Premiere Publications from The Triological Society

Read all three of our prestigious publications, each offering high-quality content to keep you informed with the latest developments in the field.

**The Laryngoscope**

FOUNDED IN 1896

Editor-in-Chief: Michael G. Stewart, MD, MPH

The leading source for information in head and neck disorders.

[Laryngoscopy.com](http://Laryngoscopy.com)

**Investigative Otolaryngology**

Editor-in-Chief: D. Bradley Welling, MD, PhD, FACS

Rapid dissemination of the science and practice of otolaryngology-head and neck surgery.

[InvestigativeOto.com](http://InvestigativeOto.com)

**ENTtoday**

A publication of the Triological Society

Editor-in-Chief: Alexander Chiu, MD

Must-have timely information that Otolaryngologist-head and neck surgeons can use in daily practice.

[Enttoday.org](http://Enttoday.org)

WILEY
Arachnoid Cysts of the Internal Auditory Canal: An Underappreciated Entity?

Omer J. Ungar, MD; Madeline Franck, MS; Joseph B. Nadol, MD; Felipe Santos, MD

INTRODUCTION
The internal auditory canal (IAC) is the site of diverse space-occupying pathologies, the most common of which is vestibular schwannoma (VS). Other uncommon pathologies include meningiomas, facial schwannomas, cavernomas, hemangiomas, epidermoids, lipochoristomas, metastases, and arachnoid cysts (ACs). The clinical presentation, as in vestibular schwannoma, most commonly includes sensorineural hearing loss (SNHL), tinnitus, followed by disequilibrium, dizziness, vertigo, and less commonly, facial weakness, synkinesis, or spasm.

AC is a rare benign space-occupying lesion, accounting for less than 0.5% of all tumors of the cerebellopontine angle (CPA). These benign cysts are situated between abnormal duplications of arachnoid membranes (true AC) or splitting of a single membrane (pseudo AC). The development of this duplication or splitting is not fully understood and is possibly the result of congenital malformation (primary), trauma, subarachnoid hemorrhage, meningitis, and rarely, after radiation for VS (secondary). The content of an AC is composed of cerebrospinal fluid (CSF), and the protein concentration has been reported as higher than in the ventricular system, likely due to absence of CSF circulation. Several theories hypothesize why ACs expand over time, the most accepted of which are the osmotic gradient between the cisternal and cystal CSF, CSF entrapment due to a slit-valve mechanism, and independent CSF production by the cyst wall.

The aim of this study was to present our case series of ACs, focusing on temporal bone (TB) histopathological findings and correlation with clinical presentation.

MATERIALS AND METHODS
We identified all cases in the Massachusetts Eye and Ear Infirmary TB collection with an AC. Identification of the arachnoid membrane, which is typically composed of closely packed oval cells with pale nuclei and small nucleoli, in at least one section was used as an inclusion criteria. The clinical history was collected during life through enrollment in the National Institute on Deafness and Other Communication Disorders (NIDCD), National Temporal Bone, Hearing and Balance Pathology Resource Registry. After death (average postmortem time = 18 hours, range, 2–90 hours), the TBs were prepared and

Objectives/Hypothesis: To describe the histopathologic findings and clinical presentation of arachnoid cysts (ACs) within the human temporal bone.

Study Design: Retrospective cohort analysis.

Methods: An analysis of all medical records of patients diagnosed with an AC was performed. Temporal bones underwent standard processing for histologic examination. The slides were examined by light microscopy. The histologic findings were compared to premortem clinical data.

Results: Twenty-seven ACs were identified in 22 patients. Twenty ears (74%) had no identified risk factor for AC development. The median volume was 12.8 mm³. The most prevalent location of the ACs was at the fundus (16 ACs) followed by the middle portion of the internal auditory canal (IAC) (six ACs). Nine ACs were asymptomatic. Among the 18 symptomatic ACs, the most common presentation was sensorineural hearing loss (SNHL) (94%), followed by tinnitus (22%). The most affected structure was the cochlear nerve (59%), followed by the vestibular nerve (41%). The average hearing threshold was of moderately severe SNHL and speech discrimination was in the range of 50% on monosyllabic word tests. The median time interval from initial presentation to death was 12 years. No correlation was found between duration of symptoms and AC volume.

Conclusions: AC of the IAC is not uncommon. Its presentation is variable, ranging from asymptomatic to SNHL, with poor speech discrimination, tinnitus, and vertigo. This diagnosis should be kept in the differential diagnosis of retrocochlear pathologies.

Key Words: Arachnoid cyst, temporal bone histopathology, otopathology, temporal bone histology.

Level of Evidence: 4

Laryngoscope, 129:1667–1674, 2019

DOI: 10.1002/lary.27601

Laryngoscope 129: July 2019
processed as previously described. All slides were examined by a single examiner, who was blind to the clinical data until the end of the analysis, using light microscopy.

The size of each AC was measured in three perpendicular plains. The maximal dimensions of each cyst were measured using the directions parallel and perpendicular to the IAC (transverse and anteroposterior dimensions, respectively). The coronal dimension was estimated using the first and last slides in which each cyst was visible. Given that most ACs in our cohort had an ellipsoid shape defined by the cylindrical contour of the IAC, the volume of each AC was calculated using the following formula:

$$AC_{volume} = \frac{\pi}{6} \times (\text{transverse dimension}) \times (\text{AP dimension}) \times (\text{Coronal dimension})$$

Two-dimensional graphic reconstruction of the cochlea was performed using published accepted methods to quantify cellular and acellular elements.

RESULTS

Twenty-two patients were identified to have an AC. There were five bilateral cases totaling 27 ACs. The male to female ratio was 13:9. The median (range) age of death was 80 years (47–96 years). Twelve (44%) of the ACs were located on the right TB, and 15 (56%) were located on the left.

Concomitant ear disease was found in 12 subjects (55%), the most prevalent of which was Meniere’s disease (eight subjects, 67%), followed by SNHL in three subjects (25%). Only one patient had presumed idiopathic sudden SNHL (ISSNHL) without recovery. Twenty ears (74%) had no identified risk factor for AC development. Among those with identifiable risk factors for arachnoid cysts, four patients (15%) had a history of meningitis, and three patients (11%) had a history of significant head trauma. Bilateral presentation was not associated with a higher prevalence of identified risk factors ($P = .621$).

Extension of the cyst’s wall to the CPA (three cysts, 11%), resulted in tearing of the medial cyst’s wall, an artifact of harvesting of the temporal bone, restricting length measurement and volume calculation (Fig. 1A). Additionally, six cysts did not exhibit the typical ellipsoid shape, but a bilobular one (22%) (Fig. 1B), thus not allowing for a reliable volume calculation and leaving 18 ACs for volume analysis. The median (range) volume was 12.8 mm$^3$ (0.1–33.9 mm$^3$), resembling normal distribution (Fig. 1C). The most prevalent location of the ACs was at the fundus (16 ACs) (Fig. 2A), followed by the middle portion of the IAC (six ACs) (Fig. 2B), and ACs extending from the porus acusticus (PA) to the fundus of the IAC (two ACs). The rest of the ACs were either located in the middle portion of the IAC extending to its fundus (Fig. 2C), located in the PA and extending to the middle portion of the IAC, or extending medially as far as the geniculate ganglion.

![Fig. 1](https://www.laryngoscope.com/)

Fig. 1. (A) Horizontal section through the left IAC of a 74-year-old female patient. The thin walls of the AC extend toward the CPA. The last audiogram is significant for 50 dB bone-conduction threshold with speech discrimination of 80%. The extension of the AC to the CPA resulted in tearing the cyst, preventing accurate volume measurement. (B) The horizontal section through the left IAC of a 78-year-old male patient. The bilobular structure of the cyst on both sides of the cochlear nerve prevents accurate volume measurement. (C) Prevalence distribution of the AC as a function of volume among our cohort showing normal distribution. The volume range was from submillimeter to 33.9 mm$^3$. AC = arachnoid cyst; CN = cochlear nerve; Co = cochlea; CPA = cerebellopontine angle; IAC = internal auditory canal; V = vestibule; VN = vestibular nerve. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]
(one AC each) (Fig. 1A and Fig. 2J, respectively). Nine (33%) ACs were occult and identified incidentally after TB processing (Fig. 2K). Among the 18 symptomatic ACs, the most common presentation (17 ears) was SNHL (94%) (Fig. 2D–F), followed by tinnitus (22%), vertigo (17%) (Fig. 2G–I), and motor facial dysfunction in two cases (11%) (Fig. 2J) (Table I).

In terms of the IAC content compression or diversion by AC, the most affected structure was the cochlear nerve (16 cases, 59%) (Fig. 2D–F), followed by the vestibular nerve (11 cases, 41%) (Fig. 2G–I). The facial nerve was affected in two cases (7%) (Fig. 2M). The AC dissected the cochlear and the vestibular nerve fibers (intraneural dissection) in five and three cases, respectively (Fig. 2L and...
The IAC walls were expanded in four cases (Fig. 2N) as a result of long-standing AC. Audiograms were available in 11 cases, with a median of 3 years from the last audiogram to death. The last available audiogram was used to better correlate with the histological findings. The median time (range) from audiogram to death was 6 years (1–14 years). The average hearing threshold of moderately severe SNHL and speech discrimination was low, in the range of the 50s, suggesting retrocochlear pathology (Fig. 3). The median (range) time interval from initial presentation to death was 12 years (2–20 years). No correlation was found between duration of symptoms and AC volume (Fig. 4). It is difficult to objectively assess the degree of cochlear or vestibular nerve compression, retraction, and dissection as a result of AC presence in the IAC. However, no identifiable morphologic features on light microscopy were associated with degree of hearing loss, speech discrimination, or labyrinthine weakness, respectively. There was no correlation found between the AC’s size and clinical presentation (Fig. 5). The demograhic, clinical, and histological data are summarized in Table II. Figure 6 depicts the audiometric and cytological findings of a representative case of a 47-year-old male with an asymmetric SNHL, and audiogram and auditory brainstem response (ABR) suggestive of retrocochlear pathology. A large AC in the left IAC pushed the vestibular nerve anteriorly, compressing it against the cochlear nerve (Fig. 6B). The resultant cochleocytogram showed a diffuse reduction of spiral ganglion cell count in the Rosenthal’s canal, as well as inner and outer hair cell absence, mainly in the basal cochlear turn (Fig. 6A). An ABR was suggestive of left retrocochlear pathology (Fig. 6C).

**DISCUSSION**

The true prevalence of ACs in the IAC is unknown. Prior to the use of high resolution magnetic resonance imaging (MRI), it was reported to be found in 0.005% to 0.5% of operations for suspected IAC neoplasm.4,18 In our cohort, 33% of ACs were asymptomatic, and the resulting symptomatic ACs were not operated on due to failure to diagnose an IAC lesion during life or because of an increased American Society of Anesthesiologists score of the candidate, who was misdiagnosed as having a schwannoma (one patient). None were diagnosed during life.

Our cohort had a high incidence of more than 50% concomitant otologic diagnoses. This may be the result of selection bias, consistent with the fact that most TB donors had some otologic condition that led them to enroll in the NIDCD National Temporal Bone, Hearing and Balance Pathology Resource Registry. The high coexistence of Meniere’s disease (8/27 TBs) with associated endolymphatic hydrops (8/8 TBs) is not understood. Several risk factors for AC development are known, including trauma, subarachnoid hemorrhage, meningitis, and rarely, following radiosurgery with marginal tumor doses of 12 to 13 Gy.9–11 According to the available clinical data, a minority of our cohort suffered one of these conditions (26%) without increased risk for bilateral presentation. An idiopathic etiology was the most common in this series.

Due to the rarity of IAC AC, published reports are limited to one to four cases each, so no gender distribution is available in the literature. In our 22-patient series, the male:female gender distribution was 13:9 (male being more susceptible). This finding is probably the result of

**TABLE I.** The Most Common Presentations Among the Symptomatic Arachnoid Cysts

<table>
<thead>
<tr>
<th>Location</th>
<th>Occult</th>
<th>Hearing Loss</th>
<th>Tinnitus</th>
<th>Vertigo</th>
<th>Facial Paresis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fundus</td>
<td>6</td>
<td>10</td>
<td>4</td>
<td>2</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Middle to fundus</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Middle</td>
<td>1</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Porus to middle</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Porus to fundus</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Geniculate</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>10</td>
<td>17</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>36</td>
</tr>
</tbody>
</table>

FIGURE

Fig. 2. (A) Horizontal section through the left IAC of a 92-year-old female patient. A fundal AC is seen to dissect the saccular branch of the vestibular nerve. (B) Horizontal section through the right IAC of a 66-year-old female patient. The AC is located in the middle portion of the IAC. The vestibular nerve is seen as it is compressed between the cyst’s wall and the cochlear nerve. (C) Horizontal section through the left IAC of an 87-year-old male patient. The AC occupies the middle portion and the fundus of the IAC, extending to the vertical crest (Bill’s bar) and IAC’s roof. (D) Horizontal section through the left IAC. The AC is seen to compress the cochlear nerve against its wall in the bony canal. (E) High magnification of Figure 1D, presenting how the nerve sheath is compressed against the bony spicule. (F) Audiogram of the same patient in Figure 2D and 2E, showing severe SNHL. Speech discrimination was 52%. (G) Horizontal section through the same TB as Figure 2B. The vestibular nerve takes an S shape between the AC and the vertical crest. (H) High magnification of Figure 2G. (I) Audiogram of the same patient presenting profound SNHL. (J) Horizontal section through the left IAC of an 80-year-old female patient. The AC is seen to extend as medial as the geniculate ganglion, causing hemifacial spasm. This was accompanied with hearing loss and reduced speech discrimination (8%). (K) Horizontal section through the left IAC of a 74-year-old female patient. A tiny fundal AC is seen. It was asymptomatic during life. The volume is ~0.1 mm³. (L) Horizontal section through the right IAC of a 53-year-old male patient. The AC is seen to dissect the cochlear nerve in the IAC’s fundus. Surprisingly, this AC was occult. (M) Horizontal section through the same TB as in Figure 2J, showing the AC to access the geniculate ganglion, causing hemifacial spasm. (N) Horizontal section through a left TB, showing an AC located in the middle portion of the IAC, expanding its volume. AC = arachnoid cyst; BB = Bill’s bar; CN = cochlear nerve; Co = Cochlea; CPA = cerebellopontine angle; FN = facial nerve; GG = geniculate ganglion; GSPN = greater superficial petrosal nerve; IAC = internal auditory canal; ME = middle ear; V = vestibule; VN = vestibular nerve. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

Laryngoscope 129: July 2019

Ungar et al.: Histopathology of Arachnoid Cysts of the IAC

1670
selection bias due to the uneven gender distribution in our TB collection (M:F = 624:545, with more males donating both TBs than women).

AC size was presented as volume rather than axis length because volumetric assessments have the greatest clinical utility, sensitivity, and accuracy in measuring tumor size.\textsuperscript{19–21} The ACs’ dimensions and volumes were measured and calculated, respectively, using three orthogonal diameters. This method is subject to overestimation, especially when the contour of the tumor is lobular or not symmetrical.\textsuperscript{19} The ACs’ volumes ranged from submillimetric to \( \sim34 \text{ mm}^3 \), showing normal distribution. These dimensions are consistent with values reported for VS.\textsuperscript{3,22} The only difference between VS and AC in terms of volume distribution is the result of TB preparation. Extension of the AC beyond the PA of the IAC will result in tearing of its wall, not allowing volume calculation using histological measurements. Consequently, the largest ACs are not enrolled in the histology-based volume presentation.

Comparison of AC volume to clinical symptoms yielded no correlation, consistent with some VS studies.\textsuperscript{3} However, other VS studies proved association between SNHL and headache risk to VS size, in contrast to other symptoms such as vertigo or tinnitus.\textsuperscript{22} In our cohort, extremely small ACs were expectedly asymptomatic, and ACs >15 mm\(^3\) presented with SNHL, tinnitus, or vertigo. However, some large (>25 mm\(^3\)) ACs were asymptomatic as well and were found incidentally during TB analysis, whereas one small AC (\( \sim6 \text{ mm}^3 \)) presented with asymmetric SNHL. Symptomatic and asymptomatic ACs were distributed uniformly along the volume axis.

Although the pathogenesis of hearing loss remains unknown, one possibility is neural compression.\textsuperscript{23} We

![Fig. 3. Average bone-conduction threshold and speech discrimination of the cohort. These data suggest retrocochlear pathology.](#)

![Fig. 4. Symptom durations of the cohort as a function of AC volume. The occult cysts are included on the left and excluded on the right, showing no correlation between size and symptom durations. AC = arachnoid cyst. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]](#)

![Fig. 5. Distribution of clinical presentation as a function of volume. The various clinical presentations distributed uniformly along the volume axis, suggesting no correlation between size and symptoms. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]](#)
### TABLE II.
Demographic, Clinical, and Histological Data

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Gender</th>
<th>Age</th>
<th>Side</th>
<th>Concomitant Ear Disease</th>
<th>Meningitis</th>
<th>Trauma</th>
<th>Volume (mm³)</th>
<th>Location</th>
<th>Occult</th>
<th>Hearing Loss</th>
<th>Discrimination (%)</th>
<th>Tinnitus</th>
<th>Vertigo</th>
<th>Facial</th>
<th>Cochlear</th>
<th>Vestibular</th>
<th>Facial</th>
<th>Time From Presentation to Death (Years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>81</td>
<td>R</td>
<td>None</td>
<td>*</td>
<td></td>
<td>11.7</td>
<td>Porus to fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>66</td>
<td>R</td>
<td>Meniere</td>
<td>*</td>
<td></td>
<td>19.7</td>
<td>Mid.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>76</td>
<td>R</td>
<td>None</td>
<td>*</td>
<td></td>
<td>12.8</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>47</td>
<td>L</td>
<td>None</td>
<td>*</td>
<td></td>
<td>8.7</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>15</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>61</td>
<td>R</td>
<td>Meniere</td>
<td>*</td>
<td></td>
<td>8</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>NA</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>82</td>
<td>R</td>
<td>InSSNHL</td>
<td>*</td>
<td></td>
<td>6.1</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>74</td>
<td>L</td>
<td>None</td>
<td>*</td>
<td></td>
<td>0.1</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>96</td>
<td>L</td>
<td>None</td>
<td>*</td>
<td></td>
<td>20</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>18</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>87</td>
<td>L</td>
<td>ISSNHL, Meniere</td>
<td>*</td>
<td></td>
<td>29.8</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>53</td>
<td>R</td>
<td>None</td>
<td>*</td>
<td></td>
<td></td>
<td>NA</td>
<td>Mid. to fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>81</td>
<td>L</td>
<td>InSSNHL, Meniere</td>
<td>*</td>
<td></td>
<td></td>
<td>NA</td>
<td>Porus to mid.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>64</td>
<td>R</td>
<td>Meniere</td>
<td>*</td>
<td></td>
<td>28.5</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>90</td>
<td>L</td>
<td>Meniere</td>
<td>*</td>
<td></td>
<td>9.6</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>72</td>
<td></td>
<td></td>
<td></td>
<td>14</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>74</td>
<td>L</td>
<td>Meniere</td>
<td>*</td>
<td></td>
<td></td>
<td>NA</td>
<td>Porus to fundus</td>
<td></td>
<td></td>
<td></td>
<td>80</td>
<td></td>
<td></td>
<td></td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>F</td>
<td>92</td>
<td>R</td>
<td>Meniere</td>
<td>*</td>
<td></td>
<td>23.3</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>88</td>
<td></td>
<td></td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>91</td>
<td>L</td>
<td>Meniere</td>
<td>*</td>
<td></td>
<td>23.3</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>86</td>
<td></td>
<td></td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>17</td>
<td>F</td>
<td>70</td>
<td>L</td>
<td>InSSNHL</td>
<td>*</td>
<td></td>
<td></td>
<td>NA</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>81</td>
<td>R</td>
<td>None</td>
<td>*</td>
<td></td>
<td>23.9</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>F</td>
<td>90</td>
<td>L</td>
<td>Otosclerosis</td>
<td>*</td>
<td></td>
<td>25.2</td>
<td>Fundus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>M</td>
<td>79</td>
<td>R</td>
<td>None</td>
<td>*</td>
<td></td>
<td></td>
<td>NA</td>
<td>Mid.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>F</td>
<td>80</td>
<td>R</td>
<td>Cong. measles</td>
<td>*</td>
<td></td>
<td></td>
<td>NA</td>
<td>Geniculate gang.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>M</td>
<td>78</td>
<td>L</td>
<td>None</td>
<td>*</td>
<td></td>
<td></td>
<td>NA</td>
<td>Mid.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Cong. = congenital; F = female; gang. = ganglion; InSSNHL = inherited nonsyndromatic sensorineural hearing loss; ISSNHL = idiopathic sudden sensorineural hearing loss; L = left; M = male; Mid. = middle; NA = not available; R = right.

A bullet symbol meaning that this individual arachnoid cyst was associated with the corresponding parameter.
failed to prove any association between AC location along the IAC and presenting symptom (Table I). We found no site of predilection within the IAC having seen them from the porus to the fundus (Fig. 2E). We also did not find a correlation between AC volume and symptom duration before and after excluding asymptomatic ACs (Fig. 4).

Regarding the mechanism by which ACs develop, we did not observe ependymal cells capable of CSF production in any of our ACs. The majority of the IAC-limited ACs were preserved well throughout TB processing, fully exhibiting the cystic wall, suggesting possible CSF entrapment due to a slit-valve mechanism.

The most common presenting symptoms were related to the cochlear division of cranial nerve (CN) VIII. The observed SNHL with reduced speech discrimination is consistent with the pattern observed with retrocochlear pathology. AC = arachnoid cyst; CN = cochlear nerve; Co = Cochlea; V = vestibule; VN = vestibular nerve; OHC = outer hair cells; IHC = inner hair cells. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

CONCLUSION

ACs of the IAC are not uncommon. Their presentation is variable, ranging from asymptomatic to SNHL,
with poor speech discrimination, tinnitus, and vertigo. This
diagnosis should be kept in the differential diagnosis of ret-
rocochlear pathologies. The resolution of current imaging
technology may not accurately capture these lesions in life.

BIBLIOGRAPHY

1. Propp JM, McCarthy BJ, Davis FG, Preston-Martin S. Descriptive epidemi-
ology of vestibular schwannomas. Neuro Oncol 2006;8:1–11.
2. Bohrer PS, Chole RA. Unusual lesions of the internal auditory canal.
3. Matthies C, Samii M. Management of 1000 vestibular schwannomas (acous-
4. Kohan D, Downey LL, Lim J, Cohen NL, Elowitz E. Uncommon lesions pre-
senting as tumors of the internal auditory canal and cerebellopontine
5. Rengachary SS, Watanabe I. Ultrastructure and pathogenesis of intracra-
6. Vaquerio J, Carrillo R, Cabezudo J, Nombela L, Bravo G. Arachnoid cysts of
7. Schachenmayr W, Friede RL. Fine structure of arachnoid cysts.
J Neuro-
pathol Exp Neurol 1979;38:434–446.
8. Robinson RG. Congenital cysts of the brain: arachnoid malformations. In:
10. Tsuda T, Ueda S, Matsumoto K. Clinicopathological study of the arachnoid
Long-term follow-up of acoustic schwannoma radiosurgery with marginal
of the cerebellopontine angle: diagnosis and surgery. Neurosurgery 1997;
13. Samii M, Carvalho GA, Schuhmann MU, Matthies C. Arachnoid cysts of the
Arachnoid cysts—report of two adult cases in the interhemispheric
fissure and over the cerebral convexity. Neurol Med Chir (Tokyo) 1982;22:
71–76.
15. Starkman SP, Brown TC, Linell EA. Cerebral arachnoid cysts. J Neu-
16. Alolado R, Weller RO, Parrish EP, Garrod D. The cranial arachnoid and
pia mater in man: anatomical and ultrastructural observations. Neu-
17. Schuknecht HF. Pathology of the Ear. 2nd ed. New York, NY: Lea and Fei-
gner; 1993.
Analysis of vestibular schwannoma size in multiple dimensions: a compar-
ative cohort study of different measurement techniques. Clin Otolaryngol
nance imaging scanner reliability for measuring changes in vestibular
21. Li D, Tsimpas A, Germanwala AV. Analysis of vestibular schwannoma size:
a literature review on consistency with measurement techniques. Clin Neu-
22. Kentala E, Pyykkö I. Clinical picture of vestibular schwannoma. Auris
23. Badie B, Pyle GM, Nguyen PH, Hadar EJ. Elevation of internal auditory
canal pressure by vestibular schwannomas. Otol Neurotol 2001;22:
696–700.
24. Nakamura M, Roser F, Mirzai S, Matthies C, Varkapic P, Samii M. Menin-
25. Verbist BM. Imaging of sensorineural hearing loss: a pattern-based
approach to diseases of the inner ear and cerebellopontine angle. Insights
26. Daniels RL, Swallow C, Shelton C, Davidson HC, Krejci CS, Harnsberger HR.