MINI REVIEW

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Tinnitus Footprints in the Cochlea

Carlos Augusto Costa Pires de Oliveira[1]*
Helga Moura Kehrle[2]
Ronaldo Campos Granjeiro[2]
Taciana Sarmento de Oliveira[2]
Maria Cristina Teixeira[2]
Jose Elias da Silva Murad[3]
Miriã Moreira Cardoso Severino[3]

ABSTRACT

The purpose of this paper is to show how temporal bone histopathology has been instrumental in adding knowledge about the origin of tinnitus in the cochlea and how it will still be useful for that purpose in the future. The papers published on this subject will be reviewed, and their contributions will be highlighted. The knowledge that is now part of the subject will be pointed out, and future research on this area will be pointed out.

Keywords: Tinnitus, Temporal bone histopathology, Endolymphatic hydrops, Cochlea origin of tinnitus, Otosclerosis.

[1]Professor of Otolaryngology - Department of Otolaryngology, Brasília University Medical School, Brasília - DF, Brazil
[2]Department of Otolaryngology, Hospital de Base, Brasília, DF, Brazil
[3]Medical Resident of Otolaryngology, Department of Otolaryngology, Hospital de Base, Brasília, DF Brazil

***Send correspondence to**
Carlos A Oliveira,
Professor of Otolaryngology - Department of Otolaryngology, Brasília University Medical School, Brasília - DF, Brazil, Tel: 6184432757. Email: cacpoliveira@brturbo.com.br

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INTRODUCTION

Tinnitus is a phantom auditory sensation, the perception of a sound in the absence of a corresponding sound source. In most cases, tinnitus is associated with hearing loss[1, 2] but whether hearing loss actually causes tinnitus has not been clarified yet. Even though tinnitus is a very common symptom among otologic patients, only I% of them have the severe disabling type that demands innovative treatments, as shown by Oliveira et al[3].

Heller and Bergman found that approximately 94% of people with normal hearing will experience tinnitus when confined in a soundproof booth for five minutes[4].

This paper reports the personal experience of the author with tinnitus studies. At the same time, it shows the importance of temporal bone histopathology in Otological Research. In 1989 the author was at the Massachusetts Eye and Ear Infirmary doing a post-Doctor fellowship in temporal bone histopathology with Professor Harold F Schuknecht. At that time Dr Schuknecht suggested the search for a histopathologic correlate for tinnitus. The clinical files of the temporal bone collection were examined looking for patients with significant tinnitus during life, patients who had tinnitus as a main complain in the otology consultation[5, 6].

Eighty-three temporal bones were selected from patients who met the criteria described above. The main histopathologic diagnosis in each bone was recorded. As a control group, the authors selected 33 bones from patients who had the same histopathologic diagnosis but had no tinnitus during life.

The following histopathologic diagnoses were found (Table 1):

-Bacterial and viral infection,

-Sudden deafness,

-Drug ototoxicity,

-Acoustic trauma,

-Sensorineural hearing loss,

-Endolymphatic hydrops,

-Otosclerosis,

-Normal histopathology,

-Developmental or hereditary disorders,

-Paget's disease,

-Degenerative disorders including presbycusis.

None of the histopathologic diagnosis was statistically significantly different between the tinnitus and the non-tinnitus group. However, of the 83 temporal bones from patients with tinnitus during life[5, 6]:

-11 had normal histopathology,

-18 had endolymphatic hydrops (EH),

-11 had otosclerosis.

Therefore 34.93% had normal histology or EH, 48.83% had normal histology or Endolymphatic Hydrops (EH) or otosclerosis as histopathologic diagnosis. In 2007 we studied 48 consecutive patients with otosclerosis measuring tinnitus intensity before and after surgery using the Visual Analog Scale (VAS) [7].

Forty four of the 48 patients reported tinnitus pre-operatively (90%). Nineteen of these patients complained of Severe Disabling Tinnitus (SDT) on VAS.

Incidence of tinnitus in otosclerosis was said to be similar as the general population. We found SDT to have a many folds higher incidence in otosclerosis than in general population[7].

EH and otosclerosis both have high incidence in temporal bones from patients who had significant tinnitus during life[8].

It may be that both cause tinnitus by changing inner ear fluids homeostasis. This is a subject that reserves further research[8].

When compared lesions of hair cells, cochlear neurons, tectorial membranes, stria vascularis, endolymph volumes between tinnitus and non-tinnitus group, the following significant features were observed[5, 6]:

Table 1: These otopathologics diagnosis were present in the following proportions in these bones.

Histopathologis Diagnosis	Tinnitus Group	Non Tinnitus Group	p-Value
Endolymphatic hydrops	18 (21.7%)	5 (15.2%)	0.4257
Otosclerosis	11 (13.3%)	5 (15.2%)	0.7891
Normal histology	11 (13.3%)	4 (12.1%)	0.8698
Drug ototoxicity	8 (9.6%)	2 (6.1%)	0.5356
Sensorineural hearing loss	3 (3.6%)	0 (0.0%)	0.2685
Sudden deafness	3 (3.6%)	0 (0.0%)	0.2685
Acoustic trauma	6 (7.2%)	3 (9.1%)	0.7352
Bacterial and viral infections	9 (10.8%)	4 (12.1%)	0.8440
Developmental or hereditary disorders	3 (3.6%)	2 (6.1%)	0.5584
Paget's disease	2 (2.4%)	1 (3.0%)	0.8493
Degenerative disorders including presbycusis	9 (10.8%)	7 (21.2%)	0.1440
Total	**83**	**33**	

-Diffuse loss of outer hair cells - $p<0.001$

-Distortion of outer hair cells (OHC) and inner hair cells (IHC) - $p<0.04$

-Focal loss of OHC and IHC - $p<0.001$

-Cochlear neurons - retrograde degeneration - $p<0.001$

-Focal total atrophy of tectorial membrane - $p<0.001$

-Endolymph volume - $p<0.32$

All these lesions were worse in the non-tinnitus group[5, 6].

In previous research, the authors correlated tinnitus annoyance, with outer hair cells dysfunction measured by otoacoustic emissions (Figures 1-3). They concluded that altered TEOAE and DPOAE results in patients with tinnitus and normal hearing suggest the involvement of cochlear dysfunction in the generation of tinnitus[9]. Tinnitus annoyance was correlated with anxiety and depression disorders in patients with tinnitus and normal hearing. No correlation was observed between OHC dysfunction evaluated by OAE tests and annoyance of tinnitus, anxiety, or depression[10].

DISCUSSION

None of the histopathologic diagnosis is statistically significant in tinnitus patients.

Normal histology of the temporal bones, endolymphatic hydrops and otosclerosis are present in 48.83% of patients with significant tinnitus[7].

Tinnitus is the most prevalent symptom in Meniere's disease. Outer hair cells lesions are always present in tinnitus patients. TEOAE and DPOAE are more often abnormal in tinnitus patients compared with non-tinnitus normal hearing patients[9, 10].

Why temporal bones of significant tinnitus patients are better preserved than those of non-tinnitus patients is intriguing[5, 6].

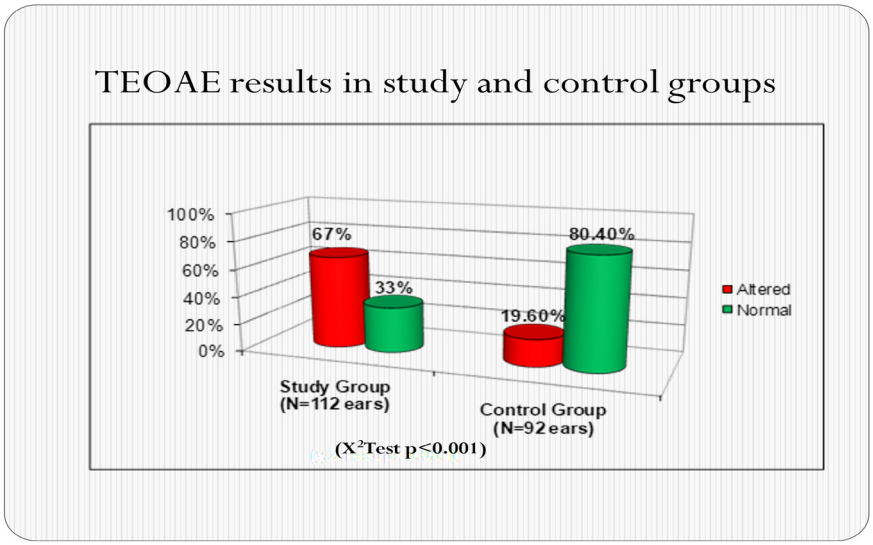

Figure 1: TEOAE results in study and control groups.

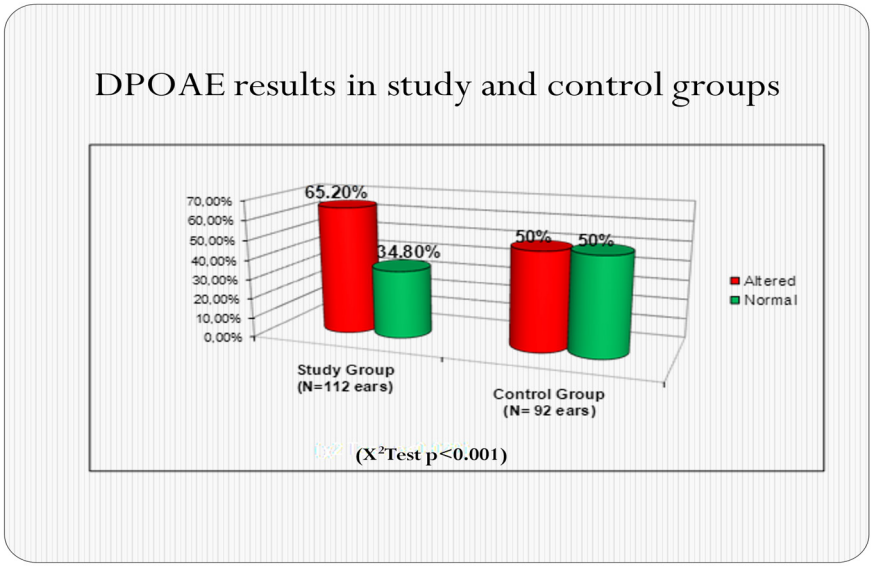

Figure 2: DPOAE results in study and control group.

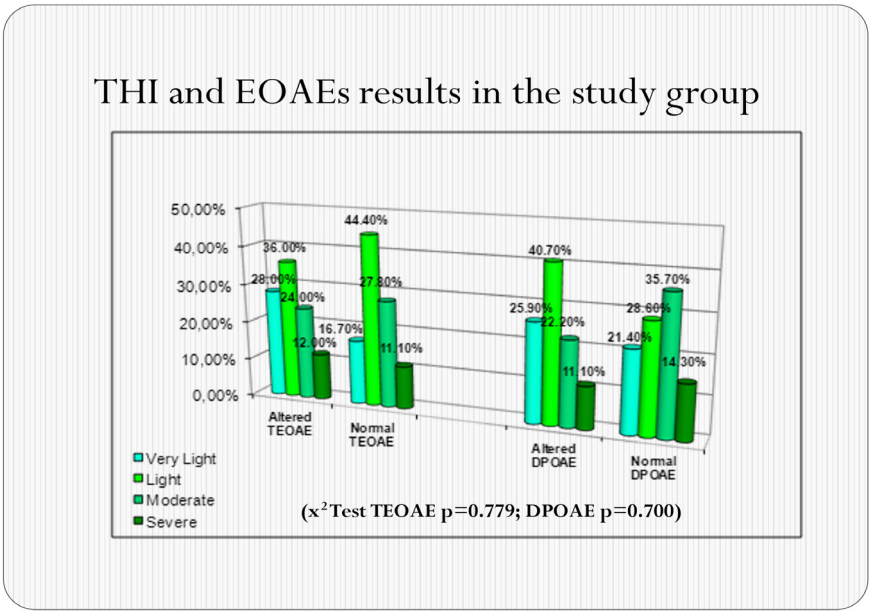

Figure 3: THI and EOAEs results in the study group.

Tinnitus annoyance does not correlate with degree of abnormality of TEOAE and DPOAE results in normal hearing patient with significant tinnitus[9].

Tinnitus annoyance correlates with degree of anxiety and depression[10, 11].

Recently we studied 10 years follow up in patients with tinnitus and normal hearing. Thirty percent of the study group with tinnitus and no hearing loss in 2009, had tinnitus and some degree of hearing loss in the follow up exam performed in 2019. Abnormal DPOAE results were more prevalent in the control group than in the study group after a ten year follow up[12].

CONCLUSION

Tinnitus, in most cases, starts in the cochlea and leaves footprints in the scale media (cochlear duct) but the degree of annoyance of the symptom is determined in the central nervous system. Abnormal results of TEOA and DPOAE as a trigger event in the cochlea for tinnitus is still questioned. The initial lesions may involve the synapses of inner hair cells and dendrites of the spiral ganglion cells (hidden hearing loss).

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