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Microvascular Decompression for Tinnitus: Significant Improvement for Tinnitus Intensity Without Improvement for Distress. A 4-Year Limit

OBJECTIVE: Microvascular compressions of the cochlear nerve can lead to tinnitus. The tinnitus initially is related to nonsynchronous signal transmission in the auditory nerve, neurophysiologically characterized by a peak II amplitude decrease. Chronic compression can lead to a focal demyelination, resulting in an increase in interpeak latency I-III with tinnitus and frequency-specific hearing loss as a consequence. Decompressing the cochlear nerve may result in improvement in tinnitus if the auditory nerve is not too damaged for recovery. The aim of the study is to find a cut-off point for this recovery based on clinical data.

MATERIALS AND METHODS: Twenty patients undergo a microvascular decompression of the vestibulocochlear nerve for unilateral intractable tinnitus. Pre- and postoperative visual analogue scale for tinnitus intensity and tinnitus questionnaires for tinnitus distress are analyzed before and after microvascular decompression.

RESULTS: Of the 20 patients studied, 10 had improvements on their tinnitus visual analogue score intensity postoperatively, 8 were unchanged, and 2 worsened. On the Tinnitus Questionnaire scores, 7 of 13 patients improved and 6 of the 13 patients worsened. If decompression is performed before the end of the 4th year of tinnitus duration, a significant tinnitus intensity improvement can be obtained ($P < .05$); after 4 years, improvement cannot be obtained ($P = .55$). However, the tinnitus distress does not seem to decrease significantly.

CONCLUSION: Microvascular decompression of the cochlear nerve can improve tinnitus intensity in selected patients if decompression is performed early, before the end of the 4th year. Tinnitus distress does not seem to change.

KEY WORDS: Cochleovestibular compression syndrome, Tinnitus, Microvascular compression, Microvascular decompression

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Microvascular compression of the cranial nerve (CN) VII–VIII complex can be the cause of several symptoms of both hyper- and hypoactive disorders, including hemifacial spasm,[1] disabling positional vertigo,[2] tinnitus,[3] otalgia,[4] and tinnitus with frequency-specific hearing loss.[5]

It has been assumed that the contact between a blood vessel and a cranial nerve must be localized at the root entry zone to produce noticeable symptoms,[6] but more recent studies indicate that vascular contact with the central segment of a cranial nerve is associated with clinical symptoms, whereas only rarely does a compression of the peripheral segment of a cranial nerve produce symptoms.[7]

ABBREVIATIONS: CN, cranial nerve; **IPL**, interpeak latency; **MRI**, magnetic resonance imaging; **TQ**, Tinnitus Questionnaire; **VAS**, visual analogue scale

A pathophysiological mechanism for microvascular compressions has been suggested as the cause of tinnitus.[8] When a blood vessel comes into contact with the auditory part of CN VIII, a disorganized signal transmission arises (ie, peak II decreases), resulting in tinnitus.[8] When the compression induces a focal demyelization, resulting in slowing down of the signal transmission, hearing loss develops at the tinnitus frequency (interpeak latency [IPL] I-III prolongs).[8] As a compensation mechanism, the signal transmission of the auditory input from the noncompressed side slows down between the cochlear nucleus and the inferior colliculus so that both

signals arrive in the inferior colliculus at the same time (De Ridder, submitted).

After a microvascular decompression, signal transmission does not normalize at the level of the auditory nerve after 1 year, hypothetically due to structural damage.[8] This could explain the prolonged or delayed clinical improvement with microvascular decompressions.

However, this model also suggests that, in time, more damage develops based on the "hammering effect" of the compressing blood vessel. This model suggests that if decompressions are to be performed, they should be performed as soon as possible.[3,5,8-12]

We analyze tinnitus pre- and postoperatively, both for tinnitus intensity, using a visual analogue scale (VAS) asking "how intense is your tinnitus?" on a scale from 0 (= not at all) to 10 (= the most intense imaginable), and for tinnitus-related distress, using a validated Tinnitus Questionnaire (TQ; score 0–80)[13] originally published by Goebel and Hiller.[14]

METHODS AND MATERIALS

The study population was selected from a database consisting of 208 patients with tinnitus who presented at the multidisciplinary TRI (Tinnitus Research Initiative) Tinnitus Clinic of the Antwerp University Hospital, Belgium. Based on a recently developed research classification[8](Table 1), the relevant associated symptoms, eg, optokinetic vertigo, ipsilateral cryptogenic hemifacial spasms and ipsilateral paroxysmal short bouts of otalgia (geniculate neuralgia), or feeling of pressure in the ear were documented. Auditory brainstem response (ABR) parameters were assessed using Møller's criteria[15] and considered abnormal if at least one of the Møller criteria was met. The patients' magnetic resonance imaging (MRI) scans were visually inspected for a vascular compression of the eighth cranial nerve.

TABLE 1. Criteria and Classification for CVCS[a]

Criteria
1. Intermittent paroxysmal spells of unilateral tinnitus lasting only seconds
2. Associated ipsilateral symptoms
a. Cryptogenic or overt HFSs
b. Otalgia with or without deep facial pain or feeling of pressure in the ear
c. Vertiginous spells: short-lasting, optokinetically induced
d. Ipsilateral hearing loss at tinnitus frequency
3. Positive MRI for vascular conflict
4. Positive brainstem auditory evoked potential, using Møller's criteria[25]
Classification
Possible CVCS: initially intermittent, unilateral tinnitus spells without associated symptoms
Probable CVCS: possible CVCS with associated symptoms (otalgia, vertigo or HFSs) or MRI
Demonstrating vascular compression of cochleovestibular nerve (using high-resolution, heavily T2-weighted CISS images) or abnormal ABR.
Definite CVCS: probable CVCS with associated symptoms and/or abnormal ABR and/or abnormal MRI.
Certain CVCS: definite CVCS that is surgically proven.

[a] ABR, auditory brainstem response; CISS, constructive interference in steady state; CVCS, cochleovestibular compression syndrome; HFS, hemifacial spasm.

Based on these criteria (ie, associated clinical symptoms, abnormal ABR, and abnormal MRI), 20 patients were classified in the definite cochleovestibular compression syndrome (CVCS) group (Table 1)[8] and in the certain CVCS group after undergoing a surgical microvascular decompression.

Twenty patients—9 men and 11 women—were included in this study. Patients' ages ranged between 40 and 72 years, with a mean of 54 years (SD = 9.51 yr) (see Table 2 for patient overview). The mean preoperative score on the VAS for tinnitus intensity was 7.80/10 (range, 4–10) (Table 2). The mean preoperative TQ score was 53.47/80 (range, 29–77). However, preoperative TQ data from 5 patients and postoperative TQ data for 2 patients were lost, and, therefore, TQ analysis was performed on only 13 patients.

Results of microvascular decompression[s3,5,8-12] and transcranial magnetic stimulations[16,17] suggest tinnitus treatment results worsen over time, with a turning point of efficacy between 3 and 5 years. We therefore opted to take 4 years as a cut-off for defining tinnitus of recent onset vs chronic tinnitus. This fits with a magnetoencephalographic study that demonstrates that after 4 years the tinnitus-generating network changes its connectivity.[18] Performing a median split on our data also gives a cut-off of 4 years.

All analyses were done with SPSS statistical software (version 15.0, Chicago, IL). We performed a Wilcoxon Z-test, due to the small sample, to verify whether there are differences in both VAS intensity and TQ between preoperative vs postoperative measurement for both the tinnitus of recent onset and chronic tinnitus groups. In addition, the Pearson correlation was calculated between tinnitus duration and postoperative VAS intensity.

RESULTS

Of the 20 patients studied, 10 had improvements on their tinnitus VAS postoperatively (7 patients with tinnitus of recent onset; 3 patients with chronic tinnitus); 8 patients had neither benefit nor worsening; and 2 patients worsened. For the tinnitus of recent onset group, a significant improvement of 23.03% (mean VAS difference = 1.67; SD = 2.27) on the VAS intensity was obtained ($Z(11) = -2.20$, $P < .05$). The mean preoperative VAS score was 7.2 (SD = 1.49); the mean postoperative VAS score was 5.58 (SD = 2.35). For the chronic tinnitus group, no significant improvement was obtained ($Z(7) = -.39$, $P = .71$). The mean preoperative VAS score was 8.63 (SD = 1.30); the mean postoperative VAS score was 8.25 (SD = 2.05).

Of the TQ scores, 7 of 13 improved (4 patients with tinnitus of recent onset; 3 patients with chronic tinnitus), and 6 of 13 worsened. For both the tinnitus with recent onset and chronic tinnitus groups, no significant improvement was obtained on the total TQ scores: Z (6, degrees of freedom) = .00, $P = 1.00$ and Z (5, degrees of freedom) = -.11, $P = .92$). For the acute tinnitus group, the mean preoperative TQ score was 57.57 (SD = 18.07)

TABLE 2. Overview of Patients[a]

Patient	Age, y	Sex	Tinnitus Duration, y	VAS		TQ	
				Preoperative	Postoperative	Preoperative	Postoperative
1	48	F	15	8	8	29	36
2	49	M	3	8	7	63	54
3	56	F	9	7	10	52	73
4	63	M	2	9	8	46	59
5	48	F	2	7	7		58
6	64	F	4	7	4	71	69
7	63	M	4	9	9	73	70
8	64	M	3	5	6	29	35
9	46	M	14	9	7	33	25
10	45	F	7	10	10	63	57
11	55	M	2	8	3		55
12	72	M	11	10	10	59	63
13	40	F	2	7	1		11
14	44	F	5	7	5		63
15	62	F	4	8	7	77	54
16	48	M	5	10	10	77	66
17	60	F	2	7	7	44	63
18	65	F	3	8	4	37	
19	48	M	1	4	4	49	
20	40	F	11	8	6		16

[a] TQ, Tinnitus Questionnaire; VAS, visual analogue scale.

and the postoperative score was 57.71 (SD = 11.91); in the chronic tinnitus group the mean preoperative TQ score was 52.17 (SD = 18.36) and the postoperative score was 53.33 (SD = 18.75).

One patient with tinnitus of recent onset and one patient with chronic tinnitus had improvement on both VAS intensity and the TQ score.

A positive correlation was found between the duration of tinnitus and postoperative VAS intensity ($r(20) = .44$, $P < .05$) (Fig. 1), indicating the longer a patient has tinnitus, the higher the postoperative score on VAS for tinnitus intensity. Although it was not a significant correlation, a positive trend was found between the duration of tinnitus and preoperative VAS intensity ($r(20) = .41$, $P = .08$)

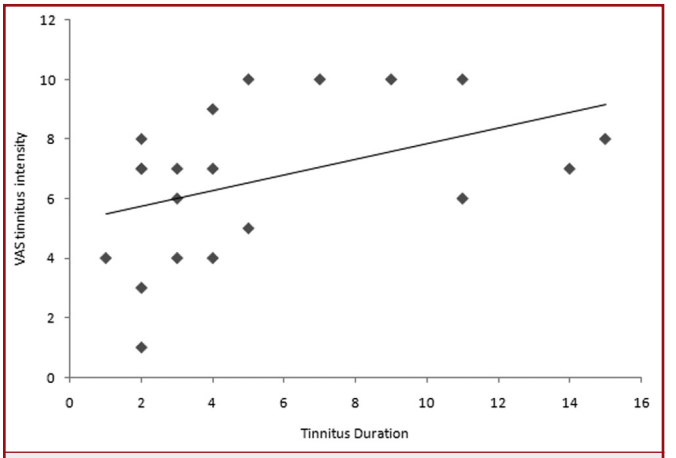

FIGURE 1. *Pearson correlation between tinnitus duration and postoperative tinnitus intensity score (VAS, visual analogue scale).*

DISCUSSION

This study shows that microvascular decompression of the auditory nerve in patients with signs and symptoms of tinnitus from vascular contact with a blood vessel can be improved only if their tinnitus has been present for less than 4 years. This is in agreement with previously published reports,[3,5,8-12] and is statistically significant. In the Møllers' study, the patients who experienced total relief and those who showed marked improvement had experienced their tinnitus for an average of 2.9 years and 2.7 years, respectively; those who showed slight improvement and those who had no improvement had experienced their tinnitus for a longer time before the operation (mean, 5.2 and 7.9 yr, respectively). In

Brookes' study, 2 patients who had no benefit from the surgery presented with long-standing tinnitus of 6 years. In Ryu's study, average tinnitus duration was 2.1 yr, too short to note a differential effect.

Based on clinical and neurophysiological data, it has been suggested that tinnitus in CVCS is related to impaired signal transmission in the auditory nerve, and that a prolonged hammering effect of the compressing vessel could lead to focal demyelination with tinnitus frequency-specific hearing loss as a consequence.[8] It has been proposed that, to become symptomatic, a vascular conflict should be located at the CNS segment (not just the root entry zone), which is the neural generator of peak II. Thus, peak II changes are expected to be the first abnormality to be noted in vascular conflicts of the auditory nerve. Evoked potentials result from a synchronized firing pattern as a reaction to a sensory stimulus.[19] The more synchronized the firing of the nerves, the higher the amplitude of the evoked potentials will be. Contact with a blood vessel may alter neural conduction (ie, decrease conduction velocity in some fibers or inactivate some fibers), causing the temporal coherence of the firing in the central segment of the auditory nerve to decrease, resulting in a decrease of the amplitude of peak II. Clinically, this may result in frequency-specific tinnitus.

In nonoperated patients harboring a microvascular compression syndrome of the vestibulocochlear nerve in the setting of tinnitus, no ABR changes could be detected during the first 2 years, but after that period a decrease of the amplitude of peak II occurred, and a prolongation of the IPL of peak I-III occurred in patients whose peak II had disappeared.[8] The rate of IPL I-III increase slows down after 10 years. IPL I-III prolongation correlates with ipsilateral hearing loss at tinnitus frequency and worsens in time. This correlates with a worsening of the tinnitus associated with the worsening of the IPL I-III.[8] Tinnitus frequency correlates to the frequency of maximal hearing loss, and the more the hearing loss at tinnitus frequency, the worse the tinnitus.[8] In operated patients, postoperative improvement of tinnitus correlates with postoperative improvement of peak II, and postoperative improvement of hearing loss at the tinnitus frequency correlates with postoperative IPL I-III improvement.[8] Thus, it was concluded that tinnitus initially is the result of impaired signal transmission at the level of the vascular contact, electrophysiologically characterized by a decrease in peak II. However, the longer the compression exists, the more damage is induced on the auditory nerve, electrophysiologically characterized by an increase in IPL I-III. This correlates with a frequency-specific hearing loss, and the perceived tinnitus is louder with more damage to the nerve. Thus, whereas initially a microvascular contact generates tinnitus by dyssynchronized signal transmission, in time the tinnitus may become more closely related to a frequency-specific hearing loss and auditory deafferentation. The natural history of CVCS is a worsening of the perceived tinnitus over time, and once hearing loss sets in it may be too late for recovery. In this study, a similar trend ($r(20) = .41$, $P = .08$) for duration of tinnitus and preoperative VAS intensity also can be noted. Tinnitus resulting from hearing loss has been shown to have the same spectral characteristics as the hearing loss.[20] Therefore it, can be suggested that tinnitus in CVCS initially results from disorganized signal transmission in the auditory nerve, neurophysiologically seen as an ipsilateral peak II decrease.[8] But once demyelination sets in, seen as IPL I-III prolongation,[8] the tinnitus might be related to the deafferentation of auditory input to the thalamocortical system, resulting in thalamocortical dysrhythmia,[21] neurophysiologically expressed as an increase in gamma band activity in the contralateral auditory cortex.[21,22]

Based on recent auditory processing data in patients in a vegetative state[23,24] and theoretical models of consciousness,[25] it has become clear that tinnitus is the result of a network activity and is not restricted to auditory cortex activation. The exact tinnitus network has not been detailed, but it includes both auditory and nonauditory areas, such as frontal, parietal, and cingulate areas.[18,26-29]

An explanation for the differences in surgical outcome between the tinnitus of recent onset and chronic tinnitus groups may be found by considering the functional architecture of the brain, which changes over time. This theory is consistent with a recent magnetoencephalography study, which revealed that the distribution of gamma in the neural network changes over time. Magnetoencephalography analyzes spontaneous magnetic activity in the brain, analogous to the way in which electroencephalography measures electrical activity. Using phase locking analysis in the 1 to 90 Hz frequency range of 1 minute of resting-state magnetoencephalography recording, functional long-range coupling in tinnitus can be analyzed. In patients with a tinnitus history of less than 4 years, the left temporal cortex was predominant in the gamma network, whereas in patients with tinnitus duration of more than 4 years, the gamma network was more widely distributed, including more frontal and parietal regions.[18] Another electoencephalographic study further revealed that the neural generator in the brain of a patient with tinnitus changes over time (Vanneste, submitted). A similar process could be operating in microvascular compression of the CN VII–VIII complex. The irritation of the blood vessel leads to reorganization of auditory nuclei in the auditory brainstem, probably induced by the expression of neural plasticity.[30] This neural plasticity may affect the entire auditory pathway, including the auditory cortex, causing reorganization[31] and creating hyperactivity in the auditory cortex, which may be clinically expressed as tinnitus.[8,9] After 4 years, however, the overactivation of auditory cortex decreases (De Ridder et al, submitted), and other brain areas that may generate tinnitus become more active, which may become independent of the initial sound initiator (ie, microvascular compression). Consequently, decompression surgery has no influence on tinnitus intensity after more than 4 years. Hence, the preoperative duration of tinnitus is important for outcome, can be similar to what has been described for trigeminal neuralgia,[32-35] and seems to be a general characteristic for chronic microvascular compressions.

The literature pertaining to microvascular decompressions for tinnitus has never evaluated whether improvement in tinnitus intensity correlates with an improvement in tinnitus distress. Our results demonstrate that even though the tinnitus intensity improves in a significant way, the clinical relevance seems to be limited,

because tinnitus-related distress does not seem to decrease significantly. This may be because the tinnitus intensity and distress components of the perceived tinnitus are not linearly correlated but demonstrate a cubic function, with VAS = ± 4 as a drop-off (Vanneste et al, submitted). These results, especially on the high VAS intensity scores, show little improvement in tinnitus distress with moderate-intensity improvements. After our surgical decompressions, the average tinnitus intensity improvement is small. The VAS improves from 7.25 to 5.58, too little to produce a dramatic improvement in distress. Due to its cubic relation, a dramatic improvement in distress can be predicted if the tinnitus intensity postoperatively drops below 4.

Therefore, it can be suggested that microvascular decompressions should be treated before the end of 4 years and may yield best results when tinnitus intensity is not higher than 5 or 6 on a VAS, due to the limited improvement that can be obtained with surgical decompression in regard to the VAS intensity. This fits with a previous study that shows tinnitus intensity seems to increase in time in microvascular compressions as more damage develops in time.[8]

CONCLUSION

Based on the pathophysiology of tinnitus in microvascular compressions, surgical decompressions should be performed before the end of 4 years. Tinnitus distress is not improved if tinnitus intensity improvement is limited.

Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

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