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An otoneurosurgical approach to non-pulsatile and pulsatile tinnitus

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Abstract. *An otoneurosurgical approach to non-pulsatile and pulsatile tinnitus. Objective*: Most treatments proposed for tinnitus are non-surgical, to such an extent that it is sometimes forgotten that a certain number of patients with tinnitus may benefit from a surgical solution. The aim of this paper is to review the possible otoneurosurgical approaches in tinnitus treatment, treating the tinnitus causally or symptomatically.
Methods: A Pubmed search on the words "surgery", "tinnitus" and "pulsatile" was performed and compared to the authors' personal experience with surgical approaches for alleviating tinnitus. The most relevant different pathologies presenting as pulsatile and non-pulsatile tinnitus are given and possible otoneurosurgical approaches for these identities summarised.
Results and discussion: Non-pulsatile tinnitus can be the clinical expression of vestibular schwannomas and other cerebellopontine angle lesions, arachnoid cysts, Ménière's disease, otosclerosis, brain tumours along the auditory pathways, Chiari malformations and microvascular compressions of the vestibulocochlear nerve.
Symptomatic improvement of non-pulsatile tinnitus can also be obtained by electrical stimulation of the cochlea, auditory nerve or cortex.
Pulsatile tinnitus can present as a venous hum resulting from benign intracranial hypertension, Chiari malformation and a high jugular bulb. Arterial-pulse-synchronous tinnitus can be caused by benign intracranial hypertension, arteria carotid stenosis, glomus tumours, vascular lesions of the petrous bone and skull base, ateriovenous malformations, aneurysms, and vascular loops inside the internal auditory canal.
Conclusion: Before people are told "to learn to live with their tinnitus" a thorough exploration of possible cause and potential surgical treatments should be provided for patients presenting with incapacitating tinnitus.

Objective

Most treatments proposed for tinnitus are non-surgical, to such an extent that it is sometimes forgotten that a certain number of patients with tinnitus may benefit from a surgical solution. The aim of this paper is to review the possible otoneurosurgical approaches in tinnitus treatment.

Tinnitus can be subdivided into two entirely different entities: pulsatile and non-pulsatile tinnitus.[1-3] It is suggested that the sounds perceived in pulsatile tinnitus are the result of venous or arterial pulsations transferred to the cochlea via cerebrospinal fluid conduction.[4-6] Pulsatile tinnitus synchronous with the heartbeat therefore seems to be related to arterial causes, pulsatile tinnitus synchronous with respiration is most likely due to venous causes. Pulsatile tinnitus in general is therefore not necessarily associated with pathology of the auditory pathways. Non-pulsatile tinnitus, on the other hand, is thought to be the result of the abnormally functioning of the auditory system and can be considered an auditory phantom phenomenon.[7,8]

As heartbeat-synchronous tinnitus is predominantly vascular in origin, almost all causes of pulsatile tinnitus can be diagnosed with magnetic resonance imaging and magnetic resonance angiography, except for what has been suggested to be the most frequent cause of pulsatile tinnitus: benign intracranial hypertension.[1-3]

Methods

A Pubmed search was performed for the words pulsatile, surgery and tinnitus in two different combinations (tinnitus and surgery, pulsatile and tinnitus and surgery). The pathologies and treatments in the literature were reviewed and the relevant literature related to the authors' personal experience. We present a summary of the results.

Results and discussion

Non-pulsatile tinnitus

Non-pulsatile tinnitus can be considered an auditory phantom phenomenon[8] resulting from auditory deprivation or deafferentation.[7] In

other words, unlike pulsatile tinnitus, non-pulsatile tinnitus is related to a pathological functioning of the auditory system. As a consequence, any lesion along the auditory tract altering its normal function can cause non-pulsatile tinnitus. Ménière's disease, vestibular schwannoma, cerebellopontine angle lesions, arachnoid cysts, microvascular compressions, Chiari malformation and brain tumours are causes of non-pulsatile tinnitus that can be treated surgically.

If no cause can be identified, and if no causal treatment can therefore be offered, a trial with permanent symptomatic electrical stimulation treatment is a possibility. This involves non-invasive stimulations directed at different targets in the auditory system (promontory stimulation, transcranial magnetic stimulation) to determine if a permanent implant could be beneficial.

In vestibular schwannoma a high-pitch tinnitus (ringing or steam from a kettle) is present in 60 to 85% of the patients.[9] Since the advent of stereotactic irradiation, vestibular schwannomas are often treated with radiosurgery, and gamma knife radiosurgery. This seems to have almost no effect on the tinnitus.[10] By contrast, microscopic surgery eliminates the tinnitus in 40 to 50% of cases.[11] Unfortunately, microsurgery also creates tinnitus in almost as many patients.[12] Gamma knife treatment has advantages over surgery as well as drawbacks. Gamma knife radiosurgery is less invasive and requires shorter hospitalisation and convalescence periods. The development of a facial paresis is extremely rare (1% at an irradiation dose of <14 Gy) and hearing can be saved in 71% of patients if the same maximum dose is respected. The technique is, however, limited to lesions less than 3 cm in size and involves a greater risk of post-treatment hydrocephalus and a very small risk for dedifferentiation into a neoplasia.

Other cerebellopontine angle lesions (CPA)[13] such as meningiomas, epidermoid tumours, lipomas, choroid plexus papillomas, epithelial cysts, teratomas, cavernomas, and haemangiomas can present with non-pulsatile tinnitus, usually with associated symptoms depending on the location of the lesion and the degree of brainstem, cerebellar or cranial nerve compression.

Arachnoid cysts are a rare cause of non-pulsatile tinnitus. They are a congenital or post-traumatic/post-inflammatory disorder[14,15] leading to vague symptoms. Arachnoid cysts producing tinnitus can occur in the CP angle, but also in the retroclival and retrocerebellar areas. Usually, symptoms of intracranial hypertension are associated with non-pulsatile tinnitus.[16,17] Surgical treatment consists of marsupialisation or excision of the cyst.[16]

Supratentorial sylvian fissure arachnoid cysts can generate tinnitus, and tinnitus suppression can be the result of marsupialisation of the cysts if they act as a mass lesion.[18] Using intrathecal contrast to verify whether an arachnoid cyst-like lesion communicates with normal CSF flow can help to ascertain whether an arachnoid cyst could act as a mass lesion and therefore be symptomatic or not. MRI sequences looking for a flow void within the cyst can be helpful as well.[19]

In Ménière's disease, no kind of surgery – whether vestibular nerve section, cochlear nerve section, endolymphatic sac surgery[20] or gentamicin injections[21] – seems to achieve more than 50% tinnitus control. This is a marginal improvement upon the 30% spontaneous disappearance rate.[22]

In otosclerosis, the rate of relief for non-pulsatile tinnitus through successful stapedectomy can be expected to be about 40 to 64%.[23,24] On rare occasions, otosclerosis also produces arterial pulsatile tinnitus due to neovascularisation at the site of stapes fusion. Stapedectomy can sometimes cure this rare and particular form of pulsatile tinnitus.[2]

A brain tumour in the auditory cortex or compressing the auditory cortex can cause ipsilateral fluctuating non-pulsatile tinnitus as the sole symptom, probably due to a direct effect on normal cortical sound processing. Removal of the lesion eliminated tinnitus in four out of five patients.[25] Tumours elsewhere along the auditory tract, for example the brainstem, rarely present with tinnitus only, usually giving rise to additional symptoms related to the closeness of other neural structures in the brainstem.

In the case of intractable non-pulsatile tinnitus, auditory brainstem implants[26] and auditory cortex stimulations can provide relief.[27-29] These treatments are based on a recently developed pathophysiological model for non-pulsatile tinnitus based on auditory deprivation or deafferentation as the initial trigger for tinnitus generation. The decrease of auditory input into the thalamocortical system induces a slowing of auditory information transfer generating slow-wave activity (delta en theta oscillations),[30,31] with a resultant decrease in lateral

inhibition[32] and a halo or edge of increased activity.[30,33] This is also called thalamocortical dysrhythmia[30] and is associated with cortical reorganisation.[34,35] The mechanism correlating gamma hyperactivity with reorganisation is synchrony. Synchronisation of the gamma band activity could induce topographical reorganisation through simple Hebbian mechanisms (cells that fire together wire together). It would therefore seem logical to try and modify this tinnitus-related auditory cortex reorganisation/hyperactivity in an attempt to suppress the tinnitus. This can be done with neuronavigation-guided TMS, a technique that can modulate cortical activity. If TMS can suppress tinnitus, the effect could be maintained by implanting electrodes at the area of signal abnormality on the auditory cortex. The first results in these patients indicate statistically-significant tinnitus suppression for unilateral pure tone tinnitus, without suppressing white or narrow band noise in TMS responders.[28] More recent trials have also suggested that narrow band tinnitus can be suppressed with novel stimulation designs (De Ridder, personal communication).

Reafferentation of the deprived auditory thalamocortical system can also be achieved with cochlear implants. Almost immediately after the introduction of cochlear implants for hearing improvement, it was noted that the electrical intracochlear stimulation led to an improvement in tinnitus in a large proportion of patients.[36,37] Multiple studies since then have replicated these results, indicating that cochlear implants inserted for hearing improvement can also modulate tinnitus,[38-42] not only unilaterally but also bilaterally in a majority of patients.[43] A recent study using cochlear implant insertion in patients with-incapacitating tinnitus and ipsilateral cophosis and contralateral preserved hearing generated similarly promising results (Van de Heyning, personal communication). Promontory stimulation as a preoperative non-invasive test in this selected group of patients seems to predict good outcomes in tinnitus suppression.

A limit to this technique is that it can only be used in patients with unilateral cophosis. This could be extended to people with high-frequency hearing loss but preserved low-frequency hearing, since a recent paper has shown that short hybrid cochlear implants can preserve low-frequency hearing.[44] Another option is to use extracochlear stimulation for tinnitus suppression. The first attempts to develop extracochlear electrical stimulation have been made.[41,45]

Pulsatile tinnitus

Benign intracranial hypertension (BIH), also known as pseudotumor cerebri, is the most frequent cause of pulsatile tinnitus. In a study by Sismanis,[2] 40% of all pulsatile tinnitus patients and almost all patients presenting with venous pulsatile tinnitus were diagnosed as having BIH. BIH is a clinical entity almost exclusively afflicting young (<40 years) overweight women. Clinical symptoms include a venous hum, headache and blurry vision. Oddly enough, the venous hum presents most often unilaterally (80%), and it can be the only presenting symptom.[2] In general, symptoms are more prominent or can be worsened when intracranial pressure is increased, as when lying down (e.g. in the morning when waking up) or bending over, when coughing or performing other manoeuvres that raise intracranial pressure. Clinically, the suspicion of BIH can be confirmed by compressing the ipsilateral jugular vein. This results in a flow arrest in the ipsilateral sigmoid sinus, putting a stop to the venous hum. When the tinnitus disappears, the headaches tend to increase. Low-frequency sensorineural hearing is present in half of the patients and disappears on ipsilateral jugular vein compression as well. Papilloedema is present on ophthalmoscopy. Recently, we have noticed a similar syndrome in patients without papilloedema and normal pressure. Magnetic resonance angiography and magnetic resonance imaging are usually negative. An empty sella, which was a feature in 25% of cases,[2] should however raise suspicion of BIH since it can be related to prolonged intracranial pressure. Diagnosis is usually confirmed by lumbar puncture (opening pressure >20 cm water). Treatment consists of weight loss, diuretics or ventriculoperitoneal or lumboperitoneal shunting. BIH is usually idiopathic, but it may be caused by venous sinus outflow obstruction[46] and it can also be seen after posterior fossa surgery (unpublished results).

The Chiari malformation is a clinical entity in which there is a tonsillar herniation into the foramen magnum. Four different types exist but only Chiari type I is still seen relatively frequently in the Western world. Between 7 and 10%[47] of these patients present with tinnitus and the tinnitus can be both non-pulsatile and pulsatile.[48] The pulsatile tinnitus most commonly consists of a venous

hum, which is likely to be caused by raised intracranial pressure, as it worsens on bending over and other Valsalva manoeuvres. The pulsations normally disappear upon ipsilateral jugular vein compression, which also results in an improvement of low sensorineural hearing loss (masking). No brainstem auditory evoked potential changes are noted in this kind of tinnitus. After surgical decompression, this form of tinnitus can disappear.[48]

Non-pulsatile tinnitus on the other hand is usually intermittent and the cause is unknown. It is either due to stretching of the cochlear nerve, for example by microvascular compression, or to brainstem traction.[49] Brainstem auditory evoked potential changes are seen in 75% of patients and consist of an IPL III-V prolongation in 100% of the patients and in 30% of IPL I-III prolongation.[49] The IPL III-V prolongation could be due to brainstem traction and/or contralateral microvascular compression;[50] the IPL I-III could be the result of ipsilateral microvascular compression.[51,52] Posterior fossa decompression consisting of opening the foramen magnum and widening the dura mater can therefore result in an improvement in non-pulsatile tinnitus in three out of four patients (De Ridder, personal communication), as in trigeminal neuralgia in patients with Chiari malformation.[53] The clinical improvement could be due to a secondary autodecompression of the vestibulocochlear nerve, analogous to what has been suggested in the surgical removal of posterior fossa tumours (De Ridder, personal communication).

A high jugular bulb can also generate a venous hum as a result of an intimate direct contact with the cochlea. As has been described for benign intracranial hypertension, the venous hum disappears upon compression of the ipsilateral jugular vein. A high jugular bulb can be diagnosed by CT imaging. Surgically ligating or lowering the jugular bulb and interposing teflon or bonewax can eliminate or reduce this form of tinnitus.[54,55]

Abnormal veins such as an abnormal posterior condylar emissary vein[56] and an abnormal mastoid emissary vein[57] have also been described as surgically treatable causes of venous pulsatile tinnitus.

Carotid stenosis is the second most frequent cause (30%)[1-3] of arterial pulsatile tinnitus after BIH. The pulsatile tinnitus should disappear upon compression of the ipsilateral, internal or common carotid artery. The diagnosis can be confirmed with sonography, MR, CT or classical angiography. Treatment of the extracranial carotid artery stenosis can consist of dilation and stenting or carotid endartrectomy. As for the rarer intracranial carotid artery stenosis, two approaches can be followed. An initial balloon occlusion test under transcranial doppler and EEG monitoring can verify whether the ipsilateral carotid artery can be sacrificed. If so, one option is to ligate the symptomatic carotid artery. The other option is to dilate and stent the intracranial portion of the carotid artery, resulting in the elimination of the arterial pulsatile tinnitus. A major problem still faced today is that stents tend to occlude, and this elegant technique will therefore remain experimental until the coagulation problems are better controlled.[58]

Glomus tumours, or paraganglioma, are predominantly encountered in women presenting with unilateral hearing loss and pulsatile tinnitus, and should therefore be differentiated from BIH. Diagnosis is confirmed by MRI and/or angiography. As glomus tumours are benign lesions growing less than 2 cm in five years, treatment options are a "wait and see" policy or embolisation and surgery.[59] If the tinnitus is incapacitating, embolisation with surgery option can be helpful.

Table 1

Surgically treatable causes of tinnitus

Pulsatile tinnitus	***Non-pulsatile tinnitus***
Venous Benign Intracranial Hypertension Chiari Malformation High Jugular Bulb **Arterial** Carotid Stenosis Glomus Tumour Vascular Lesions of Petrous Bone/ Skull Base Arteriovenous Malformation Aneurysm Microvascular Compression Benign Intracranial Hypertension	Vestibular Schwannoma (Acoustic Neuroma) Other Cerebellopontine Angle Lesions Arachnoid Cyst Ménière's Disease Otosclerosis Brain Tumour Chiari Malformation Microvascular Compression
	Symptomatic
	Cochlear implant Brainstem implant Auditory cortex implant

Other vascular lesions of the petrous bone or skull base such as hemangiopericytoma,[60] plasmacytoma,[60] and giant cell tumours[61] are also known to generate a otoneurosurgically treatable pulsatile tinnitus.

Dural arteriovenous malformations (AVM) are the best-known cause of arterial-pulse-synchronous pulsatile tinnitus. Often, AVMs result from chronic mastoiditis or other causes occluding the sigmoid-transverse sinus. As a natural repair mechanism, vascular bypasses tend to develop around the occlusion, resulting into a dural AVM. If the dural AVM. is symptomatic or if it is asymptomatic with leptomeningeal drainage, these lesions should be embolised, usually in multiple sessions. If intractable with endovascular treatment, surgical excision of the AVM and dura may be an option.[62]

Microvascular compressions of the cochlear nerve can cause incapacitating pulsatile or non-pulsatile tinnitus.[63,64] Most vascular compressions cause non-pulsatile tinnitus. Vascular compression of the cochlear nerve results in non-pulsatile tinnitus and does not cause pulse-synchronous pulsatile tinnitus, as is often believed. This is in line with other vascular compressions such as trigeminal neuralgia and hemifacial spasm, which do not result in pulse-synchronous bouts of pain in the distribution of the trigeminal nerve or pulse-synchronous hemifacial spasms either. This difficult diagnosis is based on the clinical picture and confirmed by auditory brainstem evoked potentials and magnetic resonance imaging.[65,66-68] It has recently been shown that peak II disappearance correlates causally with the presence of ipsilateral tinnitus at the frequency of hearing in MVC and IPL I-III to ipsilateral frequency specific hearing loss (De Ridder, personal communication). Results of microsurgical vascular decompressions are related to surgical delay, pre-operative hearing status, MRI imaging and gender. It would therefore appear to be the case that, if the tinnitus has been present for less than three years,[65,68] if there is a serviceable or normal hearing,[63] and if MRI shows a vascular compression[64,66,67] – in women[65] – the results of microvascular decompressive surgery can be good. Vascular compressions of the vestibulocochlear nerve are found in many asymptomatic patients (12.3%[69] and 21.4%[70]) but this discrepancy is also noticed in trigeminal neuralgia (14%)[71] and even in herniated lumbar discs (36%),[72] where this is not considered an argument to doubt the pathophysiological importance of the neural compression.

However, if the vascular loop enters into the internal auditory meatus it can generate arterial-pulse-synchronous tinnitus via CSF/bone conduction.[73] MR imaging has demonstrated that, in pulsatile tinnitus, after the exclusion of other causes, a statistically significant higher number of vascular loops are seen in the internal auditory canal by comparison with non-pulsatile tinnitus,[74] and teflon insulation of the vascular loop has resulted in the elimination of the arterial-pulse-synchronous tinnitus.[73]

Idiopathic arterial pulsatile tinnitus is seen in about 15% of maximally investigated cases.[2,3] As mentioned above, it has recently been shown that a statistically significant number of vascular loops enter the internal auditory canal from the posterior fossa by comparison with non-pulsatile tinnitus,[74] possibly further reducing the number of idiopathic pulsatile cases. Pathophysiologically, the following explanation has been suggested: the relatively sharp turn of the vascular loop in the triangular internal auditory canal creates turbulence, which generates sound waves that are concentrically irradiating. The internal auditory canal has a cave or funnel effect, guiding the sound wave towards its end, the top of the triangle where the cochlea is located. The CSF bone interface sound waves are transferred via bone conduction to the cochlea. High-frequency waves carry less energy than low-frequency waves and are therefore reflected more easily than the longer low-frequency waves. This could explain why pulsatile tinnitus matches low frequencies.

There are pathologies that mimic pulsatile tinnitus. Causes of this non-vascular pulsatile tinnitus are palatal myoclonus,[75] tensor tympani spasms,[76,77] stapedial muscle myoclonus[78] and a patulous Eustachian tube.[79] These pathologies generate neither arterial-pulse-synchronous, nor respiratory-rate-synchronous tinnitus, but tend to fluctuate in intensity (as in a stormy wind) or are perceived as clicks. Temporomandibular joint disorders, also known as Costen's syndrome, can cause fluctuating tinnitus as well, although other causes can be found for the tinnitus in most patients (90%).[80] In TMJ disorders, tinnitus arises when TMJ symptoms arise, and tinnitus worsens when clenching the teeth.[81]

Conclusion

Tinnitus actually consists of two entirely different entities with a

different pathophysiology, different clinical symptoms and different treatment. Before tinnitus patients are told “to learn to live with their tinnitus” a search for possible causes for both non-pulsatile and pulsatile tinnitus is advisable, since this can result in a otoneurosurgical causal treatment for the tinnitus. In patients with non-pulsatile tinnitus, non-invasive trials with promontory or transcranial magnetic stimulation may help in the selection of patients for a permanent implant as a treatment for tinnitus.

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