Tomography of the Vestibular Aqueduct in Ear Disease

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- A controversy exists concerning whether or not roentgenographic narrowing or nonvisualization of the vestibular aqueduct is a specific sign for Meniere's disease. Of 190 ears that were evaluated, abnormal aqueducts were seen in 42.9% of ears with Meniere's disease, 45.4% of contralateral, noninvolved ears from patients with Meniere's disease, 41.3% of ears with diseases other than Meniere's disease, 30.4% of ears with no disease, and 51.6% of normal ears. Narrowing or nonvisualization of the vestibular aqueduct is a nonspecific roentgenographic sign that is seen in disease, as well as in normal ears, and should not be used to make a diagnosis of Meniere's disease. Indications for polytomography in Meniere's disease include (1) preoperative evaluation of the aqueduct prior to endolymphatic shunt procedures and (2) exclusion of acoustic neurinomas or other organic causes of vertigo.

Since the initial description of Meniere's disease by Prosper Meniere in 1861, one problem in making the diagnosis of idiopathic endolymphatic hydrops has been the inability to rely on an objective sign that is specific for Meniere's disease. This is primarily a function of the lack of a thorough understanding of the basic disease process. Diagnoses are usually based on the following triad: (1) the classic history of well-defined spells of vertigo with absence of symptoms between attacks; (2) the characteristic fluctuating, progressive sensorineural hearing loss in the involved ear(s); and (3) the subjective sensations of tinnitus, fullness, and pressure. Audiologic evidence of hair cell dysfunction (poor speech discrimination, loss of tone, recruitment, and diplacusis), electro-nystagmographic manifestation of labyrinthine dysfunction, and laboratory exclusion of other medical diseases (hypothyroidism, syphilis, and disorders of carbohydrate and lipid metabolism) contribute to the data base that is used to aid in making a diagnosis. Alone, none of the aforementioned criteria are specific for Meniere's disease. A patient, reporting only one symptom or a combination of symptoms, may be classified as having either cochlear or vestibular Meniere's disease. When hypocycloidal polytomography became available as a practical, noninvasive method to visualize detailed temporal bone anatomy, investigations of the vestibular aqueduct began to help elucidate its role in Meniere's disease.

HISTORICAL REVIEW

Stimulated by House's endolymphatic-subarachnoid shunt operation, Clemis and Valvassori wrote a preliminary report in 1968, which correlated hearing loss (pure-tone average exceeding 25 dB ISO) with the roentgenographic appearance of the vestibular aqueduct. Of 97 ears that were examined with abnormal aqueductal configuration (25 narrow and 72 nonvisualized), 87 (90%) had abnormal hearing loss. In 1969 and 1973, the same authors found that the vestibular aqueduct was narrowed or nonvisualized in more than 50% of 1,000 patients with inner ear disorders as compared with 90% visualization of 200 control subjects.

In 1971, Brünner and Pedersen suggested that tomography of the vestibular aqueduct would be a useful supplement in diagnosing Meniere's disease and reported one case of left-sided Meniere's disease with a nonvisualized left aqueduct.

In contrast, Yuven and Schuknecht in 1972 measured the isthmus of the vestibular aqueducts in temporal bones from 19 patients with Meniere's disease and from 19 control subjects. They found that there was no statistically significant difference (P > .10) between the mean diameter of the vestibular aqueduct isthmus from patients with Meniere's disease (0.245 mm) and normal control subjects (0.262 mm). There was a significant difference (P < .025) between the mean diameter of the endolymphatic duct in patients with Meniere's disease (0.108 mm) as compared with normal subjects (0.150 mm).

Two years later, Stahle and Wilbrand, tomographically studied 86 ears from patients with Meniere's disease and 32 normal ears. In all of the normal ears, they were able to visualize the vestibular aqueduct. In contrast, of 66 ears from patients with unilateral Meniere's disease, the vestibular aqueduct could be seen in 65% of the ears from the noninvolved side and from 59% of the ears from the involved side. Similar results were obtained for 20 ears from patients with bilateral involvement in which 58% could be visualized. No correlation was found when visualization or nonvisualization of the aqueduct was compared with age, pure-tone threshold, discrimination score, or caloric response. These results differ from those of Clemis and Valvassori.

In 1974, Brünner and Pedersen roentgenographically evaluated 32 ears from patients with Meniere's disease and 43 ears from control subjects. They found that the aqueduct was abnormal in 84.4% of the patients with Meniere's disease, while abnormal in only 11.6% of the control subjects. Similar results have been reported by Rumbaugh et al.

In 1975, Oigaard et al. roentgenographically compared the aqueducts of patients with Meniere's disease with those of patients having chronic otitis media, as well as with control subjects without ear disease. The aqueduct was narrow or nonvisible in 5% of normal subjects, 56.6% of

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patients with Meniere’s disease, and 51.2% of patients with chronic otitis media. According to Wilbrand, a narrowing or nonvisualized aqueduct is not a specific diagnostic sign for Meniere’s disease.

GROSS ANATOMY

The adult vestibular aqueduct is a bony canal of the petrous bone, which contains the endolymphatic duct and sac, small blood vessels, and surrounding bone marrow. The proximal segment contains interfibrillar spaces, periosteum, and a process of cranial dura mater (Fig 1). Several authors have compared its shape with an inverted J that is composed of a proximal, ascending portion and a distal, descending straight segment. The proximal portion of the aqueduct, the internal aperture, begins at the posterior wall of the vestibule and arches through the otic capsule superiorly, medially, and slightly posteriorly; because of its embryologic development, it is relatively constant in shape. The curved proximal segment ascends parallel but anteromedial to the crus commune that is formed by the superior and posterior semicircular canals. Because the crus commune is an easily identifiable tomographic shadow, the anatomic relationship aids in the roentgenographic localization of the vestibular aqueduct.

The second portion of the aqueduct, which houses the isthmus of the endolymphatic duct, is the most narrow portion and serves as the junction between the arched proximal portion and the third section or distal, descending segment. The angle that is transcribed by the arched proximal and straight distal portions has been measured and varies from 90 to 135°. There is a negative correlation between the length of the aqueduct and the angle such that the shorter the aqueduct, the wider the angle. The variation in angle is thought to result from caudotropism forces that are exerted on the endolymphatic duct during the post-midterm growth and development of the sigmoid sinus and posterior fossa dura. For this reason, the distal, descending segment is the longest and the most variable part of the aqueductal system. It courses inferiorly and posteriorly and terminates as the external aperture on the posterosuperciliary bone on the petrous pyramid approximately 10 mm posterolaterally to the porus acusticus, 9 mm inferior to the superior petrosal sulcus, and 10 mm superior to the sigmoid sulcus. Traversing through the petrous bone adjacent to the vestibular aqueduct is a second canal, the paravestibular canalculus. As reported by Ogura and Clemis, the paravestibular canalculus is 0.12 mm wide (range, 0.1 to 0.2 mm), parallel to the vestibular aqueduct, and houses an artery and vein. Its internal aperture is located superior and medial to the internal aperture of the vestibu lar aqueduct in the medial wall of the vestibule. A small channel from the paravestibular canalculus communicates with the vestibular aqueduct at the level of the vestibule. The canalculus descends through the otic capsule first lateral to, then anteroinferior to, the aqueduct. It terminates in close proxim¬ity to the floor of the external aperture. One function is to house the blood supply for the endolymphatic duct and sac. It may serve as a pressure-equalizing mechanism between the perilymphatic space and posterior fossa. Several authors have identified it on tomograms.

ROENTGENOGRAPHIC ANATOMY

In 1965, Anson and, later in 1975, Gado and Arenberg recognized that the aqueduct frequently varied in form, position, and size. With the use of the lateral and off-lateral projections, Gado and Arenberg recognized the variation tomographically and described three roentgenographic configurations: tubular (type I), filiform or narrow (type II), and funnel shaped (type III). More importantly, these observers reasoned that the roentgenographic appearance of the aqueduct depends on the orientation of the canal to the plane of the tomographic cut. If the external aperture is widest in a plane that is parallel to the plane of the tomographic cut, it will appear as a funnel-shaped image. If it is widest in a plane that is perpendicular to the plane of the tomographic cut, it will appear more uniform in caliber, either tubular or filiform. The roentgenographic configuration of the vestibular aqueduct will greatly depend on the position of the patient’s head in reference to the plane of tomographic cut, as well as individual anatomic variation (Fig 2-4).

MATERIALS AND METHODS

Ninety-five cases (190 ears) were randomly selected from the polytomograph file at Presbyterian-University Hospital, Pittsburgh; true lateral projections were available for all cases. The off-lateral projection was not utilized. All films were viewed without any knowledge of the respective clinical diagnosis. To be consistent with the literature, tubular and funnel-shaped aqueducts were classified as “normal.” Filiform (narrow) and nonvi-

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**Fig 1.** Schematic of left membranous labyrinth and endolymphatic duct, lateral projection. Note parallel, superimposed relationship of proximal, ascending segment of endolymphatic duct to crus commune. For this reason, ascending segment is not usually seen tomographically (from Rumbaugh et al).
ualized aqueducts were considered to be "abnormal."

After the films had been examined, clinical diagnoses were correlated with the tomographic findings. As outlined by the Subcommittee on Equilibrium and Its Measurement, American Academy of Ophthalmology and Otolaryngology, a diagnosis of Meniere's disease was made if the following criteria could be documented: (1) fluctuating, progressive sensorineural hearing loss (unilateral or bilateral); (2) well-defined episodes of vertigo with absence of symptoms between attacks; and (3) tinnitus and/or a sense of fullness. No cases of suspected cochlear or vestibular Meniere's disease were included.

All tomograms were taken with a Philips Polytome with a 0.3-mm focal spot (magnification factor of 30%). Chronex-4 film and DuPont high-plus screens (nongrid technique) were used for all studies. Exposure technique was as follows: kV = 65 to 75; time = six seconds; mamp = 40 to 60; tight collimation; and 1-mm thick sections, 1 and 2 mm apart. Film processing was done at 51.6 °C by a 90-second processor (Kodak X-omat).

ANATOMIC LANDMARKS

With the use of lateral projection tomographic cuts, three measurements of each vestibular aqueduct were recorded with reference to the following landmarks (Fig 5).

External Aperture

The vertical diameter of the external aperture was measured from point A (the

Fig 2.—Lateral projection tomogram (left) and schematic (right) of left ear at level of crus commune demonstrating tubular (type I) vestibular aqueduct.

Fig 3.—Lateral projection tomogram (left) and schematic (right) of left ear at level of crus commune demonstrating filiform (type II) vestibular aqueduct.

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intersection of the superior aqueductal wall with the medial wall of the petrous pyramid to point B on the inferior aqueductal wall as determined by a line AB perpendicular to the superior aqueductal wall.

Diameter of Descending, Straight Portion

The mean diameter of the descending, straight portion of the aqueduct was measured from point C (a point equidistant between points A and E, the point of intersection of the superior aqueductal wall with the medial wall of the crus commune) to point D on the inferior aqueductal wall determined by a line CD perpendicular to the superior aqueductal wall.

Length of Descending, Straight Portion

The length of the distal, descending portion from the crus commune was measured along the straight line AE.

Mediolateral Diameter of External Aperture

This measurement was evaluated from the numbered tomographic cuts, 1 or 2 mm apart, in which the aperture was seen. For example, if the aqueduct was seen in three consecutive cuts, 1 mm apart, this was recorded as a distance of 2 mm. Obviously, this measurement is only an approximation of the actual distance.

A quantitative assessment of periaqueductal and petrous bone pneumatization was made of each ear according to four types:

Type I—no pneumatization and a small mastoid antrum

Type II—poorly pneumatized petrous bone with less than six periantral air cells

Type III—normal pneumatization with greater than six periantral air cells and greater than a 5-mm distance from the posterior attic to the posterior wall of the mastoid antrum

Type IV—hyperpneumatization of the petrous bone

Each aqueduct was measured with the use of a metric ruler (1-mm markings) and a standard viewbox. No magnifying devices were used during any part of the measurement procedure. Given the lack of clear and distinct borders of the aqueduct, which produced an inherent magnitude of error of 0.5 mm, it is thought that the above measurement procedure is not only practical but simulates the conditions under which such measurements would be made in a radiologist’s reading room.

RESULTS

A total group of 95 patients (190 ears) was studied. The mean age was 45.6 years (range, 5 to 74 years). There were 47 males, with a mean age of 42.9 years (range, 8 to 74 years) and 48 females, with a mean age of 48.1 years (range, 5 to 72 years). The patients were classified into four groups:

Group 1 consisted of patients with Meniere’s disease. Group 2 were patients with ear disease other than Meniere’s disease. Group 3 were patients with no ear disease. Group 4 were patients with normal, contralateral ears from groups 2 and 3.

Group 1

Of 95 patients, 25 had Meniere’s disease (22 unilateral and three bilateral) or 28 ears. There were 12 males, with a mean age of 60.8 years (range, 25 to 74 years) and 13 females, with a mean age of 46.5 years (range, 29 to 67 years). The mean duration of illness was 5.4 years (range, six weeks to 19 years). Of the 28 diseased ears, normal aqueducts (14 tubular and two funnel shaped) were seen in 16 ears (57.1%), and abnormal aqueducts (four filiform and eight nonvisualized) were observed in 12 ears (42.9%). Of the 28 contralateral ears from patients with Meniere’s disease, 12 ears (44.6%) had normal aqueducts, and ten ears (45.4%) were abnormal (five filiform and five nonvisualized) (Table 1).

Group 2

The group with ear disease other than Meniere’s disease consisted of 41 patients (82 ears). There were 63 diseased ears (chronic infection, vertigo, otosclerosis, sudden hearing loss,
noise-induced hearing loss, presbycus¬
toria, congenital hearing loss, and ototoxin-induced hearing loss) and 19 noninvolved, contralateral ears (Table 2). Of 63 diseased ears, 37 ears (58.7%) had normal aqueducts, and 26 ears (41.3%) had abnormal aqueducts.

**Group 3**

No ear disease was found in 29 patients (58 ears). Tomograms were taken because of head trauma, suspected acoustic neurinoma, vascular insufficiency, seizure disorder, or facial nerve paralysis (Table 3). There were 46 involved ears and 12 normal, contralateral ears. Of the 46 involved ears, 32 ears (69.6%) had normal aqueducts, and 14 ears (30.4%) had abnormal aqueducts.

**Group 4**

A combined total of 31 noninvolved, contralateral ears were studied from groups 2 and 3. Of these 31 ears, 15 ears (48.4%) had normal aqueducts, and 16 ears (51.6%) had abnormal aqueducts (Table 4).

### Aqueduct Measurement

Of 190 ears, 139 vestibular aqueducts were visualized and could be measured. The mean vertical diameter of the external aperture (line AB) was 1.3 mm (range, 0.4 to 5.0 mm) (Table 5). The diameter of the aqueduct (line CD) was 0.8 mm (0.1 to 2.0 mm). As measured from the crus commune, the mean length of the distal, descending segment (line AE) was 8.4 mm (3.5 to 19.0 mm). These are comparable with measurements found by other authors.

Of the 28 ears with Meniere's disease, 20 aqueducts were visualized. The mean vertical diameter of the external aperture, mean diameter of the aqueduct, and mean length of the distal, descending segment showed essentially no difference as compared with the other groups (Table 5).

### Pneumatization

Of 190 ears, seven ears (3.7%) were type I, 27 ears (14.2%) were type II, 142 ears (74.7%) were type III, and 14 ears (7.4%) were type IV (Table 6). There was no correlation between the configuration of the aqueduct and the degree of pneumatization.

Of the 28 ears with Meniere's disease, four ears (14.3%) were type II, 20 ears (71.4%) were type III, and four ears (14.3%) were type IV. There were no cases with absence of pneumatization as described by Wilbrand. Of the 22 contralateral ears from patients with Meniere's disease, three ears (13.6%) were type I, four ears (18.2%) were type II, 14 ears (63.6%) were type III, and one ear (4.6%) was type IV. Similar results were found for the remaining groups.

### Tomographic Cuts

As calculated from the tomographic cuts, 87 of 139 aqueducts (62.6%) could be seen over a distance of 1 mm, 38 aqueducts (27.3%) were seen over a 2-mm distance, 12 aqueducts (8.6%) were seen over a 3-mm distance, and only two aqueducts (1.4%) could be seen over a 4-mm distance.

### COMMENT

With the advent of polytomography, previous authors have used tomo-
### Table 2.—Aqueduct Configuration in Ear Disease Other Than Meniere’s Disease*

<table>
<thead>
<tr>
<th>Vestibular Aqueduct Configuration</th>
<th>Chronically Infection</th>
<th>Vertigo</th>
<th>Otosclerosis</th>
<th>Sudden Hearing Loss</th>
<th>Noise Hearing Loss</th>
<th>Presbycusis</th>
<th>Congenital Hearing Loss</th>
<th>Ototoxic Hearing Loss</th>
<th>Total Diseased Ears</th>
<th>Normal Contralateral Ears</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Tubular</td>
<td>4 (36.4)</td>
<td>13 (15.0)</td>
<td>2 (50.0)</td>
<td>3 (33.3)</td>
<td>1 (16.7)</td>
<td>2 (66.7)</td>
<td>1 (16.7)</td>
<td>2 (66.7)</td>
<td>25 (39.7)</td>
<td>8 (42.1)</td>
</tr>
<tr>
<td>Funnel</td>
<td>3 (27.3)</td>
<td>4 (15.4)</td>
<td>1 (25.0)</td>
<td>2 (22.2)</td>
<td>1 (16.7)</td>
<td>1 (33.3)</td>
<td>12 (19.0)</td>
<td>2 (10.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>7 (63.7)</td>
<td>17 (65.4)</td>
<td>3 (75.0)</td>
<td>5 (55.5)</td>
<td>2 (33.4)</td>
<td>3 (100.0)</td>
<td>37 (58.7)</td>
<td>10 (52.6)</td>
<td></td>
<td></td>
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</table>

### Table 3.—Aqueduct Configuration in Patients With No Ear Disease*

<table>
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<tr>
<th>Vestibular Aqueduct Configuration</th>
<th>Suspected Acoustic Neurinoma</th>
<th>Vascular Insufficiency</th>
<th>Seizure Disorder</th>
<th>Facial Nerve Paralysis</th>
<th>Head Trauma</th>
<th>Total With No Ear Disease</th>
<th>Normal Contralateral Ears</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Tubular</td>
<td>8 (36.3)</td>
<td>4 (100.0)</td>
<td>8 (53.3)</td>
<td>20 (43.5)</td>
<td>4 (33.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Funnel</td>
<td>6 (27.3)</td>
<td>3 (75.0)</td>
<td>1 (100.0)</td>
<td>12 (26.1)</td>
<td>1 (8.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>14 (62.6)</td>
<td>3 (75.0)</td>
<td>4 (100.0)</td>
<td>10 (66.6)</td>
<td>32 (69.6)</td>
<td>41 (41.6)</td>
<td></td>
</tr>
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</table>

### Table 4.—Aqueduct Configuration by Group

<table>
<thead>
<tr>
<th>Vestibular Aqueduct Configuration</th>
<th>Meniere’s Diseased Ear</th>
<th>Meniere’s Contralateral Ear</th>
<th>Ear Disease</th>
<th>No Ear Disease</th>
<th>Normal Contralateral Ears</th>
<th>Total Ears</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal aqueducts</td>
<td>16 (57.1)</td>
<td>12 (54.5)</td>
<td>37 (58.7)</td>
<td>32 (69.6)</td>
<td>15 (48.4)</td>
<td>112</td>
</tr>
<tr>
<td>Abnormal aqueducts</td>
<td>12 (42.9)</td>
<td>10 (45.5)</td>
<td>26 (41.3)</td>
<td>14 (30.4)</td>
<td>16 (51.6)</td>
<td>78</td>
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<tr>
<td>Total</td>
<td>28 (100.0)</td>
<td>22 (100.0)</td>
<td>63 (100.0)</td>
<td>46 (100.0)</td>
<td>31 (100.0)</td>
<td>190</td>
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### Table 5.—Aqueduct Measurements

<table>
<thead>
<tr>
<th>Source</th>
<th>Material*</th>
<th>No. of Ears</th>
<th>No. of Nonvisualized Aqueducts</th>
<th>Vertical Diameter of External Aperture, mm</th>
<th>Mean Diameter of Vestibular Aqueduct, mm</th>
<th>Length From Crus Commune to External Aperture, mm</th>
</tr>
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<tbody>
<tr>
<td>Present series</td>
<td>P</td>
<td>20</td>
<td>8</td>
<td>1.2 (0.5-3.0)</td>
<td>0.8 (0.1-2.0)</td>
<td>8.1 (5.0-13.0)</td>
</tr>
<tr>
<td>Meniere’s diseased ears</td>
<td>P</td>
<td>17</td>
<td>5</td>
<td>1.3 (0.4-3.0)</td>
<td>0.8 (0.2-2.0)</td>
<td>7.6 (5.0-13.0)</td>
</tr>
<tr>
<td>Meniere’s contralateral ears</td>
<td>P</td>
<td>46</td>
<td>17</td>
<td>1.4 (0.5-5.0)</td>
<td>0.8 (0.2-2.0)</td>
<td>8.6 (4.0-14.0)</td>
</tr>
<tr>
<td>Ear disease</td>
<td>P</td>
<td>38</td>
<td>8</td>
<td>1.5 (0.5-4.5)</td>
<td>1.0 (0.5-2.0)</td>
<td>10.0 (3.5-19.0)</td>
</tr>
<tr>
<td>No ear disease</td>
<td>P</td>
<td>18</td>
<td>13</td>
<td>1.0 (0.5-3.0)</td>
<td>0.8 (0.5-2.0)</td>
<td>7.5 (5.0-13.0)</td>
</tr>
<tr>
<td>Total</td>
<td>139</td>
<td>51</td>
<td>1.3 (0.4-5.0)</td>
<td>0.8 (0.1-2.0)</td>
<td>8.4 (3.5-19.0)</td>
<td></td>
</tr>
<tr>
<td>Gado and Arendberg*</td>
<td>P</td>
<td>10</td>
<td>1</td>
<td>1.5 (5.0)</td>
<td>. . .</td>
<td>(5.5-11.0)</td>
</tr>
<tr>
<td>Anson*</td>
<td>C</td>
<td>. . .</td>
<td>3.2 (0.6-4.5)</td>
<td>0.35 (0.25-0.5)</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>Ogura and Clemis*</td>
<td>C</td>
<td>16</td>
<td>. . .</td>
<td>. . .</td>
<td>. . .</td>
<td>7.3 (4.0-10.0)</td>
</tr>
<tr>
<td>Wilbrand et al**</td>
<td>C</td>
<td>35</td>
<td>. . .</td>
<td>. . .</td>
<td>. . .</td>
<td>6.3 (4.0-9.0)</td>
</tr>
</tbody>
</table>

*P indicates polytomogram; C, cadaver specimen.
graphic techniques in hopes of finding a specific roentgenographic sign for Meniere's disease. From the data presented, it is seen that the tomographic appearance of the vestibular aqueduct was abnormal, ie, either filiform or nonvisualized, in approximately the same percentage of patients with Meniere's disease (42.9%) as in patients with other ear diseases (41.3%), patients with no ear disease (30.4%), and normal ears (51.6%). Contralateral noninvolved ears from patients with Meniere's disease were equally abnormal (45.4%). A roentgenographically narrow or nonvisualized vestibular aqueduct may be seen in several pathologic conditions that affect the temporal bone and is not a specific sign for Meniere's disease.

The configuration of the vestibular aqueduct is a function of (1) the orientation of the aqueduct to the plane of the tomographic cut and (2) individual anatomic variation. Wilbrand et al have reported that the plane of the vestibular aqueduct varies between 31 and 65° with reference to the plane of the superior semicircular canal, which, itself, varies in its accepted 90° plane to the long axis of the petrous pyramid. Moreover, the longitudinal axis of the petrous pyramid is not always 45° to the sagittal plane of the skull. Although Gado and Arenberg were able to visualize the distal, descending segment of the aqueducts on all cadaver specimens that were examined without contrast material, an inability to visualize the aqueduct in the clinical situation is, in part, a function of head positioning. In the majority of cases, the true lateral projection with 1-mm sections, taken 1 mm apart, is the most advantageous view to be used for routine examinations of the aqueduct. In selected situations when it is imperative to know the course of the aqueduct, the off-lateral projection may be employed to increase the probability of identifying the canal. The aqueduct is seen 1 to 2 mm medial or lateral to the crus commune; the proximal segment is not seen because it has a narrow diameter and is superimposed on the crus commune.

Although one case of nonvisualization of the aqueduct caused by documented bony obliteration has been reported in Meniere's disease, it is unlikely that obliteration or stenosis is a major cause of nonvisualization. In four histopathologically confirmed cases of endolymphatic hydrops, I. Sando, MD (oral communication, April 1977) has found straightening and narrowing of the vestibular aqueduct. However, of 450 temporal bones, including ten patients with Meniere's disease, Schuknecht did not observe any narrowing or obstruction of the

### Table 6.—Pneumatization of Petrous Pyramid*

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of Ears</th>
<th>Type I</th>
<th>Type II</th>
<th>Type III</th>
<th>Type IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meniere's diseased ear</td>
<td>28</td>
<td>0</td>
<td>1 (4.3)</td>
<td>20 (71.4)</td>
<td>4 (14.3)</td>
</tr>
<tr>
<td>Meniere's noninvolved ear</td>
<td>22</td>
<td>3 (13.6)</td>
<td>1 (4.3)</td>
<td>14 (63.6)</td>
<td>1 (4.6)</td>
</tr>
<tr>
<td>Ear disease</td>
<td>63</td>
<td>2 (3.1)</td>
<td>12 (19.0)</td>
<td>45 (71.4)</td>
<td>4 (6.3)</td>
</tr>
<tr>
<td>No ear disease</td>
<td>46</td>
<td>2 (4.3)</td>
<td>3 (6.5)</td>
<td>37 (80.4)</td>
<td>4 (8.7)</td>
</tr>
<tr>
<td>Normal contralateral ear</td>
<td>31</td>
<td>0</td>
<td>4 (12.9)</td>
<td>26 (83.9)</td>
<td>1 (3.2)</td>
</tr>
<tr>
<td>Total</td>
<td>190</td>
<td>7 (3.7)</td>
<td>27 (14.2)</td>
<td>142 (74.7)</td>
<td>14 (7.4)</td>
</tr>
</tbody>
</table>

*Type I indicates no pneumatization; type II, poor pneumatization; type III, normal pneumatization; type IV, hyperpneumatization.

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![Image](http://archotol.jamanetwork.com/)

**Fig 6.—Lateral projection tomogram (left) and schematic (right) of left ear at level of crus commune demonstrating external aperture situated lower than normal in sigmoid sinus groove.**

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vestibular aqueduct. Others have reported similar findings.\textsuperscript{7,13} Even though aqueductal ossification is complete at birth, the bone remains non-Haversian or fetal type throughout life.\textsuperscript{8} It remains to be learned if there are, indeed, natural or pathologic processes that lead to narrowing and obliteration of the aqueduct.

Without utilizing magnification and complicated instruments, a simple, standardized technique for measuring the vestibular aqueduct compares favorably with the results obtained by others from both polytomograms and temporal bone specimens. Wilbrand\textsuperscript{12} and Wilbrand et al\textsuperscript{13} have found good reliability between tomographic measurements of the aqueduct and actual anatomic dimension. By using the measurement methods outlined earlier, accurate anatomic information may be obtained from the lateral polytomogram.

In contrast to previous reports,\textsuperscript{4,14} the degree of petrous bone pneumatization does not appear to influence the configuration of the vestibular aqueduct. Regardless of the pathologic condition, the majority of all ears (74.7%) had normal pneumatization. Absence of pneumatization was found in only 3.7%; there were no diseased ears from patients with Meniere's disease in this category. On the other hand, Wilbrand\textsuperscript{12} found that 74% of the patients with Meniere's disease, compared with 22% of the normal subjects, had no pneumatization and that no patients with Meniere's disease had large cell pneumatization. Additional studies will be necessary to better understand this discrepancy and to elucidate the significance of peraqueductal pneumatization.

The role of polytomography in assessing a patient with Meniere's disease is twofold. Before an endolymphatic-subarachnoid shunt procedure is performed, valuable preoperative information may be obtained concerning the size and course of the vestibular aqueduct, as well as the location of the external aperture. We have seen patients in whom the external aperture was situated lower than normal in the sigmoid sinus groove (Fig 6). If maximum information and fine detail are mandatory, it may be necessary to include 15° off-lateral projections and sections that are taken 0.5 mm apart.

A second indication for polytomography in Meniere's disease is to aid in excluding acoustic neoplasms, other cerebellopontine angle tumors, otosclerosis,\textsuperscript{10,11} otodystrophies, and primary and metastatic tumors. Routinely, anteroposterior and lateral projections should be obtained. Pluri-directional tomography may demonstrate subtle erosions of the internal auditory canal when plain film roentgenography is normal. When calcifications are present in a cerebellopontine angle mass, tomography may be of diagnostic value in helping to differentiate between acoustic neurinoma and meningioma or cartilaginous tumor. Often, the size of tumors cannot be accurately predicted by tomography, especially if the tumor is predominantly extracanalicular. Following tomography, intravenously enhanced computerized cranial tomography (CT) should be the next special procedure performed in the workup of an acoustic neurinoma; CT will detect tumors greater than 1.5 cm in diameter and has largely displaced the more invasive techniques of angiography, pneumoencephalography, and iophendylate (Pantopaque) cisternography. If positive, an operation may be undertaken without the need for further studies. Vertebral angiography may be done if an angiographic map is desired by the surgeon. If CT is negative, low-volume iophendylate cisternography is the procedure of choice to rule out a tumor less than 1.5 cm in diameter. Recently, low-dose metrizamide (Amipaque) CT cisternography has been used as a means to detect intracanalicular tumors.\textsuperscript{22,23}

References