International Tinnitus Journal 1, 139-146 (1995)

Surgical Decompression of the Eighth Nerve for Tinnitus

Peter S. Roland, M.D., William Fell, M.D., William Meyerhoff, M.D., Ph.D.
The University of Texas Southwestern Medical Center at Dallas, Dallas, Texas, USA

Abstract: This article reports two cases of vascular decompression for tinnitus, both of which were successful. The pathophysiology of vascular compression syndromes in general and specifically eighth nerve vascular compression syndromes is reviewed. The anatomy of the anterior inferior cerebellar artery and its relationship to the eighth nerve complex is discussed in detail and the literature of vascular compression syndrome reviewed. Although no unequivocal definitive test procedure is available for preoperative diagnosis of vascular compression syndrome, the presence of the following features makes the diagnosis more likely: I-III interpeak latency abnormalities on auditory brain stem response (ABR), abnormalities of stapedius reflex, unilateral sensorineural hearing loss, ipsilateral electronystagmography (ENG) abnormalities, especially spontaneous nystagmus, and radiographic visualization of a vessel contacting the eighth nerve complex.

INTRODUCTION

Tinnitus is a sound of internal origin experienced at least episodically by the majority of persons. Ninety-five percent of the population has perceived a sound at one time or another which appears to have been generated internally. The sound may be described as buzzing, hissing, roaring or screeching. It may be pulsatile or steady. In addition, tinnitus appears to be a regular, although not invariant, accompaniment of hearing loss from any etiology. As such, it is often well-tolerated and many patients with even significant hearing loss report tinnitus as only "mildly annoying". About 15 percent of the U.S. population (40,000,000) experiences tinnitus of sufficient frequency, intensity and duration so as to describe themselves as "suffering" from tinnitus. An additional three to five percent of the population reports that the symptom of tinnitus is so severe as to significantly disrupt lifestyle.[1] An even smaller subset of patients finds the symptoms so disturbing as to contemplate (and sometimes even commit) suicide as the only method of achieving relief. A variety of treatment regimens is available for the management of tinnitus and all of them appear to be helpful for some patients but none is effective for all. Improvement in hearing appears to reduce or eliminate tinnitus in many individuals. Thus removal of obstructing cerumen, repair of tympanic membrane perforations, ossiculoplasty and stapedectomy often result in reduction or elimination of tinnitus if hearing is improved. Exceptions, however, are common and some individuals experience decrease or elimination of tinnitus even though hearing remains unchanged while other individuals note deterioration of tinnitus despite hearing improvement. When amplification is a practical method of rehabilitation, it frequently leads to elimination or reduction in tinnitus. Interestingly it should be noted many individuals who use amplification regularly find that tinnitus is suppressed for many hours after the hearing aid has been removed. Practically speaking, amplification is one of the most useful methods of tinnitus control in individuals whose hearing loss is sufficiently severe to benefit from this modality.

Although some individuals report their tinnitus to be worse in the presence of loud noises, the majority of tinnitus sufferers report diminution of the subjective intensity of tinnitus in the presence of background noise. Conversely, tinnitus is experienced as most severe in quiet environments. Consequently, difficulty initiating sleep is a common complaint of tinnitus sufferers. The use of ambient background noise to "mask" tinnitus and thereby diminish its severity is commonplace. Many tinnitus sufferers, having recognized the relationship between background noise and diminution of tinnitus, use masking techniques regularly without ever having been told to do

Reprint requests: Peter S. Roland, M.D., Associate Professor, Department of Otolaryngology, U.T. Southwestern Medical Center at Dallas, 5323 Harry Hines Blvd., Dallas, TX 75235-9035, USA

so. Monotonous, repetitive noises like the sound of the ventilation system, air conditioner, refrigerator, or fan are most effective since they are not likely to capture the attention of the patient or significant other. Although the television and radio are often effective, they are often disruptive to either the patient or significant other, whose attention is recurrently drawn to them. Nowadays, the use of audio-cassettes or CDs which have captured the sound of the ocean on the beach, wind in the trees, or rain on the roof are widely used for tinnitus suppression. In individuals who awaken as soon as the tape or disc is over, the use of a machine which artificially generates such noises can be very helpful. A more formal application of the same masking technique is the "tinnitus masker." This device looks like a hearing aid but rather than amplifying ambient sounds, it produces a steady narrow band noise matched specifically to the frequency of the patient's subjective tinnitus. Such devices appear to be helpful in five to fifteen percent of severe tinnitus sufferers.[2]

Avoidance of caffeine, nicotine, salt, and stimulants also appears to have a beneficial effect on tinnitus for many individuals.[1] Although a large variety of medicines, vitamins and dietary supplements have been utilized in the management of tinnitus, clinical data exists to support only a few.

All of the sedative-hypnotic group of medications appear to be helpful in tinnitus suppression for many individuals.[3] The best studied and most useful appear to be benzodiazepine derivatives. At least for short periods of time, benzodiazepines appear to significantly decrease tinnitus in many patients. Tolerance, however, is likely to develop and require increasing dosage. The benzodiazepines share with other sedative-hypnotic drugs the disadvantage of producing both psychological and physical dependence. Such dependency increases with increasing duration of use and increasing daily dosage. Consequently, these medications are impracticable for most individuals. Since tinnitus is a chronic condition, long term use of medications is inevitable and tolerance promotes the use of increasingly high daily doses. Few physicians feel that the patient's best interest is served by suppressing tinnitus at the expense of producing addiction.[4,5]

Although some evidence exists regarding the effectiveness of the para-aminobenzoic acid and aminoacyl group of local anesthetics, their use once popular, appears to have waned. Anti-convulsive medications, such as Tegretol®, Mysoline®, and Primidone® have been used in the treatment of tinnitus with varying success.[5,6,7] Many theories of tinnitus origin involve concepts of hyper-excitability in various parts of the nervous system which makes the mechanism of action of anticonvulsant medications very appealing. Tinnitus appears to share some common characteristics with other "central pain states" such as causalgia and phantom limb pain which sometimes respond to anticonvulsant medications. Tricyclic antidepressants, especially Amitriptyline, have also been found useful.[3] The molecular structure of tricyclic antidepressants and anticonvulsants of the Tegretol group is extremely similar. It is unclear whether the benefit to tinnitus patients from such medications occurs as a consequence of their antidepressant effect or as a consequence of a neuroleptic effect. Amitriptyline appears to be effective in some chronic pain states by mechanisms other than those related to improving depression. There seems to be a direct effect on peripheral nerves that is of therapeutic benefit. Surely in some individuals the medicine is useful because it combines an antidepressant and neuroleptic effect.

A variety of psychologically based treatment regimens is helpful in some individuals. These approaches include all of the psychological modalities used in treating depression, including psychodynamically based, insight oriented therapies, counselling, hypnotherapy and biofeedback. Biofeedback, especially, appears to be useful in a significant number of individuals and is generally well-accepted.[1,5]

None of these treatment modalities, however, is effective for all patients and some patients appear to benefit from none of them. When non-surgical conservative management fails and the patient's life is intolerable, consideration should be given to surgical intervention. Patients arriving at this treatment option are not commonand represent less than 0.1% of tinnitus sufferers. Several surgical treatment options, however, have been explored. Lempert (1946) reported that tympanic neurectomy reduced tinnitus in two-thirds of sufferers.[8] Lempert's observation was partially confirmed by Tsyganov in 1968.[9] Also, numerous studies have shown that between 50 and 95 percent of individuals will have significant reduction of tinnitus following surgical sectioning of the eighth nerve close to the brain stem.[10-13] Labyrinthectomy appears to be less effective. When vertigo due to an injured labyrinth requires destruction of the inner ear, eighth nerve section may be a better choice than labyrinthectomy in individuals who complain of bothersome tinnitus.[10-14]

In anacusic patients cochlear implantation appears to suppress tinnitus in a significant number and the operation has been done solely for the purpose of suppressing tinnitus in patients who would not be good implant candidates.[15] Vascular decompression of cranial nerve VIII may also play a role in the treatment of selected tinnitus sufferers.

Case Presentation

Case 1. A 50-year-old male noticed the onset of high pitched tinnitus in his left ear associated with mild hearing loss. He had no other otologic or neurologic symptoms. The tinnitus produced such extreme anxiety that he was essentially incapacitated. A variety of non-surgical modalities were tried, all to no avail. Hearing was normal in his right ear but the left ear manifested a high frequency neurosensory hearing loss. Auditory brain stem response audiometry revealed increased I-III interpeak interval on the left side compared to the right. He underwent a left suboccipital craniotomy. The eighth nerve was found to be compressed by the posterior inferior cerebellar artery. Autologous muscle was interposed between the offending vessel and the eighth nerve. He experienced almost complete relief of the tinnitus. Audiogram performed 18 months postoperatively showed improved hearing. Both his pure tone thresholds and his discrimination score were significantly better. Postoperative auditory brain stem response audiogram showed a return to normal of his I-III interpeak latency.

Case 2. A 44-year-old man developed increasingly severe high frequency tinnitus in his right ear associated with mild hearing loss. He had no other neurological or neurotological symptoms. Neurological and psychiatric examinations were normal. He failed all forms of non-surgical management and remained incapacitated by his tinnitus. Audiometric evaluation showed normal hearing in the left ear but a high frequency loss in the right. He had rollover in the right ear. Stapedius reflexes were absent in the right ear but present in the left. Electronystagmography, ABR, and magnetic resonance imaging were within normal limits.

A right suboccipital craniotomy was performed. Muscle was placed between a compressing branch of the anterior inferior cerebellar artery and the eighth nerve. Postoperatively the patient's tinnitus has improved markedly and he is able to function normally. Rollover disappeared and stapedial reflexes returned to normal 12 months postoperatively.

Pathophysiology

Vascular compression of cranial nerves in the subarachnoid space is a well-established cause of certain types of peripheral cranial nerve dysfunction.[16-22] Displacement of the vessel causing compression has led to elimination or reduction of symptoms in many patients. The success of these operations leads one to ask: Is it not possible that tinnitus might result from vascular compression of the auditory portion of the eighth nerve? Although a relatively large series of tinnitus patients who underwent vascular decompression has been reported by Møller, et al., their observations have not been independently confirmed.[23] The two cases we are reporting in this article support the notion that vascular decompression can produce clinical improvement in tinnitus patients.

The pathophysiology of tinnitus remains poorly understood. There is little or no agreement on its etiology and, indeed, its site of origin remains quite controversial. There is increasing suspicion that more than one site of origin and more than one pathophysiologic mechanism is involved. Several authors have postulated cochlear etiologies.[13,24] Central mechanisms have also been proposed.[24,25]

More thoroughly studied than vascular compression of the eighth nerve is vascular compression of the seventh nerve producing hemifacial spasm. Initially hemifacial spasm was believed to be caused by direct physical effects of the compressing vessel on the facial nerve. Based on histopathologic study of excised segments of compressed facial nerve, it was hypothesized that motion and pressure from the compressing vessel eroded the central myelin sheath and allowed neuronal axons to contact each other directly.[19-20,26-28] The exposed axonal fibers would permit current to spread from a single axon to involve many others and produce mass movement of the face.[14,17] Histopathologic evidence, however, does not support demyelinization as an important part of the pathologic process in cases involving the eighth nerve. Schwaber and Whetsell have examined six nerves from patients with eighth nerve vascular compression syndrome. None had demyelinization.[29] The predominant pathologic finding in all cases was endoneurial fibrosis very similar to that seen after vestibular neuronitis.[30-32] Six nerves were evaluated from persons with other conditions than vascular compression (four had classic Ménière's). All had normal myelin. Two had slight to moderate endoneurial fibrosis.[29]

A second, alternative, hypothesis suggested that hemifacial spasm occurred as a consequence of abnormally enhanced central neuronal activity within the facial motor nucleus of the brain stem.[17,19] The latter hypothesis has received the most support on the basis of intraoperative electrophysiologic observations. Moreover, studies in the rat by Møller's group in Pittsburgh have demonstrated that chronic stimulation of the facial nerve can enhance spontaneous activity in the facial nucleus.[33] Thus even though the site of abnormal neural hyperactivity may be within the brain stem, evidence exists that such a hyperactive state can be produced by irritation of the peripheral nerve distal to its exit from the brain stem. Claussen has recently used brain electrical activity mapping of vestibular evoked responses in patients with

acoustic tumors to show excitatory activity over broad areas of the central nervous system. This finding appears to reflect an abnormal form of increased spontaneous activity. Thus, tinnitus from acoustic tumors appears to be characterized by a state of central disinhibition over broad cortical areas.[25] Gerken, in our laboratory, has shown that cochlear injury can affect central portions of the auditory system.[34] The effectiveness of anticonvulsant agents, tricyclic antidepressants, para-aminobenzoic acid derivatives and benzodiazepines could all be explained on the basis of central inhibition. Benzodiazepine derivatives are known to profoundly decrease spontaneous neuronal activity in the vestibular nuclei as well as many other areas of the brain.

Schwaber and Whetsell have combined the results of their own histopathologic study with the evidence for central nervous system involvement. They have developed a hypothesis in which vascular compression is not the primary pathological event.[29] They propose that an inflammatory process (specifically, neuronitis) first produces neural injury. In most cases, they hypothesize normal axons are insulated from "current leak" from injured or regenerating axons by prominent endoneural fibrosis. When this process is incomplete, chronic auto-stimulation of the nerve may result. Their hypothesis accounts for the marked motion intolerance so characteristic of vertigo produced by vascular compression. Activity in the vestibular nerve is increased during movement which, logically, would increase the likelihood of "current leak". Schwaber and Whetsell believe that the motion transmitted to the nerve by a pulsating artery exacerbates activity within the injured nerve. The combination of chronic "auto-stimulation" and stimulation from the compressing vessel eventually results in re-organization of the vestibular nuclei. Because the vestibular nuclei receives impulses from both the oculomotor nuclei and the proprioceptive system, re-organization of the vestibular nuclei could account for the marked enhancement of the oculovestibular reflex (sensitivity to movement within the patient's visual field) chronic disequilibrium and other characteristic clinical features.[29] We see additional advantage to their hypothesis. It would help answer the vexing question as to why vascular compression of the eighth nerve produces prominent vertigo in some patients; prominent tinnitus in other patients, prominent hearing loss in yet other patients, and no symptoms at all in some patients. The relative prominence of vertigo, tinnitus, or hearing loss would depend on the parts of the nerve injured by viral infection which would determine areas subject to the development of post-injury endoneural fibrosis. If the viral insult was principally to vestibular neurons, then vertigo is the logic consequence of the injury. If the virus initially infected auditory fibers, then tinnitus and/or hearing loss might be the principle symptom. By this hypothesis, symptomatology is the consequence of the initial viral insult and not the compressing vessel which serves only to produce or amplify symptoms based on the pre-existing underlying pathologic morphology.

The principle vessel found in association with the seventh and eighth cranial nerves within cerebellopontine angle is the anterior inferior cerebellar artery (AICA). In 72 percent of cases it arises as a single trunk from the basilar artery. In 12 percent of cases it arises as two separate branches from the basilar artery. In 6 percent of cases it does not have a separate origin from the basilar artery but rather, arises as a branch of the posterior inferior cerebellar artery. In about 10 percent of cases a branch of AICA is given off as a separate cerebellar-labyrinthine artery.[27] From this secondary loop arises the labyrinthine artery (or internal auditory artery), which lies on the gutter of the eighth nerve, and usually divides into three terminal branches: the vestibular, the cochlear, and the vestibulocochlear arteries. AICA invariably loops away from the brain stem laterally toward the meatus of the internal auditory canal, and then loops back toward the brain stem. That portion of the AICA which begins at the basilar artery and courses around the pons is the "premeatal segment." The portion of the AICA associated with the porous acoustic is termed the "meatal segment," and that portion of the artery distal to the eighth nerve bundle, which courses medially to supply the pons and cerebellar peduncle, is the "postmeatal segment." The meatal segment loops outward toward the porous acoustic and often into the IAC.[35] When a secondary cerebellar-labyrinthine artery is present, it generally arises from the afferent (premeatal) loop and curves laterally toward the internal auditory meatus and then back toward the brain stem.[27] Cerebellar branches can arise from either the afferent or efferent portion of the principle or secondary artery.

The main branch of AICA passes under the seventh and eighth cranial nerves in 25% to 50% of individuals, between the seventh and eighth cranial nerves in 35% to 50% of individuals, and above the seventh and eighth cranial nerves in 5% to 10% of individuals.[27,36-38] Similarly, the secondary loop (if there is one) may pass below, between, or above the nerves. Any of these options would be available to the second AICA artery, if more than one arise from the basilar artery. There are generally two or more internal auditory arteries associated with the eighth cranial nerve as it passes through the internal auditory meatus.[27,37,39]

The meatal segment of most AICA loops reach their maximum lateral extent and turn back toward the brain stem within the cerebellopontine angle, just medial to the meatus of the internal auditory canal. This appears to be the case in between 30% and 60% of individuals. In

approximately 25% of cases, the artery loops immediately adjacent to the orifice of the internal auditory canal. AICA loops into the internal auditory meatus between 15% and 50% of cases.[27,35,37-39] The variation in arterial anatomy makes possible a very large variety of anatomic configurations. When the venous anatomy is taken into consideration, then the possible relationships between vessels and nerves in the cerebellopontine angle and internal auditory canal is almost endless.

Cranial nerves exit the brain stem surrounded by glial tissue of central neural origin. This sleeve of central glial elements is accompanied by pia mater. At some point within the subarachnoid space, the central glial sheath terminates. At the glial-Schwann cell junction, where the oligodendroglial myelination ends, the Schwann cell ensheathment begins. Schwann cells and the oligodendroglia of the central nervous system both serve to form and maintain myelin. As the nerve passes through this transition point, a ring of pia mater surrounds the nerve. Neural axons passing through this ring have been noted to undergo constriction.[27] Some authors have noted diminution of myelin at the transition zone, and thickening of the myelin sheath as the nerve passes peripherally. The transition zone occurs at different distances from the brain stem for different cranial nerves. The fourth, seventh, ninth, and tenth cranial nerves, for example, all have transition zones less than 1.5 mm from the brain stem. The eighth nerve has the longest central glial segment, with a transition zone about 8 mm peripheral to the nerve's exit from the brain stem.[27,39]

The average distance between the brain stem and the porus acusticus is between 9 mm and 10 mm.[27,40-41] Therefore, the glial-Schwann celljunction occurs close to the meatus of the internal auditory canal. Tallan noted a very even distribution of Schwann cells along peripheral nerves with neither an increase nor decrease of Schwann cells around the transition zone.[40] Loops of AICA or of its branches appear to reach their lateral-most extent close to the transition zone of the vestibular and cochlear portions of the eighth cranial nerve.

Jannetta, Ouaknine, and others have hypothesized that arterial loops may become elongated on a congenital basis or as a result of aging, indurated by atherosclerotic plaque or fixed by arachnoid adhesions.[27,42-43] Such changes would explain the development of vascular compression syndromes later in life.

Clinical Presentation

Vertigo as a consequence of vascular compression of the eighth nerve is a much more clearly defined entity than is the clinical manifestation of tinnitus from the same etiology. Balance disturbance caused by vascular compression of the eighth nerve is a disorder that has been best characterized by McCabe.[22,44] Relatively constant vertigo, markedly exacerbated by position change and with severe motion intolerance are its defining characteristics. The syndrome can produce complete disability.

Vascular compression of the eighth cranial nerve causing vestibular symptoms has been studied carefully by several authors. Leclercq et al. have reported 10 patients with vertigo due to vascular compression, all of whom had hearing loss and caloric weakness.[45] Brookler and Hoffman have reported five patients, four of which had unilateral caloric weakness, and five of whom had sensorineural hearing loss.[46] Bertrand reported five patients, all with caloric weakness and spontaneous nystagmus.[47] Applebaum and Valvasori reported 15 patients, 14 of which had spontaneous nystagmus and all of whom had sensorineural hearing loss.[43] McCabe and Harker's series reported seven out of eight patients with spontaneous nystagmus and all with sensorineural hearing loss.[22] Wiet reported six patients, all with sensorineural hearing loss.[48] Schwaber has recently reported 63 patients who had symptomatology suggestive of vascular compression. Ninety-three percent of them had an abnormal electronystagmography and more than 75 percent had spontaneous nystagmus. Eighty percent of Schwaber's patients had sensorineural hearing loss.[49] Sakaki et al. operated on 18 patients with eighth nerve symptoms for relief of vertigo. All had a significant hearing loss. Fifteen of the 18 had unilateral caloric weakness. The presence or absence of spontaneous nystagmus was not reported.[50]

Review of the literature thus reveals a fairly broad consensus that individuals with eighth nerve compression have sensorineural hearing loss and ENG abnormalities. Spontaneous nystagmus is a relatively uncommon finding on electronystagmography and the frequency with which it is found in individuals with vascular compression would appear to make the present of spontaneous nystagmus a very useful diagnostic finding. Spontaneous nystagmus is most commonly associated with an acute vestibular lesion. In the majority of such cases, the etiology of the vestibular insult is clear. The clinical setting and symptomatology is rarely such as to suggest the presence of vascular compression syndrome. The relative infrequency with which spontaneous nystagmus is identified in patients with chronic vestibular disorders and its rather common occurrence in individuals with apparent vascular compression of the eighth nerve would appear to make the presence of spontaneous nystagmus the most specific diagnostic abnormality for eighth nerve vestibular compression syndrome.

A variety of audiometric tests have been developed over the years to identify vascular compression of the eighth nerve preoperatively. Many of these are no longer in use, but both stapedius reflex testing and auditory brain stem

testing remain widely utilized and are readily available. In Schwaber's series of 63 patients, a third had abnormalities of stapedius reflex testing, as did our Case 2. Auditory brain stem response testing is the audiometric test procedure most commonly used to identify retrocochlear pathology.[49] Using conventional criteria of abnormality, Wiet reported that three out of five patients with vascular compression syndrome had an abnormal ABR.[48] However, Brookler and Hoffman reported that only one out of five of their patients had abnormal ABR testing and Applebaum and Valvasori reported a normal ABR in all 15 of their patients.[43,48,51] The ABR has been championed as the diagnostic study of choice for establishing vascular compression of the eighth nerve by Møller et al. However, the criteria of abnormality utilized by the Pittsburgh group is considerably more sensitive than that used by others. The criteria used by the Pittsburgh group have been clearly outlined by Møller.[18,23]

1. A difference in the interpeak latency between I and III of 0.2 milliseconds (0.16 milliseconds see if wave II is low or absent) was taken as an indication of involvement of the eighth nerve on the side of the longest interpeak latency.
2. A difference between the interpeak latency of III and V of 0.2 milliseconds between the two sides was taken as an indication of brain stem compression **contralateral** to the side of the **longer** interpeak latency.
3. Absolute values of interpeak latency I to III exceeding 2.3 milliseconds and of interpeak latency III to V exceeding 2.2 milliseconds in patients with normal audiograms were regarded as abnormal.
4. Wave II amplitude < 33% of contralateral side.

In their study of 41 patients operated on for disabling positional vertigo, the Pittsburgh group reported that 35 patients had an abnormal ABR. Of these 41 patients, 30 achieved cure or had significant improvement after vascular decompression.

No reports of how frequently individuals without complaints of vertigo might meet these ABR criteria has been published. Thus the sensitivity and specificity of this test procedure appears to be unknown. In Schwaber's series of 63 patients, he evaluated each patient using these same criteria. Seventy-five percent were abnormal using the Møller criteria. However, two of the 13 patients who had no evidence for vascular compression on surgical exploration had positive ABR test results and three of the 11 patients who did have vascular compression on surgical exploration had negative ABR tests.[49] In a series of 18 patients who had surgery for microvascular decompression of the eighth cranial nerve for vertigo Sakaki et al. reported that ABR testing was normal in 11. Three patients had no response due to the severity of their hearing loss, and four had increased wave I-III inner peak latencies. The criteria they used for establishingABR abnormality, however, is not reported.[50]

In 1985, Applebaum and Valvassori reported 10 patients in whom a vascular loop was seen within the internal auditory canal on air contrast CT cysternography.[43] Patients were evaluated prior to the introduction of MRI scanning, and air contrast CT's were performed to rule out acoustic tumors. Not surprisingly, all of these patients had auditory and vestibular symptoms, since it was such symptoms that precipitated the cysternogram in the first place. However, it is significant that in no case was a loop seen on the contralateral, asymptomatic side. In 1989 McCabe and Gantz reported positive air contrast CT cysternography in all 29 patients with surgically proven vascular loops.[44] They used intravenous contrast material to highlight the vascular structures at the time of air contrast cysternography. They did note three patients who had loops in the contralateral internal auditory canal. In Schwaber's study of 63 patients, some had routine contrasted CT scans, some had air contrasted cysternography, and others had magnetic resonance imaging. A total of 29 patients (a little less than 50%) had abnormalities detected on radiographic imaging, suggestive of vascular loops.[49] Parnes retrospectively reviewed 100 normal MRI scans. He was able to identify a vessel in contact with the eighth nerve complex in 12.5% of scans. When both the nerve and vessel could be visualized on the same screen, contact was apparent in 50%. Radiographic confirmation of nerve-vessel contact is not sufficient evidence for vascular compression syndrome.[52] Schwaber and Hall believe that routine gadolinium enhanced MRI scanning is the study of choice for evaluating patients with suspected cranial nerve compression syndrome.[49]

The largest series of vascular decompressions for tinnitus has been performed by the Pittsburgh group. This series was reported by Marguerita Møller and A.R. Møller in several articles. Most recently they reported the results of surgical therapy in 74 patients operated on over a 10-year period. All patients had a preoperative evaluation which included audiometric pure tone testing with speech discrimination and immittance audiometry including stapedius reflex testing. Brain stem auditory evoked potentials were obtained using 2000 Hz tone bursts or clicks. The same criteria for abnormality was used for tinnitus patients as for patients with disabling positional vertigo. Seventy of the patients had abnormal brain stem auditory evoked potentials. Many had hearing loss and abnormal acoustic reflexes as well. Of the 92 patients who underwent microvascular decompression, 18 percent had dramatic improvement in their tinnitus (five were

totally free of tinnitus). Sixteen percent showed improvement as defined by the ability to return to the activities of daily life. Eleven percent had slight improvement. Forty-nine percent of patients had no improvement but only two patients were worse.

Precise correlations between preoperative auditory testing and outcome are not reported in the Pittsburgh study. However, duration of tinnitus was significantly longer in individuals who had slight or no improvement (5.2 years and 7.9 years) than in individuals who had excellent or marked improvement (2.9 years and 2.7 years).[23] In a subgroup of 19 of these patients compound action potentials were monitored intraoperatively. Results were compared with intraoperative monitoring of eighth nerve potentials from patients with similar hearing loss but no tinnitus. No systematic differences could be found.[53] It appears that, in this large series of patients, vascular decompression for tinnitus was less successful than is vascular decompression for trigeminal neuralgia or hemifacial spasm. Moreover, improvement may take up to a year. The reason for failure is unclear. The most likely explanations, as suggested by the author, are that vascular compression, although present, was not the cause of the patient's tinnitus, or that injury to the central nervous system by prolonged peripheral injury was irreversible.

CONCLUSION

Vascular decompression of cranial nerve VIII can improve or eliminate tinnitus and reversal of preoperative retrocochlear findings seems to confirm that these abnormalities were a direct consequence of compression of the eighth nerve by the offending vessels. It seems clear that intracranial vascular decompression of the cochlear-vestibular nerve can relieve tinnitus in a significant number of properly selected patients. Diagnosis depends on a complete evaluation, including audiometry, ENG, ABR, and radiography. No unequivocal criteria for surgical intervention exist. The presence of retrocochlear findings on audiometric testing, abnormalities of stapedius reflex, abnormal auditory brain stem response audiometry, spontaneous nystagmus or unilateral weakness on ENG, unilateral neurosensory hearing loss, and abnormal imaging studies, all support the diagnosis of vascular compression syndrome. In those patients who are desperate for relief from tinnitus, it seems reasonable to discuss this form of surgical intervention when clinical history and laboratory evaluation support the diagnosis of vascular compression syndrome.

REFERENCES

1. Meyerhoff WL, Shrewsbury DW. Tinnitus. In: Diagnosis and Management of Hearing Loss. WL Meyerhoff (ed.) Philadelphia: W.B. Saunders Co., 1984, pp 95-104.

2. Vernon J, Schleuning A: Tinnitus: A new management. Laryngoscope 88:413-419, 1978.

3. Dobie RA, Sakai CS, Sullivan MD, et al: Antidepressant treatment of tinnitus patients: Report of a randomized clinical trial and clinical prediction of benefit. Am J Otol 14(1):18-23, 1993.

4. Silverstein H, Haberkamp T, Smouha E: The state of tinnitus after inner ear surgery. Otolaryngol Head Neck Surg 95:438-441, 1986.

5. House JW. Panel on tinnitus control: Management of the tinnitus patient. Ann Otol Rhinol Laryngol 90:597-601, 1981.

6. Melding PS, Goodey RJ, Thorne PR: The use of intravenous lignocaine in the diagnosis and treatment of tinnitus. J Laryngol Otol 92:115-21, 1978.

7. Shea JJ, Emmett JR, Mays K, et al: Medical treatment of tinnitus. Ann Otol Rhinol Laryngol 90:601-606, 1981.

8. Lempert J: Tympanosympathectomy: Surgical technique for the relief of tinnitus aurium. Arch Otolaryngol 43:199-212, 1946.

9. Tsyganov AI: Anesthesia of the tympanic plexus in the treatment of subjective tinnitus. Arch Otolaryngol 87:127-131, 1968.

10. Dandy WE: Surgical treatment of Ménière's disease. Surg Gynecol Obstet 72:421-425, 1941.

11. Barrs DM, Brackmann DE: Translabyrinthine nerve section: Effect on tinnitus. J Laryngol Otol (suppl) 9:287-293, 1984.

12. Silverstein H: Partial or total eighth nerve section and the treatment of vertigo. Ann Otol Rhinol Laryngol 86:47-60, 1978.

13. Pulec JL: Cochlear nerve section for intractable tinnitus. ENT Journal July, 1995, pp 468-476.

14. Gardner G: Neurologic surgery and tinnitus. J Otolaryngol (suppl) 9:311-318, 1984.

15. Souliere CR, Kileny PR, Zwolan TA, Kemink JL: Tinnitus suppression following cochlear implantation. A multifactorial investigation. Arch Otolaryngol Head Neck Surg 118:1291-1297, 1992.

16. Møller AR: The cranial nerve vascular compression syndrome: I. A review of treatment. Acta Neurochir (Wien) 113:18-23, 1991.

17. Møller AR: The cranial nerve vascular compression syndrome: II. A review of pathophysiology. Acta Neurochir (Wien) 113:24-30, 1991.

18. Møller MB: Results of microvascular decompression of the eighth nerve as treatment for disabling positional vertigo. Ann Otol Rhinol Laryngol 99:724-729, 1990.

19. Ruby JR and Jannetta PJ: Hemifacial spasm. Ultrastructural changes in the facial nerve induced by neurovascular compression. Surg Neurol 4:369-370, 1975.

20. Iwakuma T, Matsumoto A, Nakamur, N: Hemifacial spasm: Comparison of the three different operative procedures in 110 patients. J Neurosurg 57:753-756, 1982.

21. Schuknecht HF: Pathology of the Ear, 2nd Edition, Philadelphia: Lea & Febiger, 1993.

22. McCabe BF, Harker LA: Vascular loops as a cause of vertigo. Am Otol Rhinol Laryngol 92:542-543, 1983.

23. Møller MG, Møller AR, Jannetta PJ, Jho HD: Vascular decompression surgery for severe tinnitus: Selection criteria and results. Laryngoscope 103:421-427, 1993.

24. Jastreboff PJ: Phantom auditory perception (tinnitus): Mechanisms of generation and perception. Neurosci Res 8:221-254, 1990.

25. Claussen C-F, Schneider D, Koltchev Ch: On the functional state of central vestibular structures in monaural symptomatic tinnitus patients. The International Tinnitus J. 1(1):5-12, 1995.

26. Jannetta PJ, Hackett E, Ruby JR: Electromyography and electron microscopic correlates in hemifacial spasm treated by microsurgical relief of neurovascular compression. Surg Forum 21:449-451, 1970.

27. Ouaknine GE: Microsurgical anatomy of the arterial loops in the ponto-cerebellar angle and the internal acoustic meatus. In: The Cranial Nerves. Samii M, Jannetta PJ (eds). New York: Springer, 1981, pp 378-390.

28. Kumagami H: Neuropathological findings of hemifacial spasm and trigeminal neuralgia. Arch Otolaryngol 99:160-164, 1974.

29. Schwaber MK, Whetsell WO: Cochleovestibular nerve compression syndrome. Ii. Vestibular nerve histopathology and theory of pathophysiology. Laryngoscope 102:1030-1036, 1992.

30. Schuknecht HF, Kitamura K: Vestibular neuritis. Ann Otol Rhinol Laryngol 90 (Suppl) :1-19, 1981.

31. Ylikoski J, House WH: Morphologic findings in the vestibular nerve from patients with vestibular neuritis. Am J Otol 2:28-35, 1980.

32. Belal A, Ylikoski J: Pathologic significance of Ménière's symptom complex: a histopathologic and electron microscopic study. Am J Otolaryngol 1:275-284, 1979.

33. Sen CN, Møller AR: Signs of hemifacial spasm created by chronic periodic stimulation of the facial nerve in the rat. Exp Neurol 98:336-349, 1987.

34. Gerken GM, Solecki JM, Boettcher FA: Temporal integration of electrical stimulation of auditory nuclei in normal hearing and hearing impaired cat. Hear Res 53:101-112, 1991.

35. Rhoton AL Jr, Tedeschi H: Microsurgical anatomy of acoustic neuroma. Otolaryn Clinic N Amer 25(2):257-294, 1992.

36. Sunderland S: The arterial relations of the internal auditory meatus. Brain 68:23-27, 1945.

37. Mazzoni A, Hansen CC: Surgical anatomy of the arteries of the internal auditory canal. Arch Otolaryngol 91(2):128-135, 1970.

38. Salah S, Bock FW, Perneczky A, et al: Vascular anatomy of the cerebellopontine angle. In: Koos WT, Bock FW and Spetzler RF (eds) Microneurosurgery. Thieme, 1978, pp 70-71.

39. Boussens J, Caille JM, Koehler R, et al: A propos de la vascularisation du conduit auditif interne. Cah ORL (Montpellier) 7:763-774, 1972.

40. Tallan EM, Harner SG, Beatty CW: Does the distribution of schwann cells correlate with the observed occurrence of acoustic neuromas? Am J Otol 14(2):131-134, 1993.

41. Lang J: Facial and vestibulocochlear nerve, topographic anatomy and variations. In: The Cranial Nerves. Samii M, Jannetta PJ (eds) Heidelberg: Springer-Verlag, 1981, pp 363-377.

42. Barker FG, Jannetta PJ, Bissonette DJ, et al: Microvascular decompression for hemifacial spasm. J Neurosurg 82:201-210, 1995.

43. Applebaum EL, Valvassori G: Internal auditory canal vascular loops: Audiometric and vestibular system findings. Am J Otol (suppl) Nov, 1985, pp 110-113.

44. McCabe BF, Gantz B: Vascular loop as a cause of incapacitating dizziness. Am J Otol 10:117-20, 1989.

45. Leclercq TA, Hill CL, Grisoli F: Retromastoid microsurgical approach to vascular compression of the eighth cranial nerve. Laryngoscope 90:1011-1017, 1980.

46. Brookler KH, Hoffman RA: Acoustic neuroma or vascular loop? Am J Otol 1:32-36, 1979.

47. Bertrand RA, Milina P, Hardy J: Vestibular syndrome and vascular anomaly in the cerebellopontine angle. Acta Otolaryngol 83:187-194, 1977.

48. Wiet RJ, Schramm, DR, Kazan, RP: The retrolabyrinthine approach and vascular loop. Laryngoscope 99:1035-1040, 1989.

49. Schwaber MK, Hall JW: Cochleovestibular nerve compression syndrome. I. Clinical features and audiovestibular findings. Laryngoscope 102:1020-1029, 1992.

50. Sakaki T, Morimoto T, Miyamoto Kyoi K, et al: Microsurgical treatment of patients with vestibular and cochlear symptoms. Surg Neurol 27:141-146, 1987.

51. Applebaum EL, Valvassori, GE: Auditory and vestibular system findings in patients with vascular loops in the internal auditory canal. Ann Otol Rhinol Laryngol 92 (Suppl 112): 63-69, 1984.

52. Parnes LS, Shimotakahara SG, Pelz D, et al: Vascular relationships of the vestibulocochlear nerve on magnetic resonance imaging. Am J Otol 11:278-281, 1990.

53. Møller AR, Møller MB, Jannetta PJ, et al: Compound action potentials recorded from the exposed eighth nerve in patients with intractable tinnitus. Laryngoscope 102:187-197, 1992.