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Tinnitus and otosclerosis

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Abstract. *Tinnitus and otosclerosis. Objective*: To establish the origin of tinnitus in patients with otosclerosis.
Methodology: We performed a literature review to identify the characteristics of tinnitus in the general population and among patients with otosclerosis.
Results: Characteristics of tinnitus among patients with otosclerosis differ from those among the general population, suggesting mechanisms other than those implicated in presbycusis. Considerable improvements in the prevalence of tinnitus following corrective surgery demonstrate the limited contribution of diffuse labyrinth lesions. Middle ear and possibly secondary inner ear dysfunction produced by stapes blockage appear to be major etiopathogenic factors in the development of tinnitus in patients with otosclerosis.
Conclusion: The conductive hearing deprivation produced by otosclerosis is associated with limited and reversible modifications in the central auditory pathway that are linked to tinnitus.

Introduction

Tinnitus is a common disorder, particularly in patients with otosclerosis. However, a number of questions remain. Are the characteristics of tinnitus in patients with otosclerosis similar to those of persons in the general population? How do symptoms evolve after stapes surgery? What is the origin of the symptoms? Is tinnitus more frequent in cases of associated sensorineural hearing loss (SNHL), suggesting labyrinth involvement in otosclerosis as often postulated? We conducted a literature review in order to determine the answers to these pertinent questions.

Materials and methods

A literature search was initiated to elucidate the characteristics, evolution, and etiology of tinnitus in the general population and in patients with osteosclerosis.

Results and discussion

Tinnitus in the general population versus those with otosclerosis

A diagnosis of tinnitus should be considered if symptoms are present every day for more than 5 minutes per day. The prevalence of tinnitus in the general population is about 10%[1-3] and increases with age from 5.5% in persons younger than 40 years of age to 14.5% in those older than 60 years.[3] Individuals exposed to noise are at greater risk for tinnitus. Of patients presenting with tinnitus before the age of 40 years, 16.5% have been exposed to noise and of those presenting after the age of 60 years, 30% have been exposed to noise.[2] Tinnitus is more common in men than in women: 15% of patients 55-65 years of age are men, whereas 9% are women. Interestingly, pregnancy significantly increases the incidence of tinnitus. Twenty-five percent of pregnant women report tinnitus as compared to 11% of non-pregnant women.[4]

There is no clear link between the measured intensity of tinnitus and its perceived severity.[5] The greater degree of SNHL, the higher the prevalence of tinnitus. The risk increases by 11% for each 10 dB hearing loss increment in pure tone average in the better ear.[6] Audiogram configuration modifies tinnitus prevalence: 20% in cases of high-frequency, steeply sloping audiograms; 12% in cases of gently sloping audiograms; and 6% in cases of flat configurations.[3] This observation appears to be related to the "edge effect" theory. The normal auditory area adjacent to a deafferentation area may show a decrease in lateral inhibition, resulting in an increase in activity and therefore a greater risk of tinnitus.[7]

Tinnitus presents differently in patients with otosclerosis. In these patients, the prevalence is much

Table 1

Prevalence and severity of tinnitus before surgery and the frequency of improvement or remission after surgery in patients with otosclerosis

Authors	Pre-operative tinnitus	Pre-operative severe tinnitus	Post-operative improvement	n
Sobrinho *et al.*[10]	91%	40%	91%	48
Lima *et al.*[15]			95%	23
Oliveira *et al*[8]	91%	40%	91%	48
Gritswood *et al.*[13]	65%			1014
Gersdorff *et al.*[12]			96-64%	50
Ayache *et al.*[11]	74%	25%	88%	65

n: the number of patients evaluated in the cited publications.

higher than in the general population, between 65% and 91% (Table 1), and it is not age dependent.[8-13] In contrast with the general population, women with otosclerosis present with tinnitus more frequently than do men with otosclerosis. One distinguishing characteristic of tinnitus in otosclerosis is its perceived severity. Tinnitus is life disabling in 25-40% of patients with otosclerosis as compared to 1% of the general population.[8-12]

Factors promoting tinnitus in otosclerosis

Different factors influence the prevalence and intensity of tinnitus in patients with otosclerosis (Table 2). A large pre-operative air-bone gap (ABG) is associated with more severe tinnitus before surgery and better post-operative improvement.[9,10,13] A smaller post-operative ABG and more developed SNHL (which indicate higher bone conduction levels) are both associated with less severe tinnitus.

The expected evolution of tinnitus after surgery is shown in Table 1. The majority of patients show some improvement after surgery. Gersdorff *et al.*[12] reported less improvement in cases of stapedectomy than stapedotomy, but Ayache *et al.*[11] found no difference between the two techniques.

Tinnitus in otosclerosis does not appear to be associated with the degree of footplate pathology,[11,13] pitch, or loudness.[11,12]

Etiology of tinnitus in otosclerosis

Tinnitus in otosclerosis could originate in the middle ear, cochlea, or central auditory pathway. We present various mechanisms that could favor the development of tinnitus in patients with otosclerosis:

1. Hearing loss profile

Hearing masks physiological tinnitus experienced by more than 90% of normal hearing subjects placed in a soundproof room.[14] Conductive hearing loss produced by stapes blockage results in hearing deprivation and, consequently, loss of the masking effect of hearing. Thus, progressive conductive hearing loss allows the tinnitus to reach increasingly perceptible levels.

This hypothesis explains the link between the ABG and tinnitus symptoms, as well as the considerable improvements seen after surgery and their relationship with post-operative ABG size. This hypothesis also corroborates reports that 80% of chronic otitis media sufferers experience improvement in tinnitus after tympanoplasty.[15]

The effect of ABG is only observed up to 40 dB.[13] At higher

Table 2

Influence of air-bone gap (ABG) and bone conduction levels on the severity of tinnitus and the influence of other factors (F: female; M: male) on the prevalence of tinnitus (T) in patients with otosclerosis

Author	Pre-operative ABG	Bone conduction levels	Post-operative ABG	Other factors
Sobrinho *et al.*[10]	If large: better post-surgical improvements in T	If low: more severe T	If small: less severe T	
Oliveira *et al.*[8]	If large: better post-surgical improvements in T		If small: less severe T	More prevalent in F than M
Gritswood *et al.*[13]	>T if <40 dB <T if >40 dB	If high: less severe T		More prevalent in F than M No link with footplate lesions No age effect

levels, cranial transfer with direct cochlear stimulation probably plays a role.

2. Stapes ankylosis

Stapes blockage disturbs middle ear muscle function, inducing more isometric than isotonic muscular contractions and probably a change in somatosensory receptor function. It is known that tinnitus can be caused by somatosensory modulations of the auditory system and is commonly encountered in jaw, masticator muscle, and neck disorders.[16,17] It is possible that isometric middle ear muscle contractions produce tinnitus in the same way.

3. Labyrinth lesions

It is widely maintained that tinnitus in otosclerosis is produced by labyrinth lesions.[13] However, this hypothesis is less probable in the 90% of patients with otosclerosis who experience improvements after surgery, as stapedotomy and stapedectomy do not modify diffuse labyrinth lesions.[9-10,18] Furthermore, this hypothesis does not explain the decrease in tinnitus prevalence in cases of SNHL or the lack of link with the degree of footplate involvement.[13]

4. Mechanical wave alteration

Stapes blockage modifies traveling waves in the cochlea. A normal stapes is pushed inward and outward during sound vibrations, giving rise to a traveling wave along the basilar membrane. The pressure difference between the round and oval windows is small and the cochlear fluid is incompressible.[19] In case of stapes blockage, sound vibrations can stimulate the basilar membrane by bone conduction or round window coupling, and basilar membrane movements and inner ear pressure distribution will be affected. Consequently, stapes blockage may alter inner ear pressure homeostasis, as observed to a greater degree in hydrops. This disturbance could modify hair cell electromotility and/or is associated with a sustained higher firing rate of inner hair cells, as seen following exposure to loud noise, salicylate absorption, or a Meniere attack.[1,20] These pathological situations are known to produce very disturbing tinnitus. This hypothesis may explain the high prevalence of disturbing tinnitus in patients with otosclerosis, 40% versus 1% in the general population.[8-12] In this hypothesis, the tinnitus has a mechanical origin localized in the inner ear and there is no SNHL. Surgery repairs this defect and alleviates the tinnitus.

5. Inner ear damage

A decrease in cochlear input because of hair cell loss, with secondary loss of inhibition in the central auditory pathway, can also cause tinnitus.[1,7,21] In this hypothesis, SNHL is present. Curiously, in patients with otosclerosis, SNHL is associated with less severe tinnitus.[13] How can this be explained?

Hair cell loss stops abnormal synaptic stimulation thereby decreasing the higher firing rate thought to be associated with inner ear mechanical obstacles (see above). This decrease in firing rate inversely mitigates the tinnitus.

This hypothesis could explain the lack of an age effect on tinnitus in patients with otosclerosis. Indeed, in the general population, presbycusis is associated with hearing deprivation and secondary central auditory pathway hyperactivity, giving rise to increased tinnitus over time. In patients with otosclerosis, presbycusis decreases the supposed higher firing rate by hair cell loss. The effects of hair cell loss and higher firing rates cancel each other out with age. It is also possible that conductive hearing loss protects subjects against noise toxicity.

6. Central perception

Tinnitus is a central auditory perception. Peripheral sensory deafferentation or modulation modifies the central auditory pathway: hyperactivity secondary to loss of inhibition, as in phantom limb pain.[7]

These data indicate that surgery can usually alleviate tinnitus symptoms in patients with otosclerosis. Our observations suggest that central auditory pathway plasticity is less marked and/or more reversible in patients with conductive hearing loss than in patients with SNHL. However, the sex effect remains unclear. Among patients with otosclerosis, tinnitus is present more often in women, while the opposite is true in the general population.

Prospective studies are needed to evaluate the different mechanisms. These studies should evaluate electrocochleography before and after surgery, the correlation between tinnitus and middle ear resonance frequency, and investigate presynaptic potentials, like the cochlear microphonics that are increased in cases of auditory neuropathy. The role played by genetic mutations in the manifestation of tinnitus symptoms should also be evaluated.[22,23]

Conclusion

Our findings on the prevalence of tinnitus and its characteristics

before and after surgery allow us hypothesize on the etiopathogenesis of tinnitus in patients with otosclerosis. The considerable improvements observed after surgery indicate that tinnitus is not usually caused by labyrinth lesions, that it generally originates from a mechanical dysfunction in the middle or inner ear, and is associated with more limited and/or more reversible central auditory pathway hyperactivity.

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