THINKING ABOUT TINNITUS

Carlos A. Oliveira, M.D., Ph.D.

ABSTRACT

In 1989, I did a thorough search of the medical records and histopathologic features of the temporal bones in the collection of the Massachusetts Eye and Ear Infirmary. During their lifetimes, these patients had had significant tinnitus. The goal was to find a histopathologic correlate for tinnitus in the cochlea. Although no such correlate could be found, some thoughtprovoking facts were verified. In this paper I want to share these thoughts hoping that others will follow their suggested investigational lines.

Key words

Tinnitus, Cochlea, Histopathology.

Goodhill in 1952¹ stated that tinnitus is a symptom common to many otologic diseases. Vernon in 1972² added that it may also be prevalent among persons with normal hearing. Tinnitus is said to be objective when the sound perceived by the patient is detectable by others in the vicinity of the ear (i.e., vascular bruits, vascular tumors). Subjective tinnitus refers to sound which is heard *only* by the patient and cannot be detected objectively. It is by far the commonest form of this condition.3 Central tinnitus means non-lateralizing "head sounds" probably generated centrally to the cochlea. Peripheral tinnitus means sound which can be heard unilaterally or bilaterally and is generated in the inner, middle, or external ears.

Tinnitus generated from the middle and external ears usually has an identifiable cause: cerumen impaction, eustachian tube dysfunction, or glomus jugulare tumors. They are objective in nature. Inner-ear-generated tinnitus, in contrast, is extremely common in many different otologic diseases and in persons with normal hearing and to date we have no hard data regarding its cause or causes.

Hilding in 1953⁴ dissected cochleas under the operating microscope and described four insertions for the tectorial membrane: origin in the limbus, along Hensen's stripe, on the hair cells, and on an outer insertion on Hensen's cells. As he found the external insertion to be easily detached, he theorized that loud sounds could free the outer insertion, thus leading to increased pressure by the tectorial membrane on the hair cells. Permanent tinnitus would then result. This has been the first attempt to find a pathologic correlative for tinnitus reported in the literature.

In 1989, I conducted a thorough search at the Massachusetts Eye and Ear Infirmary (MEEI) of the medical records and the histopathology of temporal bones of all patients who had had tinnitus as a significant complaint. The goal was to find a histopathologic correlation for cochlea-generated tinnitus. The results of this work, reported in 1990,⁵ were entirely negative: no pathologic correlation for cochlea-generated tinnitus could be found at the light microscopy level.

Before reaching the final format of that investigation as reported, I spent many hours studying the data from many different perspectives. None of these analyses led to any hard scientific conclusion-that is why they were not reported in the original work. However, some very thought-provoking clues did appear from these attempts at organizing the data. In this paper I describe some of the ways I looked at the data and discuss the thoughts they suggested to me. I do hope these thoughts will inspire others to follow the indicated investigational avenues.

FINDINGS

Of 730 clinical records on file at the MEEI temporal bone collection (those of fetuses and children up to 2-year-old were excluded), 93 patients (12.7%) had had tinnitus mentioned in the clinical history.

When I studied the temporal bone histopathology of these 93 patients, 10 were excluded because there was evidence of compression of the cochlear nerve on the internal auditory canal by neoplasms (7), pulsatile tinnitus resulting from the presence of glomus jugulare tumors in the middle ear (2) and one in which tinnitus disappeared with compression of the ipsilateral common carotid artery.

I then looked at the remaining 83 cases searching for inner ear histopathologic features regardless of the presence of other findings (e.g., middle ear infection, otosclerosis). The results of this preliminary study are summarized in Table I. There were 37 histopathologically normal inner ears (44.5%), 23 inner ears of tinnitus patients had endolymphatic hydrops (27.7%) and the remaining 23 cases had varying degrees of hair-cell loss, strial atrophy, neuronal loss, tectorial membrane defects, and loss of dendrites. Interestingly, 13 cases with normal inner ear histopathology (35.1%) also had rotatory vertigo recorded in their clinical histories (see Table I). Of the 23 patients, 19 who had endolymphatic hydrops (82.6%) also had a history of rotatory vertigo.

It is necessary to point out that the data are presented by number of patients rather than number of ears because no significant differences existed between the two ears of the same patient except in the severity of histopathologic findings. In patients with unilateral hydrops, the tinnitus was present in the hydropic ear. Generally speaking, preservation of the sensory and neural elements was good in the hydropic ears but some degree of sensory and neural losses did overlap with the presence of endolymphatic hydrops.

Next I selected 13 patients of the 83 whose medical record was very clear in identifying the presence of unilateral tinnitus, stating which ear was involved and in some of them, giving a reasonable account of the behaviour of the symptom during their lifetimes. I then carefully compared the histopathologic findings in the tinnitus with th-ose in the non-tinnitus ear. Table II summarizes these findings.

There was better preservation of sensory and neural elements in the tinnitus ear in three patients, endolymphatic hydrops was present in the tinnitus ear *only* in four and bilaterally in two cases, and there was eosinophilic precipitate in the endolymph of the tinnitus ear in only one patient. There was no difference in histopathologic findings among tinnitus and nontinnitus ears in six patients.

One of these 13 patients caught my attention in a special way. The patient's records were complete regarding tinnitus and described

the evolution of this symptom during life very precisely. This was a 68-year-old woman who had had vertigo and sensory neural hearing loss in her left ear at age 42. Her right ear was unaffected. At age 45 (1954) she started aspirin therapy (10 tablets a day) for arthritis, at which point tinnitus appeared in her right (better) ear but not in the left (partially deafened ear). Hearing in her right ear deteriorated with time so that by 1958 she had severe sensory neural hearing loss in both ears, but worse in the left. She continued taking the aspirin until 1971, but the tinnitus in the right ear was no longer present. The histopathologic study of her temporal bones showed bilateral severe hydrops and better preservation of hair cells and neuronal population in her right ear (tinnitus ear).

The temporal bones were processed for histopathologic examination in the usual way, embedded in celloidin and cut in 20-micronthick slices. Every tenth section was stained with hematoxilin and eosin, mounted on glass slides and studied by light microscopy.

CONCLUSIONS

Table I shows that 72.2% of the temporal bones from tinnitus patients showed either normal inner ears (44.5%) or endolymphatic hydrops. The remaining bones (27.7%) showed varied degrees of sensory and/or neural losses in the inner ear including strial atrophy, tectorial membrane lesions, hair cells, and neuronal losses.

Endolymphatic hydrops does not cause tinnitus; yet there seems to be a link between tinnitus and endolymphatic hydrops. This link is suggested again by the data in Table II: 4 of 13 patients with unilateral tinnitus had endolymphatic hydrops only in the tinnitus ear. The only way to establish such a link is to postulate a common cause for inner-ear-generated tinnitus and endolymphatic hydrops. Most of the tinnitus patients had had normal inner ears at the light microscopic level, suggesting that tinnitus comes before the development of endolymphatic hydrops. If we consider that 35% of the patients with tinnitus also had vertigo then we come closer to Ménière's disease itself. What is the primary event which leads to tinnitus, vertigo, and eventually to

Table I.

Summary of Histopathologi	c Findings*			
	number of	percentage	patients	percentage of patients
	patients		with vertigo	with vertigo
Normal inner ear	37	44.5	13	35.1
Endolymphatic hydrops	23	27.7	19	82.6
Varied degrees of				
sensory and neural	23	27.7	0	0
losses				

*Total of 83 patients with tinnitus

endolymphatic hydrops cannot be ascertained, but we do know it is not detectable at the light microscopy level. A good hypothesis would be that this event is linked with production/ absorption of endolymph. Some theories on the pathophysiology of Ménière's disease6-9 propose a primary cause in the central nervous system which, through the autonomic nervous system, would affect the micro-circulation in the inner ear causing faulty inner-ear-fluid homeostasis. Although the data we gathered do support such a line of thinking, it is certainly far from confirming the hypothesis. Ultrastructural studies of inner ears from tinnitus patients certainly can lead to further clues regarding the primary event leading to tinnitus. More basic experiments on cochlear physiology focusing on inner-ear-fluid pro-duction and absorption are needed before we can progress further on this matter. We simply lack basic knowledge on mechanisms of inner-ear homeostasis.

Table II shows that 23% (3 of 13) of

patients with unilateral tinnitus showed better preservation of sensory and neural elements than in the non-tinnitus ear of the same patient. The reverse situation was not found in any of the 13 unilateral tinnitus cases. The case of the patient I reported in detail caught my attention because tinnitus provoked by aspirin intake developed in the better ear and not in the partially deafened one. Furthermore, as the better ear became progressively worse, tinnitus disappeared even though aspirin intake continued. These findings suggest that tinnitus is an early symptom of cochlear dysfunction and that as damage to sensory and neural elements progresses, the symptom tends to disappear. This idea is by no means new, because it has been expressed by many authors in the past.¹⁰⁻¹³ Of course, this view would be in harmony with the idea that a change in cochlear homeostasis, possibly involving production and/or absorption of endolymph, leads to tinnitus first and later to endolymphatic

Table II.

Comparison of Histopathologic Findings Between Tinnitus and Non-tinnitus Ears of 13 Patients with Unilateral Tinnitus

	Tinnitus ear	Non-tinnitus ear	Both ears
Better preservation of			
sensory and neural	3	0	
elements			
Endolymphatic hydrops	4	0	2
Eosinophilic			
precipitate in	1	0	0
endolymph			
No difference between		6	
ears			

1) Two patients with no differences between ears had bilateral endolymphatic hydrops.

2) One patient with better preservation of sensory and neural elements also had endolymphatic hydrops only in the tinnitus' ear.

hydrops in Ménière's disease.

Vernon¹⁴ states that only 15 of 1200 patients seeking relief for tinnitus in his clinic had a previous history of Menière's disease. This led him to believe that tinnitus in this disease disappears as the pathologic process "burns itself out". Longitudinal studies of the evolution of tinnitus in Ménière's disease patients would prove or disprove Vernon's assumption and the ideas I have put forward earlier. Such a study would take many years to be completed but is perfectly feasible.

Finally I want to emphasize that the findings I described in this paper do suggest these ideas but by no means do they establish these theories as scientific facts. I believe, however, that the research lines they point to are worth pursuit and I hope this paper will stimulate others to do so.

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Requests for reprints of this article should be addressed to: Carlos A. Oliveira, M.D., Ph.D., SHIS QL 22 Conjunto 4 Casa 9, Brasília-DF-71650-145, Brasil