# Hidden Hearing Loss: Revisiting the Significance of Word Recognition Scores in Standard Audiometric Evaluations

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*Editor's Note: Many recent reports have failed to find what the media has colloquially referred to as "hidden hearing loss" and some reports note that while this may occur in lower mammals, it does not occur in humans. Howver, a recent publication by Grant et al., Predicting neural deficits in sensorineural hearing loss from word recognition scores. Scientific reports. (2022) has reignited our interest in "hidden hearing loss." We caught up with one of the authors of this report, Dr. Stephane Maison, for both a summary of this important research, as well as a more general Question and Answer section that immediately follows the reference section of this summary.*

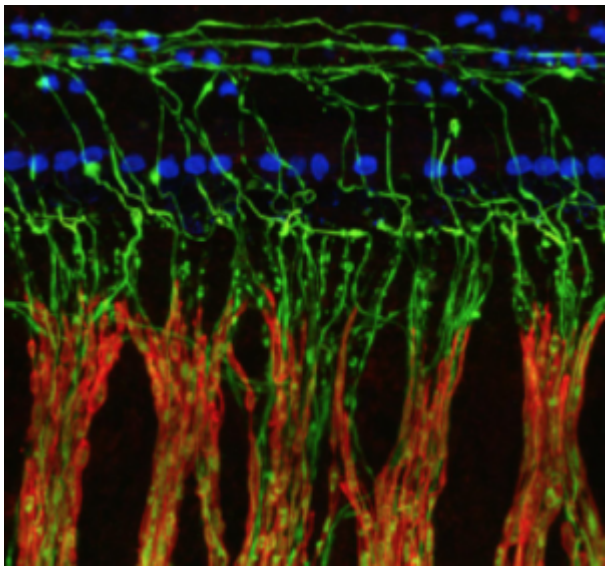


**Photo is courtesy of Dr.Stephane Maison.**

For the past decade, a large number of studies from animal models and human temporal bones on aging have shown that outer hair cell loss can be preceded by cochlear nerve degeneration (CND), whereby auditory-nerve fibers (ANFs) are disconnected from inner hair cells (for review, see.[1] Remarkably, the most sensitive ANFs to aging have high thresholds and low-spontaneous

discharge rates. Consequently, the loss of their unique synaptic connections with inner hair cells does not affect hearing thresholds until it becomes extreme (>80% loss), including when measured behaviorally on the audiogram. Therefore: (1) CND can be present in patients even if audiometric thresholds are within normal limits, and (2) CND is likely to be extensive in the presence of hearing loss.

Due to this insensitivity of the audiogram to CND, this phenomenon has been coined *hidden hearing loss*; a poor choice of words considering that the loss of these synaptic connections leads to poorer auditory processing, which may translate into a variety of *quite apparent* perceptual abnormalities, including one very familiar to all audiologists and widely reported by patients (with or without hearing loss): speech intelligibility difficulties in noisy environments.[2–6] CND may also be key to the genesis of other perceptual anomalies associated with sensorineural hearing loss (SNHL), including hyperacusis and tinnitus.[7–13]

Thus, the gold standard for hearing evaluations in adults that includes a pure-tone audiogram combined with a suprathreshold word-recognition task in quiet is inadequate to fully assess CND's hearing impairments. Bearing this in mind, we chose to revisit the significance of traditional audiometric data in a retrospective study recently published in *Scientific Reports,*[14] testing the hypothesis that deficits in word recognition may underlie CND.

To do so, we compared the *measured* word scores obtained at PBmax from nearly 48,000 patients to the word scores each patient should obtain as *predicted* by the Speech Intelligibility Curve (SIC) associated with their audiogram. The SIC is a speech performance/intensity function derived from hearing thresholds combined with a transfer function appropriate to the speech material. In other words, this sigmoidal curve describes the predicted word score a patient should obtain as a function of the speech presentation level.[15] Since the speech material is presented at suprathreshold levels (optimally at PBmax), word recognition performance is unrelated to speech *audibility*. Therefore, differences between measured and predicted word recognition score (WRS) reflect deficits in speech *intelligibility* that may arise from CND.

Unsurprisingly, WRS deficits were minimal in a cohort of patients with conductive hearing loss. Indeed, the SIC assumes hearing loss filters out speech sounds (as expected with a conductive component). However, in the same cohort, a lack of large WRS among the oldest patients suggested that cognitive decline was not a major contributor to speech intelligibility deficits assessed in traditional hearing evaluations.

On the other hand, WRS deficits increased significantly with age and degree of hearing loss in patients with presbycusis. Moreover, these deficits in speech intelligibility were even greater in SNHL etiologies known or suspected to cause greater nerve loss than age-related hearing loss, including in patients with Ménière's disease, patients with vestibular schwannoma, and patients after sudden sensorineural hearing loss. Altogether, these results were consistent with the idea that CND significantly contributes to the loss of speech intelligibility in SNHL.

We then compared our data of speech intelligibility deficits from different SNHL etiologies with the existing histopathological data on CND in human temporal bones from donors with presbycusis only and donors with vestibular schwannomas[16] and *Ménière's* disease.[17] As a result, we could estimate the relationship between word score as assessed in standard hearing evaluations and CND.

CND must exceed 60% before word scores start to fall below 90%. Past the 60% CND mark, word scores drop rapidly with neural loss.

This predictive model of CND based on word scores could be a lot more sensitive and informative if future hearing evaluation protocols incorporate more challenging listening tasks (e.g., speech in noise; time-compressed words with reverberation). In addition, defining biomarkers of CND is key if audiologists want to identify candidates for and track the efficacy of emerging treatments meant to provide better speech intelligibility to both non-traditional hearing aid candidates and hearing aid users.

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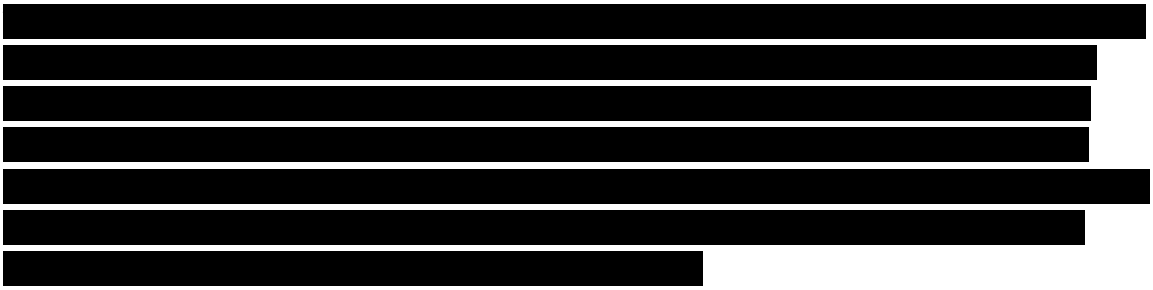

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## Q&A

1. The study suggests that cochlear nerve degeneration strongly drives discrepancies between predicted and measured speech scores. This implies that we could use discrepancies between predicted and measured speech scores to estimate cochlear nerve degeneration. However, speech measures can be quite variable with single listeners in the clinic versus 96 000 listeners in a large study (especially when using short word lists). Do you think there is a place for using discrepancies between predicted and measured speech scores to estimate cochlear nerve damage in a clinical setting?



2. Your data show that low-frequency hearing tended to decline more rapidly in older female than male listeners. Do you have any ideas as to why this may be?



[redacted]

3. It is fascinating that the data do not show any evidence for a relationship between cochlear nerve damage and reported acoustic over-exposure, except for people with thresholds in the severe range. On the other hand, the data do show evidence of cochlear nerve degeneration for people with 4 kHz notches in the audiogram.
    1. Is reported noise exposure simply too unreliable to help estimate the effects of noise?

    [redacted]

    2. Is it safe to assume that humans with noise-induced synaptopathy will likely have thresholds shifts as well (unlike animals in laboratory studies with carefully titrated noise exposures)?

    [redacted]

    1. What clinical measures would you suggest for estimating synaptopathy or cochlear nerve degeneration?

    [redacted]

4. For listeners scoring 99% and above, the mean audiogram was roughly 15 dB to 2 kHz, sloping to 25 at 4 kHz and 35 dB at 8 kHz. Do you think this merits a reconsideration of the range that we consider to be normal hearing?

[redacted]

5. Many research articles are now questioning whether “hidden hearing loss” actually exists in

humans, despite evidence of its existence in lower mammals. Do you think that this study firmly establishes this phenomenon in humans?

[redacted]

[redacted]