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Clinical Article

Frequency specific hearing improvement in microvascular decompression of the cochlear nerve

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Summary

Background. Microvascular compressions of the cochlear nerve can lead to hearing loss. Due to the tonotopic organization of the cochlear nerve any focal compression of the cochlear nerve will result in a frequency specific hearing loss. Decompressing the cochlear nerve could result in a frequency specific hearing improvement, without improving overall hearing.

Method. Thirty one patients underwent microvascular decompression operations of the vestibulocochlear nerve for vertigo or tinnitus. Preoperative audiograms were substracted from postoperative audiograms obtained 2 years after microvascular decompression. The frequencies of maximal hearing improvement postoperatively were determined.

Findings. Of the 31 patients studied, 19 had improvements of 5 dB or more at one or more frequencies postoperatively, and 15 patients had improvements of 10 dB or more. Three patients had improvements of 25 dB or more postoperatively. The postoperative hearing improvement was frequency-specific and related to the anatomical location of the vascular contact on the auditory nerve. The improvement of hearing becomes diluted when the difference between pre- and postoperative hearing thresholds are averaged over all audiometric frequencies. We therefore present results for each frequency that was tested.

Conclusions. Microvascular decompression of the cochlear nerve can improve hearing in selected patients. The improvement seems too small to justify decompressive surgery for the sole purpose of hearing improvement, but it could be considered if associated short vertigo spells, ipsilateral tinnitus, otalgia and cryptogenic hemifacial spasm are present. Decompression should be performed early, before BAEP changes become noticeable. 3D-MRI could become a valuable tool for selecting good surgical candidates.

Keywords: Cochleovestibular compression syndrome; hearing impairment; hearing loss; microvascular compression; microvascular decompression; tonotopy.

Introduction

Microvascular compressions of cranial nerves are known to be involved in the cause of several hyperactive and hypoactive disorders such as trigeminal neuralgia, glossopharyngeal neuralgia, hemifacial spasm, disabling positional vertigo, tinnitus and otalgia [12].

It has been assumed that the contact between a blood vessel and a cranial nerve must occur at the root entry zone (REZ) in order to produce noticeable symptoms [12] but more recent studies indicate that vascular compression of the central segment of a cranial nerve [7], which is characterized by a structurally less resistent anatomical organization is associated with clinical symptoms and only rarely does a compression of the peripheral segment of a cranial nerve produce symptoms [34]. The central segment of cranial nerves differs in length [16, 40, 42] and the length of the central segment and the incidence of the associated microvascular compression syndrome are correlated [7]. Since the central segment of the vestibulocochlear nerve is the longest of all cranial nerves the incidence of the cochleovestibular compression syndrome (CVCS) can be expected to be the highest of all microvascular compression syndromes, estimated at 8–9/100.000 [7].

Microvascular compression of the vestibulocochlear nerve has been associated with attacks of vertigo known as disabling positional vertigo (DPV) [5, 11, 13, 25] and tinnitus [13, 14, 24, 35]. A case report has associated vascular compression of the cochlear nerve with sudden deafness [32]. Some patients with presumed vascular contact with the auditory nerve secondary to hemifacial spasm have unusual dips in their audiograms [23].

In this paper, we show evidence that blood vessels which are compressing the cochlear nerve, anywhere along its cisternal segment, i.e. its central nervous system segment, are associated with hearing impairment, which can be ameliorated by microvascular decompression of the vestibulocochlear nerve, performed as treatment of vertigo and tinnitus.

We hypothesize that vascular compression of the (vestibulo) cochlear nerve can cause frequency-specific hearing loss and that successful decompression of the auditory nerve can partly restore hearing. Okamura *et al.* [29] have shown that hearing improvement of more than 5 dB was achieved in 64% of patients in whom they performed microvascular decompression for cochlear symptoms (tinnitus and hearing loss) [29].

Recently it has been suggested that the human cochlear nerve has a tonotopic structure [8], as has been shown for the cochlear nerve in the cat [3]. The vestibulocochlear nerve is rotated in its course from the cochlea to the brainstem anatomically [39] and functionally [8]. The cochlear nerve is located anterior to the inferior vestibular nerve in the internal auditory canal and at the brainstem it is located posteriorly of the facial nerve. The facial nerve is anterior to the superior vestibular nerve, superior to the cochlear nerve, and anterior to the cochlear nerve close to the brainstem. The intermediate nerve remains in between the cochlear nerve and facial nerve along its cisternal trajectory.

Due to this tonotopic organization it may be expected that focal contact with a blood vessel will cause the greatest hearing loss in a specific and narrow frequency range. Vascular contact with nerve fibers that are tuned to 1,000 Hz would be expected to cause the greatest hearing loss at 1000 Hz with some effect at 500 Hz and 1500 Hz.

Materials and methods

Thirtyone patients were operated upon by one of the authors (H.R.) using microvascular decompression of the VIIIth nerve for vertigo or tinnitus. Patients were only operated on if three conditions were fulfilled: 1. MRI or air-CT showed a vascular conflict with the vestibulocochlear nerve, 2. no other neuro-otological explanation for the symptoms could be found and 3. that medical treatment with carbamazepine, microcirculation enhancing medication, steroids and high osmolarity drugs were ineffective in reducing the symptoms. Pre- and postoperative audiograms were digitalized, subtracted from each other and normalized to the contralateral audiogram to evaluate the improvement of hearing at specific frequencies after a microvascular decompression of the vestibulocochlear nerve. The difference between the contralateral pre- and postoperative audiograms was subtracted from the difference between the ipsilateral pre- and postoperative audiograms. Postoperative audiograms were obtained two years after the operation. Results were included in the study irrespective of the changes in the patients' tinnitus or vertigo.

Surgery

A classical retrosigmoid approach via a 2×2.5 cm craniectomy with the patient in lateral position was used to approach the vestibulocochlear nerve. The nerve was examined carefully along its entire cisternal segment for vascular compression. Any offending artery was gently displaced from the nerve and adhered to the adjacent dura with a small piece of oxycellulose soaked in Biobond® (Yoshitomi Pharmaceutical Industries Ltd, Osaka, Japan) or fibrin glue. Veins which were in close contact with the vestibulocochlear nerves were coagulated and transsected, if no decompression could be envisaged safely (Table 1).

Results

Of the 31 patients studied, 19 (61.3%) had improvements of 5 dB or more in one or more frequencies postoperatively, and 15 patients had improvements of 10 dB or more (Table 1, Fig. 1). Overall, postoperative hearing improvement was evenly distributed among the different frequencies (Fig. 2). Three patients had improvements of 25 dB or more postoperatively (Tabel 1, Fig. 1). Improvement postoperatively averaged over all frequencies was not statistically significant ($P > 0.2$, Wilcoxon test).

In order to minimize measuring errors between pre- and postoperative audiometry, the subtracted audiograms (preoperative minus postoperative) were normalized (Table 1) by comparison with the results of the operated side. This normalization would cause false positive improvement on the operated side if hearing in the non-operated side deteriorated. No major differences are noted however between such normalized audiograms and the difference between pre- and postoperative audiograms without normalization (Table 1). Only the results obtained in case #5 could be regarded a false positive result (of the surgery) due to the normalization. No major differences are seen when 5 dB improvements or 10 dB improvements are taken as thresholds in relation to the site of compressions (Table 1).

The improvement in hearing that is achieved is related to the anatomical location of the vascular contact on the auditory nerve. Decompression at the postero-inferior side of the cochlear nerve (Fig. 3a) leads to the greatest mean improvement for low frequencies (10 dB at 250 Hz; SD = 4 dB) and 6.25 dB mean improvement for 500 Hz (SD: 2.5 dB); hearing loss was present

Table 1. *Postoperative improvement of hearing threshold [6]*

#	Case #	Age and gender	Side	Tinnitus or vertigo resolved	Hearing improved	Hearing improvement (dB)	Hearing improv. normalized	Preop–postop (max dB improvement)	Preop–postop normalized	Compressive bloodvessel	Site of compression
1	1	61F	R	−	−					AICA + PICA	PI
2	3	47M	L	+	+	25	35	1000	1000	AICA	PS
3	4	24M	R	+	−					AICA	PI
4	5	53M	L	+	+	5	0	8000	4000–8000	SCA	PS
5	7	53M	L	−	−					AICA	PS
6	8	53M	R	+	+	25 + 20	30 + 20	500 + 2000	500 + 2000	AICA	PS + PI
7	9	31F	L	+	+	5	5	250–500	500	AICA	PI
8	10	52F	L	+	−					AICA	PS
9	12	47F	R	+	+	35 + 35	30 + 30	250–500 + 4000–8000	250–500 + 4000–8000	AICA	PS + PI
10	13	54M	R	−	u	u	u			AICA	
11	14	68F	R	+	+	10	10	8000	4000–8000	PICA	PS
12	15	42F	R	+	+	10	10	4000	4000	PICA	PS
13	16	46M	L	+	−					AICA	
14	17	46F	R	+	+	10	10	1000	1000	Chor plex	
15	18	38F	R	+	−					AICA	
16	21	53F	L	+	−					AICA	
17	23	43M	L	+	+	15	35	2000–8000	4000–8000	AICA	PS
18	25	64M	L	+	−					AICA	
19	29	49M	L	+	+	15	15	2000–8000	2000–8000	Vein	PS
20	30	50F	R	+	+	10	15	8000	8000	AICA	PS
21	31	67F	R	+	+	25 + 30	25 + 25	250–500 + 4000–8000	250–500 + 4000–8000	AICA + PICA	PS + PI
22	33	73F	L	+	+	10	10	250–1000	250–500	VA	PI
23	34	44M	R	+	+	15	20	2000	2000–4000	Vein	PI
24	35	52M	L	+	+	5	15	4000	4000–8000	AICA	PS
25	36	48M	L	+	+	15	10	250–2000	250–1000	VA	PI
26	38	42M	R	+	+	10	5	8000	8000	AICA	PS
27	39	58M	L	+	+	5	5	4000	2000–4000	AICA	PS
28	40	61M	L	−	−					VA	
29	41	42F	R	+	−					VA	
30	42	45M	L	+	+	15	10	500	500	AICA + vein	PI
31	43	44F	L	+	−					AICA + vein	

F Female, *M* male, *R* right, *L* left, + symptom improvement, − no symptom improvement, *u* unavailable, *AICA* Anterior Inferior Cerebellar Artery, *PICA* Posterior Inferior Cerebellar Artery, *SCA* Superior Cerebellar Artery, *VA* Vertebral Artery, *chor plex* choroid plexus? *PS* posterosuperior, *PI* postero-inferior.

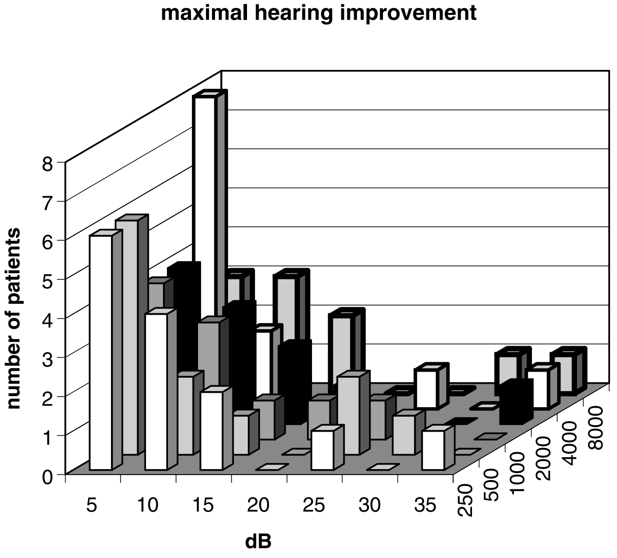

Fig. 1. Distribution of maximal frequency-specific hearing improvement postoperatively plotted both for intensity and frequency. □ 250, ▨ 500, ▨ 1000, ■ 2000, ◘ 4000, ▣ 8000

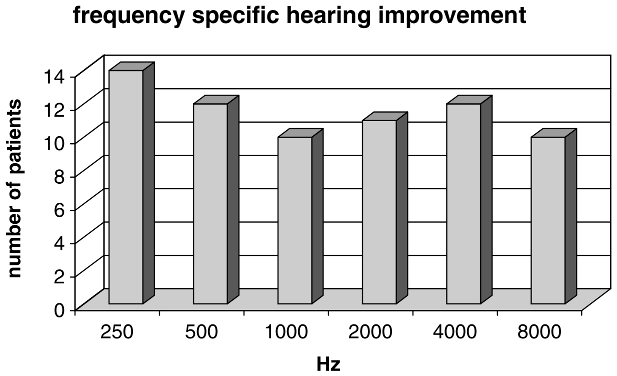

Fig. 2. Distribution of frequency-specific hearing improvement postoperatively. All frequencies improved more or less equally when site of compression is not taken into consideration ($P > 0.2$, Wilcoxon test)

postoperatively at high frequencies. The mean hearing loss at 4000 Hz was 6.25 dB (SD: 11 dB) at 8000 Hz it was 2.5 dB (SD = 5 dB). The improvement is only statistically significant for 250 and 500 Hz when the vascular contact was at the postero-inferior side of the auditory nerve (Student t-test: 250 Hz: $P = 0.016$, 500 Hz: $P = 0.015$, 1000 Hz: $P = 0.08$, 2000 Hz: $P = 0.21$, 4000 Hz: $P = 0.34$, 8000 Hz: $P = 0.39$)

Decompression at the posterosuperior aspect of the cochlear nerve (Fig. 3b) resulted in less improvement of hearing. The mean improvement was 3.75 dB for both 4000 and 8000 Hz (SD: 5 and 8 dB respectively) and a mean improvement of 2 dB (SD: 6 dB) for 250 Hz and 1.25 dB for 500 Hz (SD: 7.5 dB). The improvement was significant (Student t-test) for 4000 Hz only at this site of compression (250 Hz: $P = 0.27$, 500 Hz: $P = 0.57$, 1000 Hz: $P = 0.48$, 2000 Hz: $P = 0.33$, 4000 Hz: $P = 0.03$, 8000 Hz: $P = 0.13$).

No peri-operative mortality occurred. In one patient however a temporary hearing deterioration developed (case 31).

Discussion

Microvascular compression as the cause of hearing impairment

The present study shows that microvascular decompression of the auditory nerve in patients with hearing loss can improve hearing. This indicates that vascular contact with the auditory nerve can cause hearing loss. The improvement after vascular decompression occurs in a limited frequency range and the frequency with the

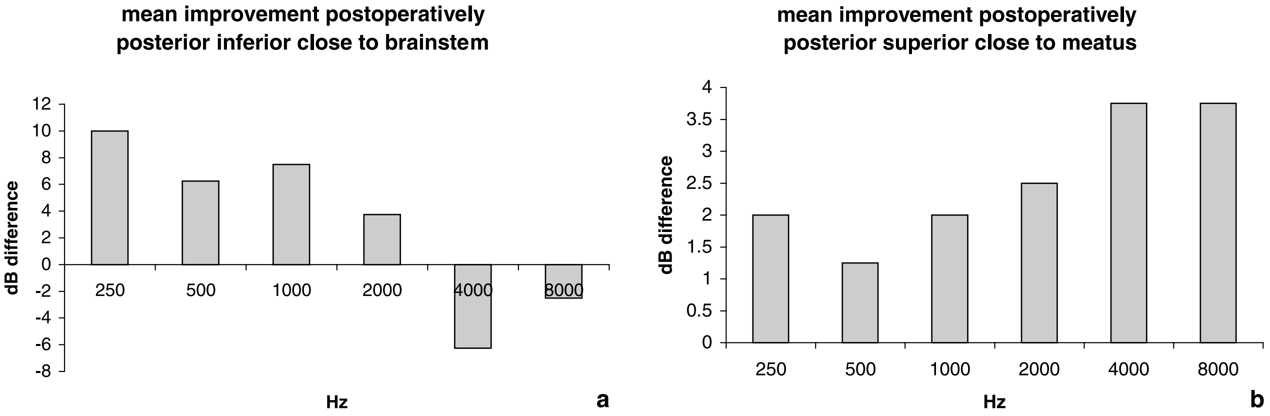

Fig. 3. (a) Frequency-specific hearing improvement postoperatively with consideration of site of compression. Data for compression at the posterior inferior part of the cisternal segment of the cochlear nerve, close to the facial nerve. The improvement is only statistically significant for 250 and 500 Hz (Student t-test: 250 Hz: $P = 0.016$, 500 Hz: $P = 0.015$). (b) Frequency-specific hearing improvement postoperatively with consideration of site of compression. Data for compression at the posterior superior part of the cisternal segment of the cochlear nerve, close to the internal auditory meatus. The improvement was significant for 4000 Hz only (Student t-test: $P = 0.03$)

greatest improvement is related to the anatomical location of the vascular contact, and it is in agreement with the presumed tonotopic organization of the auditory nerve based on studies in animals (cat) [3]. Since the improvement of hearing only occurs in a narrow frequency range the measured improvement of hearing becomes diluted when averaged over all audiometric frequencies.

The fact that vascular compressions create frequency specific forms of hearing loss might be a reason why poor correlation has been found between vascular compression of the auditory nerve and auditory symptoms [5, 17], and that both postmortem studies [15, 18, 41] and imaging studies [30] have shown that vascular contacts with the vestibulocochlear nerve can exist without producing symptoms and signs.

Our data demonstrate that a postero-inferior compression results in low frequency hearing loss and a postero-superior compression to a high frequency hearing loss. Furthermore the hearing improvement in the low frequencies seems to be better than in the high frequencies. Why there is a difference between the amount of hearing improvement between the two groups in not known. One can only speculate: it has been suggested that in humans, nerve fibers originating at the hook of the cochlea, transmitting high frequency sounds, have a smaller diameter than nerve fibers originating at the base, transmitting low frequency sounds [9], similarly to what has been demonstrated in mammals [1–3]. The larger fiber diameter of the low pitch fibers might make them more resistant to vascular compression [44] possibly resulting in less permanent damage and allowing a better functional recovery following decompression.

Diagnostic difficulty

Hearing impairment from vascular compression of the cochlear nerve is rare and its diagnosis is mainly based on history, brainstem auditory evoked potentials and MRI.

History: Hearing impairment that occurs together with symptoms of vascular compression of the vestibular nerve, the intermediate nerve or facial nerve, is more likely to be caused by vascular contact with the auditory nerve, especially if it is frequency specific. For example Moller *et al.* [23] noted that some patients with a hemifacial spasm had hearing loss that occurred in narrow frequency ranges at low and mid-frequencies. Frequency specific hearing loss that is associated with short spells of vertigo [4], optokinetically induced vertigo spells [25], or tinnitus [24, 33], or ipsilateral spells of otalgia [4, 25] (geniculate neuralgia) is likely to be caused by close contact between the auditory nerve and a blood vessel.

In this series of 31 patients, 4 (13%) patients did not show any measurable hearing improvement postoperatively nor benefit of any of the symptoms for which they were operated upon (case# 1, 7, 13, 40), indicating that vascular contact might not have been the cause of their hearing impairment and other symptomatology, even though a vascular conflict was noted during the operation.

The reason why 11 patients (35%) did not have any hearing improvement, even though they did benefit from an improvement of either tinnitus, vertigo or both might be that only the cochlear part of the vestibulocochlear nerve was damaged too much to recover after microvascular decompression. In this series of 31 patients, 29 patients suffered from tinnitus, in 20 of whom it disappeared or markedly decreased postoperatively (6). In six of nine patients in whom the tinnitus did not ameliorate hearing was severely impaired (>60 dB) (case# 5, 8, 18, 21, 25, 43) suggesting that the cochlear nerve was damaged too much to recover [8, 33]. This hypothesis is supported by the fact that hearing loss after microvascular compressions deteriorates over time (31).

Brainstem auditory evoked potentials (BAEP) can provide important diagnostic information regarding vascular contact with the auditory nerve. BAEP measures the conduction time of the cochlear nerve [20–22]. The use of BAEP in the diagnosis of a vascular contact with the auditory nerve is based on the knowledge about the neural generators of the BAEP [20–22]. Peak I is generated by the distal segment of the cochlear nerve and peak II originates from the central segment in the cistern. Peak III has more complex generators, mainly the cochlear nucleus. Since vascular compression of the auditory nerve resulting in hearing loss is usually located in the cisternal segment of the auditory nerve, it will cause an increase of the latency of peak II and all subsequent peaks of the BAEP [24, 25, 29, 37]. Increased interpeak latency (IPL) between peak I and III therefore suggests that the proximal portion of the auditory nerve if affected [22, 38]. It seems reasonable to assume that the degree of increased conduction time is related to the severity of the effect on the nerve from the vascular contact. A large degree of slowing of conduction might therefore signify an irreversible stage of cochlear nerve damage [10].

MRI: High resolution heavily T2 weighted MRI images (CISS) has a high sensitivity and specificity for demonstrating microvascular compressions [6, 19, 31, 36]. Newer techniques, involving 3D MRI reconstructing techniques [26, 28, 43] seem to be even more promising in locating the exact site of compression of the cochlear nerve. Correlation between the exact site of vascular compression and specific frequency hearing loss, using this 3D imaging technique is being verified [27]. This finding might add to the diagnostic probability that close contact between a blood vessel and the vestibulocochlear nerve can be the cause of the hearing loss.

Clinical benefit of the decompression

Decompression of the cochlear nerve is capable of improving a frequency specific hearing impairment in 65% of patients suffering a cochleovestibular compression syndrome, and deteriorates hearing temporarily in 13% in this series. The limited clinical benefit of 10 dB does not suggest a microvascular decompression is justified for the sole purpose of improving progressive hearing loss. It has been stated before that in the setting of a microvascular compression of the vestibulocochlear nerve hearing deteriorates progressively in time and tinnitus and vertigo worsen as well [33]. It has also been demonstrated that in the setting of a microvascular compression of the vestibulocochlear nerve tinnitus becomes intractable after 3–4 years. Furthermore tinnitus does not seem to improve if hearing has become afunctional (>60 dB hearing loss) in this series. As in the majority of patients hearing improves, albeit in a frequency specific manner, in stead of deteriorating, these data add to the idea that microvascular decompressions of the vestibulocochlear nerve should be performed as early as possible and not be postponed until hearing loss becomes irreversible.

Conclusion

Microvascular decompression of the cochlear nerve can decrease hearing loss at specific frequencies in selected patients. The observed improvement of hearing seems too small to justify an MVD operation if the sole purpose was hearing improvement but it might halt and even ameliorate slightly progressive hearing deterioration in patients with short vertigo spells, ipsilateral tinnitus, otalgia and hemifacial spasm. Decompression should be performed early, before BAEP changes (increased IPL I–III) become noticeable. 3D-MRI might become a valuable tool for selecting good surgical candidates.

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Comments

As you know, cranial nerve surgery has become one of my major specialties and consequently I read this paper with interest. In a series of 31 patients the authors have demonstrated moderate hearing improvement in 19 cases. The surgery was carried out with the intention of treating tinnitus or vertigo which was successful in 27/31 cases.

This type of microvascular decompression surgery is not widely practised in Europe, particularly not in the UK where this article will be especially educational. Whilst it has already been shown that modest hearing improvement can be achieved by decompressing the vestibulocochlear nerve, this paper is the first to describe the different frequencies which can be specifically affected by compression. The authors reasonably suggest that this is due to the tonotopic organisation of the axons.

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The purpose of this study was to show evidence that blood vessels that are in contact with the cochlear nerve are associated with hearing impairment, which can be ameliorated by microvascular decompression of the vestibulocochlear nerve, performed as treatment of vertigo and tinnitus. Preoperative audiograms were substracted from postoperative audiograms obtained 2 years after microvascular decompression. One of the main findings of the study is that microvascular decompression of the cochlear nerve can improve hearing in selected patients.

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