Medial acoustic neuromas: clinical and surgical implications

Clinical article

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Object. Medial acoustic neuroma is a rare entity that confers a distinct clinical syndrome. It is scarcely discussed in the literature and is associated with adverse features. This study evaluates the clinical and imaging features, pertinent surgical challenges, and treatment outcome in a large series of this variant. The authors postulate that the particular pathological anatomy with its arachnoidal rearrangement has a profound implication on the surgical technique and outcome.

Methods. The authors conducted a retrospective analysis of 52 cases involving 33 women and 19 men who underwent resection of medial acoustic neuromas performed by the senior author (O.A.) over a 20-year period (1993–2013). Clinical, radiological, and operative records were reviewed, with a specific focus on the neurological outcomes and facial nerve function and hearing preservation. Intraoperative findings were analyzed with respect to the effect of arachnoidal arrangement on the surgeon’s ability to resect the lesion and the impact on postoperative function.

Results. The average tumor size was 34.5 mm (maximum diameter), with over 90% of tumors being 25 mm or larger and 71% being cystic. Cerебellar, trigeminal nerve, and facial nerve dysfunction were common preoperative findings. Hydrocephalus was present in 11 patients. Distinguishing intraoperative findings included marked tumor adherence to the brainstem and frequent hypervascularity, which prompted intracapsular dissection resulting in enhancement on postoperative MRI in 18 cases, with only 3 demonstrating growth on follow-up. There was no mortality or major postoperative neurological deficit. Cerebrospinal fluid leak was encountered in 7 patients, with 4 requiring surgical repair. Among 45 patients who had intact preoperative facial function, only 1 had permanent facial nerve paralysis on extended follow-up. Of the patients with preoperative Grade I–II facial function, 87% continued to have Grade I–II function on follow-up. Of 10 patients who had Class A hearing preoperatively, 5 continued to have Class A or B hearing after surgery.

Conclusions. Medial acoustic neuromas represent a rare subgroup whose site of origin and growth patterns produce a distinct clinical presentation and present specific operative challenges. They reach giant size and are frequently cystic and hypervascular. Their origin and growth pattern lead to arachnoidal rearrangement with marked adherence against the brainstem, which is critical in the surgical management. Excellent surgical outcome is achievable with a high rate of facial nerve function and attainable hearing preservation. These results suggest that similar or better results may be achieved in less complex tumors.

Key Words • medial acoustic neuroma • vestibular schwannoma • arachnoid plane • cerebellopontine angle • hypervascularity • brain tumor • hearing loss • facial nerve • oncology

Abbreviations used in this paper: AICA = anterior inferior cerebellar artery; CN = cranial nerve; IAC = internal auditory canal; PICA = posterior inferior cerebellar artery.

In 1992, Tos et al. coined the term “medial acoustic neuroma” to describe a clinical entity of an extrameatal vestibular schwannoma “without tumor mass located laterally in the internal auditory canal.” Proteicaceous CSF, rather than tumor, fills the lateral canal. They suggested that the distinguishing clinical findings of large size, relatively preserved hearing on initial presentation, and more severe impairment of the cerebellum, brainstem, and tri-
Since that time, there have been very few reports on the subject. \(^{9,31,34}\) The geminal nerve might be explained by the medial origin. \(^{34}\) Here, we describe our experience with a large series of medial acoustic neuromas, in which we found these tumors to be frequently cystic, often hypervascular, overwhelmingly large in size, and very adherent to the brainstem due to focal absence of the typical arachnoidal duplication. We draw on our intraoperative observations and existing theories on the site of origin of acoustic neuromas and adjacent arachnoidal anatomy to postulate a pattern of growth as it relates to the regional arachnoid layers and cisterns. This growth pattern helps define the symptom complex of the medial acoustic neuroma, its particular surgical considerations, and its impact on outcome.

Methods

Fifty-two patients were identified who underwent surgery for medial acoustic neuroma performed by the senior author (O.A.) between 1993 and 2013. In all cases, the tumors fulfilled the criteria for medial acoustic neuroma, based on preoperative MRI and CT images. These findings were corroborated by clinical features and confirmed by intraoperative observation.

We performed retrospective analysis of these cases through review of medical records, including pre- and postoperative imaging studies, operative findings, follow-up evaluations, and outcomes. We excluded patients with neurofibromatosis Type 2 and those who had undergone prior radiation therapy.

All patients underwent a complete neurological evaluation with specific attention to the integrity and function of the cranial nerves (CNs). Facial nerve function was graded before and after surgery and at each follow-up visit, based on the House-Brackmann scale. \(^{8}\)

Hearing was assessed before and after surgery and graded based on the Committee on Hearing and Equilibrium guidelines for the evaluation of hearing preservation in acoustic neuroma. \(^{18}\) Audiograms were obtained preoperatively in all cases, and postoperatively for patients with expected serviceable hearing. Evaluation of swallowing and vocal cord function and dedicated neuro-ophthalmology evaluation were performed when dictated by the clinical findings and patients’ symptoms.

Imaging evaluation included preoperative MRI and CT scan, with a dedicated temporal bone sequence to evaluate the internal auditory canal (IAC) (Fig. 1). Magnetic resonance imaging was obtained in all patients postoperatively, at 3 months’ follow-up, and annually thereafter.

Surgical Considerations

Several particular considerations pertain to the surgical technique in resection of medial acoustic neuromas: tumor adherence to the brainstem and facial nerve; displacement of the facial nerve and anterior inferior cerebellar artery (AICA); involvement of multiple CNs due to the typically giant size of the tumor; difficulty of dissection due to frequently cystic components; common hypervascularity; and absence of canal enlargement, which increases the angulation or “kinking” of the nerve at the porus, a known point of vulnerability, which in turn increases the risk of injury to the facial nerve. \(^{11,35}\)

Patients underwent surgery in the supine position. A transmastoid retrosigmoid approach was performed as described, where the transverse and sigmoid sinuses are skeletonized and reflected outward, minimizing the need for cerebellar retraction. \(^{7}\) Neuronavigation and neurophysiological monitoring are applied, including monitoring of somatosensory evoked potentials, brainstem auditory evoked responses, and electromyography, for any potentially involved CNs, from CN III to CN XII. Release of CSF from the cisterna magna permits relaxation of the cerebellar hemisphere and allows for direct and wide exposure of these frequently giant tumors.

Initial tumor resection is performed after using a neurostimulator to locate a safe entry into the tumor. The tumor is debulked internially with an ultrasonic aspirator and the tumor capsule is dissected carefully along the arachnoidal planes (Fig. 2A). The lower CNs are identified along with the posterior inferior cerebellar artery (PICA) at the inferior pole of the tumor (Fig. 2B). In medial acoustic neuromas, multiple layers of arachnoid facilitate intraarachnoid dissection and safe preservation of these structures. At the superior pole, similar multiple arachnoid layers facilitate the dissection of the trigeminal nerve (Fig. 2C). Coagulation is avoided around all nerves, and hemostasis is obtained through pulse irrigation. A diamond drill is used to open the posterior lip of the IAC and inspection for any intracanalicular extension of the tumor is performed (Fig. 2D). Frequent stimulation is used to locate the facial nerve. Particular attention is paid to the severe angulation of the facial nerve at the entry to the IAC where, due to the absence of tumor in the canal, the nerve is markedly “kinked” and adherent, more so than would be in a similarly sized nonmedial acoustic neuroma. The AICA likewise courses along the wall of the tumor, frequently ventrally and inferiorly displaced (Fig. 2E). As dissection approaches the brainstem, the intraarachnoid plane is followed along the superior and inferior poles. The multiple arachnoid layers present superiorly and inferiorly subside medially, and dissection becomes more difficult. If no plane is identified, the surgeon might be forced to perform intracapsular resection of tumor, leaving a thin capsule that is severely adherent to the brainstem (Fig. 2F). If a plane of dissection can be identified, complete resection ensues with a quest for cure. An endoscope with 0° and 30° lenses is introduced to inspect the field—particularly inside the meatus, under the tentorium, along the trigeminal nerve, and ventral to the brainstem to ensure the absence of residual tumor.

Results

Clinical Findings

Fifty-two adult patients (33 women, 19 men) were identified with medial acoustic neuromas. The patients’ ages ranged from 19 to 74 years (average 43 years, median 45 years). The peak tumor incidence was in the 4th decade of life, with 24 patients being 40 years or younger.
and two-thirds of those being female. The mean duration of follow-up was 23 months (range 1–132 months).

The tumor size ranged from 13 mm to 53 mm in maximum diameter, with an average size of 34.5 mm (median 35 mm) (Table 1). Forty-seven patients (90.4%) had tumors of 25 mm or larger. Thirty-seven tumors (71%) had a cystic appearance on MRI. Hydrocephalus was present in 11 patients (21%), 3 of whom underwent placement of a ventriculoperitoneal shunt. Four patients had undergone a previous surgery at another institution and presented with recurrent tumor.

The most common initial symptoms were progressive hearing loss (88%), unsteady gait (38%), headaches (35%), facial numbness (35%), and dizziness (33%). Other symptoms are listed in Table 2.

Table 3 details preoperative CN deficits. Twenty-nine patients (56%) had trigeminal nerve involvement, most commonly involving all 3 branches. Facial weakness was present in 7 patients, 4 of whom had undergone a previous surgery. Forty-six patients reported hearing impairment prior to surgery, while 6 reported no preoperative hearing deficit. On formal audiogram testing, 10 patients had Class A, 10 patients had Class B, and 6 patients had Class C hearing prior to surgery (Table 4).

Surgical Findings and Outcome

All patients underwent microsurgical resection through the transmastoid retrosigmoid approach with the aim of total resection. There were no mortalities. Fifty-six percent of the tumors were found to be severely adherent to the brainstem. Seventeen (42.5%) of 40 patients had specific annotation of hypervascularity in the operative report. Due to these complicating factors, intracapsular dissection was performed on the tumor at its adherence to the brainstem, which was depicted as enhancement on postoperative MRI in 18 cases (35%, Fig. 3). However, only 3 patients had documented tumor growth within the follow-up period.
Table 5 details facial nerve function prior to surgery and on long-term follow-up. Among patients with good facial nerve function on initial presentation, only 1 had permanent House-Brackmann Grade VI facial paralysis on long-term follow-up and subsequently underwent facial nerve reanimation. Another patient with intact facial function before surgery had House-Brackmann Grade VI paralysis early postoperatively, but only had 4 months of follow-up at the time of this study. Transient postoperative facial nerve dysfunction was noted in 28 cases (immediate in 25 cases, delayed in 3 cases); the patients underwent gold weight implantation to facilitate eyelid closure. The average length of time from surgery to improvement of facial nerve function was just over 7 months. In half of the patients with transient dysfunction, facial nerve function improved within 3 months. In 1 patient with recurrent tumor and preoperative facial dysfunction, CN VII was grafted using a posterior auricular nerve.

Of 10 patients with Class A hearing before surgery, functional hearing (Class A or B) was preserved in 5 of 9 patients with postoperative audiograms (Table 4). CSF leak was noted in 7 cases, with 4 requiring surgical repair. One patient had postoperative meningi-
Medial acoustic neuroma

A rare variant of acoustic neuroma occupies the cisternal compartment with no extension into the lateral IAC and is termed “medial acoustic neuroma.” The medial origin and intracisternal growth of these tumors allow them to reach a considerable size before patients present with symptoms of brainstem compression, CN involvement, cerebellar dysfunction, and hydrocephalus. Some degree of hearing is frequently present despite a large tumor size. Indeed, more than 90% of the tumors in our series were large or giant.

We present a large series documenting the clinical characteristics of the syndrome and revealing the frequency of hypervascularity and cystic nature of the tumors as well as the younger age of the patients, all of which are factors conventionally associated with surgical difficulties and worse outcome across all acoustic neuromas. Notwithstanding the natural limitations of a retrospective methodology, including selection bias, non-adjudicated outcome analysis, varying duration of follow-up, and advancements in techniques, we postulate that the origin and growth of these tumors in relation to adjacent arachnoid confer a distinct set of presenting symptoms and surgical challenges, and we describe our technical maneuvers to optimize management.

Discussion

A rare variant of acoustic neuroma occupies the cisternal compartment with no extension into the lateral IAC and is termed “medial acoustic neuroma.” The medial origin and intracisternal growth of these tumors allows them to reach a considerable size before patients present with symptoms of brainstem compression, CN involvement, cerebellar dysfunction, and hydrocephalus. Some degree of hearing is frequently present despite a large tumor size. Indeed, more than 90% of the tumors in our series were large or giant.

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Tumor Origin, Arachnoid Planes, and the Medial Tumor

The exact site of origin of schwannomas and the precise interplay between schwannoma growth and arachnoid anatomy has been the subject of considerable debate. It is commonly held that acoustic neuromas originate at the transition zone between the neuroglial and ensheathing Schwann cell elements (Obersteiner-Redlich zone), but there is only scant support for this theory in the literature. Yåsargil et al. posited that tumors originate epi-arachnoidally, slowly pushing the adjacent arachnoid medially until it folds in on itself, creating the “double layer.” Tarlov supported a modified version of this epi-arachnoidal theory. The safest dissection is achieved when working in between the arachnoid layers.

Debate aside, the point of origin of the tumor dictates the pattern of the arachnoidal rearrangement. Various studies have addressed the precise site of schwannoma

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**TABLE 2: Presenting symptoms among 52 patients with medial acoustic neuromas**

<table>
<thead>
<tr>
<th>Presenting Symptom</th>
<th>No. of Cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>progressive hearing loss</td>
<td>46 (88)</td>
</tr>
<tr>
<td>ataxia</td>
<td>20 (38)</td>
</tr>
<tr>
<td>headache</td>
<td>18 (35)</td>
</tr>
<tr>
<td>facial numbness or tingling</td>
<td>18 (35)</td>
</tr>
<tr>
<td>dizziness</td>
<td>17 (33)</td>
</tr>
<tr>
<td>tinnitus</td>
<td>10 (19)</td>
</tr>
<tr>
<td>facial weakness</td>
<td>7 (13)</td>
</tr>
<tr>
<td>memory impairment</td>
<td>5 (10)</td>
</tr>
<tr>
<td>dysarthria</td>
<td>3 (6)</td>
</tr>
<tr>
<td>diplopia</td>
<td>3 (6)</td>
</tr>
<tr>
<td>dysphagia</td>
<td>3 (6)</td>
</tr>
<tr>
<td>facial pain</td>
<td>2 (4)</td>
</tr>
<tr>
<td>hemifacial spasm</td>
<td>1 (2)</td>
</tr>
</tbody>
</table>

**TABLE 3: Cranial nerve and cerebellar involvement on presentation**

<table>
<thead>
<tr>
<th>Presenting Sign</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN V deficit</td>
<td>29*</td>
</tr>
<tr>
<td>CN VI deficit</td>
<td>3</td>
</tr>
<tr>
<td>CN VII deficit</td>
<td>7†</td>
</tr>
<tr>
<td>CN VIII deficit</td>
<td>46</td>
</tr>
<tr>
<td>CN IX/X deficit</td>
<td>2</td>
</tr>
<tr>
<td>CN XI deficit</td>
<td>1</td>
</tr>
<tr>
<td>CN XII deficit</td>
<td>2</td>
</tr>
<tr>
<td>nystagmus</td>
<td>8</td>
</tr>
<tr>
<td>cerebellar dysfunction</td>
<td>19</td>
</tr>
</tbody>
</table>

* Deficit of V1, V2, and V3 in 17 cases; V1 and V2 in 4 cases; V2 and V3 in 3 cases; only V1 in 2 cases, only V2 in 2 cases, and only V3 in 1 case.
† Due to previous surgery in 4 cases.

**TABLE 4: Hearing function before and after resection of medial acoustic neuroma**

<table>
<thead>
<tr>
<th>Preop Class</th>
<th>Postop Hearing Function Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (n = 10)†</td>
<td>A 3 2 1 3</td>
</tr>
<tr>
<td>B (n = 10)†</td>
<td>3 5</td>
</tr>
<tr>
<td>C (n = 6)</td>
<td>1 5</td>
</tr>
<tr>
<td>D (n = 26)</td>
<td>26</td>
</tr>
</tbody>
</table>

* Values represent the number of patients with the specified class of hearing. On audiogram, patients with Class A function exhibited ≤ 30 dB of pure-tone thresholds and ≥ 70% speech discrimination; those with Class B function had > 30 dB but ≤ 50 dB of pure-tone threshold and ≥ 50% speech discrimination; those with Class C function had > 50 dB pure-tone threshold and ≥ 50% speech discrimination; and those with Class D function had < 50% speech discrimination with any level of pure-tone threshold.
† Postoperative data were not available for all patients.
Histopathological analysis of intracanalicular schwannomas demonstrated that tumors arose anywhere along the course of CN VIII from the glial-Schwann cell transition zone to as lateral as the termination in vestibular and auditory end organs. Autopsy studies demonstrate a glial-Schwann cell transition zone medial to the porus in over half of patients, allowing acoustic neuromas to originate medial to the porus.

This intracisternal birth allows free growth to a large size prior to symptomatic presentation, leading to the distinct clinical findings of medial acoustic neuromas. The intracisternal growth also leads to particular rearrangement of arachnoid layers surrounding the tumor that is different from the pattern of the typical acoustic neuroma. In this pattern, while multiple arachnoid layers exist in the superior and inferior poles as a contribution from the trigeminal cistern, the lateral pontomesencephalic cistern, and the pontomedullary cistern, duplication of arachnoid layers is absent against the brainstem. This growth pattern and anatomical arrangement creates several surgical challenges (Fig. 4), as addressed below.

**Size**

Medial tumors are large at presentation (Fig. 5).

**TABLE 5: Facial nerve function prior to surgery and at latest follow-up, as quantified by House-Brackmann grade**

<table>
<thead>
<tr>
<th>Grade at Latest Follow-Up</th>
<th>I (n = 36)</th>
<th>II (n = 4)</th>
<th>III (n = 7)</th>
<th>IV (n = 1)</th>
<th>V (n = 0)</th>
<th>VI (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (n = 45)</td>
<td>35</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>II (n = 1)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III (n = 4)</td>
<td></td>
<td></td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV (n = 0)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V (n = 0)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI (n = 2)</td>
<td></td>
<td></td>
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</tbody>
</table>

* Values represent the number of patients with the specified House-Brackmann grade.
Larger size of acoustic neuromas has long been associated with increased neurological impairment and surgical risk. In addition to CN involvement, larger tumors compress and displace the brainstem, distorting the usual view of the brain-tumor interface; draining veins in large and giant tumors may be unusually distended and fragile, increasing the risk of hemorrhage. When tumors are stratified by size, those larger than 3 cm (20%–22%) are associated with higher complication rates and longer hospital stays than those under 3 cm (6%–9.6%). In our series, no mortality or unexpected adverse neurological events were encountered. Furthermore, no cerebellar infarction or vascular injury of the AICA or PICA was observed.

**Hypervascularity**

“Hypervascular schwannomas” have been described as a distinct entity, associated with the observation of rich abnormal tumor vessels leading to excessive intraoperative bleeding and adverse outcomes. An unusually high percentage of the medial acoustic neuromas in our series were hypervascular, as specifically mentioned in the operative report. Hypervascular acoustic neuromas are supplied by the vertebrobasilar system and demonstrate numerous intratumoral arteriovenous shunts as well as early filling of large draining veins on angiography. In contrast, typical acoustic neuromas are more commonly supplied by small meningeal feeding vessels from the external carotid arteries.

Interestingly, our review of case reports of hypervascular (schwannomas) revealed that their radiographic attributes fulfill the description of medial acoustic neuroma. Given the frequent finding of hypervascularity in our case series, we speculate that medial acoustic neuromas derive a similar blood supply from the vertebrobasilar system. The growth pattern of the medial tumors into the cerebellopontine cistern, compressing the surface of the brainstem and the petrosal surface of the cerebellum, results in a tendency to parasitize multiple feeding vessels from the AICA and PICA. Lacking the arachnoid layer, the plane of dissection becomes difficult to delineate, leading to further manipulation of the vessels and pial surface and carrying additional risk of vascular injury, with consequent life-threatening cerebellar and brainstem infarcts. However, although the hypervascular nature adds to the operative complexity, awareness of and caution against these factors prevented the adverse events described in previous reports.

**Cystic Prevalence**

Estimates of the prevalence of cystic lesions range from 5.7% to 48% of all acoustic neuromas. Cystic acoustic neuromas are reported to have faster growth rates, lower total resection rates, and poorer surgical outcome, most notably, with respect to facial nerve function. This may be due to vulnerability of the splayed facial nerve separated from dissection by the thin peripherally located cyst capsule. Cystic tumors have been speculated to arise from focal degeneration of tumor cells, recurrent intratumoral microhemorrhages, or coalescence of microcysts. Matrix metalloproteinase-2 (MMP-2) has also been implicated in the genesis of cysts and may contribute to their adherence to the facial nerve. The large size and hypervascularity of medial acoustic tumors may therefore predispose to cystic degeneration and consequent surgical risks.

**Hearing Preservation**

A distinguishing finding in medial acoustic neuromas is the presence of hearing function despite large tumor size, with some patients having perfect preoperative hearing. Hearing preservation was not thought to be feasible.
due to the large size of tumors in the original series of medial acoustic neuromas. The feasibility of preserving hearing has subsequently been reported. Our finding shows that preservation of hearing is an achievable goal in these patients, especially if they had good hearing before surgery.

While direct compression of the cochlear nerve is likely to contribute to hearing loss in acoustic tumors, an imperfect correlation between size, hearing levels, and preserved nerve fibers suggests that other mechanisms of hearing loss are involved. A recent histopathological study of unresected unilateral acoustic neuromas showed that degenerative changes in the inner ear and atrophy of the stria vascularis and spiral ligament were present in addition to cochlear nerve degeneration when compared with control specimens. Tumor adherence to the cochlear nerve in the IAC as well as increases in IAC pressure from tumor may be responsible for these changes, which have been shown to adversely affect hearing outcome.

The absence of significant intracanalicular extension may provide an opportunity to preserve hearing despite the typically large size of the medial tumor variant. If hearing is present and preservation is achievable, then the surgical approach for these tumors should be chosen accordingly, with the translabyrinthine approach being less favorable. While the middle fossa approach allows for preservation of hearing, it is disadvantageous for tumors with significant caudal extension. To allow for maximal surgical exposure during resection of these large, cystic, hypervascular tumors, while minimizing cerebellar retraction, we found the transmastoid retrosigmoid approach to be superior and highly recommended.

Facial Nerve Function

Owing to their large size, cystic component, hypervascularity, and unusual adherence, the facial nerve is extremely vulnerable in medial acoustic neuromas. Further compounding the risk is the severe kinking of the facial nerve at the porus due to the sharp angulation of the nerve as it exits the normal horizontal course from the meatus to an abrupt and steep displacement by the tumor (Fig. 6B).

Reported rates of good postoperative facial nerve function (House-Brackmann Grade I–II) in medial tumors range from 66% to 78%. In our series, 87% of patients who had Grade I–II facial function prior to surgery continued to have Grade I–II function at follow-up, consistent with other authors’ success of achieving approximately 80% Grade I–II facial nerve function in patients with large tumors (> 3 cm). The rate of permanent complete facial paralysis in patients with intact preopera-

![Fig. 6. Schematic illustration of a medial acoustic neuroma in transverse (A) and coronal (B) views, as compared with a typical acoustic neuroma in transverse (C) and coronal (D) views. Note the absence of the double arachnoid layer against the brainstem as well as the increased angulation of the facial nerve complex entering the IAC in the medial acoustic neuroma.](image-url)
con-}

tive facial function and long-term follow-up was approxi-
mately 2%. In fact, the high rate of facial nerve function
preservation in this series of large medial tumors with
difficult anatomical features would seem to substantiate a
microsurgical approach to smaller tumors as well.

Conclusions

We highlight the distinct clinical findings of medial
acoustic neuromas and expand the original description,
noting that these tumors are frequently giant, cystic, hy-
pervascular, and adherent to the brainstem and that they
predominantly affect younger patients. Despite these
multiple high risk factors, understanding of the patho-
logical anatomy guides surgical considerations to achieve
safe and curative resection, with a low risk of permanent
facial nerve deficit and high rate of hearing preservation.

Disclosure

The authors report no conflict of interest concerning the mate-
rials or methods used in this study or the findings specified in this
paper.

Author contributions to the study and manuscript preparation
include the following. Conception and design: Al-Mefty, Dunn.
Acquisition of data: Al-Mefty, Dunn, Bi, Erkmen, Hasan, Tang,
Pravdenkova. Analysis and interpretation of data: Al-Mefty, Dunn,
Bi, Erkmen, Kadri, Pravdenkova. Drafting the article: Al-Mefty,
Dunn, Bi, Erkmen, Kadri, Hasan. Critically revising the article: all
authors. Reviewed submitted version of manuscript: all authors.
Approved the final version of the manuscript on behalf of all authors:
Al-Mefty. Statistical analysis: Dunn, Bi. Administrative/technical/
material support: Tang. Study supervision: Al-Mefty.

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