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Preventing Sensorineural Hearing Loss: A Review of Modifiable Risk Factors, Early Screening, and Neuroprotective Strategies

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ABSTRACT

Sensorineural hearing loss (SNHL) is by far the most prevalent cause of reported hearing loss globally, and is primarily caused by damage to the cochlea, auditory nerve and/or central auditory pathways through various mechanisms. The evidence suggests that the vast majority of the time, these factors are associated with SNHL as a result of modifiable risk factors, including exposure to loud noise, ototoxic chemicals and drugs, certain vascular/metabolic conditions (diabetes), and other health challenges. The result is a major opportunity for developing primary and secondary preventive initiatives. Potential strategies for reducing or eliminating the risk of SNHL include community awareness and health campaigns, community screening programmes to detect early stages of SNHL, measuring risk for those who are at high risk of developing SNHL (by addressing their modifiable risk factors), and by providing early intervention opportunities through the introduction of cochlear implants and/or the application of surgical techniques to protect residual hearing. Somewhat paradoxically, although advances continue to be made towards understanding the impact of modifiable determinants and preventive activities; there still remains a disconnect between the clinical practice and prevention programmes on the population level. Combining epidemiological risk assessment and early intervention through screening, addressing risk factors through lifestyle changes and surgical protection, and enabling prevention policies for modifiable risk factors should be effective in reducing the incidence of, and burden associated with, SNHL. Future research efforts and coordinated public health strategy should and must be encouraged, to make implementable, scalable and evidence-based preventive measures available to the population throughout their lives.

1. Introduction

Sensorineural hearing loss (SNHL) (which accounts for 90% of all reported cases of hearing loss) is defined by any hearing loss as a result of damage to the cochlea, auditory nerve, or central nervous system [1][2]. SNHL differs from conductive hearing loss, where sound is not able to reach the inner ear because of damage to the middle ear structures [3], and mixed hearing loss, which involves both sensorineural and conductive damage [4]. According to Podury et al., hearing loss is an important issue because it can lead to both psychological and social issues, such as isolation, depression, or strained social networks [5]. SNHL are usually treated by neurosurgeons and ENT using hearing aids, cochlear implants, medications, etc [6][7].

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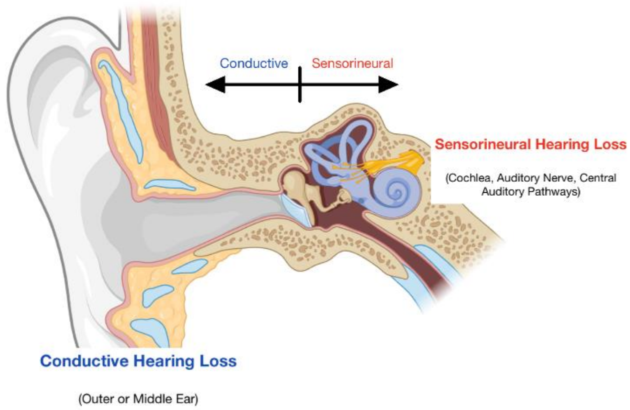

Fig. 1 - Anatomic localization of hearing loss across the auditory pathway. This schematic highlights the distinction between conductive and sensorineural hearing loss. Conductive hearing loss arises from pathology in the outer ear or middle ear that impairs sound transmission to the cochlea. Sensorineural hearing loss reflects dysfunction within the inner ear and neural elements, including the cochlea and the vestibulocochlear nerve, resulting in impaired transduction of mechanical sound energy into neural signals.

A large amount of the global population suffers from both sensorineural hearing loss and disabling hearing loss. WHO estimates over 430 million currently have disabling hearing loss, and 700 million will have a disabling hearing loss by 2050 if preventive measures are not taken [8]. An estimated 60% of all child hearing loss is due to preventable factors; therefore, there is a significant opportunity for prevention over the course of a person's life [8]. A recent meta-analysis reporting on the prevalence of hearing loss in adult, estimates of 31.0% for any form of hearing loss and 15.9 % for disabling hearing loss caused by the environment, lifestyle choices (e.g., noise exposure) and/or medical conditions (e.g., cardiovascular disease) [9] Children and adolescents represent a large population living with hearing loss worldwide (tens of millions under 20 years old have HL), as evidenced by research findings on global public health issues related to auditory impairments in this age group [10].

There are many modifiable risk factors associated with developing and progressing sensorineural hearing loss, many of which can be addressed through the implementation of preventive medicine approaches. Exposure to loud noises (either from work or play) is among the largest preventable contributors to the development of disabling hearing loss; one estimate is that noise exposure at work accounts for 16% of disabling hearing loss in adults [11]. There are indications that hazardous noise levels exist at both work and play for a majority of the population, based on national survey data [11]. Specific ototoxic substances, including specific classes of antibiotics, certain chemotherapeutic agents, and various industrially used chemicals, can lead to irreversible damage to hair cells in the cochlea [2]. As a result, these exposures link the use of medications and the exposure to chemicals found in the environment as relevant contributors to avoidable declines in hearing ability [2]. Other health conditions present at the same time as systemic health problems (e.g., cardiovascular disease, diabetes, etc.) may be associated with an increase in the risk of developing sensorineural hearing loss [12][13].

Although there are several modifiable factors associated with childhood and adult sensorineural hearing loss and a high prevalence of this type of hearing loss globally, there is still a disconnect between clinical and public health interventions; specifically, the focus is often on providing reactive treatment as opposed to implementing preventive strategies. This is shown by the fact that many types of hearing loss in both adults and children could have been prevented through the combined efforts of public health and clinical interventions. Therefore, there is an urgent need for developing synthesis of research related to prevention and developing guidelines on prevention based on the available research [14]. Additionally, integrating factors from each specialty (ear, nose, and throat) and neurosurgery (e.g., early detection of inner ear/auditory nerve pathology; optimising how we perform surgery to prevent iatrogenic injury; and providing lifestyle risk reduction options) would provide an opportunity to improve prevention practices within each clinical specialty. Based on the combined information from the epidemiology of sensorineural hearing loss, modifiable risk factors, and prevention strategies, a review based on narrative literature review has laid the foundation for bringing together both clinical practice and policy to reduce the burden associated with sensorineural hearing loss.

2. Epidemiology and Risk Factors

As per the Global Burden of Disease Study of 2019, Sensorineural Hearing Loss (SNHL) added significantly to standard hearing impairment worldwide affecting an estimated 1.57 billion people worldwide (i.e., about 1 in 5 people). This estimate includes all severities of standard hearing impairment (e.g., mild, moderate, severe, profound). As of 2019, around 403.3 million people had moderate or complete standard hearing impairment and will require assistive technology or rehabilitation; this means that a significant portion of standard hearing impairment in both adults and children can be attributed to SNHL. According to the same estimates, the number of people with standard hearing impairment is projected to reach approximately 2.45 billion by 2050 due to population aging, compounded environmental exposures, and increased noise exposure due to modern lifestyles (this poses a public health issue). The estimates above indicate that 430 million people affected by standard hearing impairment (including 34 million children) have a disabling condition; in addition, children with a standard hearing impairment are particularly at risk regarding communication, educational attainment and developmental trajectory [8][15].

Noise exposure is the second leading cause of acquired sensorineural hearing loss worldwide, with noise-induced hearing loss occurring due to repeated and extreme sound exposure, following age-related degeneration as a major cause of hearing loss by a far margin (Figure 1) [11]. Approximately 5% of the world's population suffers from noise-induced hearing loss (NIHL), either in the workplace, home or as a form of recreation [11]. Occupational noise accounts for a significant portion of this burden, with an estimated 16% globally of disabling hearing loss attributable to noise at work, making it a major risk for workers' rights related to their hearing health [16]. There are common recreational exposures that place individuals at risk, including the use of personal listening devices at unreasonably high levels of volume and frequent exposure to loud sounds at entertainment establishments [17]. A conservative estimate indicates that at least one billion young individuals in the world today are at high risk of developing hearing loss from unsafe listening behavior [17]. Thus, there are several public health interventions that can be implemented to help eliminate or reduce acquired sensorineural hearing loss, including controlling noise, promoting safe listening practices and promoting occupational hearing conservation as modifiable risk factors for developing in the future [18].

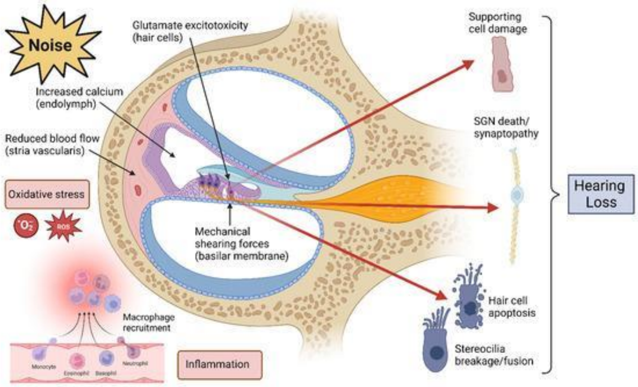

Fig. 2 - Schematic representation of noise-induced hearing loss. Image reproduced from Natarajan et al. (2023) [19] under the Creative Commons Attribution (CC BY) license (https://creativecommons.org/licenses/by/4.0/).

There are many other medications and toxic chemicals besides acoustic exposures that may cause SNHL due to direct cochleotoxic/neurotoxic properties. Currently, over 600 medicational agents have reported potential ototoxicity including aminoglycoside antibiotics; platinum-based anti-cancer medications such as cisplatin/carboplatin; certain loop diuretics; and many anti-inflammatory medications [20]. According to meta-analyses, the prevalence rate of hearing loss related to exposure to aminoglycosides is approximately 40.6% in exposed populations [21]. The highest risk of development occurs in instances such as multidrug-resistant tuberculosis treatment with aminoglycosides, whereas the range for development of cisplatin-induced hearing loss is 40% to 60% based on prior doses and schedule of administration [21]. The presence of loud noise adds to the occupational risk and prevalence of SNHL occurring from chemical exposure in the United States, where studies have indicated that approximately 13% of workers have reported chemical exposure (ototoxic) and approximately 16% have reported noise exposure in the past year; overall, approximately 28% of workers reported having an occupational noise exposure history [22]. Identification of and prevention of toxic exposures through the use of surveillance, monitoring, and substitution strategies is the best way to prevent diseases such as SNHL. Therefore, these exposures are excellent targets for intervention in workplace safety and health.

Even though noise and ototoxic risks are modifiable, the degree and presence of SNHL (sudden sensorineural hearing loss) can also be caused by biological and health conditions, which determine the amount of risk and presence of SNHL as well as the person's susceptibility. Cardiovascular factors such as diabetes, hypertension and dyslipidemia have been shown to increase an individual's odds of experiencing sudden SNHL [23]. It has been suggested that the possible presence and effect(s) of microvasculature, possibly through impaired blood flow, could have a negative impact on cochlear function due to reduced nutrient delivery to the cochlea, ultimately leading to hearing decline [23]. There is evidence that smoking and secondhand smoke exposure can cause shifts in hearing thresholds, with particular emphasis placed on the additive nature of other metabolic risk factors such as obesity involved with this, indicating the interaction of both systemic vascular and inflammatory processes may play a role in these events [24]. Age is the most common non-modifiable risk factor associated with age-related hearing loss; thus, there is a need for individuals to avoid additional exposure to other causative factors that could lead to hearing loss [11]. Early identification of potentially high-risk individuals and the continued implementation of public health prevention measures can assist in preventing hearing loss as well as identifying individuals with hearing loss.

3. Pathophysiology of Hearing Loss

Sensorineural Hearing Loss (SNHL) is defined as hearing loss that results from injury to the cochlea (also known as the inner ear) or the auditory nerve. It occurs as a result of such damage either to the hair cells or to their associated neurons in the cochlea [25]. In a clinic or as physiology, SNHL is clinically and physiologically distinguished from conductive hearing loss, which is caused by dysfunction in the outer or middle ear, thus displaying an air-bone gap when examining through audiometry [26]. Pathophysiologically, SNHL has traditionally been categorized into primary peripheral causes (the absence/dysfunction of hair cells, the organ of Corti and/or spiral ganglions), versus primary functional causes to the central nervous system, as this has implications for the way that both diagnosis and prevention occur [27]. In humans, cochlear hair cells do not regenerate in adults, hence any sensory-cell death associated with acquired (SNHL) cannot be reversed; due to this biological limitation, prevention is emphasised in both the clinical setting and the public health domain [25].

The cochlea has multiple converging injury pathways that molecularly and cellularly cause death of hair cells and neurons. One of these well-defined pathways involves oxidative stress from reactive oxygen species (ROS), which induce lipid peroxidation, DNA damage and activation of apoptotic pathways in hair cells that occur after noise exposure [28]. Another important pathway is excitotoxicity, where excessive glutamate released at the inner hair cell — auditory nerve synapse causes depolarization and damage to the afferent fibers, leading to loss of synapses even when the hair cells remain intact [25]. Mitochondrial dysfunction plays a central role in many of these pathways due to the increased production of mitochondrial ROS, which not only amplifies cellular injury, but depletes the energy supply necessary for maintaining hair cell homeostasis and therefore links metabolic stressors (like aging and diabetes) with the susceptibility of the cochlea [27]. These molecular pathways exist not just in the context of noise trauma but also contribute to drug-induced ototoxicity (e.g., aminoglycosides and cisplatin), hence the mechanistic overlap can provide numerous common targets for future pharmacological prevention strategies [25].

Cochlear synaptopathy can occur shortly after exposure to loud noise, causing loss of connections from the inner hair receptors of the cochlea to the spiral ganglion neurons that transmit sound information to the brain. These changes can affect sound-processing ability considerably, even when the individual can hear pure tones via standard audiometric testing [29]. Cochlear synaptopathy therefore generates "hidden hearing loss" because many cochlear synaptopathies only express themselves under conditions of increased background noise [30]. The loss and degradation of the cochlear mechanisms over time will result in a loss of clarity, hearing in adverse conditions, and the precise perception of speech in adverse listening conditions [30]. With time, further neural degeneration will occur and degradation of peripheral mechanisms will result in long term neural decline [29]. The loss of cochlear function will have an effect on brain structures that process auditory information; these auditory structures will adapt and reorganize following cochlear insult but may lead to maladaptive perception of sound and ultimately include tinnitus, hyperacusis and other auditory-processing disorders; therefore, the ENT and neurosurgical perspectives are important for audiological rehabilitation and prevention of cochlear-neural loss [27].

Knowing how these processes work gives a definitive indication of when to implement prevention strategies. In particular, the time period immediately after a cochlear injury has occurred (when there is a presence of oxidative stress, excitotoxic synaptic damage, and mitochondrial dysfunction) represents the time that we have available to implement therapeutic options such as antioxidant therapy, glutamate receptor antagonists, or other neuroprotective methods to prevent a permanent loss of hearing [31]. The same information about the mechanistic pathways explains and supports the same level of noise reduction, employer noise exposure restrictions, monitoring the use of ototoxic drugs, and control of cardiovascular and metabolic co-morbidities as the main prevention strategies to limit the cumulative cochlear injury that occurs across the lifespan for a large population [25]. Additionally, because synaptopathy can occur before threshold shifts occur, prevention also supports the development of better and more sensitive screening methods (e.g., suprathreshold ABR metrics, extended high-frequency audiometry, speech-in-noise testing) for at-risk groups so that intervention can be implemented before the irreversible loss of cochlear function due to degeneration occurs [29]. Finally, understanding the underlying mechanisms of hearing loss is currently driving progress in the development of new and innovative ways to research and develop ways to repair cochlear damage, keep the hair cells healthy, and create ways to preserve cochlear function. As these new forms of treatment are developed, clinicians and public health professionals will be in a better position to develop appropriate screening practices and procedures to reduce the risk of developing irreversible hearing loss as new scientific findings are developed [32].

4. Screening and Early Detection

Early Hearing Detection and Intervention Programs are developed to identify babies with or at risk of developing a permanent hearing loss before the symptoms arise. The purpose of the EHDI program is to allow for a definitive diagnosis and timely intervention for those identified as having hearing loss before any symptoms develop through population-based screening efforts [33]. The Joint Committee on Infant Hearing (JCIH) has framed early identification as a secondary preventive measure to improve the future language, communication skills, literacy, and psychosocial development of those babies identified early by reducing the amount of time spent without access to auditory information [33]. Research shows that due to the developmental development of cortical regions due to auditory stimulation during the infant's critical window of development, those who have had delayed identification of their hearing loss compared to those who had their hearing loss identified at an earlier time show measurable decreases in both cognitive and language development [34]. Therefore, the EHDI program is not just a way to provide early diagnosis; it also represents an effective method to reduce future developmental and educational impairments through a proactive way to minimize the impact of auditory deprivation on children before they reach an educationally required age [33]. Otoacoustic emissions (OAE) and automated auditory brainstem response (AABR) testing are commonly used in children in addition to early hearing loss tests. The screening occurs within the first few days after the child is born. This is to diagnose any potential congenital and/or early-onset sensorineural hearing loss in infants [35]. Incidence estimates based on numerous studies have indicated that congenital/early-onset sensorineural hearing loss will occur in 1-3 out of every 1,000 infants who are born healthy at a term pregnancy. The rates of occurrence of this condition are much greater among those infants who are in the NICU and other high-risk health categories [35]. The JCIH 1-3-6 goals (additional finding goals of screening at one month of age, diagnostic audiological evaluations at 3 months of age, and enrollment for intervention services at six months of age) are operational goals outlined in EHDI programs used to evaluate how well children are being identified in a timely manner and then referred to receive the appropriate services [33][36]. Prior to universal screening programs being conducted in every country, we have an overwhelming amount of empirical evidence that only those infants who were selected for a hearing evaluation based on their risk of congenital auditory impairment were being picked up in the screening process. This, together with the evidence that there was a large cohort of infants who were diagnosed with a hearing loss that had not been identified, were significant motivating factors in many countries adopting universal newborn hearing screening programs [35].

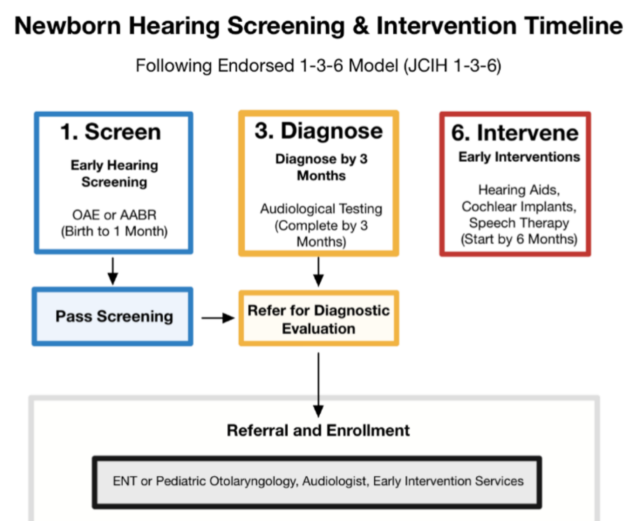

Fig. 3 - Newborn hearing screening and early intervention pathway using the JCIH 1 3 6 framework. This figure summarizes the recommended timeline for early detection and management of congenital or early onset hearing loss, emphasizing hearing screening by 1 month of age (typically with otoacoustic emissions or automated auditory brainstem response), completion of diagnostic audiologic evaluation by 3 months for infants who do not pass screening, and initiation of early intervention services by 6 months. The diagram also highlights key decision points, including pass versus referral for diagnostic evaluation, and the transition to coordinated specialty follow up and enrollment in early intervention services to optimize speech, language, and developmental outcomes.

Abbreviations: JCIH, Joint Committee on Infant Hearing. EHDI, Early Hearing Detection and Intervention. OAE, otoacoustic emissions. AABR, automated auditory brainstem response.

The practice of routinely screening adults with audiometric equipment is generally focused on adult populations deemed at higher risk for the development of hearing deficits due to either short-term hazardous workplace noise exposure (e.g. truck drivers) or long-term hazardous workplace, military personnel, people receiving ototoxic medications, older adults at risk of developing age-related hearing deficits, etc. Screening can help to identify hearing deficits early so that professionals can counsel patients about ways to prevent further hearing loss and make changes in patients' environment to reduce additional hazardous exposures [37]. Acquired sensorineural hearing loss associated with noise exposure and unsafe listening behaviour is a leading preventable cause of acquired sensorineural hearing loss worldwide, and estimations based on large population studies show that there are hundreds of millions of individuals at risk for acquiring hearing loss caused by unsafe listening behaviours alone [17][38]. Periodic screening of workers for hearing impairment can enable preventative referrals to intervention services such as hearing protection, duty-time modification, medication review or referral for amplification or rehabilitation services [37]. When adult screening is framed within the lens of preventative medicine, the recognition that hearing deficits may develop, often without symptoms, allows for the reduction of the downstream consequences related to hearing deficits, such as the decline in communication, increase in social isolation and additional cognitive load on the brain, all of which may interact with dementia risk [17].

In addition to issues associated with access to screenings, loss to follow-up following an initial referral, and limited resources in low-resource settings as detractors from the effectiveness of current screening programs, gaps existed which necessitated the development of scalable solutions to ensure quality without compromising the reach of services provided [39]. Multiple recent studies have demonstrated that tele-audiology and validated portable/automated audiometry devices provide screening and, in some cases, diagnostic services at a non-inferior level as compared to traditional methods; thus allowing for the creation of community-based and remote-region-based screening programs [39]. The initial evidence indicates that AI-assisted signal processing and automated analysis of acoustic signals improve throughput and assist in identifying patterns which create a need for further referrals. Although these tools still require prospective assessment and the implementation of quality assurance frameworks, they support preventive-medicine models with integrated referral systems and overall population risk-reduction approaches and promote earlier, equitable detection of hearing loss [40].

5. Preventative Interventions

According to a number of studies, Noise-Induced Hearing Loss (NIHL) is the 2nd most prevalent cause of sensorineural hearing loss in people worldwide, and is estimated to be found in approximately 5% of people globally; thus, environmental noise is an appropriate target for primary prevention of hearing loss [41]. Based on recommendations made by OSHA and NIOSH, chronic sound exposure at levels greater than or equal to 85 dB(A) have a direct correlation to permanent hearing loss if a person does not take advantage of hearing protection devices [42]. Research using population based data estimates that between 7 and 21% of disabling adult hearing loss can be attributed directly to occupational noise exposure, with variations across regional boundaries [41]. Among the various interventions to minimize adult hearing loss due to occupational noise, engineering controls, administrative exposure limits, and consistent usage of certified hearing protection devices are the bases for primary prevention, and are likely to have moderate levels of evidence supporting their positive effect on reducing short term noise exposure [42].

Pre-clinical and early-stage human studies have looked at the use of drugs to combat the effect of oxidative stress and other pathways involved with damage caused by exposure to excess noise on the cochlea [43]. Antioxidants, including alpha-lipoic acid, NAC, vitamins C and E, beta-carotene, and newer types of treatments like ebselen which chemically resembles endogenous glutathione peroxidase have been studied [43]. In some studies, they report significant reductions in temporary and permanent threshold shifts in individuals after a controlled exposure to noise, although this has been reported with mixed and inconsistent evidence [43]. Although there are some interesting immunological rationales to date none of the stated pharmacological prevention strategies are recommended for use in general practice because of study design issues, small sample sizes and inconsistencies with study results [44].

Beyond direct noise exposure, systemic health factors such as smoking, obesity, diabetes, and hypertension have been associated with accelerated hearing loss trajectories in longitudinal cohort analyses [45]. Although individual effect sizes are modest in some cohorts, multiple large studies indicate that clustering of cardiovascular risk factors correlates with worse audiometric thresholds over time, suggesting a role for lifestyle modification in preventative strategies [45]. Smoking has been specifically linked with increased odds of hearing impairment in epidemiologic data, likely via microvascular and inflammatory pathways affecting cochlear perfusion [46]. Comprehensive public health approaches that target tobacco cessation, glycemic control, weight management, and blood pressure optimization may therefore have ancillary benefits in reducing the incidence and progression of sensorineural hearing loss [45].

The focus of secondary prevention is to identify signs of hearing loss early, and initiate treatment before significant loss of hearing occurs [41]. Conducting regular audiometric tests among people who are at risk of developing hearing problems (for example, construction workers or young people who are typically exposed to excessive noise for recreational use) provides a means of implementing preventative treatment options (such as educating patients about exposure to noise and changing or eliminating exposure to noise) before substantial intervention is needed [42]. The collaboration of early intervention programs with occupational health services and primary care practice will promote more consistency of behaviour change and assistance with the progression of temporary thresholds from work exposure reduction [42]. In order to provide equal access to secondary prevention of hearing loss, consideration must be given to identifying areas of social determinants of health (such as access to hearing healthcare providers, affordable hearing protection equipment, and educational materials on the prevention and management of hearing loss) [41].

6. Surgical and Device Interventions

By identifying and interrupting disease progression prior to the occurrence of irreversible cochlear or neural injury, surgical approaches can help preserve hearing. In treating lesions that could potentially cause damage to the auditory structures (for example, small vestibular schwannomas or progressive cholesteatoma), the surgeon may choose to utilize hearing-preserving surgical approaches or may perform the surgery at an earlier point in time in order to reduce the potential for cochlear or cochlear nerve injury due to chronic compression/inflammation [47]. The concept of hearing-preservation surgery allows the re-classification of certain surgical procedures, ENT and skull base procedures, into the category of primary-preventive surgical procedures when both timing and technique can prevent downstream sensorineural degeneration [48]. In clinical practice, clinicians should document baseline audiometry, counsel patients with regard to the preventive intent of the selected surgical interventions versus the rehabilitative intent of those surgical interventions, and provide patients with outcome data linking earlier, less traumatic resections with increased long-term hearing retention [47].

Iatrogenic sensorineural hearing loss is a recognized, often preventable complication of otologic and lateral skull-base surgeries and arises through mechanisms such as direct mechanical trauma, vascular compromise, thermal injury from drilling, and vibration/pressure transmission to the cochlea [49]. Experimental and clinical evidence implicates drilling-related vibration and acoustic energy as contributors to postoperative SNHL after middle ear and mastoid procedures, motivating strategies that reduce drill speed, use irrigation and burr selection, and minimize bony vibration near the cochlea (mechanical/vibration data; research on drilling injury). To mitigate these risks, operative planning increasingly emphasizes minimally traumatic corridors, refined burr technique, and strict hemostasis to preserve cochlear perfusion and reduce thermal injury (systematic analyses of causal factors). Finally, preoperative risk stratification (anatomic variants, prior otologic disease, baseline hearing level) combined with documented intraoperative protective steps helps convert what would otherwise be iatrogenic injury into an anticipated and minimized risk — a core tenet of preventive surgical practice [49].

7. Adopted Protection Strategies in Various Countries

The implementation and effectiveness of national and regional hearing protection policies are markedly different. However, research on cross-national comparisons has shown that through the use of standardised screening processes and regulations these areas can see notable increases in early detection of hearing loss and lower rates of downstream disabilities.

UNHS greatly increases the chance that infants will be detected with permanent bilateral hearing loss before nine months of age if they live in a region where UNHS is performed as opposed to a region where there is no UNHS program (Figure 4) [50][51]. In a pooled analysis of over one million newborns, infants born in an area with UNHS are at least three times as likely to be detected with permanent bilateral hearing loss before nine months of age than newborns born in an area where there is no UNHS (relative risk of 3.28, 95%CI 1.84–5.85) [50].

Fig. 4 - Image depicting newborn hearing screening. Reproduced from Liannadavis [51] under the CC BY-SA license (https://creativecommons.org/licenses/by-sa/4.0).

Despite this clear benefit, global coverage of UNHS remains uneven: surveys across 158 countries indicate that only about one-third of newborns worldwide are born in regions achieving ⩾85 % screening coverage, with 38 % of countries reporting minimal or no screening at the national level [52]. In high-coverage programs, such as widespread initiatives in the United States, most newborns receive hearing screening before hospital discharge, often achieving near-universal (>95 %) coverage; however, gaps remain in timely follow-up and intervention after screening, highlighting that implementation quality is as critical as coverage [53]. Comparative data from Europe affirm that established UNHS programs achieve screening coverage exceeding 90 %, with structured protocols employing otoacoustic emissions and automated auditory brainstem response to detect hearing impairments early, although detailed national outcome data are limited in the literature [54].

There are differences in regulatory strength and enforcement of occupational noise risk reduction strategies. Many articles show that between 7% and 21% of hearing loss in workers may be due to occupational exposure to noise, with lower proportions reported in more developed countries where stronger regulations and enforcement may have provided some preventive benefit [55]. Cochrane reviews show that there is very limited evidence of the effectiveness of workplace interventions beyond regulation (e.g. engineering controls, hearing protection programs) in preventing hearing loss due to occupational noise. They highlight the uncertainty in the extent to which non-pharmaceutical interventions reduce measured hearing loss, and indicate that more rigorous evaluations of these interventions in actual practice are needed [42]. Frameworks requiring an employer to implement hearing conservation programmes relative to an employee's exposure level (e.g. requiring programmes at noise exposure levels of ⩾85 dB(A)) provide the basis for structural efforts to promote prevention; however, compliance and evaluation of results would differ between industry and jurisdiction [56].

The issue of noise exposure from recreational activities is still not addressed adequately by the national governments, although estimates from systematic reviews suggest that 0.67-1.35 billion young people are at risk of developing hearing disorders through the use of unsafe listening methods and there is a large gap in access to national policy-level guidelines for safe listening practices [17].

8. Conclusion

In conclusion, SNHL is a significant health issue that is prevalent throughout the world, and it has both public health and clinical ramifications. A Narrative Synthesis (NS) analysis of existing literature found that a significant proportion of SNHL is caused by preventable environmental factors (e.g., hazardous noise exposure and ototoxic agents), which offer opportunities to implement primary prevention programs through regulation, educational programs and the use of personal protective devices (PPDs). The current practice of early detection initiatives and routine screening allows for the identification of subclinical impairments and serves as the basis for implementing secondary prevention initiatives, which may assist in slowing down progression and maintaining communication abilities. Through surgical procedures and the development of assistive device technology, the preservation of remaining auditory function, as well as the prevention of iatrogenic harm, can be achieved. There are still gaps in the implementation of these methods, revealing the need to better incorporate preventive measures into the practice of ENT and neurosurgery, and the need to expand these models of prevention to include broader public health policies. The most effective means of reducing the overall burden of SNHL, as well as its associated negative impact on an individual's quality of life, is the implementation of a comprehensive model focused on prevention that addresses both the reduction and potential elimination of modifiable risk factors, early detection, and the use of clinical and surgical safeguards, as well as public health interventions at the population level.

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