

## *Editorial*

# Is there a place for microvascular decompression?

**A. R. Møller**

University of Texas at Dallas, School of Behavioral and Brain Sciences, Texas, USA

The best-known microvascular compression disorders are hemifacial spasm (HFS), trigeminal neuralgia (TGN), and glossopharyngeal neuralgia (GPN) involving cranial nerves VII, V and CNIX [7, 9, 26]. Vascular contact with nervous intermedius and CNIII have been associated with geniculate neuralgia [18], and vascular contact with CNIII has been associated with cyclic oculomotor spasm and palsy [15], respectively.

Vascular contact with the cochlear-vestibular nerve (CNVIII) has been associated with disabling positional vertigo (DPV) [10, 31] and tinnitus [22, 23, 30].

Studies have indicated that the anatomical location of the physiological abnormality in patients with HFS is the central nervous system [19, 27] rather than the respective cranial nerve as was earlier assumed [9, 32] supporting the hypothesis of Ferguson [7]. This indicates that expression of neural plasticity is involved in creating both hyperactivity and re-routing of information that are the signs of HFS [19] and that the vascular contact with a cranial nerve is only one of several factors that are all necessary to produce symptoms [19]. Involvement of the central nervous system is also evident from disorders such as TGN that can be effectively treated by MVD operations as well as by medication that is likely to act on the central nervous system [8].

Re-routing of information may explain abnormal sensations such as the abnormal awareness of head movements in patients with DPV, similar to hyperalgesia and allodynia in central pain disorders [21]. Re-routing of auditory information through the non-classical ascending pathways in some patients with tinnitus [29] may open subcortical connections to limbic structures [21, 22, 24] and may explain why affective signs and symptoms such as phonophobia and depression often accom-

pany severe tinnitus. Other studies have shown evidence of increased activation of limbic structures in patients with tinnitus [17].

Many forms of tinnitus are phantom disorders [11] that are referred to the ear while the anatomical location of the physiological abnormalities that cause the tinnitus in fact is the central nervous system where the abnormalities are caused by expression of neural plasticity. Many forms of tinnitus have similarities with central neuropathic pain [21, 25].

The selection of patients for MVD of CNVII, CNV and CNIX depends almost entirely on the patient's history and assessment of the patients' symptoms and history is also essential for selecting patients with symptoms from the CNVIII for MVD operations. While HFS, TGN and GPN have distinct diagnostic characteristics, the diagnosis of DPV is less distinct and careful selection of patients for MVD operations for DPV is necessary to achieve a similar effectiveness of the MVD operation for DPV as for HFS and TGN. The symptoms of DPV are similar to other several disorders of the vestibular system and it is especially challenging to determine which side that is affected in patients with DPV.

In addition to the patients' history, two different objective methods have been described for selecting patients with DPV and tinnitus for MVD operations. One makes use of electrophysiological methods (ABR) [30, 31] and the other makes use of imaging methods [3, 6]. The fact that vascular contact may occur without symptoms [34] would seem to make imaging methods less effective in diagnosis. Other tests may be negative, thus in one study, 43% of patients with severe DPV had normal ENG [31].

With proper selection of patients, the cure rate for DPV is approximately 80% [3, 31] thus similar to that of HFS and TGN, which have cure rates of approximately 85% [1, 2]. Several investigators have reported that the patients who were operated for DPV required more than one operation.

The diversity of disorders that produce similar symptoms is even greater for tinnitus than for DPV and may be one of the reasons for the low cure rate of tinnitus. Tinnitus is a large group of disorders with diverse and poorly defined pathophysiology, and the forms of tinnitus that respond to MVD operations have yet not been defined to an extent that makes it possible to establish definite selection criteria. Thus not only does bilateral tinnitus not respond to MVD operations but also many forms of unilateral tinnitus does not respond to MVD treatment.

An important indicator of vascular contact with the auditory nerve is abnormalities in the ABR (IPL I–III prolonged and absence of peak II) but also auditory neuropathy of other causes have similar signs and can cause tinnitus but such forms of tinnitus may not respond to MVD operations.

It is a general problem to differentiate between symptoms that are caused by disorders of the ear and those of the auditory-vestibular nerve. Symptoms of disorders such as vestibular or auditory neuropathy of various causes may be difficult to distinguish from symptoms of vascular compression.

Cure rates for tinnitus have been reported to be in the order of 40% [33, 5, 14, 30]. There is general agreement that the MVD operation is much less efficient in treating patients who have had tinnitus for more than 4 years than for patients who have had tinnitus for a shorter period [30]. The fact that the success of MVD operations in men and women differs considerably (29% and 53% respectively) is another difference from disorders of other cranial nerves that can be effectively cured by MVD with little differences in outcome for men and women.

Vascular compression disorders typically have unilateral symptoms. Bilateral tinnitus does not respond to MVD operations [35], thus similar to bilateral spasm of the face (blepharospasm) that does not respond to MVD of CNVII. (Bilateral tinnitus is not associated with the abnormalities in the ABR that are typical for unilateral tinnitus that respond to MVD treatment [35]).

The lower success rate of MVD operations of the cochlear-vestibular nerve than obtained in patients with vascular compression of other cranial nerves may be

related to anatomical differences between CNVIII and CNVII, CNV and CNIX. The CNVIII has a much longer segment of central myelin than the other cranial nerves [16].

Medical treatment is an option for TGN at least in its early phase, but no such treatment has been found for HFS. Medical treatment (diazepam) of DPV is effective in some patients and similarly, treatment with other benzodiazepines such as Xanax can give some patients relief from tinnitus [13]. However, doubt has been presented regarding the validity of these trials because of their nature (open label). Some investigators have found carbamazepine effective [4]. Patients with tinnitus are also treated by masking sounds, and a program known as tinnitus retraining therapy (TRT) [12] has gained some success.

While the outcome of treatment of HFS, TGN and GPN is relatively easy to evaluate, the results of MVD of the cochlea-vestibular nerve is more difficult to assess quantitatively. Many patients may have some vestibular symptoms after treatment and in particular, patients who are treated for tinnitus can have different degrees of improvement from total relief to a situation where the tinnitus is present but no longer bother them.

## Conclusion

Studies agree that patients with DPV improve after MVD operations at nearly the same rate as those with HFS and TGN. MVD operations must therefore be regarded to be an option when medical treatment is insufficient to provide relief of symptoms or carry unacceptable side effects. The fact that DPV can be totally disabling, essentially keeping the patient bedridden for most of the time must be taken into account when assessing the risk/benefit ratio of MVD operations.

Severe tinnitus can prevent an individual to work and naturally implies a severe reduction in the quality of life, and it can cause patients to commit suicide. The much lower success rate for MVD for tinnitus must be considered when advising patients about MVD operations. Since the likelihood of favorable outcome for both tinnitus and DPV depends on the selection criteria that are used, appropriate diagnostic work-up is essential.

MVD operations on the auditory-vestibular nerve requires substantial experience by the surgeon and such operations carries higher risk of hearing loss than MVD operations of other cranial nerves and the use of intra-operative monitoring [20, 28] of neural conduction in the auditory nerve is essential to reduce these risks. In the

hands of experienced surgeons, other risks such as brain-stem strokes are small but do exist.

The question whether there is a place for MVD operations for the cochlear-vestibular nerve must therefore be answered with a conditional yes.

## References

- Barker FG, Jannetta PJ, Bissonette DJ *et al* (1996) The long-term outcome of microvascular decompression for trigeminal neuralgia. *N Eng J Med* 334: 1077–1083
- Barker FG, Jannetta PJ, Bissonette DJ *et al* (1995) Microvascular Decompression for Hemifacial Spasm. *J Neurosurg* 82: 201–210
- Brackmann DE, Kesser BW, Day JD (2001) Microvascular decompression of the vestibulocochlear nerve for disabling positional vertigo: the House Ear Clinic experience. *Otol Neurotol* 22: 882–887
- Brandt T, Stedding S, Daroff RB (1994) Therapy for benign paroxysmal positioning vertigo, revisited. *Neurology* 44: 796–800
- Brookes G (1996) Vascular-Decompression Surgery for severe tinnitus. *Am J Otol* 569–576
- De Ridder D, Ryu H, Møller AR *et al* (2004) Functional anatomy of the human cochlear nerve and its role in microvascular decompressions for tinnitus. *Neurosurgery* 54: 381–388
- Ferguson JH (1978) Hemifacial spasm and the facial nucleus. *Ann Neurol* 4: 97–103
- Fromm G (1991) Medical treatment of patients with trigeminal neuralgia. In: Fromm GH, Sessle BJ (eds) *Trigeminal neuralgia*. Butterworth-Heinemann, Boston, pp 133–144
- Gardner WJ (1962) Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. *J Neurosurg* 947–958
- Jannetta PJ, Møller MB, Møller AR (1984) Disabling positional vertigo. *New Engl J Med* 1700–1705
- Jastreboff PJ (1990) Phantom auditory perception (tinnitus): mechanisms of generation and perception. *Neurosci Res* 8: 221–254
- Jastreboff PJ, Jastreboff MM (2000) Tinnitus Retraining Therapy (TRT) as a method for treatment of tinnitus and hyperacusis patients. *J Am Acad Audiol* 11: 162–177
- Johnson RM, Brummett R, Schleuning A (1993) Use of Alprazolam for relief of tinnitus. *Arch Otolaryngol Head & Neck Surg* 119: 842–845
- Ko Y, Park CW (1997) Microvascular decompression for tinnitus. *Stereotactic & Functional Neurosurg* 68: 266–269
- Kommerell G, Mehdorn E, Ketelsen UP *et al* (1988) Oculomotor palsy with cyclic spasms: electromyographic and electron microscopic evidence of chronic peripheral neuronal involvement. *Neuro-Ophthalmol* 8: 9–21
- Lang JJ, Ohmachi N, Lang JS (1991) Anatomical landmarks of the rhomboid fossa (Floor of the 4th ventricle), its length and its width. *Acta Neurochir (Wien)* 113: 84–90
- Lockwood A, Salvi R, Coak M *et al* (1998) The functional neuroanatomy of tinnitus. Evidence for limbic system links and neural plasticity. *Neurology* 50: 114–120
- Lovely TJ, Jannetta PJ (1997) Surgical treatment of geniculate neuralgia. *Am J Otol* 18: 512–517
- Møller AR (1993) Cranial nerve dysfunction syndromes: pathophysiology of microvascular compression. In: Barrow DL (ed) *Neurosurgical topics book 13, 'surgery of cranial nerves of the posterior fossa'*, chapter 2. American Association of Neurological Surgeons, Park Ridge, IL, pp 105–129
- Møller AR (1995) Intraoperative neurophysiologic monitoring. Harwood Academic Publishers, Luxembourg
- Møller AR (2005) Neural plasticity and disorders of the nervous system. University of Cambridge Press, Cambridge (in press)
- Møller AR (2003) Pathophysiology of tinnitus. In: Sismanis A (ed) *Otolaryngologic Clinics of North America*. W.B.Saunders, Amsterdam, pp 249–266
- Møller AR (1984) Pathophysiology of tinnitus. *Ann Otol Rhinol Laryngol* 93: 39–44
- Møller AR (2003) *Sensory systems: anatomy and physiology*. Academic Press, Amsterdam
- Møller AR (1997) Similarities Between Chronic Pain and Tinnitus. *Am J Otol* 18: 577–585
- Møller AR (1998) Vascular compression of cranial nerves. I: history of the microvascular decompression operation. *Neurol Res* 20: 727–731
- Møller AR, Jannetta PJ (1984) On the origin of synkinesis in hemifacial spasm: results of intracranial recordings. *J Neurosurg* 61: 569–576
- Møller AR, Møller MB (1989) Does intraoperative monitoring of auditory evoked potentials reduce incidence of hearing loss as a complication of microvascular decompression of cranial nerves? *Neurosurgery* 24: 257–263
- Møller AR, Møller MB, Yokota M (1992) Some forms of tinnitus may involve the extralemniscal auditory pathway. *Laryngoscope* 102: 1165–1171
- Møller MB, Møller AR, Jannetta PJ *et al* (1993) Vascular decompression surgery for severe tinnitus: Selection criteria and results. *Laryngoscope* 103: 421–427
- Møller MB, Møller AR, Jannetta PJ *et al* (1993) Microvascular decompression of the eighth nerve in patients with disabling positional vertigo: selection criteria and operative results in 207 patients. *Acta Neurochir (Wien)* 125: 75–82
- Nielsen V (1984) Pathophysiological aspects of hemifacial spasm. Part I. evidence of ectopic excitation and ephaptic transmission. *Neurology* 418–426
- Okamura T, Kurokawa Y, Ikeda N *et al* (2000) Microvascular decompression for cochlear symptoms. *J Neurosurg* 93: 421–426
- Sunderland S (1948) Microvascular relations and anomalies at the base of the brain. *J Neurol Neurosurg Psychiatry* 11: 243–257
- Vasama JP, Møller MB, Møller AR (1998) Microvascular decompression of the cochlear nerve in patients with severe tinnitus. Preoperative findings and operative outcome in 22 patients. *Neurol Res* 20: 242–248

Correspondence: Aage R. Møller, University of Texas at Dallas, School of Behavioral and Brain Sciences, GR 41, P.O. Box 830688, Richardson, TX 75083-0688, USA. e-mail: amoller@utdallas.edu