

RESEARCH ON THE EFFECTS OF INDUSTRIAL NOISE ON PIECES OF HUMAN NECROPSY. CASE REPORT

CĂLIN MOHOR¹

¹“Lucian Blaga” University of Sibiu

Keywords: industrial noise, local effects, organ of Corti, necropsy sections

Abstract: We started from the premise that people who have worked for a long time in environments with industrial noise may come up with histopathological changes of the auditory analyzer. By analogy with the study conducted on laboratory animals, which outlined the changes at the level of the organ of Corti after prolonged exposure to industrial noise, I have sought to highlight the same changes in the auditory analyzer of workers exposed to industrial noise, who have worked in a weaving factory, using for this purpose a case study.

Cuvinte cheie: zgomot industrial, efecte locale, organul Corti, piese de necropsie

Rezumat: S-a pornit de la premisa că la persoanele care au lucrat o perioadă îndelungată în mediu cu zgomot industrial pot apare modificări histopatologice marcate la nivelul analizatorului auditiv. Prin analogie cu studiul efectuat pe animale de laborator, care a evidențiat modificări la nivelul organului Corti după expunerea îndelungată la zgomot industrial, am căutat să evidențiem aceleași modificări și la nivelul analizatorului auditiv al muncitorilor expuși la zgomotul industrial, care și-au desfășurat activitatea în țesătorie, utilizând pentru aceasta un studiu de caz.

INTRODUCTION

Multiple studies on the effects of industrial noise on the health of workers from a weaving factory have presented various researches. These references have highlighted the results of the research and not the histopathological features or aspects of the pathogenesis. That is why, I seemed useful to present an experimental research on the changes produced by industrial noise to the reception organ, the Corti's organ, on pieces of human necropsy.

To determine the level of changes on the organ of Corti, I needed histopathological examination of the spiral organ of Corti consisting of sensory hearing cells. Exposure to high intensity noise leads to damage of the stereocils of the ciliate cells, which have an essential role in the mechanical sound transduction and transformation into electric stimulus that induces the appearance of hearing loss. That is why, in the current paper, I will track changes in the organ of Corti to globally understand and explain the occurrence of hearing loss and deafness. By presenting the results I seek to emphasize the correlations between the exposure to industrial noise and the appearance of deafness and hearing loss.

Working hypothesis

Literature data suggests that exposure to industrial noise determines the occurrence of changes in the inner ear by age.

Hearing disorders are an increasing public health problem impacting on the quality of life, with permanent consequences over the staff's exposed to noise.

In literature, there is not much data on changes in the organ of Corti, assessed by histopathological examinations in human subjects exposed to industrial noise. Therefore, through this study, I have tried to make a parallel between changes in animals exposed to the experience of industrial noise and human subjects that operated in the environment of industrial noise.

In a recent study, carried out on a large number of subjects with changes in hearing, Gates and his colleagues (2000) (1) observed that the changes produced by the noise in the cochlea are even more accentuated if exposure to noise has been extended. This issue of hearing loss due to noise exposure has significance for public health (Gates 2000, Rosehall Gates 2003, Lee 2005) (1,2,3) given the high prevalence of exposure to noise. Concerns about the long-term effects of exposure to noise in the inner ear are heightened by the increasing prevalence of hearing loss in relation to noise, from a younger age all (Wallhagen, 1997, National Institute of health 2000, Folmer, 2002).(4,5) However addressing exposure to noise in human studies is difficult.

Nadol, in 1988, had highlighted the degeneration of internal and external ciliate cells on human temporal bones.(6)

The Hu's study, from 2002 and Nicotera, 2003, both showed that exposure to high intensity noise, for a short period of time caused damage to the cells by necrosis and apoptosis.

Study of Bohne and Clarke in 1982, conducted on experimental animals exposed to noise for 24 hours over a period of 6 months, could not highlight significant changes at a histopathological examination of the organ of Corti.

In 2006, Henderson had shown, in its study, a relationship between prolonged exposure to high intensity noise and loss of contact at the level of the organ of Corti between Deiters' support cells and ciliate cells.(7)

It started from the premise that people who have worked for a long time in environments with industrial noise may come up with important changes of the histopathological auditory analyzer. By analogy with the study conducted on laboratory animals, which outlined the changes at the level of the organ of Corti after prolonged exposure to industrial noise, we have sought to highlight the same changes in the auditory analyzer of workers exposed to industrial noise, who have worked in a weaving factory, using for this purpose a case study.

¹Corresponding author: Călin Mohor, Str. Lucian Blaga, Nr. 2A, Sibiu, România, E-mail: calinmohor@gmail.com, Tel: +40745 528270
Article received on 10.02.2014 and accepted for publication on 10.04.2014
ACTA MEDICA TRANSILVANICA June 2014;2(2):274-278

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METHODS

The research starts on the temporal bone harvested from patient I.M., female, in 2009, who had passed away from a heart disease at the age of 60. The patient was monitored over the past 5 years. Patient's monitoring and the collection of temporal bone was done with the patient's approval during life.

Figure no. 1. The left temporal bone harvested from the human subject



Heredo-collateral antecedents: irrelevant.

Personal physiological history: menarche at 16 years, 4 pregnancies, 2 births, 2 abortions, physiological menopause at 52 years.

Personal history of illnesses: childhood diseases, high blood pressure stage II, chronic ischemic cardiomyopathy, grade II obesity and hydrostatic varicose veins at the lower limbs, bilateral hearing loss.

Living conditions – appropriate

Professional anamnesis: the subject has worked for 42 years at the „Red Silk” factory in Cisnădie.

Occupation: weaver.

Occupational length at the time of death: 39, followed by 3 years of professional pension.

Professional way: the subject had graduated a vocational school with professional practice on the weaving machine. The subject has never changed its workplace.

Technological process: loom weaving.

Operations fulfilled by the weaver: 3 shifts, work shift I between 7 - 15 o'clock, work shift II between 15 – 23 o'clock, work shift III between 23 – 7 o'clock. The activity was held in the weaving workshop, about 1 m away from the weaving machine.

Workplace characteristics: the weaving workshop has about 650 sqm of surface in which 30 weavers were operating, natural and artificial lighting, natural ventilation through the natural opening of doors and windows.

Professional nuisances: a) physical factors: noise, air currents, lighting.

b) physico-chemical factors:
textile dust.

Means of collective protection: sound-absorbant and phono insulating surfaces.

Individual protection means: helmets, ear protectors.

Work equipment: overalls, scarves.

Symptoms at work: irritability, fatigue, decreased ability of auditory perception, tachycardia.

Disorders present in other workers: fatigue, asthenia, headache, migraine, syncope, irritability, depression, decreased ability of auditory perception, balance disorders, tachycardia, palpitations, visual disturbances.

Periodical medical examination: clinical examination, general, ophthalmology, ENT, neurological, psychological, audiogram, cardiology exam, electrocardiogram.

Pathological antecedents: bilateral hearing loss-1999, bilateral deafness-2006.

Occupational exposure through objectification of noise measurements in various areas of textile showed values of 85-107 dB.

Clinical-stage: the patient hears during the hearing loss period;

-at the stage of deafness she does not hear: instability, insomnia, tinnitus, dizziness, tachycardia

Paraclinical examinations: - liminary tonal audiometry performed in standardized conditions (gives an indication of the type of deafness, hearing impairment, the frequencies concerned)

Study methodology

Liminary tonal audiometry conducted in 2002, 2009.

Description of method

Methodology for investigating auditory function of the worker exposed to industrial noise, liminary tone audiometry technique.

Liminary - that aims to establish the lower threshold of audibility (limit of audibility, or lowest sound intensity that causes an audible sensation).

Tone - because audiometer tones are pure tones (not complex sound or human voice).

Audiometric testing was performed, according to the quality standard required by EU rules, Sibiu County Hospital, the Otolaryngology Department.

The patient was exposed to noise by a pure tone generator with frequencies: 125, 250, 500, 1000, 2000, 4000 and 8000 Hz, with a pure tone transmission to the inner ear of the subject investigated including microphone headset that is put on the auricle air transmission path (CA) and which is placed on the mastoid vibrator (bone driving path (CS) to a moderator of the intensity of the sound, with a signal (which indicates the perception of the subject sound - the flare).

I observed the basic conditions for conducting audiometry, namely:

- Subject investigated had an ear rest for 16 hours without exposure to noise.
- The room in which it audiometric testing must be soundproofed.
- Pipes hearing of the subject investigated were free, without cerumen plugs.

It started with the right ear, then with the left ear and then I continued with gradually increasing intensity of the sound in the headset. The results were recorded graphically - audiogram, separately for the right ear and left ear separately for air conduction and bone conduction. Normally, air conduction (AC) is in full lines and bone conduction (CO) dashed. In interpreting the results of audiometric we took into account:

- Frequency of occurrence of auditory deficit (e.g. 500, 1000, 2000 or 4000 Hz).
- The intensity of the auditory deficit, i.e. hearing loss expressed in dB.
- The behaviour of the two types of leadership (and CO).
- Age of the subject investigated.
- Definition of hearing loss and deafness professional.

RESULTS

The results of audiometric interpretation

A) Audiogram Interpretation: bilateral perceptible hearing loss -2002.

I.M., 50 years old, female weaver weaving textile enterprise section.

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Seniority: Total - 32 years , the noise - 32 years.

Noise levels from 85-107 dB current job.

No permanent earplugs wearing.

Date of the audiometry – 2002.

Diagnosis: professional hearing loss with permanent hearing loss 61 dB at a frequency of 4000 Hz (UD) and 56 dB at a frequency of 4000 Hz (U.S.). After correction for presbycusis, hearing loss remains UD with permanent hearing loss of 23 dB .

B) Interpretation audiogram - 2009. Bilateral perceptive deafness

I.M., 57 years old, female weaver weaving textile enterprise section.

Seniority: Total - 39 years , the noise - 39 years.

Noise levels from 85-107 dB current job.

No permanent earplugs wearing.

Date of the audiometry – 2009.

Diagnosis: occupational deafness UD with permanent hearing loss of 47 dB at average frequencies of 500, 1000 and 2000 Hz and permanent hearing loss 63 dB at 4000 Hz, after correction for presbycusis - deafness UD with permanent hearing loss of 35 dB.

Occupational deafness U.S. with permanent hearing loss of 45 dB in average frequencies 500, 1000 and 2000 Hz and permanent hearing loss 58 dB at 4000 Hz, after correction for presbycusis - deafness UD with permanent hearing loss of 33 dB

Histopathological examination of the reception organ (the Corti ganglione)

a) Harvesting – the harvesting has been made by the pathologist together with the author of the paper in the prosecture chamber using the scalpel, chisel, saw, immediately after the death of the subject, in order to avoid any deterioration of the tissues in the inner ear. Tissue had been taken from the temporal bone that contains the middle and internal ears along with some surrounding tissue to avoid macroscopic lesions at the level of the piece, with the medial and lateral sagittal sections, and front and rear sections. The entire fragment of piece was washed with saline solution under slight pressure and then inserted into 20% formalin for about 2 hours. After that the piece was easily cut and brought to the optimum size (the inner ear) for further processing. The thickness of the piece was about 5 mm.

b) Fixing – the fixation is the second operation in the technological flow to obtain a permanent histological preparation.

c) Washing – the purpose of washing is to stop the process of fixation, by removing the fixing agent. Washing was done with tap water, after fixation with formalin, left to run continuously across the pieces. Washing was immediately followed by dehydration and inclusion, the paraffin block being the environment in which the pieces are best preserved.

d) Decalcification – in order to obtain thin sections of calcificated tissues (bone), proper decalcification of the parts was needed. There are chemical methods in which insoluble calcium salts are extracted, either by transforming into soluble compounds with mineral acids or organic, chelating agents, or by means of the ion exchange resins or physical methods, by electrolysis. The decalcifier agent used here was EDTA.

e) Parts processing - it can be done in several ways: retinal detachment with the freezing microtome, freezing-inclusion in paraffin wax, which causes overall hardening of the parts very rich in collagen, inconvenient which was eliminated by using butyl alcohol as clarification medium.

f) Inclusion - The inclusion of the parts, after washing and fixation, is an operation that is done in order to create optimal conditions for cutting thin micrometral sections,

transparent, with flat sides, so that they can be properly examined in transmitted light microscope. Masses of inclusion are anhydrous (paraffin, celoidines, certain plastics) and aqueous masses (gelatin).

g) Sectioning – severing, or polygonal microtomia, is a difficult operation. Hystological sections shall be carried out with the help of the mycotome, a high-precision device. Mycotome sections usually measure 5 microns.

The process is followed by display, because paraffin sections present a series of creases which make the parts inappropriate for microscopic examination, and glueing, so that during different operations of staining, dewaxing or impregnation, the sections won't come apart.

h) Staining or coloration – the impregnation process comprises certain conditions and reagents needing to be installed in a special support – the coloration battery - which includes staining dyes, differentiators, mordants, washing liquids etc., as well as hydrocarbon benzenic baths and various concentrations of alcohols, for the dewaxing and the preparation of sections necessary for installation in various media.

In our study we used hematoxiline-eosin color, and Giemsa.

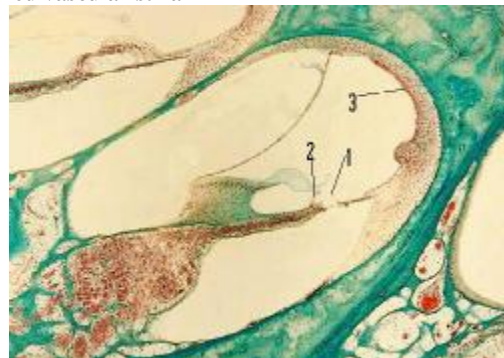
i) Fitting - this stage of permanent microscopical preparation aims to protect fragile section from deterioration, conserving the same color as long as possible and to provide a uniform and transparent environment, optimum for the examination at the mycroscope with transmitted light. The assembling of sections is done between the studying blade and the smaller blade, in different mediums of mounting fluid, but which subsequently solidify.

j) Labelling of histological pieces – the parts will have to be labelled, noting the coloration used, fixative, species and diagnosis on the label.

Results of the histopathological examination

Particularity the case: Histopathological examination of the harvested case showed changes in the organ of Corti, of the internal and external ciliate cells, loss of cells of Deiters and Hensen, both resulting in flattening the organ of Corti and the destruction of the vascular stria on a subject who has worked for a long time in an environment with intense industrial noise in weaving, and who was diagnosed with hearing loss and then deafness, declared as occupational disease.

Figure no. 2. Section in the inner ear, of the human temporal bone harvested from a 60-year-old subject, noticing the almost entire destruction of the internal and external ciliate cells in the organ of Corti, loss of cells of Deiters and Hensen, both resulting in flattening the organ of Corti, vascular stria destruction. Giemsa-Coloration. 1 – destroyed external ciliate cells, 2 destroyed internal ciliate cells, 3-modified vascular stria



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These confirm the changes described in the literature and in our study, in conjunction with the degradation of the receptor organ of Corti from subjects exposed to noise for a long period.

Figure no. 3. Section in the inner ear, of the human temporal bone harvested from the 60 year old subject, the almost total destruction of the internal and external ciliate cells in the organ of Corti, loss of cells of Deiters and Hensen, both resulting in flattening the organ of Corti, Coloration Giemsa, 1- destroyed external ciliate cells, 2-destroyed internal ciliate cells, 3,4-destroyed supportive cells.

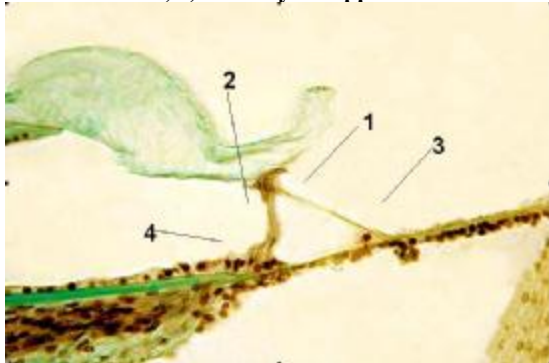


Figure no. 4. Section in the inner ear, of the human temporal bone harvested from the 60 year old subject, the almost total destruction of the internal and external ciliate cells in the organ of Corti, loss of cells of Deiters and Hensen, both resulting in flattening the organ of Corti, 40 x. 1- destroyed external ciliate cells, 2-destroyed internal ciliate cells, 3-destroyed supportive cells



DISCUSSIONS

The correspondence between changes in hearing loss occurring on the audiogram and cellular damage that occur at the organ of Corti could have been thus revealed.

However, what has been discovered in connection with changes in the organ of Corti in animals exposed to the intense industrial noise corresponds with the changes that occur in human subjects exposed to industrial noise for a long time. Similar changes occurred involving the need for an effective prophylaxis of human subjects exposed to industrial noise in order to minimize the impact of industrial noise on the status of their health.

Nadol, in 1988, a study carried out in human temporal bones, revealed the absence of ganglion of Corti and destroying nerve cell destruction ciliates with internal and external.

The correlation between the audiometric data and post-mortem analysis of the cochlea of human subjects and animals exposed to industrial noise of high intensity provides convincing evidence that the degeneration of the internal and

external ciliate cells plays an extremely important role in many cases of hearing loss (Johnson, 1997). In most cases, the external ciliate cells degenerate first, followed by the internal ciliate cells (Schuknecht, 1976; Spongr, 1997). They've also noticed the destruction of the vascular stria.

The cochlear pathology associated with hearing loss after exposure to industrial noise, encompasses the entire cell systems of the cochlea (j. Schacht, Popper, Fay).

Cooper and Owen, in 1976, and Kryter, 1985, have developed a statistical study that showed that the interaction between age and exposure to industrial noise leads to progression of hearing loss on human subjects. Gates, 2000, and 2003, Rosenhall confirmed in a study on two batches of human subjects, that subjects of the same age exposed to industrial noise had a significant loss of hearing in subjects of the same age non-exposed to noise.

Joni Linthium and Fred Doherty Jr., from the House Ear Institute Eccles, conducted a histological study of the spiral ligament in cochlear otosclerosis, trying to establish a role between ion transporter and neurosensory hearing loss pathogenesis.

The authors have studied 79 temporal bones of patients with cochlear otosclerosis, from 59 donor patients. The average age of death was 71 years (47-90).

All the bones were placed in formaldehyde, decalcified with EDTA, placed in wax and sectioned to 20 microns. Every tenth section was coloured with hematoxylin and examined under a microscope by the blade. Intermediate sections were preserved for immunohistochemical studies.

The cochlea was examined by Dr. Joni Docherty as they've chased the degree of hyalinisation of the spiral ligament and the degree of atrophy, increasing sections of 73.25 times mounted and by measuring the coiled and vascular streak along hyalinisation trails. To rule out other causes of neurosensory hearing loss, they've rated number of cells and ganglions of ciliate cells. In the 31 temporal bone with the organ of Corti normally populated by ciliates and spiral lymph cells, audiometric data were compared with those of a hyalinized ligament and atrophied vascular stria used to detect if it has to do with the neurosensory hypoacusis

Frequencies of 500, 1000, and 2000 hertz have been used. Transport of molecules expression Na-K-ATP-ASE, connexin 26, carbonic anhydrase, as well as cell markers such as S-100 and vimentin were evaluated by means of hystoimunochemistry compared with a side wall and one with normal cochlear otosclerosis.

The cochlear segment presented a calcified and hyalinised spiral ligament on an otospongious lesion. The vascular stria, the organ of Corti, peripheral neurons were not modified. It was a 50 Db loss of hearing.

A correlation between the hyalinised ligament and the atrophied vascular stria was used in conjunction with neurosensory hearing loss. Out of 298 cochlear spires measured, 116 showed varying degrees of hyalinisation. 64 of 75 of the bones had hyalinisation of the posterior medium coil, 11 at the anterior middle coil, 13 at the base and 28 at the top of the coil. Temporal bones, in 34 cases with normal organ of Corti, the ciliate cells population had been above 75%, spiral ganglions over 50% while the audiometric data was available for review. An association with statistic importance is between the hyalinisation of the spiral ligament and the atrophy of the vascular stria inside the posterior and anterior apical spire of the cochlea. Also, through the association between both hyalinisations and atrophy of the strias, the average threshold of conduction through the bone was significant in the case of these spires.

There are no significant immunohistological differences between the otosclerotic cochlea and the normal cochlea. In fact, all immunohistological tests did not come up with new things. Some temporal bones with otosclerosis showed declines of NA-K-ATP-ASE, HORSES, and conexine channel 26 in comparison with normal cochlea. This study demonstrates that hyalinisation of the spiral ligament is associated and vascular stria atrophied and deafness may result in both cases. These data demonstrate that the function and structure of the spiral ligament are essential for the survival of the vascular stria. Audiometric data presented showed that the spiral ligament is required for the normal function of the vascular stria, as its hyalinisation is associated with atrophy of the vascular stria and neurosensory hearing loss. The loss of ion transport channels can be due to cochlear otosclerosis and can contribute to the dysfunctions of the vascular stria and ligament.

Significance: - there have been many theories concerning the cause of neurosensory deafness as if it was a cause of cochlear otosclerosis. Often, however, there is evidence of ciliate cell degeneration or neurons to explain the hearing loss of this kind (1944, Guild Schuknecht, Hinojosa and Marion 1983 1987). 29 pairs of temporal bone derived from patients with cochlear implant only 6 (21%) had the cochlear otosclerosis as alleged cause of the total loss of hearing. Research in this case has focused on the dysfunction of the coil as a possible cause of deafness. More preliminary studies have been inconclusive so far, but they can instead show how the ionic transport system is affected by the dysfunction of the spiral ligament.

The cochlea is a complex biological system that requires an increased energy consumption to correctly encode and transmit endocochlear potential, ranging from the vascular stria, the basilar membrane, internal and external ciliate cells to the acoustic nerve fillets. The cochlea is vulnerable to exposure to industrial noise with all cellular systems, support cells, vascular intake, sensory and nervous cells.

Henderson, Bauer, Brozoski, Rybak showed human subjects exposed to industrial noise had suffered from neurosensory hearing loss due to destruction of the ciliate cells, internal and external changes of the inner ear and ischemia.

CONCLUSIONS

1. The industrial noise exposure is the more prolonged the more emergence of cases of occupational hearing loss is more common, as well as the occurrence of deafness (above 20 years old).
2. Almost total destruction of the internal and external hair cells in the organ of Corti could have been observed, as well as loss of support Deiters and Hensen cells, both leading to a flattening of the organ of Corti.
3. What was found in the organ of Corti regarding the changes in animals exposed to intense industrial noise corresponds to the changes that occur in human subjects exposed to industrial noise for a long time.
4. The correspondence between changes occurring in the audiogram and cellular damage that occur in the organ of Corti has been highlighted.
5. Similar changes occurred involve the need for a prophylaxis of human subjects exposed to industrial noise to minimize the impact of industrial noise on their health.
6. Prolonged exposure to industrial noise on long time may constitute in a representative percentage, an etiopathogenic factor favouring the occurrence of occupational disease or work-related diseases (auditory fatigue, hearing loss, deafness etc.).

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