. G. (2020). Association between SSNHL and Thyroid Diseases. *International Journal of Environmental Research and Public Health 2020, Vol. 17, Page 8419*, *17*(22), 8419. https://doi.org/10.3390/ijerph17228419

Klein, L., Handzel, O., Shilo, S., Oron, Y., Abu Eta, R., Muhanna, N., & Ungar, O. J. (2023). *Is sudden sensorineural hearing loss an otologic emergency? Evidence-based cutoff for optimal treatment initiation for sudden unilateral sensorineural hearing loss: A case series and meta-analyses*. *Otology & Neurotology, 44*(3), 216–222. https://doi.org/10.1097/MAO.0000000000003808

Konadath, S., Chatni, S., Lakshmi, M. S., & Saini, J. K. (2017). Prevalence of communication disorders in a group of islands in India. *Clinical Epidemiology and Global Health*, *5*(2), 79–86. https://doi.org/10.1016/j.cegh.2016.08.003

Kuhn, M., Heman-Ackah E, S., A Shaik, J., & C Roehm, P. (2011). Sudden sensorineural hearing loss: a review of diagnosis, treatment, and prognosis. *Trends in Amplification*, *15*(3), 207–216. https://doi.org/10.1177/1084713811408349

Kuo, C. Y., Chung, C. H., Wang, C. H., Chien, W. C., & Chen, H. C. (2019). Increased incidence in hospitalised patients with sudden sensorineural hearing loss: a 14-year nationwide population-based study. *International Journal of Audiology*, *58*(11), 769–773. https://doi.org/10.1080/14992027.2019.1627009

Kurioka, T., Sano, H., Furuki, S., & Yamashita, T. (2020). Long-term administration of vitamin B12 and adenosine triphosphate for idiopathic sudden sensorineural hearing loss: A retrospective study. *PeerJ*, *8*, e10406. https://doi.org/10.7717/peerj.10406

Kwak, C., Seo, Y. J., Yoon, C. Y., Lee, J. H., & Han, W. (2022). The value of having an initial word recognition score for a precise prognosis of idiopathic sudden sensorineural hearing loss. *Auris Nasus Larynx*, *49*(4), 554–563. https://doi.org/10.1016/j.anl.2021.10.005

Lee, H., Sohn, S. I., Jung, D. K., Cho, Y. W., Lim, J. G., Yi, S. D., Lee, S. R., Sohn, C. H., & Baloh, R. W. (2002). Sudden deafness and anterior inferior cerebellar artery infarction. *Stroke*, *33*(12), 2807–2812. https://doi.org/10.1161/01.STR.0000038692.17290.24

Li, J., & Ding, L. (2020). Effectiveness of Steroid Treatment for Sudden Sensorineural Hearing Loss: A Meta-analysis of Randomized Controlled Trials. *Annals of Pharmacotherapy*, *54*(10), 949–957. https://doi.org/10.1177/1060028020908067

Li, X., Cao, Z., Chen, F., Yang, D., & Zhao, F. (2023). Sensorineural Hearing Loss in Autoimmune Diseases: A Systematic Review and Meta-analysis. *The Journal of International Advanced Otology*, *19*(4), 277–282. https://doi.org/10.5152/iao.2023.22991

Lim, E. J., Shin, J. H., Kim, T. H., Park, J. H., & Kim, S. H. (2019). Correlation between Auditory Brainstem Response Characteristics and Hearing Recovery in Sudden Deafness. *Korean Journal of Otorhinolaryngology-Head and Neck Surgery*, *62*(8), 435–441. https://doi.org/10.3342/kjorl-hns.2018.00843

Lin, C.-F., Lee, K.-J., Yu, S.-S., & Lin, Y.-S. (2016). *Effect of Comorbid Diabetes and Hypercholesterolemia on the Prognosis of Idiopathic Sudden Sensorineural Hearing Loss*. *126*, 142–149. https://doi.org/10.1002/lary.25333

Lin, H. C., Chou, Y. C., Wang, C. H., Hung, L. W., Shih, C. P., Kang, B. H., Yeh, W. Y., & Chen, H. C. (2017). Correlation between auditory brainstem response and hearing prognosis in idiopathic sudden sensorineural hearing loss patients. *Auris Nasus Larynx*, *44*(6), 678–684. https://doi.org/10.1016/j.anl.2017.01.004

Lin, W., Xiong, G., Yan, K., Yu, W., Xie, X., Xiang, Z., Wu, J., Ge, Y., & Wang, Y. (2022). Clinical Features and Influencing Factors for the Prognosis of Patients With Sudden Deafness. *Frontiers in Neurology*, *13*, 905069. https://doi.org/10.3389/fneur.2022.905069

Mandavia, R., Joshi, N., Hannink, G., Ahmed, M. N., Parmar, D., Di Bonaventura, S., Gomes, P., Iqbal, I., Lyles, J., Schilder, A. G. M., & Mehta, N. (2024). A Prognostic Model to Predict Hearing Recovery in Patients With Idiopathic Sudden Onset Sensorineural Hearing Loss. *JAMA Otolaryngology–Head & Neck Surgery*, *150*(10), 896–906. https://doi.org/10.1001/jamaoto.2024.2598

Mathews, S., Dalawayi, S., Gangwar, V., & Vadivu, A. (2020). Evaluation and management of sudden sensorineural hearing loss in Indian naval scenario - A prospective cohort study. *Journal of Marine Medical Society*, *22*(2), 128. https://doi.org/10.4103/jmms.jmms_17_20

Mattox, D. E., & Simmons, F. B. (1977). Natural history of sudden sensorineural hearing loss. *The Annals of Otology, Rhinology, and Laryngology*, *86*(4 Pt 1), 463–480. https://doi.org/10.1177/000348947708600406

McCabe, B. F. (1979). Autoimmune sensorineural hearing loss. *The Annals of Otology, Rhinology, and Laryngology*, *88*(5 Pt 1), 585–589. https://doi.org/10.1177/000348947908800501

Merchant, S. N., Durand, M. L., & Adams, J. C. (2008). Sudden Deafness: Is It Viral? *ORL; Journal for Oto-Rhino-Laryngology and Its Related Specialties*, *70*(1), 52. https://doi.org/10.1159/000111048

Mirian, C., & Ovesen, T. (2020). Intratympanic vs Systemic Corticosteroids in First-line Treatment of Idiopathic Sudden Sensorineural Hearing Loss: A Systematic Review and Meta-analysis. *JAMA Otolaryngology–Head & Neck Surgery*, *146*(5), 421–428. https://doi.org/10.1001/jamaoto.2020.0047

Mokhatrish, M., Baek, W., Nam, G. S., & Cho, S. Il. (2023). Tinnitus characteristics in patients with idiopathic sudden sensorineural hearing loss and acute tinnitus. *Laryngoscope Investigative Otolaryngology*, *8*(5), 1384. https://doi.org/10.1002/lio2.1137

Moscicki, R. A., San Martin, J. E., Quintero, C. H., Rauch, S. D., Nadol, J. B., & Bloch, K. J. (1994). Serum Antibody to Inner Ear Proteins in Patients With Progressive Hearing Loss: Correlation With Disease Activity and Response to Corticosteroid Treatment. *JAMA: The Journal of the American Medical Association*, *272*(8), 611–616. https://doi.org/10.1001/jama.1994.03520080053043

Nakashima, T., Sato, H., Gyo, K., Hato, N., Yoshida, T., Shimono, M., Teranishi, M., Sone, M., Fukunaga, Y., Kobashi, G., Takahashi, K., Matsui, S., & Ogawa, K. (2014). Idiopathic sudden sensorineural hearing loss in Japan. *Acta Oto-Laryngologica*, *134*(11), 1158. https://doi.org/10.3109/00016489.2014.919406

Nogueira-Neto, F. B., Gallardo, F. P., De Barros Suzuki, F. A., & Penido, N. D. O. (2016). Prognostic and Evolutive Factors of Tinnitus Triggered by Sudden Sensorineural Hearing Loss. *Otology & Neurotology : Official Publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*, *37*(6), 627–633. https://doi.org/10.1097/MAO.0000000000001049

Nosrati-Zarenoe, R., Arlinger, S., & Hultcrantz, E. (2007). Idiopathic sudden sensorineural hearing loss: results drawn from the Swedish national database. *Acta Oto-Laryngologica*, *127*(11), 1168–1175. https://doi.org/10.1080/00016480701242477

Okada, M., Parthasarathy, A., Welling, D. B., Liberman, M. C., & Maison, S. F. (2021). Idiopathic Sudden Sensorineural Hearing Loss: Speech Intelligibility Deficits Following Threshold Recovery. *Ear and Hearing*, *42*(4), 782–792. https://doi.org/10.1097/AUD.0000000000000987

Olex-Zarychta, D. (2020). Hyperbaric Oxygenation as Adjunctive Therapy in the Treatment of Sudden Sensorineural Hearing Loss. *International Journal of Molecular Sciences 2020, Vol. 21, 21*(22), 1–14. https://doi.org/10.3390/ijms21228588

Oulghoul, O., Labani, L., Chehbouni, M., Benhoummad, O., Rochdi, Y., & Raji, A. (2021). Management of Sensorineural Deafness in Temporal Bone Trauma: A Report of 23 Cases. *Global Journal of Otolaryngology*, *23*(5), 1–5. https://doi.org/10.19080/gjo.2021.23.556123

Padmakumar, V., Ramesh Kumar, E., & Ramakrishnan, V. R. (2019). A Prospective Study on Temporal Bone Involvement in Polytrauma Patients and the Effect of Early Diagnosis on Hearing Loss. *Indian Journal of Otolaryngology and Head & Neck Surgery*, *72*(1), 30. https://doi.org/10.1007/s12070-019-01723-w

Park, H., Lee, Y., Park, M., Kim, J., Na, B., & Shin, J. (2010). Short-term changes of hearing and distortion product otoacoustic emissions in sudden sensorineural hearing loss. *Otology and Neurotology*, *31*(6), 862–866. https://doi.org/10.1097/MAO.0b013e3181e8fb47

Park, H. M., Jung, S. W., & Rhee, C. K. (2001). Vestibular diagnosis as prognostic indicator in sudden hearing loss with vertigo. *Acta Oto-Laryngologica, Supplement*, (545), 80–83. https://doi.org/10.1080/oto.121.533.80.83

Plontke, S. K., Girndt, M., Meisner, C., Fischer, I., Boselt, I., Lohler, J., Ludwig-Kraus, B., Richter, M., Steighardt, J., Reuter, B., Bottcher, C., Langer, J., Pethe, W., Seiwerth, I., Jovanovic, N., Großmann, W., Kienle-Gogolok, A., Boehm, A., Neudert, M., … Rahne, T. (2024). High-Dose Glucocorticoids for the Treatment of Sudden Hearing Loss. *NEJM Evidence*, *3*(1). https://doi.org/10.1056/EVIDoa2300172

Pogson, J. M., Taylor, R. L., Young, A. S., McGarvie, L. A., Flanagan, S., Halmagyi, G. M., & Welgampola, M. S. (2016). Vertigo with sudden hearing loss: audio-vestibular characteristics. *Journal of Neurology*, *263*(10), 2086–2096. https://doi.org/10.1007/s00415-016-8214-0

Portmann, M., Dauman, R., & Aran, J. M. (1985). Audiometric and electrophysiological correlations in sudden deafness. *Acta Oto-Laryngologica*, *99*(3–4), 363–368. https://doi.org/10.3109/00016488509108924

Psillas, G. (2023). Hyperbaric Oxygen Therapy in the Treatment of Sudden Sensorineural Hearing Loss. *Journal of Clinical Medicine*, *12*(4), 1515–1515. https://doi.org/10.3390/jcm12041515

Psillas, G., Rizou, A., Rachovitsas, D., Tsiropoulos, G., & Constantinidis, J. (2019). Hearing outcome of low-tone compared to high-tone sudden sensorineural hearing loss. *International Archives of Otorhinolaryngology*, *23*(1), 65–69. https://doi.org/10.1055/s-0038-1657789

Quaranta, N., De Ceglie, V., & D'Elia, A. (2016). Endothelial Dysfunction in Idiopathic Sudden Sensorineural Hearing Loss: A Review. *Audiology Research*, *6*(1), 151. https://doi.org/10.4081/audiores.2016.151

Rauch, S. D. (2008). Idiopathic Sudden Sensorineural Hearing Loss. *New England Journal of Medicine*, *359*(8), 833–840. https://doi.org/10.1056/NEJMcp0802129

Rybak, L. P., & Ramkumar, V. (2007). Ototoxicity. *Kidney International*, *72*(8), 931–935. https://doi.org/10.1038/sj.ki.5002434

Rybak, M., Lomaestro, B., Rotschafer, J. C., Moellering, R., Craig, W., Billeter, M., Dalovisio, J. R., Levine, D. P., & Reilly, C. (2009). Therapeutic monitoring of vancomycin in adult patients: A consensus review of the American Society of Health-System Pharmacists, the Infectious Diseases Society of America, and the Society of Infectious Diseases Pharmacists. *American Journal of Health-System Pharmacy*, *66*(1), 82–98. https://doi.org/10.2146/ajhp080434

Saba, E. S., Swisher, A. R., Ansari, G. N., & Rivero, A. (2023). Cardiovascular Risk Factors in Patients With Sudden Sensorineural Hearing Loss: A Systematic Review and Meta-analysis. *Otolaryngology - Head and Neck Surgery (United States)*, *168*(5), 907–921. https://doi.org/10.1002/ohn.163

Sajid, T., Sajid, F., Sajid, Z., Naqvi, S. R. Q., Ali, S. M., & Shah, M. I. (2021). MAGNITUDE OF DIABETES MELLITUS IN PATIENTS OF SUDDEN SENSORINEURAL HEARING LOSS AND ITS EFFECT ON CORTICOSTEROID THERAPY. *PAFMJ*, *71*, S626–S629. https://doi.org/10.51253/PAFMJ.V71ISUPPL-3.7109

Sakashita, T., Minowa, Y., Hachikawa, K., Kubo, T., & Nakai, Y. (1991). Evoked Otoacoustic Emissions from Ears with Idiopathic Sudden Deafness. *Acta Oto-Laryngologica*, *111*(S486), 66–72. https://doi.org/10.3109/00016489109134984

Schreiber, B. E., Agrup, C., Haskard, D. O., & Luxon, L. M. (2010). Sudden sensorineural hearing loss. *The Lancet*, *375*(9721), 1203–1211. https://doi.org/10.1016/S0140-6736(09)62071-7

Selvanayagam, D. L., & Mathialagan, A. (2021). A review on hearing loss caused by occupational hazard and trauma. *International Journal Of Community Medicine And Public Health*, *8*(8), 4116. https://doi.org/10.18203/2394-6040.ijcmph20213051

Seo, H. W., Chung, J. H., Byun, H., Jeong, J. H., & Lee, S. H. (2020). Effect of Diabetes on the Prognosis of Sudden Sensorineural Hearing Loss: Propensity Score Matching

Analysis. *Otolaryngology - Head and Neck Surgery (United States)*, *162*(3), 346–352. https://doi.org/10.1177/0194599820901359

Shupak, A., Zeidan, R., & Shemesh, R. (2014). Otoacoustic emissions in the prediction of sudden sensorineural hearing loss outcome. *Otology and Neurotology*, *35*(10), 1691–1697. https://doi.org/10.1097/MAO.0000000000000627

Spear, S. A., & Schwartz, S. R. (2011). Intratympanic steroids for sudden sensorineural hearing loss: A systematic review. *Otolaryngology - Head and Neck Surgery*, *145*(4), 534–543. https://doi.org/10.1177/0194599811419466

Stachler, R. J., Chandrasekhar, S. S., Archer, S. M., Rosenfeld, R. M., Schwartz, S. R., Barrs, D. M., Brown, S. R., Fife, T. D., Ford, P., Ganiats, T. G., Hollingsworth, D. B., Lewandowski, C. A., Montano, J. J., Saunders, J. E., Tucci, D. L., Valente, M., Warren, B. E., Yaremchuk, K. L., & Robertson, P. J. (2012). Clinical practice guideline: Sudden hearing loss. *Otolaryngology - Head and Neck Surgery*, *146*(SUPPL.3). https://doi.org/10.1177/0194599812436449

Sun, X. M., Zhuang, S. M., Xiao, Z. W., Luo, J. Q., Long, Z., Lan, L. C., Zhang, H. Q., & Zhang, G. P. (2022). Autoimmune thyroiditis in patients with sudden sensorineural hearing loss. *Laryngoscope Investigative Otolaryngology*, *7*(2), 571–577. https://doi.org/10.1002/lio2.755

Tripathi, P., & Deshmukh, P. (2022). Sudden Sensorineural Hearing Loss: A Review. *Cureus*. https://doi.org/10.7759/cureus.29458

Tsai, Y. Te, Chang, I. J., Hsu, C. M., Yang, Y. H., Liu, C. Y., Tsai, M. S., Chang, G. H., Lee, Y. C., Huang, E. I., Lin, M. H., & Luan, C. W. (2020). Association between Sudden Sensorineural Hearing Loss and Preexisting Thyroid Diseases: A Nationwide Case-Control Study in Taiwan. *International Journal of Environmental Research and Public Health*, *17*(3). https://doi.org/10.3390/ijerph17030834

Tseng, H. W., Hsu, C. Y., Chen, C. H., Chu, Y. C., & Liao, W. H. (2025). A novel audiogram model for predicting the prognosis of sudden sensorineural hearing loss. *Journal of the Chinese Medical Association*. https://doi.org/10.1097/JCMA.0000000000001299

Tsuzuki, N., & Wasano, K. (2024). Idiopathic sudden sensorineural hearing loss: A review focused on the contribution of vascular pathologies. *Auris, Nasus, Larynx*, *51*(4), 747–754. https://doi.org/10.1016/j.anl.2024.05.009

Waissbluth, S., Sepúlveda, V., & Urzúa, P. (2022). Sudden sensorineural hearing loss: Recovery rates according to audiometric patterns. *Acta Otorrinolaringológica Española*, *73*(6), 346–355. https://doi.org/10.1016/j.otorri.2021.07.005

Wang, S., Wang, K., Zhang, J., Sun, D., Wang, S., Wang, K., Zhang, J., & Sun, D. (2024). The Relationship between Sudden Sensorineural Hearing Loss and Serum Sex Hormone

Levels in Perimenopausal Women. *Clinical and Experimental Obstetrics & Gynecology 2024 51 (10)*, *51*(10), 219. https://doi.org/10.31083/j.ceog5110219

Wang, C. Te, Huang, T. W., Kuo, S. W., & Cheng, P. W. (2009). Correlation between audiovestibular function tests and hearing outcomes in severe to profound sudden sensorineural hearing loss. *Ear and Hearing*, *30*(1), 110–114. https://doi.org/10.1097/AUD.0b013e318192655e

Wang, Y., Wang, L., Jing, Y., Yu, L., & Ye, F. (2020). Association Between Hearing Characteristics/Prognosis and Vestibular Function in Sudden Sensorineural Hearing Loss With Vertigo. *Frontiers in Neurology*, *11*, 579757. https://doi.org/10.3389/fneur.2020.579757

Watanabe, T., & Suzuki, M. (2018). Analysis of the audiogram shape in patients with idiopathic sudden sensorineural hearing loss using a cluster analysis. *Ear, Nose and Throat Journal*, *97*(7). https://doi.org/10.1177/014556131809700706

Wei, W., Sun, Y., You, Y., & Yan, K. (2026). Association between tinnitus and hearing recovery in sudden sensorineural hearing loss: A meta-analysis. *Otology & Neurotology, 47*(2), e151–e157. https://doi.org/10.1097/MAO.0000000000004709

Wilson, W. R., Byl, F. M., & Laird, N. (1980). The efficacy of steroids in the treatment of idiopathic sudden hearing loss. A double-blind clinical study. *Archives of Otolaryngology (Chicago, Ill. : 1960)*, *106*(12), 772–776. https://doi.org/10.1001/archotol.1980.00790360050013

Xenellis, J., Karapatsas, I., Papadimitriou, N., Nikolopoulos, T., Maragoudakis, P., Tzagkaroulakis, M., & Ferekidis, E. (2006). Idiopathic sudden sensorineural hearing loss: prognostic factors. *The Journal of Laryngology & Otology*, *120*(9), 718–724. https://doi.org/10.1017/S0022215106002362

Xu, R. Z., Guo, R. C., Wijesinghe, P., Joshua, T. G., Ayub, A., Lee, M., & Nunez, D. A. (2024). A Retrospective Comparative Chart Review of Hearing Recovery in Neural and Sensory Type Sudden Sensorineural Hearing Loss Patients. *The Journal of Laryngology & Otology*, *138*(11), 1090–1094. https://doi.org/10.1017/S0022215124000781

Yamada, S., Kita, J., Shinmura, D., Nakamura, Y., Sahara, S., Misawa, K., & Nakanishi, H. (2022). Update on Findings about Sudden Sensorineural Hearing Loss and Insight into Its Pathogenesis. *Journal of Clinical Medicine*, *11*(21), 6387. https://doi.org/10.3390/jcm11216387

Yang, J., Liu, Y., Yu, L., & Duan, M. (2024). Editorial: Sudden deafness. *Frontiers in Neurology*, *15*, 1520018. https://doi.org/10.3389/FNEUR.2024.1520018/TEXT

Yoon, C. Y., Kong, T. H., Lee, J., Seo, Y. J., & Ahn, J. J. (2023). Epidemiology of idiopathic sudden sensorineural hearing loss in the era of big data. *European Archives of Oto-Rhino-Laryngology : Official Journal of the European Federation of Oto-Rhino-*

Laryngological Societies (EUFOS) : Affiliated with the German Society for Oto-Rhino-Laryngology - Head and Neck Surgery, *280*(5), 2181–2190. https://doi.org/10.1007/s00405-022-07693-0

Yu, H., & Li, H. (2018). Association of Vertigo With Hearing Outcomes in Patients With Sudden Sensorineural Hearing Loss: A Systematic Review and Meta-analysis. *JAMA Otolaryngology–Head & Neck Surgery*, *144*(8), 677–683. https://doi.org/10.1001/jamaoto.2018.0648

Zhao, Y., Yu, C., Sun, H., Xie, F., Shen, J., Li, X., Song, X., Luo, W., He, J., & Han, Z. (2025). Exploring the causal link between serum 25-hydroxyvitamin D concentrations and idiopathic sudden sensorineural hearing loss: Insights gained from a Mendelian randomization study involving two independent samples. *PLOS ONE*, *20*(5), e0322898. https://doi.org/10.1371/journal.pone.0322898

Zheng, H., Liu, M., Fu, H., & Zhao, L. (2023). Serum fat-soluble vitamin levels may be related to sudden sensorineural hearing loss. *Acta Oto-Laryngologica*, *143*(7), 576–581. https://doi.org/10.1080/00016489.2023.2220365

Zheng, Z., Shen, Y., Xia, L., Xiao, L., Sun, Y., Wang, H., Chen, Z., Wu, Y., Shi, H., He, J., Feng, Y., & Yin, S. (2021). Thyroid-Related Hormone Levels in Clinical Patients With Moderately Severe-to-Profound Sudden Sensorineural Hearing Loss: A Prospective Study. *Frontiers in Neurology*, *12*. https://doi.org/10.3389/fneur.2021.753270

Zhu, Y., Bing, D., Wang, D., Zhao, L., Yin, Z., Zhang, Q., Guan, J., & Wang, Q. (2020). Analysis of thyroid dysfunction in patients with sudden sensorineural hearing loss. *World Journal of Otorhinolaryngology - Head and Neck Surgery*, *7*(1), 1–8. https://doi.org/10.1016/j.wjorl.2019.02.002

Zou, T., Xu, J., Lu, H., & Yu, M. (2023). The Relationship between the Characteristics of Tinnitus and the Hearing Curative Effect of Sudden Deafness. *Audiology and Neurotology*, *28*(4), 239–245. https://doi.org/10.1159/000528444

Appendix I

ALL INDIA INSTITUTE OF SPEECH AND HEARING, MYSURU – 570 006

Master's Dissertation in Audiology

Content Validation Form

Development and Validation of a Prognostic and Assessment Checklist for Sudden Sensorineural Hearing Loss (SSNHL)

Dear Sir/Madam,

I am Shruti Darshana Mishra, a student of the 2nd year M.Sc. Audiology programme at the All India Institute of Speech and Hearing (AIISH), Mysuru. I am currently conducting a dissertation under the guidance of Dr. N. M. Mamatha, Assistant Professor in Audiology, Department of Audiology, AIISH. The dissertation is on the topic "Development and Preliminary Validation of a Prognostic and Assessment Checklist for Sudden Sensorineural Hearing Loss (SSNHL). "

As part of this work, I have developed a checklist that includes items related to clinical assessment as well as prognostic evaluation of SSNHL. I humbly request your expert evaluation of the content validity of this checklist. Your feedback and suggestions for modification will be highly appreciated and, will help a lot in the refinement of the instrument. The checklist items are presented below, in the same format provided. Kindly review each item and indicate your responses under the respective evaluation parameters. If you have any additional comments, or suggestions for modification, addition, or removal, you may note them in the space given at the end of the form.

Thank you for your time and kind expert contribution.

Sl. No.	Questions	Response Options	Relevant		Repetitive	Complex	Ambiguous	Clarity		Accuracy		
			Yes	No				Yes	No	Yes	Optional	No
Section A: Core Prognostic Indicators												
1	When did you first notice signs of hearing loss? / When did you first seek medical	< 3 days / 7–14 days / >14 days										

Sl. No.	Questions	Response Options	Relevant		Repetitive	Complex	Ambiguous	Clarity		Accuracy		
			Yes	No				Yes	No	Yes	Optional	No
	consultation after noticing the hearing loss?											
2	What is your age?	<40 yrs / 40–60 yrs / >60 yrs										
3	Is the hearing loss progressive or static (i.e., has it been getting worse since it first started)?	Yes / No										
4	Is the hearing loss present in one ear or both ears?	One ear / Both ears										
5	Did you experience vertigo/imbalance/nausea with or after the hearing loss?	Yes / No										
Associated Features												
6	Is there any onset of a ringing sensation in your ear since the hearing loss began, or was it present before?	Yes / No										
7	Is there a sense of fullness or pressure in the affected ear?	Yes / No										
8	Do you have a family history of hearing loss?	Yes / No										
Section B1: Respiratory / Viral Infections												
9	Have you had a cold, sore throat, cough, or fever in the last 4	Yes / No										

Sl. No.	Questions	Response Options	Relevant		Repetitive	Complex	Ambiguous	Clarity		Accuracy		
			Yes	No				Yes	No	Yes	Optional	No
	weeks before your hearing loss?											
10	Any previous episodes of fever with rashes (mainly around the mouth and ear canal) recently?	Yes / No										
11	Were you diagnosed with any haemorrhagic fever recently (e.g., Dengue, Lassa fever, etc.)?	Yes / No										
12	Any history of blood transfusion?	Yes / No										
13	Any lingering fatigue or weakness since the infection?	Yes / No										
14	Were you diagnosed with COVID-19? If yes, when?	Yes / No; if yes, specify date										
Section B1: Trauma												
15	Did you have any instances of sudden loud noise exposure?	Yes / No										
16	History of ear surgery previously?	Yes / No										
17	Was the hearing loss preceded by severe coughing, sneezing, straining, or nose-blowing?	Yes / No										
18	Any instance of head injury (especially	Yes / No										

Sl. No.	Questions	Response Options	Relevant		Repetitive	Complex	Ambiguous	Clarity		Accuracy		
			Yes	No				Yes	No	Yes	Optional	No
	involving the petrous area)?											
Section B1: Autoimmune												
19	Do you feel unexplained extreme tiredness?	Yes / No										
20	Do you have frequent unexplained pains or swelling in joints (past 3 months or more)?	Yes / No										
21	Have you had hearing loss episodes affecting both ears (simultaneously or alternating)?	Yes / No										
22	Any vision-related changes (around 3 months)? (e.g., blurring, double vision, partial loss etc.)	Yes / No										
Section B1: Ototoxic												
23	Have you taken medication for infections (TB, pneumonia, kidney infections), cancer, or long-term pain / arthritis?	Yes / No										
24	Do you work around chemicals or fumes?	Yes / No										
Section B1: Vascular												
25	Do you have any cardiac problem?	Yes / No										

Sl. No.	Questions	Response Options	Relevant		Repetitive	Complex	Ambiguous	Clarity		Accuracy		
			Yes	No				Yes	No	Yes	Optional	No
26	Do you have high cholesterol?	Yes / No										
27	Do you have hypertension (high blood pressure)?	Yes / No										
28	Do you smoke or use tobacco?	Yes / No										
29	Any recent stress, dehydration, or lack of sleep before the hearing loss?	Yes / No										
Section B1: Neurological												
30	Do you have a history of migraine or severe recurring headaches?	Yes / No										
31	Before or during your hearing loss, did you notice any numbness, tingling, or weakness on one side of the face / body?	Yes / No										
32	Any history of fainting, blackout, or sudden weakness?	Yes / No										
Section B1: Metabolic												
33	Have you been diagnosed with diabetes or pre-diabetes?	Yes / No										
34	Do you have thyroid issues?	Yes / No										
35	Do you have irregular periods or any diagnosed hormonal condition (PCOS,	Yes / No										

Sl. No.	Questions	Response Options	Relevant		Repetitive	Complex	Ambiguous	Clarity		Accuracy		
			Yes	No				Yes	No	Yes	Optional	No
	endometriosis, infertility treatment, menopause symptoms)?											
Section B2: Red Flag / Atypical Clinical History												
36	Gradual / fluctuating hearing loss or ringing in one ear before the sudden episode?	Yes / No										
37	Long-standing imbalance or veering to one side?	Yes / No										
38	Any facial numbness or weakness?	Yes / No										
39	Do you have long-standing headaches or pressure in the back of your head?	Yes / No										

Note: All items require a response under each evaluation parameter. For "Relevant" and "Grammatically Correct," please mark Yes or No. For "Accuracy," mark the most appropriate option: Useful / Optional / Not Useful. For "Repetitive," "Complex," and "Ambiguous/Confusing," please mark only if the issue applies to that item. Response options for Section A items vary by item as indicated; all Section B items have Yes/No responses unless otherwise specified.

General Suggestions and Comments:

Expert's Name: Designation:

Date:

Signature:

Appendix II

Assessment and Prognostic indicator checklist for Sudden sensorineural Hearing loss (SSNHL)

All India Institute of Speech and Hearing,

Manasagangothri, Mysore- 570006

Name: Case number:

Age/Gender: Contact No.:

Occupation:

Instructions:

This questionnaire is designed for structured clinical evaluation of patients presenting with Sudden Sensorineural Hearing Loss (SSNHL). It assists in estimating prognostic risk and identifying possible etiological contributors through a structured weighted scoring framework.

The form begins with demographic details (Name, Age, Gender, Address, Case Number), which are recorded for identification purposes only and are not included in scoring.

The questionnaire consists of two parts:
***Part A** includes major prognostic indicators.*
***Part B** includes clinical and etiological factors and is divided into two subsections. B1 (Risk-modifying factors) includes variables that influence prognosis and are scored. B2 (Etiological factors) includes identifiable causes or triggers, recorded as Yes/No, and not included in the prognostic score.*

All questions are closed-ended. Each response carries a predefined weighted score ranging from 0 to 3, depending on clinical significance. Select the appropriate response and record the corresponding score. Only one response should be selected per question unless otherwise specified. Below is an example for clarification.

Sl. No.	Questions	Response	Score
1	Did you experience vertigo/imbalance/nausea with hearing loss?	☐ Yes ☐ No	3 / 0

If the patient reports vertigo, assign **3**. If absent, assign **0**.

*Sum all item scores in **Part A** to obtain the prognostic classification score (Good / Moderate / Poor). Record **Part B** item scores individually for descriptive risk profiling. Part B scores are not included in the prognostic classification score.*

Section A: Core Prognostic Indicators

Sl. No.	Questions	Response	Score
1.	What was the time interval (in days) between the onset of hearing loss and initial medical consultation?	☐ < 3 days ☐ 3–7 days ☐ 7–14 days ☐ > 14 day	1 2 3 4
2.	What is the age category?	☐ < 40 years ☐ 40–60years ☐ > 60 years	1 2 3
3.	*After* the initial hearing loss, did your hearing continue to worsen?	☐ Yes ☐ No	1/0
4.	At the time of onset, was the hearing loss present in one ear or both ears?	☐ One ear ☐ Both ears	1 2
5.	Did you experience vertigo/imbalance/nausea *with* hearing loss?	☐ Yes ☐ No	1/0
6.	Did you undergo hearing testing for this episode of sudden hearing loss? If yes, what was the severity of hearing loss in the affected ear at the time of initial testing? (If both ears are involved, consider the worst ear.)	☐ Mild–Moderate ☐ Moderately severe ☐ Severe-Profound	1 2 3

	If no testing was done, how severe did your hearing loss feel at its onset?	☐ Mild (difficulty hearing soft sounds) ☐ Moderate (difficulty understanding speech) ☐ Severe (unable to hear most sounds)	1 2 3
7.	What was the audiogram configuration observed in the affected ear during the initial audiological evaluation?	☐ Ascending/U-shaped ☐ Descending /Flat ☐ Profound / Total loss	1 2 3
		Total score	

Section B: Clinical & Etiological factors

Sl. No.	Questions	Response	Score
Sub Section B1: Risk Modifying Factors			
Associated Features			
1.	At the time the hearing loss began, did you experience ringing in the same ear?	☐ Yes ☐ No	1 / 0
2.	Did you experience a feeling of fullness/pressure in the affected ear?	☐ Yes ☐ No	1 / 0
3.	Is there a history of congenital or unexplained early-onset hearing loss in your immediate family (parents or siblings)?	☐ Yes ☐ No	1 / 0
Viral/other Infections			

4	Did you experience any recent viral illness (Mumps, cold, sore throat, runny nose or flu) within 4 weeks *before* hearing loss? Specify -	☐ Yes ☐ No	1 / 0
5	Did you develop a fever with rash or painful blisters around the ear or face within 4 weeks before hearing loss?	☐ Yes ☐ No	1 / 0
6	Were you diagnosed with dengue, chikungunya, or any other severe viral fever requiring medical treatment or hospitalisation in the past *3 months*?	☐ Yes ☐ No	1 / 0
7	Any history of blood transfusion within the past 6 months *before* hearing loss?	☐ Yes ☐ No	1 / 0
8.	Did you receive a confirmed diagnosis of COVID-19 within 4 weeks *before* the onset of hearing loss?	☐ Yes ☐ No	1 / 0
Autoimmune			
9.	Did you experience severe fatigue *without fever* or infection within 4 weeks *before* hearing loss?	☐ Yes ☐ No	1 / 0
10.	Do you frequently have unexplained pains or swelling in joints (hands, wrists, knees, ankles) for ≥3 months?	☐ Yes ☐ No	1 / 0
11.	Did you experience any new vision changes (such as blurred or double vision) at the time your hearing loss began?	☐ Yes ☐ No	1 / 0
Toxic			
12.	Do you have a history of prolonged use of medications for kidney dysfunction, serious infections (e.g., tuberculosis), cancer, or chronic pain/arthritis *before* hearing loss?	☐ Yes ☐ No	1/ 0
13.	Does your occupation involve regular exposure to chemicals or fumes (e.g., paints, fuels/petrols, pesticides, factory smoke, etc.)?	☐ Yes ☐ No	1 / 0
Vascular			

14.	Do you have a history of cardiovascular disease (e.g., ischemic heart disease, arrhythmia) *before* hearing loss?	☐ Yes ☐ No	1 / 0
15.	Were you diagnosed with high cholesterol or prescribed medication to control cholesterol levels?	☐ Yes ☐ No	1 / 0
16.	Were you diagnosed hypertensive, or *are you currently* on any antihypertensive medication?	☐ Yes ☐ No	1 / 0
17.	Have you ever regularly smoked or used tobacco products?	☐ Yes ☐ No	1/ 0
18.	Do you consume alcohol regularly?	☐ Yes ☐ No	1 / 0
19.	Significant stress, dehydration, or lack of sleep in the days *preceding* hearing loss?	☐ Yes ☐ No	1 / 0
Neurological			
20.	Any history of migraine or severe recurring headaches *before* hearing loss?	☐ Yes ☐ No	1 / 0
21.	At the time of hearing loss, did you experience sudden one-sided weakness (face, arm or leg), numbness, slurred speech, double vision, or difficulty walking?	☐ Yes ☐ No	1 / 0
22.	Were you diagnosed with a stroke/mini-stroke or any cerebrovascular condition (including brain MRI findings)?	☐ Yes ☐ No	1/ 0
Metabolic			
23.	Are you diagnosed as diabetic/pre-diabetic? If yes, are you on medication for it regularly?	☐ Yes ☐ No	1/ 0
24.	Are you suffering from a thyroid disorder?	☐ Yes ☐ No	1 / 0
25.	Have you ever been diagnosed with a vitamin (Vitamin D or B12) deficiency, or prescribed treatment for it?	☐ Yes ☐ No	1 / 0

26.	Have you been diagnosed with a hormonal or menstrual disorder, and are you currently receiving any treatment for it?	☐ Yes ☐ No	1 / 0
Atypical History			
27.	Before this sudden episode, did you have persistent fluctuating hearing problems or ringing in the same ear?	☐ Yes ☐ No	1/ 0
28.	Before this current episode (excluding hearing changes at the same time), have you ever experienced recurrent or progressive hearing loss affecting the other ear during a separate time in the past?	☐ Yes ☐ No	1/ 0
29.	Any history of persistent imbalance or tendency to sway to one side for >4 weeks before hearing loss?	☐ Yes ☐ No	1 / 0
		Total Score (Sec A+B) =	/
Sl. No.	**Questions**	**Response**	
Sub Section B2: Etiological Factors			
Trauma induced			
1.	Were you exposed to any loud sound before the hearing loss was noticed?	☐ Yes ☐ No	
2.	Did you undergo any ear surgery in the affected ear *before* the onset of hearing loss?	☐ Yes ☐ No	
3.	Did you sustain a head injury involving loss of consciousness or require hospitalisation *before* the onset of hearing loss?	☐ Yes ☐ No	
Pressure / Triggering Event			
4.	Did the hearing loss occur *following* sudden pressure changes like air travel, diving or heavy straining?	☐ Yes ☐ No	

Appendix III

INFORMED CONSENT

I have been informed about the study titled "Development and Preliminary Validation of a Prognostic and Assessment Checklist for Sudden Sensorineural Hearing Loss (SSNHL)" being carried out at the All India Institute of Speech and Hearing, Mysuru (AIISH). The purpose and procedure of the study have been explained to me clearly. I understand that my participation involves answering questions related to my hearing condition and allowing the researcher to use relevant clinical and audiological information for research purposes.

I understand that my participation in this study is voluntary and that I am free to withdraw from the study at any time without affecting my treatment or clinical services in any way.

I have been assured that all information collected from me will be kept confidential and used only for academic and research purposes.

I hereby give my consent to participate in this study.

Participant's Signature: _____ Date: _____

Name of Participant: _____

Researcher's Signature: _____

Appendix IV

ಮಾಹಿತಿ ಹೊಂದಿದ ಒಪ್ಪಿಗೆ ಪತ್ರ

ನಾನು "ಡೆವಲಪ್ಮೆಂಟ್ ಅಂಡ್ ಪ್ರಿಲಿಮಿನರಿ ವ್ಯಾಲಿಡೇಶನ್ ಆಫ್ ಎ ಪ್ರೋಗ್ನೋಸ್ಟಿಕ್ ಅಂಡ್ ಅಸೆಸ್ಮೆಂಟ್ ಚೆಕ್‌ಲಿಸ್ಟ್ ಫಾರ್ ಸಡನ್ ಸೆನ್ಸೊರಿನ್ಯೂರಲ್ ಹಿಯರಿಂಗ್ ಲಾಸ್ (SSNHL)" ಎಂಬ ಅಧ್ಯಯನದ ಕುರಿತು ಮಾಹಿತಿ ಪಡೆದಿದ್ದೇನೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಮೈಸೂರುದಲ್ಲಿರುವ ಅಖಿಲ ಭಾರತೀಯ ವಾಕ್ಶ್ರವಣ ಸಂಸ್ಥೆ (AIISH) ಯಲ್ಲಿ ನಡೆಸಲಾಗುತ್ತಿದೆ. ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ವಿಧಾನವನ್ನು ನನಗೆ ಸ್ಪಷ್ಟವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದಕ್ಕಾಗಿ ನನ್ನ ಶ್ರವಣ ಸಮಸ್ಯೆಗೆ ಸಂಬಂಧಿಸಿದ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸುವುದು ಹಾಗೂ ಅಗತ್ಯವಿರುವ ವೈದ್ಯಕೀಯ ಮತ್ತು ಶ್ರವಣ ಪರೀಕ್ಷಾ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಲು ಅನುಮತಿಸುವುದು ಒಳಗೊಂಡಿರುತ್ತದೆ ಎಂಬುದನ್ನು ನಾನು ಅರ್ಥ ಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ಸಂಪೂರ್ಣವಾಗಿ ನನ್ನ ಸ್ವಇಚ್ಛೆಯ ಮೇಲಾಗಿದ್ದು, ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಕಾರಣ ನೀಡದೇ ಅಧ್ಯಯನದಿಂದ ಹೊರಬರುವ ಹಕ್ಕು ನನಗಿದೆ. ಇದರಿಂದ ನನ್ನ ಚಿಕಿತ್ಸೆಗೆ ಅಥವಾ ಆಸ್ಪತ್ರೆಯ ಸೇವೆಗಳಿಗೆ ಯಾವುದೇ ತೊಂದರೆ ಉಂಟಾಗುವುದಿಲ್ಲ ಎಂಬುದನ್ನು ನಾನು ತಿಳಿದುಕೊಂಡಿದ್ದೇನೆ.

ನನ್ನಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಹಾಗೂ ಅದನ್ನು ಕೇವಲ ಶೈಕ್ಷಣಿಕ ಮತ್ತು ಸಂಶೋಧನಾ ಉದ್ದೇಶಗಳಿಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ ಎಂಬ ಭರವಸೆ ನನಗೆ ನೀಡಲಾಗಿದೆ.

ಆದುದರಿಂದ, ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನನ್ನ ಸ್ವಇಚ್ಛೆಯಿಂದ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಸಹಿ: ದಿನಾಂಕ:

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಸಂಶೋಧಕರ ಸಹಿ: