Radiographic Analysis of Cochlear Nerve Vascular Compression

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Objectives: We analyzed whether radiographically demonstrated anterior inferior cerebellar artery (AICA) vascular compression of the cochleovestibular nerve in asymmetric hearing loss could be correlated to either the symptomatic ear or to cochlear nerve diameter.

Methods: We undertook a retrospective case-control study in which patients were enrolled into a database if audiometry demonstrated asymmetry of 20 dB at one frequency, asymmetry of 10 dB at two frequencies, or a difference of 20% on word recognition scores. If AICA vascular contact was demonstrated on subsequent magnetic resonance imaging of the cerebellopontine angle, patients were included in the study. Patients with vestibular schwannoma or Meniere’s disease were excluded. The AICA contact was graded by a blinded neuroradiologist according to criteria proposed by McDermott et al. The cross-sectional area of the cochlear nerve was measured.

Results: Symptomatic ears could be correlated to a decreased cochlear nerve diameter, but not to the degree of AICA penetration into the internal auditory canal.

Conclusions: AICA vascular compression of the cochleovestibular nerve does not appear to correlate to hearing loss or to cochlear nerve diameter. The finding of decreased cochlear nerve diameter in symptomatic ears implies an alternative mechanism for asymmetric hearing loss.

Key Words: cochlear nerve, cochleovestibular nerve, magnetic resonance imaging, nerve compression syndrome, sensorineural hearing loss, unilateral hearing loss.

INTRODUCTION

The pathophysiologic role of vascular compression of cranial nerves is better defined for cranial motor nerves than it is for cranial sensory nerves. The commonly cited finding, in the year 1875, of a vertebral artery aneurysm in contact with the facial nerve of a patient with hemifacial spasm served as the index case of a model for hemifacial spasm, and the model has been successfully extended to trigeminal neuralgia. The efficacy of surgical intervention in these clinical entities led to additional hypotheses regarding a role for vascular compression in glossopharyngeal neuralgia, geniculate neuralgia, vertigo/dysequilibrium, and unilateral auditory symptoms such as tinnitus and hearing loss. Except for the case of trigeminal neuralgia, surgical decompression for sensory cranial nerve syndromes generally has not enjoyed wide acceptance and is not widely implemented.

A role for vascular compression of the vestibulocochlear nerve (VCN) continues to be the subject of investigation for symptoms such as hearing loss, tinnitus, vertigo, and dysequilibrium. Earlier studies relied upon methods such as air-contrast computed tomography, electrophysiological testing, and histopathologic studies to establish contact between the anterior inferior cerebellar artery (AICA) and the VCN, whereas more recent ones take advantage of the widespread availability and superior imaging capabilities of magnetic resonance imaging (MRI). The AICA follows a highly variable course and is frequently seen in contact with the VCN in asymptomatic patients, and therefore its role in the causation of a proposed “anterior inferior cerebellar artery syndrome” remains controversial.

To address the problem of vascular compression in unilateral auditory symptoms, Chadha and Weiner recently published a meta-analysis of studies in which MRI was used to establish contact between the AICA and the VCN. The two studies that explored unilateral hearing loss and were included in this meta-analysis were those of De Carpentier et al and McDermott et al. The findings of Chadha and Weiner suggest a high likelihood of AICA-VCN contact in unilateral pulsatile tinnitus (80 times more likely) and suggest that subjects with unilateral hearing
loss were twice as likely to have a vascular loop in contact. With specific reference to unilateral hearing loss, these authors observed that although the AICA could contribute to hearing loss, it would rarely be the only cause. We note that the majority of the subjects Chadha and Weiner\(^5\) used for their meta-analysis were drawn from the study of McDermott et al,\(^7\) and that a study by van der Steenstraaten et al\(^6\) that sought to confirm the findings of McDermott et al\(^7\) was excluded from consideration.

Although an electrophysiologic test such as that of the auditory brain stem evoked response (ABR) could make a case for a retrocochlear hearing loss caused by a vascular loop, only the study by De Carpentier et al\(^6\) incorporated both the ABR and MRI. Thus, the association between AICA-VCN contact and hearing loss remains an unresolved question.

Cochlear nerve diameter is known to have a strong positive correlation to spiral ganglion cell count. Nadol and Xu\(^9\) observed that imaging techniques could be used to infer spiral ganglion cell count by measurement of cochlear nerve dimensions. If asymmetric hearing loss is a primarily cochlear process, decreased diameter of the cochlear nerve might be radiographically detectable. Conversely, the finding of both asymmetric cochlear nerve diameter and vascular compression of the nerve could imply an inflammatory process such as that posited by Schwab and Whetsell.\(^10\) The objective of our study was to attempt to correlate cochlear nerve diameter as measured by MRI either to hearing loss or to AICA compression of the VCN.

**PATIENTS AND METHODS**

This study received an exemption from our university’s Institutional Review Board. Subjects were identified by searching billing records from 1999 to 2008 for ICD-9 codes 389.16 (asymmetric hearing loss), 389.10 (sensorineural hearing loss, unspecified), and 388.43 (impairment of auditory discrimination). Subjects with cerebellopontine angle tumors (vestibular schwannoma or meningioma, or Menière’s disease) were excluded. Patients whose workup included only ABR without MRI were excluded, as were those for whom MRI results were not available for direct review. The MRI reports mentioning vascular loops were evaluated by a neuroradiologist, and subjects were included in the study if the images confirmed vascular loop contact with the cochlear nerve. The neuroradiologist was blinded to the clinical information regarding symptoms and audiometric data.

Subjects were included if they were sent for retrocochlear workup for audiometric results showing a sensorineural asymmetry of 10 dB or more at two frequencies, an asymmetry of 20 dB or more at one frequency, or a greater than 20% discrepancy in word recognition scores. Those with other indications for retrocochlear workup, such as unilateral tinnitus or stapedial reflex abnormalities, were excluded. For each subject, the ear with the hearing loss was classified as the symptomatic ear and the contralateral ear was treated as the control.

Thin-slice T2-weighted MRI images employing 3-dimensional Fourier transform constructive interference in steady state were evaluated in the axial plane. The AICA vascular loops were then graded according to the Chavda classification: type I loops (Fig 1A) contacted the cochlear nerve in the cerebellopontine angle but did not enter the internal auditory canal (IAC), type II loops (Fig 1B) entered the IAC and extended less than 50% of its total length, and type III loops (Fig 1C) extended greater than 50% into the total length of the IAC.\(^7\) Vascular loops in the cerebellopontine angle that compressed the cochlear nerve but did not approach the IAC were graded type 0. Because of the objective nature of grading AICA vascular loops with the Chavda classification, a single neuroradiologist was used. The AICA diameter was also classified as “large” if it was greater than that of the facial nerve, and “small” if the diameter of the facial nerve was greater.

Cochlear nerve cross-sectional area was measured in the cranial-caudal and anterior-posterior dimensions at the midpoint of the IAC by use of the Vitrea Caliper tool in GE Centricity Radiology Solutions software (GE, Fairfield, Connecticut, Fig 2). Nerves were considered to be an ellipse, and cross-sectional area was calculated as area = \(\pi \times (\text{radius of the long axis}) \times (\text{radius of the short axis})\).

Statistical analysis of the results proceeded by use of the \(\chi^2\) test to determine whether the symptomatic ear was associated with a smaller cochlear nerve diameter. An analysis of variance was used to compare cochlear nerve size to the Chavda grade of the corresponding AICA loop. Fisher’s exact test was used to determine whether there was an association between the grade of the AICA loop and symptomatic hearing loss. Fisher’s exact test was also used to see whether larger vascular loops were more likely to be on the symptomatic side. A \(t\)-test was used to determine whether larger loops were associated with a smaller nerve caliber.

**RESULTS**

A total of 53 patients ranging in age from 19 to 78 years were used in this study, providing 106 ears for analysis. There were 33 men (62%) and 20 women...
(38%) in the study group. Fifty-one of the 53 patients had bilateral AICA vascular loops. Table 1 shows the grade distribution of AICA loops and their sides in relation to the symptomatic ear.

When comparing sides within individual subjects, we found a statistically significant association (p = 0.0344, χ²) between the relatively smaller cochlear nerve and a relatively greater degree of hearing loss (Table 2).

There was no statistically significant correlation between the grade of the AICA loop and the cross-sectional area of the ipsilateral cochlear nerve (Table 3). Ears with type III loops showed a tendency toward a decreased average nerve caliber, but the difference was not statistically significant.

No association was found within individual subjects when we compared the difference in grade of the vascular loops and the difference in cross-sectional area between ears (Table 4).

There was no statistically significant association with higher-grade vascular loops on the same side as the symptomatic ear. No significant trend was found for AICA loops larger than the ipsilateral facial nerve to be on the symptomatic side. No significant difference was found between the relative size of the AICA loop and the ipsilateral facial nerve and the average cochlear nerve diameter.

<table>
<thead>
<tr>
<th>TABLE 1. CHAVDA GRADE² DISTRIBUTION OF AICA LOOPS AMONG EARS</th>
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<tbody>
<tr>
<td><strong>Type</strong></td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td><strong>Type 0</strong></td>
</tr>
<tr>
<td><strong>Type I</strong></td>
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</table>

AICA — anterior inferior cerebellar artery.
TABLE 2. RELATIVE COCHLEAR NERVE SIZE COMPARED TO RELATIVE HEARING LOSS

<table>
<thead>
<tr>
<th>Symptomatic?</th>
<th>No</th>
<th>Yes</th>
</tr>
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<tbody>
<tr>
<td>Smaller nerve size?</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>Yes</td>
<td>12</td>
<td>19</td>
</tr>
</tbody>
</table>

Table is based on left ear's being compared to right ear within each individual patient \( (\chi^2 = 4.4725; p = 0.0344) \).

DISCUSSION

Although there are numerous analyses of vascular cross compression (VCC) of the cochleovestibular nerve in the literature, whether symptoms of hearing loss or vertigo can arise from this relationship remains controversial. A major factor contributing to the lingering doubt surrounding symptomatic compression of cranial nerve VIII (CN VIII) is the existence of vascular loops in many asymptomatic patients. In the group of patients studied, one of the inclusion criteria was MRI evidence of contact of the cochlear nerve with a vascular loop. Of the general population, evidence of contact between vascular loops and CN VIII has been shown in 7% by computer tomographic cisternography, 11 12% by postmortem dissections, 12 and 14% to 34% by MRI. 6,13 These prevalence rates suggest that vascular contact alone can be considered a normal anatomic variant and not a pathological condition. The difficulty in differentiating vertigo due to VCC from that due to other central and peripheral causes and the difficulty in definitively determining which ear is affected lend additional skepticism to the acceptance of microvascular decompression (MVD) for the treatment of vascular compression of CN VIII.

Contact of vascular loops at cranial nerve root entry zones (REZs), defined as the junction between central and peripheral myelin, has been thought to be responsible for compression syndromes such as trigeminal neuralgia and hemifacial spasm. The REZ is considered to be the region most susceptible to vascular compression. 4 In addition, the more proximal central myelin segment of cranial nerves has also been shown to be more susceptible to compression than the peripheral segment. 14-16 Additional support comes from a demonstrated correlation between the length of cranial nerve central segments and the incidence of a corresponding microvascular compression syndrome. 14 The trigeminal nerve has an REZ of only 0.5 to 1 cm from the pons. 17 The REZ of CN VIII, however, can be located as distally as within the IAC. This location suggests that CN VIII could be susceptible to AICA compression along its entire intracranial length, from the brain stem well into the IAC.

The pathophysiology of VCC is not completely understood, although several theories have been postulated. Compression of the vascular loop can result in impaired perfusion of the cochlea and vestibule, leading to otologic symptoms. 18 Another theory is that pulsatile irritation can cause demyelination of the nerve at the REZ. 19 Demyelination could cause decreased conduction velocity, leading to the formation of false synapses and ephaptic transmission of nerve potentials. 20 One article suggested that vascular loops and cranial nerve symptoms were coincidental and that a diffuse neuritis is responsible for symptoms. 10 Contact alone would not be sufficient for symptoms, and an episode of vestibular neuritis leading to axonal loss and swelling is required to establish attachment between the nerve and the vessel. 10 It has been suggested that the primary abnormality lies in the nucleus of the cranial nerve, and that vascular compression is secondary. 16 Other theories include a common developmental abnormality leading to both the formation of a vascular loop and cochlear malfunction. 5

There is substantial current evidence supporting VCC for other cranial nerve compression syndromes and several older reports of successful use of MVD to treat patients with otologic symptoms. Those questioning VCC of CN VIII have hypothesized that the relief of symptoms after MVD may be simply due to nerve trauma from manipulation, 21 cognitive dissonance, 22 or a placebo effect. However, the concept of VCC as a cause of trigeminal

<table>
<thead>
<tr>
<th>Type</th>
<th>N</th>
<th>Mean Area ( (\text{mm}^2) )</th>
<th>SD ( (\text{mm}^2) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>2</td>
<td>0.99</td>
<td>0.07</td>
</tr>
<tr>
<td>1</td>
<td>63</td>
<td>0.91</td>
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</tr>
<tr>
<td>2</td>
<td>30</td>
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<tr>
<td>3</td>
<td>11</td>
<td>0.89</td>
<td>0.29</td>
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Analysis of variance, \( F \) value = 0.1 and \( p = 0.9589 \).
neuralgia and hemifacial spasm faced initial doubt before gradually gaining widespread acceptance. A study of patients with hemifacial spasm due to VCC and simultaneous otologic symptoms revealed 2 cases of concomitant compression and 3 cases with an offending AICA loop or arteriole in contact with CN VIII.\(^2^3\) One study found a significant association between type II and type III AICA loops and hearing loss, but it did not examine the relationship with cochlear nerve diameter.\(^7\) Although several individual studies did not find a statistically significant association between symptoms and compression, a recent meta-analysis of vascular loops causing otologic symptoms revealed that patients with unilateral hearing loss were twice as likely to have vascular loops in contact with CN VIII than those with nonpulsatile tinnitus.\(^3\) The same meta-analysis also showed that patients with pulsatile tinnitus were 80 times more likely to have a vascular loop in contact with CN VIII than those with nonpulsatile tinnitus.\(^3\) Despite the mixed results of smaller individual studies looking at vascular loops and unilateral otologic symptoms, the meta-analysis with a larger number of subjects and greater statistical power suggests that vascular impingement is associated with unilateral otologic symptoms.

In our view, any attempt to attribute hearing loss to a compressive effect of the AICA on the cochlear nerve would ideally need to establish at least two preconditions: the first is that the hearing loss should show audiometric criteria of a retrocochlear cause, such as a decrease in word recognition scores, PIPB rollover, or increased latencies between waves I and V on ABR testing. The second would be to exclude cochlear causes of hearing loss, whether at the organ of Corti, at the stria vascularis, or within the spiral ganglion. In the case of the former precondition, the article by De Carpentier et al.\(^6\) cited in the meta-analysis by Chadha and Weiner,\(^5\) included ABR data, but Chadha and Weiner\(^5\) concluded that there was no correlation between vascular loops and any of the outcome measures used, including “sidedness” of the hearing loss.

It is the latter precondition, the exclusion of cochlear causes of hearing loss, that our study sought to address. Evidence correlating the diameter of the cochlear nerve to spiral ganglion cell counts, and the interdependence of inner hair cell and spiral ganglion cell populations mediated by neurotrophic factors, support the notion that cochlear hearing loss is reflected in decreased cochlear nerve cross-sectional area. Although Nadol and Xu\(^9\) described a positive correlation between surviving spiral ganglion cell counts and cochlear nerve diameter, they noted that there was significant overlap between the absolute diameters in deaf patients and those in normal controls. Our hypothesis depends, rather, on a relative measurement of the diameters within single patients, with the asymptomatic side serving as the control. The use of contralateral ears as a control may introduce some bias, because 51 of the 53 studied patients had bilateral vascular loops. Although these contralateral ears may be more prone to developing vascular compression, we think that interaural comparison provides the most appropriate control by minimizing the presence of confounding variables that would appear in a study looking at different individuals. In our series, we find that cochlear nerve diameter is decreased in asymmetric hearing loss, and we infer that in most cases cochlear processes are implicated. We could establish no statistically significant relationship between the extent of IAC vascular loop penetration and cochlear nerve diameter, nor could we establish a correlation between vascular loop penetration of any degree and the sidedness of hearing loss. For AICA loops larger than the ipsilateral facial nerve, we found no significant association with average cochlear nerve diameter or with being on the symptomatic side.

In conclusion, the absence of compelling evidence for a role in hearing loss of vascular compression of the cochlear nerve was further underscored by the results of our study. In attempting to elucidate the causes of isolated asymmetric sensorineural hearing loss, we believe it would be more fruitful to continue investigations into cochlear processes. Our study also suggests that although the radiographic assessment of cochlear nerve diameter in cochlear implant candidates has not been widely advocated, measurements of the cochlear nerve using currently available software can be a viable Investigational method with potential applications elsewhere in auditory research.

Acknowledgment: We thank Dr Ren Chen of the Biostatistics Core at the University of South Florida Office of Clinical Research for all of her help with the statistical analysis for this study.

REFERENCES

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