Speech-in-noise testing as a marker for noise-induced hearing loss and tinnitus

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Abstract. Speech-in-noise testing as a marker for noise-induced hearing loss and tinnitus. Noise-induced hearing loss and tinnitus are auditory complaints that often co-occur. Often, there is no immediate indication of changes in the pure tone audiogram. Patients can still have clinically normal hearing thresholds while clearly experiencing reduced speech comprehension. This might be explained by the process of neurodegeneration of the innervated dendrites of the auditory nerve fibres and secondary degeneration of spiral ganglion neurons. Subsequent maladaptive neuronal plasticity of the central auditory system can induce tinnitus. Standard hearing testing is no longer sufficient in these patients. Therefore more complex tasks, such as speech-in-noise tests, might be valuable extensions to the standard hearing tests. We carried out a prospective investigation of the influence of tinnitus upon speech comprehension in noise and the effectiveness of speech-in-noise testing, using the Flemish version of the digit triplet test (DTT). Thirty-seven patients with mild noise-induced hearing loss, tinnitus complaints and clinically normal pure tone thresholds completed the DTT and filled in two tinnitus enquiries. A statistically significant (p=0.026) correlation between the averaged high PTA₁,²,₄kHz and the averaged SRT across ears on the DTT was found. There also seems to be a slight influence of tinnitus onset on the SRT score.

Introduction

In 1999, the US National Institute of Deafness and Other Communication Disorders recorded an increase of noise-induced hearing loss (NIHL) in the population which reached up to 33% of all hearing loss cases, with a prevalence second only to presbyacusis.¹ Through a cross-sectional analysis of demographic and audiometric data from 1988−1994 in comparison with 2005−2006, Shargorodsky et al.² found a significant increase in the prevalence of hearing loss in US adolescents.³ It is possible that this finding of a significant increase in high-frequency hearing loss between the 1988−1994 and 2005−2006 time periods may indicate an increase in NIHL. Typically, NIHL is caused by long-term repeated sound exposure, or by short exposure to sounds of very high intensity. Often, this hearing loss can be associated with complaints of tinnitus, hyperacusis and reduced speech understanding, especially in noise.⁴ A short episode of hearing loss and tinnitus caused by (mild) acoustic trauma mostly has a good prognosis and can recover within 2−3 weeks.⁵ It generally results from reversible damage to the stereocilia of the hair cells, leading to a temporary threshold shift.⁶ Chronic noise exposure will predominantly lead to a destruction of the outer hair cells (OHCs), the connected synapses to the spiral ganglion neurons, and, only at a later stage, damage to the inner hair cells (IHC). OHCs are connected with efferent auditory nerve fibres, and function as a nonlinear amplifier in response to soft sounds, thereby increasing the sensitivity of the hearing organ and playing an important role in speech understanding in noise.⁷ IHCs are the true auditory sensory cells. Their synapses and connection to the auditory nerve fibres are of high importance in assuring the dynamic range and temporal information of the auditory signal. Loss of IHCs is possible, although this rarely occurs in the case of NIHL. A combination of OHC and IHC damage has been noted. The 3500 IHCs in the cochlea rarely die from NIHL, however. Instead, the innervated dendrites of the auditory nerve fibres undergo neurodegeneration.⁸ Even in mild acoustic trauma, 50% of the hair cell synapses can be lost. Neurodegeneration of the innervated dendrites of the auditory nerve fibres takes place with secondary degeneration of spiral ganglion neurons in the high frequency cochlear region.⁹ Animal studies have also shown that noise trauma induces synapse and progressive neuron loss, which in its turn has been linked to impaired speech perception in noisy environments.¹⁰ For more details on the underlying
Materials and methods

Participants

Study participants (n = 37) were patients who presented at the Department of Otorhinolaryngology, Head and Neck Surgery of the University Hospitals Leuven. They complained of reduced speech understanding and tinnitus, mostly caused by chronic noise exposure or mild acoustic trauma. All subjects presented with normal pure-tone hearing thresholds or mild high frequency threshold elevation. Only participants with a PTA at the frequencies 0.5, 1, 2 kHz and 1, 2, 4 kHz <15 dB HL were included. Conductive or mixed hearing loss cases were excluded, as well as subjects with asymmetrical hearing loss. Only patients between 18 and 45 years old participated, in order to minimize potential cognitive influences. All participants gave their written informed consent. The local medical ethical committee approved this study.

Procedures

All subjects were investigated by an ENT physician and underwent a routine clinical examination with an otomicroscope, to exclude outer or middle ear pathologies. Subsequently, a standard pure tone audiometry was conducted at the frequencies 125, 250, 500, 1000, 2000, 4000, 6000 and 8000 Hz. Audiograms were measured in a soundproof booth with a Madsen Astera audiometer and a calibrated TDH39 (Telephonics) headphone.

The DTT was conducted on a PC or a tablet with a calibrated Sennheiser HDA200 (PC) or DD45 headphones with Peltor caps (tablet) in a room with limited background noise. The audiologist gave short instructions for the test and placed on the headphones. Next, the patient received test instructions once more on the screen, followed by five practice items. Then the left and right ears were tested successively, with 27 test items for each ear. The test follows an adaptive procedure, and starts at a signal-to-noise ratio (SNR) of 0 dB, with the noise fixed at 65 dB SPL and the speech varying in steps of 2 dB.

Patients with a conductive or mixed hearing loss did not undergo the DTT because of a cumulative, negative effect of the air bone gap (ABG) on the SRT. In these cases, a tympanometry and/or acoustic reflex measurement was conducted to seek
an explanation for the ABG and possibly also the cause of the tinnitus.

In addition, a tinnitus analysis was conducted, and the tinnitus questionnaire (TQ)\(^6\) and tinnitus handicap inventory (THI)\(^7\) were filled in independently by most of the patients (Table 1).

A flowchart of the various procedural steps is presented in Figure 1.

### Results

**Tinnitus questionnaires**

The age of the study participants ranged between 18 and 44 years, with a mean age of 28 years. All participants had tinnitus complaints, with onsets varying from acute (≤ 1 month) to more than 1 year. Tinnitus severity (based on the TQ) was mild in the majority of subjects; in addition, no or only a mild handicap (based on the THI) was experienced in most subjects. There was a strong correlation of \( r = 0.72 \) \((p < 0.001)\) between the TQ and the THI scores, as shown in Figure 2.

**Pure tone audiometry**

All participants had normal hearing, based on the low (0.5-1-2 kHz) and high (1-2-4 kHz) frequency hearing threshold averages. At higher frequencies of 6 and 8 kHz, thresholds of more than 25 dB HL occasionally occurred. Elevated thresholds at 6 kHz may relate to calibration issues using the TDH39 headphones (unpublished data from a KU Leuven Master’s thesis). There were no obvious asymmetric threshold curves. A summary of the hearing thresholds for left and right ears is shown in Figure 3.

**Speech-in-noise testing: digit triplet test**

All participants performed the digit triplet test bilaterally. When we consider an SRT score deviating by more than 2 SDs of the reference

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### Table 1

Overview of patient characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Mean = 28y, SD = 7.6</td>
</tr>
<tr>
<td>PTA(_{1,2,4\text{KHz}})</td>
<td>Mean = 5 dB HL, SD = 3.9 dB</td>
</tr>
<tr>
<td>SRT</td>
<td>Mean = -10.9 dB, SD =0.7 dB</td>
</tr>
<tr>
<td>Tinnitus side</td>
<td>Left: n = 3 (8%) Right: n = 8 (22%) Bilateral: n = 12 (57%) Unknown: n = 5 (13%)</td>
</tr>
<tr>
<td>Tinnitus onset</td>
<td>≤ 1 month: n = 9 (24%) &gt;1 - &lt;12 months: n = 13 (35%) ≥ 12 months: n = 15 (41%)</td>
</tr>
<tr>
<td>TQ score</td>
<td>Mild: n = 17 (46%) Moderate: n = 5 (13.5%) Severe: n = 2 (5.5%) Unknown: n = 13 (35%)</td>
</tr>
<tr>
<td>THI score</td>
<td>No handicap: n = 6 (16%) Mild: n = 5 (13.5%) Moderate: n = 5 (13.5%) Severe: n = 1 (3%) Unknown: n = 20 (54%)</td>
</tr>
</tbody>
</table>

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![Figure 1](image1.png)

Flowchart of procedural steps (in a patient with hearing loss and/or tinnitus after noise exposure).

![Figure 2](image2.png)

Correlation between scores on the THI and the TQ.
value (SRT = -11.3 dB, SD = 0.7 dB), 14% (n = 5) of the subjects with detection thresholds within clinically normal hearing norms failed the speech-in-noise (SPIN) test. All participants with a failed SPIN-test had tinnitus complaints for more than 1 year. In addition, they mentioned that their tinnitus was bilaterally present. If we expand the pass/fail criterion to an SRT deviating by more than 1 SD of the reference value, as many as 35% (n=13) of the subjects failed the speech-in-noise test.

Spearman's rho rank correlation coefficients showed a weak, but statistically significant ($p = 0.026$) correlation of $\rho = 0.37$ between the averaged high PTA$_{1,2,4}$ kHz and the averaged SRTs (Figure 4).

There were no significant correlations between the SRTs and the scores on the tinnitus questionnaires. When dividing the dataset into three groups, based on tinnitus onset (i.e., $\leq$ 1 month, $>$ 1$-$< 12 months vs. $\geq$12 months), subjects who had reported tinnitus complaints over a longer time period tended to have slightly worse SRTs (no significant group difference). This tendency is shown in Figure 5.

**Discussion**

Hearing disabilities in the presence of a normal audiogram have often been reported. These perceptual difficulties are also known as hidden hearing loss.\(^1\) In 1955, Schuknecht et al.\(^1\) discovered that synapse loss, known as synapthopathy, does not present with a threshold shift. Synapses die off before hair cells, but spiral ganglion neuron degeneration is slow, since the cell body and central axons remain. Recent findings have also suggested cochlear damage without elevation of hearing thresholds.

Several studies on humans with a history of significant noise exposure, but with normal hearing threshold levels, have shown poorer speech recognition ability in noise compared to controls; effects on auditory spectro-temporal resolution were also demonstrated.\(^4\) These effects may be related to damage to IHCs and/or auditory neurons in spite of normal hearing thresholds, since auditory sensitivity primarily reflects the state of the OHCs. There is a change in synapse number and structure after noise exposure,\(^5\) even when the noise exposure causes only a temporary hearing threshold shift. This loss of dendrite connections may result in a slow degeneration of spiral ganglion neurons in the high frequency region of the cochlea. More specifically, the low-sensitivity neurons, which are responsible for the signalling of loud sounds, are damaged. This process may explain the reduced speech recognition performance in noise.\(^8,9\)
SPIN-testing for NIHL and tinnitus

Secondary to the degeneration of the afferent dendrites of auditory fibres, spiral ganglion cells undergo neurodegeneration as shown after glutamate-induced excitotoxic trauma in vitro, after intense tone exposure or after long-term mild trauma. Intact or healed IHCs may be associated with a deterioration of active transmitter release sites from the IHC synapse. A larger extent of deafferentation may trigger tinnitus; a lesser extent of deafferentation may instead be linked to hyperacusis. In some individuals, both complaints may exist. These subtle changes can influence brain responses. Schaette and McAlpine have suggested that a reduction in neural input can lead to a compensatory increase of neural gain in the auditory brainstem. Due to an increase in the spontaneous activity of the auditory neurons, tinnitus can occur.

On the other hand, cochlear damage does not induce tinnitus in some cases. Therefore, it is believed that maladaptive neuronal plasticity of the central auditory system, triggered by preceding cochlear damage and/or deafferentation, is one of the general neurophysiological models explaining the selective occurrence of tinnitus after cochlear damage.

From a clinical point of view, a high number of patients present with hidden hearing loss. Therefore, further hearing investigation is important in order to improve diagnosis. The authors examined whether the DTT would be an effective addition to standard hearing testing. Earlier research has shown that the DTT is a reliable test for detecting different degrees of high-frequency hearing loss, even when this would not be noticed in pure tone audiometry. It also has the advantage of being a quick and automated self-test that does not need to be conducted in a soundproof booth. Noise intolerance (which is frequently present within tinnitus subjects) was not reported during the DTT in these subjects, due to the comfortable noise volume during the test. Regarding aggravation, the DTT seems robust. This could be important for acoustic trauma that has occurred in work-related conditions (with insurance involvement etc.).

A statistically significant correlation was confirmed between the high PTA1,2,4 kHz and averaged SRT across ears in noise on the DTT within this study population. The correlation, however, was weak. This might be explained by the homogeneity of the population. There were strong age and audiometric restrictions, primarily to enable a focus on the added value of the DTT.

Earlier studies have already shown a significant increase in SRT scores in tinnitus subjects with normal hearing, both in quiet and in noise, compared to a normal hearing age-matched control group without tinnitus complaints. In a small group of tinnitus patients with normal hearing (n = 6) and unilateral tinnitus, Moon et al. also found a

![Figure 4](image)  
**Figure 4** Correlation between the averaged PTA1,2 and 4 kHz (dB HL) and the averaged SRT (dB SNR).

![Figure 5](image)  
**Figure 5** Boxplots of the averaged SRT for different tinnitus onset categories.
significantly worsened SRT score in comparison to a normal control group, using an open-set test with spondee words in the presence of speech-shaped steady noise. In addition, the patient group with hearing loss and unilateral hearing loss showed a decrease in SRT under all test conditions. It is possible that noise that is only presented at the side of the tinnitus (in case of unilateral tinnitus) could create an increase of speech understanding, due to a reduced masking effect.23 These findings suggest that tinnitus may affect the central auditory system as a central masker, when patients are engaged in listening to speech in the presence of background noise.

In our population, there also seemed to be a slight negative effect of tinnitus onset, related to the start of hearing problems, on the SRT. From a clinical perspective, these data also suggest a negative influence of worsened SRT on the prognosis of tinnitus. The authors suggest that tinnitus patients with worsened SRTs should receive active counselling and will require further therapy. Recent evidence shows a clear benefit from cognitive behaviour therapy.24 In this study, 245 patients received a stepped-care specialized tinnitus approach, compared to 247 patients receiving usual care (including tinnitus retraining therapy). Patients showed a significant decrease in tinnitus severity and impairment.

Conclusion

Standard hearing testing does not always suffice in the detection of mild acoustic trauma or in explaining tinnitus complaints. The DTT seems to be a valuable addition to the standard test protocol. Information about relevant subclinical changes is gained in a rapid and efficient way. It is possible that more active treatment options for tinnitus complaints could be developed based on these findings, once validated in future research.

Take-home messages

Tests for noise-induced hearing loss, with or without tinnitus, should include more complex tasks such as speech-in-noise testing.

1. Normal pure tone audiometry cannot exclude underlying deafferentiation.
2. Digit triplet testing allows rapid and reliable testing for patients with NIHL and tinnitus.
3. A longer period of tinnitus has been associated with a trend towards worse SRTs.

Acknowledgements

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References

14. Smoorenburg GF. Speech reception in quiet and in noisy conditions by individuals with noise-induced hearing


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