

## Case Report

# Synaptopathy Following Noise Trauma - A Case Report

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## Abstract

**Case history:** A 41-year-old patient suffered from hearing loss and tinnitus on his right ear, following a car accident with airbag deployment. Hearing loss recovered partially, Tinnitus and difficulties in speech discrimination sustained.

**Findings:** Audiometry showed typical high frequency hearing loss (40 dB) and tonal tinnitus (8kHz). However, DPOAE and ABR-potentials (Wave III and V) were completely normal, but there was no detectable CAP in ECochG.

**Discussion:** These findings indicate a recovery of the initial hair cell damage, whereas the synaptic transformation remains reduced and a slight hearing loss persists. This phenomena has been described as hidden hearing loss in the newer literature.

**Conclusion:** Noise trauma initially results in hair cell damage, but after recovery hearing loss can persist that can be due to synaptic lesions in the first neuron.

## Keywords

- Noise trauma
- Synaptopathy
- Hidden hearing loss
- Tinnitus

## INTRODUCTION

In general noise trauma results in the damage of outer hair cells, mostly in the 4-6 kHz frequency range [1]. However, latest research hints to the fact, that noise exposure can also lead to lesions in the first synapse and other neuronal changes, either additionally or even with intact hair cells [2]. There is little data available supporting this diagnosis for humans. However, the fact that lesions can persist or even exist with normal audiometric findings has a high medico-legal and socio-economic impact. We present a case that seems to proof the fact of retro cochlear lesions after noise trauma.

## CASE PRESENTATION

The 41 year old male patient suffered from a fatale vehicle crash (collision with a wild bore), without own misbehaviour. Both frontal air bags deployed with a high intensity noise. Symptoms were tinnitus and subjective hearing loss with ear pressure, but without signs of vertigo, there were no other injuries. ENT-examination (otoscopy) showed no clinically relevant findings; audiogram was normal on the left side, right sided there was a high-frequency hearing loss starting from 2 kHz up to 50 dB at 6 kHz. Tinnitus was measured at 6 kHz with a loudness of 50 dB. After high dose systemic steroid injections (500 mg prednisolone) hearing recovered to 40 dB, ear pressure vanished and subjectively hearing improved, but not to normal. Because of a persisting tinnitus intratympanic steroid therapy followed and resulted in a further though not complete recovery.

Due to persisting tinnitus complaints the patient was admitted to our clinic.

## Audiometric findings

We saw normal audiometric findings on the left side, but a hearing loss on the right side starting from 3 kHz up to 55 dB at 8 kHz. Monosyllable speech understanding was 95 % at 65 dB, tinnitus was measured at 8 kHz with an intensity of 60 dB (10 dB above threshold) (Figure 1). Speech discrimination in noise (Oldenburg sentence testing - OLSA) was normal in both ears (-4 dB for a 50% threshold of discrimination).

Distortion products of otoacoustic emissions (DPOAE) showed bilaterally sufficient emissions for all frequencies from 100 to 8000 Hz, indicating normal function of the inner ear outer hair cells even on the right ear (Figure 2).

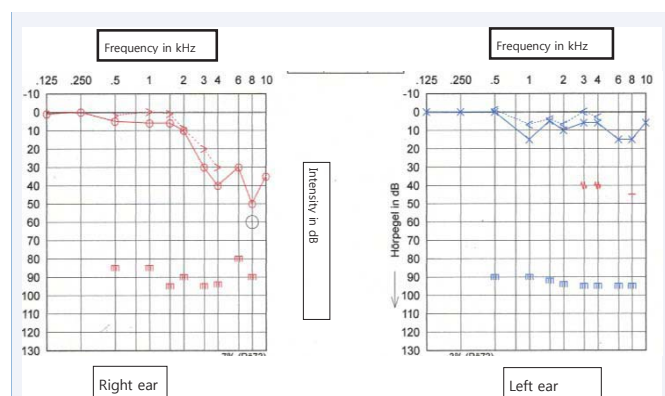
BERA findings showed normal latencies and amplitudes for wave III and V down to 50 dB intensities bilaterally, but in the electrocochleography (extratympanically recorded with alternating clicks) there was no proper compound action potential (CAP) on the right ear even for a 90 dB stimulus (Figure 3).

## Evaluation of diagnostic findings

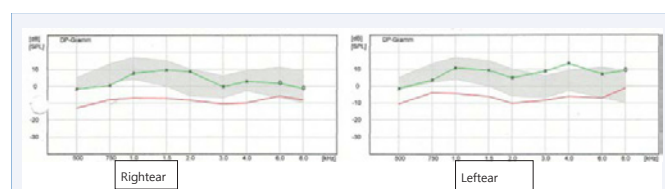
With our patient outer hair cells completely recovered after initial defect, but there was a detectable insufficiency of the first cochlear neuron, detected by a very weak CAP, whereas other brainstem potentials appeared to be normal. This hints to a cochlear synaptopathy [3], caused by the noise trauma.

## Therapy

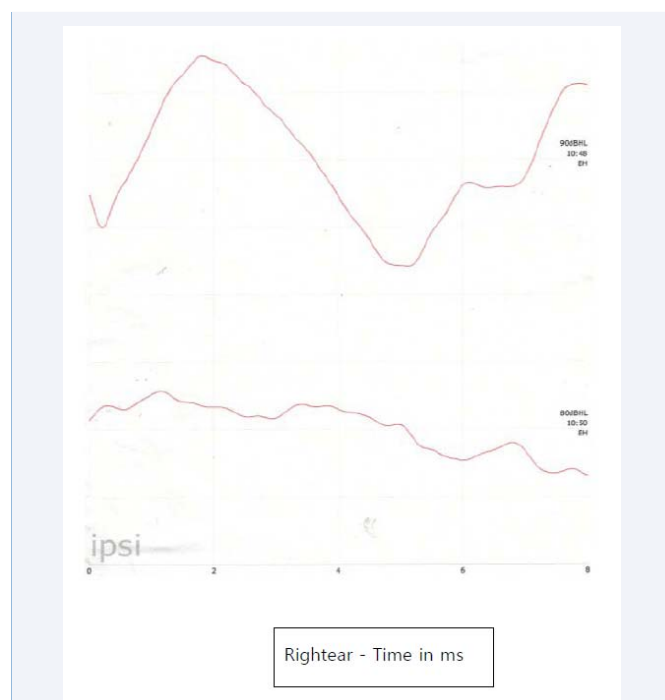
Patient was supplied with a unilateral (right) hearing aid and



**Figure 1** Audiogram of a patients 12 month after noise trauma (air-bag deployment): high frequency hearing loss starting from 3 kHz, tinnitus ( O ) measured at 8 kHz with 60 dB, 10 dB above subjective threshold, normal discomfort levels ( ■ ).



**Figure 2** DPOAE-recordings of the patient with high frequency hearing loss: Completely normal DPOAE bilaterally, even in the high frequencies for intensities down to 40 dB SPL (here: 80 dB) – (grey mark= normal DP-Gram; green line= patients recordings; red line= random noise).



**Figure 3** ECoG of this patient (right ear), recorded extratympanically with alternating clicks, 90 and 80 dB SPL: No detectable compound action potential (CAP)– (Wave III and wave V in BERA-recording was normal).

treated with a specific audiototherapy [4]. 4 weeks later tinnitus was still detectable but much less bothering; the subjective hearing threshold had improved for 10 dB only for 8 kHz, other frequencies and objective findings (BERA and DPOAE) remained unchanged.

## DISCUSSION

Airbag deployment in a car crash produces high sound pressure levels of 145-165 dB for an extremely short time [5], resulting in possible noise trauma; for the driver (left side drive) the risk for the right ear is slightly higher [6]. Our patient was driving alone, but all airbags deployed, the right airbag was more voluminous and led to a higher noise-burden on this side.

Hearing loss following noise trauma, especially after car accidents and especially when the victim is not responsible for the accident can lead to a great degree of psychosomatic burden and even persistence of a psychogenic hearing loss [7].

Therefore the first conclusion after finding audiometric hearing loss with normal outer hair cell function was this diagnosis. However, speech discrimination did not fit into this diagnosis [8,9], but the absence of the CAP in our ECoG-recording hinted to a deficiency in the cochlear synapsis.

Even if a noise trauma initially can lead to outer hair cell injury, after recovery (supported by steroid therapy) hearing loss can persist due to retrocochlear lesions regarding the synaptic communication between sensory inner hair cells and subsets of cochlear nerve fibres [10]. In animal models this has been proven [9,11], for humans this could only be detected after high level noise exposures [12].

Other researchers however did not find any change in subjective audiometric testing and brain stem potentials after noise exposure, mainly after exposition to high levels of music in concerts and after using stereo devices [13,14]. Definitely the exposition of our patient after air-bag deployment was considerably higher, leading to initial outer hair cell damage. Also the exposition for his right ear was higher than for the left ear. After recovery, a certain degree of hearing loss sustained, and the only objectively measurable deficiency was the loss of a proper CAP. However, there is a limitation regarding the question, whether ECoG-recording is suitable for the detection of cochlear dysfunction when hearing loss is present. Because even the 90 B stimulates did not show any reproducible CAP, we consider this a hint to cochlear synaptic insufficiency.

Also it seems unclear why more complex audiometric tasks like discriminations of sentences in random noise appeared to be normal, whereas the hearing threshold showed a high-frequency hearing loss, that recovered slightly with audio-therapy. Therefore it should be interesting, whether this presumably synaptic hearing loss can be reversed, especially with proper acoustic stimulation [15]?

A control examination after one or two years should answer this question.

## CONCLUSION

High levels of noise trauma can lead to hearing loss due to damage of outer hair cells. Adding to this, retrocochlear structures

and mainly the extremely fast and vulnerable synapses between inner hair cells and the first neurone can be damaged by noise exposure, resulting in high frequency hearing loss. Possibly this hearing loss can recover after proper acoustic stimulation.

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