Rigshospitalet

UNIVERSITY OF COPENHAGEN

PRIMARY NEURAL DEGENERATION FROM NOISE EXPOSURE

– AN INVISIBLE NOISE-INDUCED HEARING IMPAIRMENT

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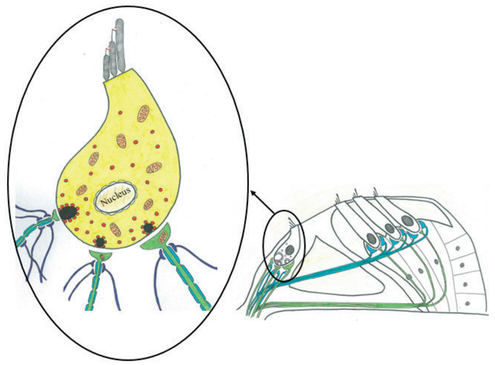
This study was conducted at Copenhagen University Hospital "Rigshospitalet" where office area and test facilities were at my disposal.

INTRODUCTION

Noise exposure has been found to produce primary, permanent hair cell loss within minutes to hours and rapid, but reversible damage to nerve. Loss of spiral ganglion cells (SGCs) however was not found for weeks to months after exposure. This is why we until now have accepted hair cells as the primary damaged targets from noise exposure.

Today new studies on mammals (mice and guinea pigs) show that loss of spiral ganglion nerve fibers from noise exposure can be extensive and permanent, even when there is no hair cell damage (Kujawa & Liberman 2009; Lin et al., 2011; Furman et al., 2013). This suggests that the peripheral type I spiral ganglion neurons can be primary damaged targets from noise exposure. The research from Furman et al., (2013) further suggests that the afferent fibers with low spontaneous rate firing and high thresholds are more vulnerable to noise exposure.



WHY IS THIS PROBLEMATIC?

Such neuronal damage could be of serious impact to an individual´s everyday communication!

The measurement tools used today to screen for a NIHL might be insufficient!

These studies revealed primary synaptic damage to IHC areas (synaptic ribbons) and postsynaptic nerve damage causing reduced wave I amplitudes of suprathreshold ABR while preserving threshold sensitivity, DPOAEs and normal hair cell structure. Thus this damage can be present without affecting thresholds measured with pure-tone audiometry. Assuming low spontaneous rate fibers are more prone to damage from noise exposure also suggests that such nerve damage would cause a hearing impairment degrading suprathreshold processing ability alone. It has been suggested that LSRFs are important for the processing of auditory stimuli in the presence of high-level background noise, as these fibers have shown greater resistance to the limitations of saturation from high noise levels (Costalupes et al., 1984). A great deal of human communication or listening tasks take place in the presence of background noise. Such neuronal damage could thus be of serious impact to an individual´s everyday communication. If these assumptions are true, the measurement tools used today to screen for a NIHL might be insufficient.

RESEARCH QUESTIONS

Are the results from experiments on mice and guinea pigs transferrable or applicable to humans?

1. Is it possible that neuronal damage might be primary damage from excessive noise exposure, and thus occurs and affects our hearing before it is measurable on pure-tone audiograms?
2. Will suprathreshold measurements like speech recognition threshold in noise (SRTN) and ABR at suprathreshold level show deviations in terms of lower amplitude of wave I (µV) and need of a (smaller) better dB SNR to obtain SRTN in the results from noise-exposed individuals, compared to a control group, while normal thresholds from pure-tone audiograms are present in both groups?
3. Should suprathreshold measurements like SRTN and ABR at suprathreshold level be included in the test battery, when screening for a noise-induced hearing loss?

METHOD

To address these research questions, two groups were recruited and tested: one test group of noise-exposed individuals, and one control group, considered a non-exposed group. For both groups a basic hearing evaluation was completed (basic audiometry procedure) after which they were tested with suprathreshold measurements (SRTN and ABR). Results were compared and analyzed to determine if significant deviations were present in the noise-exposed group from the control group.

STEP 1 - Baseline measurements

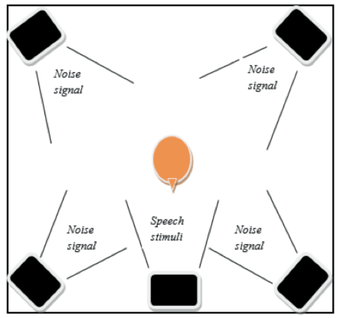
Pure-tone audiometry, speech recognition threshold and discrimination score were measured as a first step to ensure normal hearing sensitivity ≤ 20 dB HL and normal processing of speech of all participants in both groups.

STEP 2 - Speech recognition threshold in noise

This test was employed to measure the dB SNR necessary for the subject to understand 50% of the speech signal in the presence of background noise.

Test setup and apparatus

The speech material was presented binaurally in the sound-field environment of the double-walled sound-proof booth, using speakers to present the speech and noise signal. The speech material "DANTALE II" (Ardenkjær & Josvassen, 2001; Wagener et al., 2003) was used. The test was conducted using the instruction set and procedure recommended by Hansen & Ludvigsen (2001). The speech signal was presented from a front loudspeaker (0° azimuth). Simultaneously noise fixed at a level of 70 dB SPL was presented from four loudspeakers, two placed at ± 45° and two ± 135° azimuths. All subjects were seated at the exact same position in the sound booth to obtain equal test conditions for all participants.



This figure depicts the SRTN test setup. Four loudspeakers presenting noise: two placed at ± 45° and two at ± 135° azimuth. One loudspeaker at 0° azimuth presented the speech signal (sentences). All loudspeakers were placed at a distance of more than 1 meter to the listener, pointing towards the subject and were placed approximately in the same height as the head position of the subjects.

STEP 3 – Auditory brainstem response

The Interacoustics ABR system (EP15/EP25) "the Eclipse" was used to perform the ABR measurement. Etymotic Research (ER-3A) insert earphones were the transducers used for this measurement. Sensing surface electrodes measured the response.

Recording Parameters (settings) of the ABR measurement	
Stimulus	Click
Stimulus rate	16.1 per sec.
Stimulus level	90 dB nHL
Stimulus polarity	Alternating
High pass filter (for input amp.)	33 Hz 6/oct.
Low pass filter (for input amp.)	4000
Measuring time/time window)	0.0 – 20.0 ms
Sweeps	4000

STEP 4 – statistical analysis

The statistical non-parametric method "Mann-Whitney *U*" was employed to examine if any differences found between the groups were of statistical significance. Pearson´s *r* was employed to investigate if a correlation between the amplitudes of wave I and the SRTN results could be confirmed

RESULTS

For speech recognition threshold in noise a significant difference between the two groups is indicated ($p < 0.001$). The data and calculations thus clearly indicate that the noise-exposed test subjects need significantly smaller difference in dB SNR than the non-exposed subjects in the control group, in order to understand 50% of the speech signal.

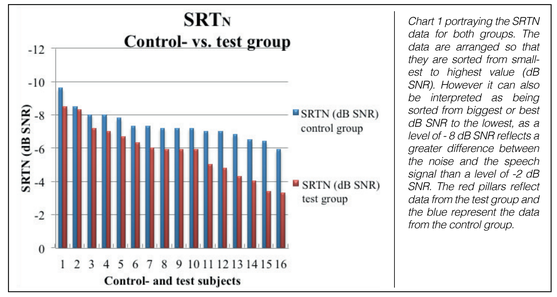


Chart 1 portraying the SRTN data for both groups. The data are arranged so that they are sorted from smallest to highest value (dB SNR). However it can also be interpreted as being sorted from biggest or best dB SNR to the lowest, as a level of - 8 dB SNR reflects a greater difference between the noise and the speech signal than a level of -2 dB SNR. The red pillars reflect data from the test group and the blue represent the data from the control group.

No significant difference was found between the groups in regards to right ear amplitudes of wave I. However for the left ear amplitudes the noise-exposed test subjects have significantly lower wave I amplitudes than the non-exposed control group. A significant difference is indicated between the two groups ($p < 0.05$).

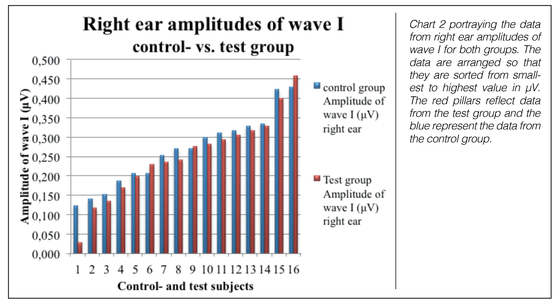


Chart 2 portraying the data from right ear amplitudes of wave I for both groups. The data are arranged so that they are sorted from smallest to highest value in µV. The red pillars reflect data from the test group and the blue represent the data from the control group.

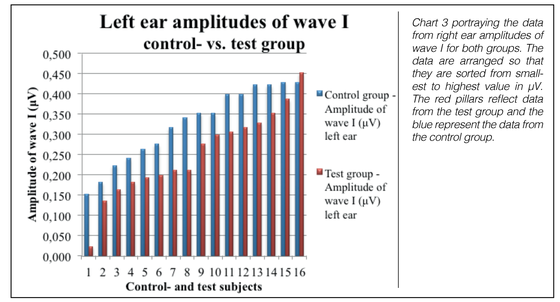


Chart 3 portraying the data from right ear amplitudes of wave I for both groups. The data are arranged so that they are sorted from smallest to highest value in µV. The red pillars reflect data from the test group and the blue represent the data from the control group.

Mann-Whitney *U* was also calculated using only the ears stated as being most exposed by twelve test subjects and their twelve matched control subjects. A significant difference between the two groups was verified ($p < 0.025$). The calculation indicates that the noise-exposed test subjects have significantly lower wave I amplitudes.

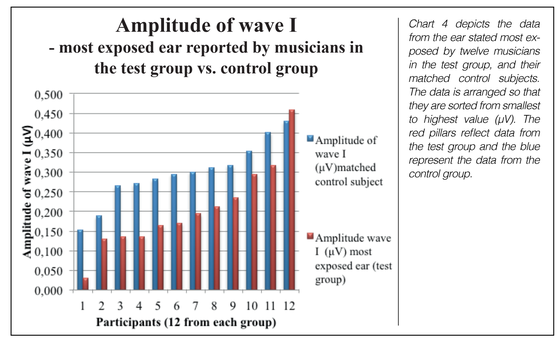


Chart 4 depicts the data from the ear stated most exposed by twelve musicians in the test group, and their matched control subjects. The data is arranged so that they are sorted from smallest to highest value (µV). The red pillars reflect data from the test group and the blue represent the data from the control group.

CONCLUSION

Noise-exposed test subjects required a significantly better dB SNR ratio to obtain SRTN despite normal threshold sensitivity. In other words they had significantly reduced SRTN compared to the non-noise exposed control group. These results provide evidence that repeated excessive noise exposure on human individuals causes reduced speech recognition threshold in noise in the absence of elevated pure-tone thresholds. Significantly reduced wave I amplitudes to suprathreshold stimuli were also confirmed in the test group, despite normal pure-tone sensitivity when using data from the ear reported as most exposed. This too can support the finding from animal studies. It provides support that noise exposure to humans causes reduced synchronised firing from the peripheral afferent SGCs in the absence of elevated pure-tone thresholds.

This study on suprathreshold processing ability of noise-exposed human subjects suggests that the results of noise-induced primary neural degeneration found in experiments on noise-exposed mice and guinea also apply to humans.

Overall these results offer evidence of noise-induced primary neural and synaptic degeneration. The study supports the finding that this primary neural degeneration has a trend for selective loss of LSRFs. Furthermore it supports that excessive noise exposure to humans can cause primary neuronal damage that affects suprathreshold hearing ability before it is measureable on pure-tone audiograms. Such neuronal damage from noise exposure is invisible to the test of pure-tone audiometry. Thus the audiometric threshold measurement does not reflect the entire impairment and truth about noise-induced hearing impairment .These findings strongly recommend a need to include measurements of SRTN, when screening for NIHL, in order to obtain a more accurate overview of the potential consequences from noise exposure.

References

Ardenkjær-Madsen, R., & Josvassen, J. L. (2001). Forslag til en ny dansk sætningsbaseret audiovisuel taleaudiometrisk test i støj. MA thesis in Speech and Hearing Sciences, University of Copenhagen.

Costalupes, J. A., Young, E. D., & Gibson, D. J. (1984). Effects of continuous noise backgrounds on rate response auditory nerve fibers in cat. J neurophysiol 51:1326-1344.

Furman, A.C., Kujawa, S.G., & Liberman M.C. (2013). Noise induced cochlear neuropathy is selective for fibers with low spontaneous rates. J neurophysiol 110:577-586.

Hansen, M., & Ludvigsen, C. (2001). Dantale II – danske Hagerman sætninger. Danske tale audiometrimateriale. Værløse: DTAS.

Kujawa, S. G., & Liberman, M. C. (2009). Adding Insult to injury: Cochlear Nerve Degeneration after "Temporary" Noise-Induced Hearing Loss. J Neurosci 29:14077-14085.

Lin, H. W., Furman, A. C., Kujawa, S.G., & Liberman, M. C. (2011). Primary neural degeneration in the guinea pig cochlea after reversible noise-induced threshold shift. J Assoc Res Otolaryngol 12:605-616.

Wagener, K., Josvassen, J. L., & Ardenkjaer-Madsen, R. (2003). Design, optimization and evaluation of a Danish sentence test in noise. International Journal of Audiology; 42:10-1

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